

**S826\_P2**

**Exploring depression**

**About this free course**

This free course is an adapted extract from the Open University course S826 Introduction to mental health science [www.open.ac.uk/postgraduate/modules/s826](http://www.open.ac.uk/postgraduate/modules/s826?LKCAMPAIGN=ebook_&MEDIA=ou).

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[http://www.open.edu/openlearn/science-maths-technology/exploring-depression/content-section-0](http://www.open.edu/openlearn/science-maths-technology/exploring-depression/content-section-0?LKCAMPAIGN=ebook_&amp;MEDIA=ol)

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## Introduction

Start of Box

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](https://www.open.edu/openlearn/science-maths-technology/exploring-anxiety/content-section-0?active-tab=description-tab).

End of Box

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](http://www.open.ac.uk/postgraduate/modules/s826) (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.

Start of Box

**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).

End of Box

Start of Box

**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.

End of Box

Start of Activity

**Activity 1 The experience of depression**

Allow 60 minutes

Start of Question

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.

Start of Media Content

Video content is not available in this format.

**TED Talk: Andrew Solomon – The Secret We Share**

[View transcript - TED Talk: Andrew Solomon – The Secret We Share](" \l "Session1_Transcript1)

End of Media Content

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?

End of Question

[View discussion - Activity 1 The experience of depression](" \l "Session1_Discussion1)

End of Activity

## 1.1 Understanding depression − key issues

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

Start of Activity

**Activity 2 Understanding depression: key issues**

Allow approximately 1 hour

Start of Question

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.

Start of Media Content

Audio content is not available in this format.

**BBC World Service ‘Discovery’ − Depression part 1**

[View transcript - BBC World Service ‘Discovery’ − Depression part 1](" \l "Session1_Transcript2)

End of Media Content

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?

End of Question

[View discussion - Part](" \l "Session1_Discussion2)

Start of Question

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.

Start of Media Content

Audio content is not available in this format.

**BBC World Service ‘Discovery’ − Depression part 2**

[View transcript - BBC World Service ‘Discovery’ − Depression part 2](" \l "Session1_Transcript3)

End of Media Content

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?

End of Question

[View discussion - Part](" \l "Session1_Discussion3)

End of Activity

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

Start of ITQ

* 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?

End of ITQ

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.

Start of Box

**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.

End of Box

## 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).

Start of Table

**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 disordera | 694.8 | 689.9 | 1171.7 | 1161.2 |
| Dysthymiab | 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.

End of Table

Start of ITQ

* 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).

End of ITQ

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.

Start of Box

**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.

End of Box

## 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](http://www.who.int/classifications/icd/revision/en/)) 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’](https://www.nice.org.uk/guidance/cg90) published October 2009, replacing [CG23](https://www.nice.org.uk/guidance/cg23) published in December 2004) still referred to both the ICD-10 and DSM-IV criteria. The NICE (CG90) guideline stated that:

Start of Quote

…..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)

End of Quote

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’](https://www.nice.org.uk/guidance/ng222), 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).

Start of Box

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

(based on ICD-10, 2003)

Start of Quote

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

End of Quote

**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).

End of Box

Start of Box

**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).

End of Box

Start of Box

**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.

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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.

End of Box

### 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:

Start of Quote

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)

End of Quote

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:

Start of Quote

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)

End of Quote

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:

Start of Quote

… 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)

End of Quote

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

Start of Quote

… 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)

End of Quote

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.

Start of Activity

**Activity 3 What is depression?**

Allow 60 minutes

Start of Question

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.

Start of Media Content

Audio content is not available in this format.

**Oxford University Podcast − What is depression?**

[View transcript - Oxford University Podcast − What is depression?](" \l "Session2_Transcript1)

End of Media Content

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?

End of Question

*Provide your answer...*

End of Activity

Start of ITQ

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

End of ITQ

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.

Start of Box

**Box 8 Psychological theories and models of depressiona**

* **Behaviourist theory**: classical conditioningb (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 conditioningb; 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).

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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.

End of Box

**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

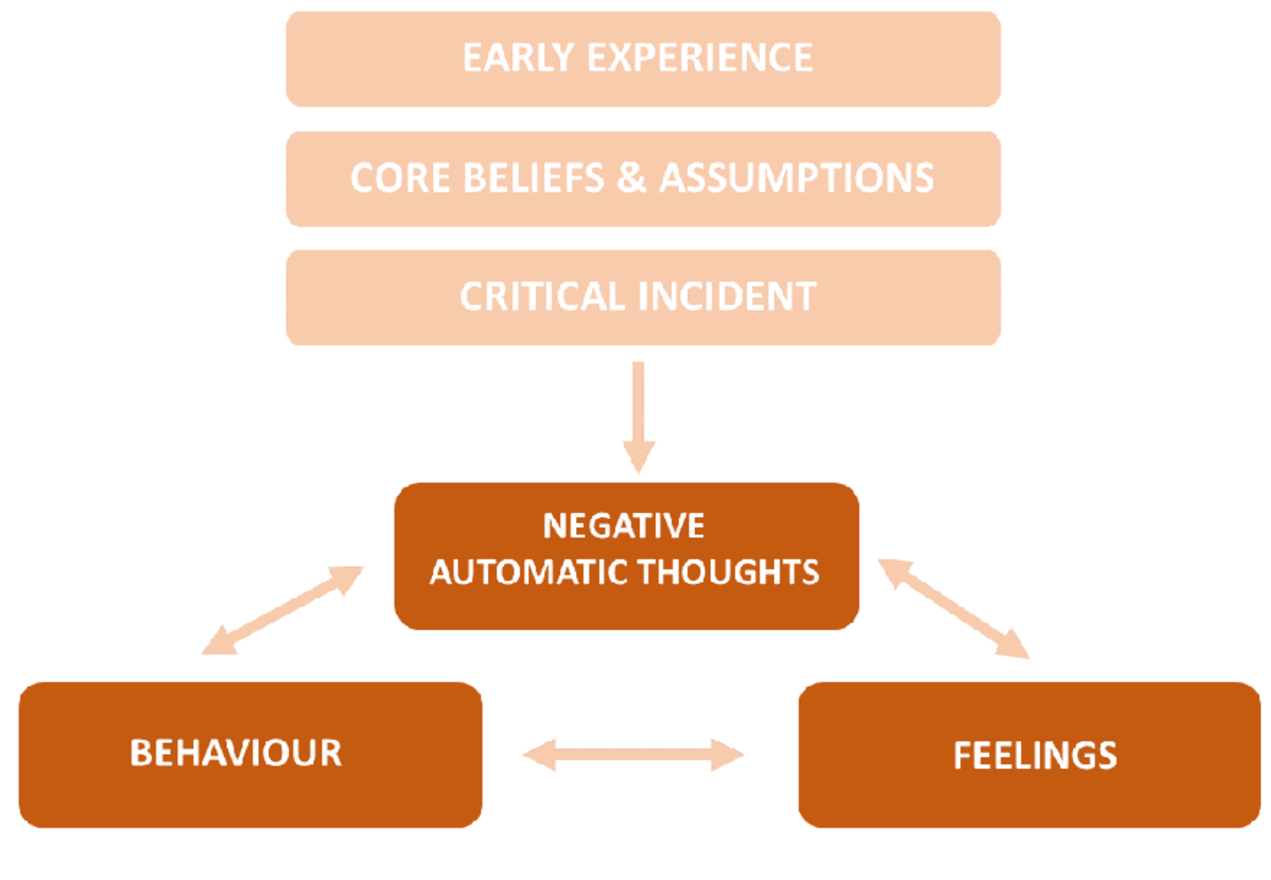
Psychodynamic theories view depression in terms of inwardly directed anger, loss of self-esteem or self-worth, egotistic or excessive narcissistic or personality demand, or deprivation in mother-child relationship (loss or rejection by a parent). Freud’s (1917) psychoanalytic theory is an example of 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).

Start of Figure



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

End of Figure

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

Start of Box

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

Start of Table

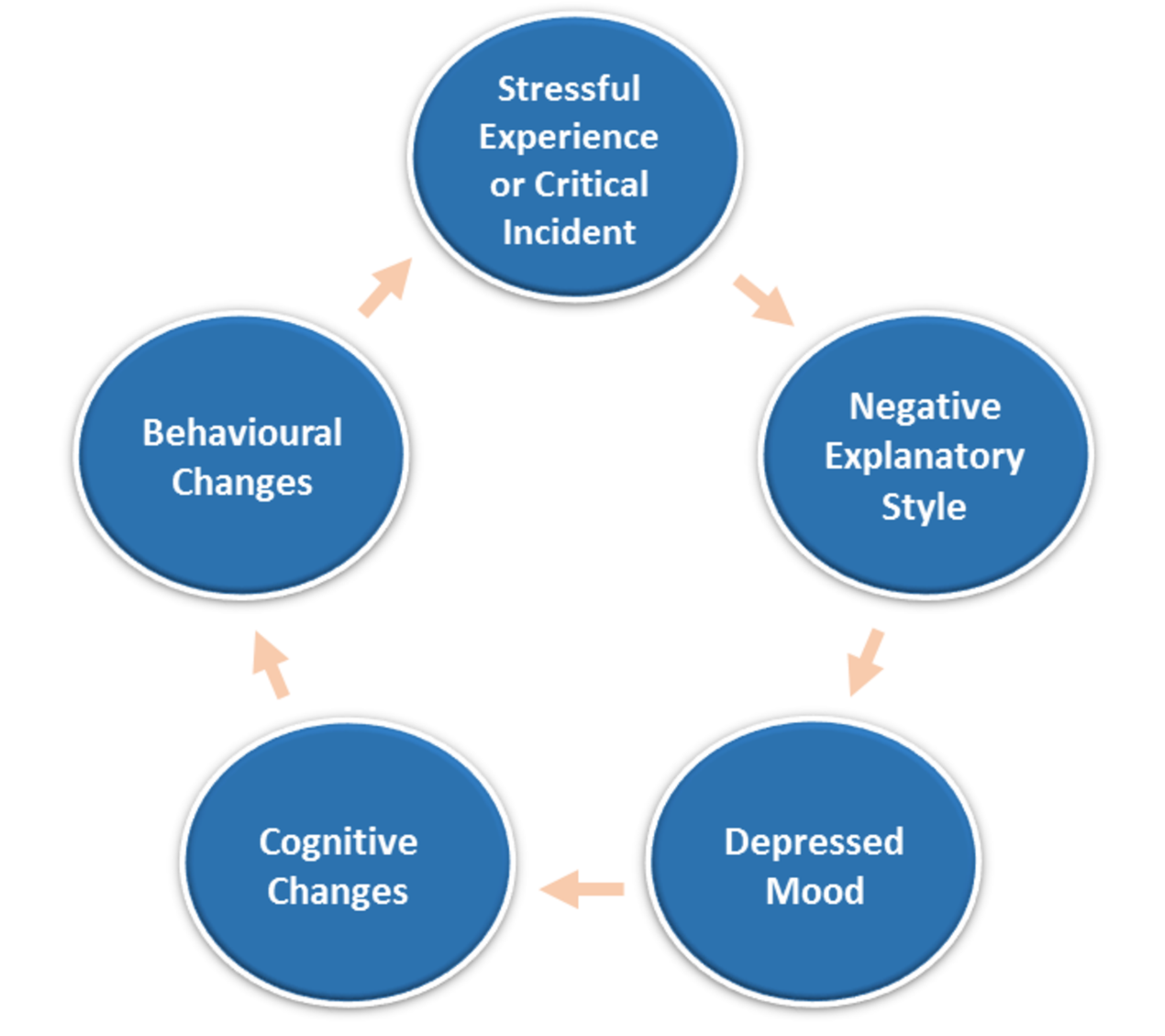
|  |  |
| --- | --- |
| **Dichotomous (‘all or nothing’) thinking** | Looking at things in absolute (‘black or white’) categories with no middle ground, e.g. ‘If I fall short of perfection, I’m a total failure’. |
| **Overgeneralisation** | Generalising from a single negative experience and viewing this 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). |

End of Table

End of Box

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).

Start of Figure



**Figure 2** Depression ‘cycle’ based on the cognitive model of depression

End of Figure

**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.

Start of Box

**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.

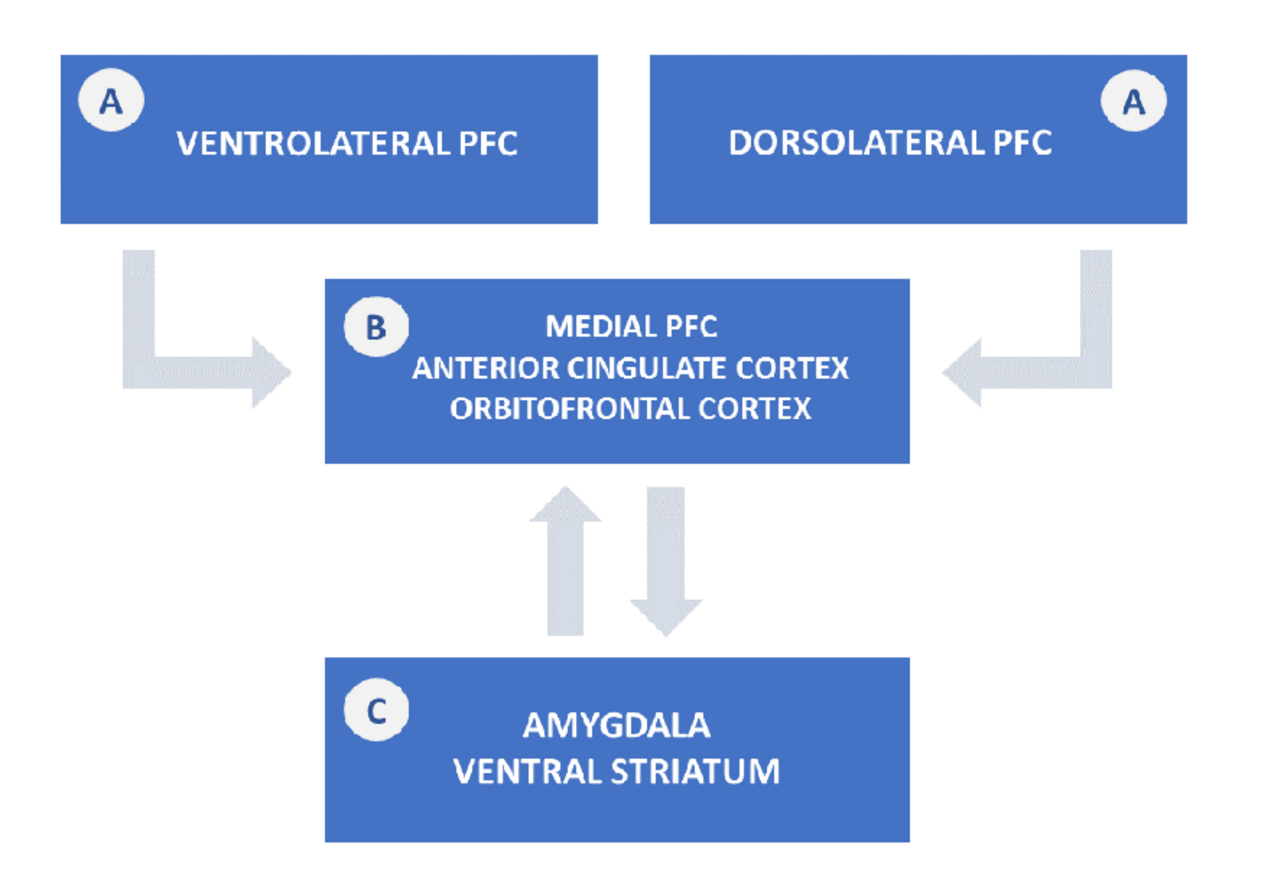
**Stress-based models:** learned helplessness, chronic mild stress, social defeat stress.

**Others:** genetically engineered mice; olfactory bulbectomy.

End of Box

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).

Start of Figure



**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.

End of Figure

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.

Start of Activity

**Activity 4 How is depression treated?**

Allow 60 minutes

Start of Question

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.

Start of Media Content

Audio content is not available in this format.

**Oxford University Podcast − How is depression treated?**

[View transcript - Oxford University Podcast − How is depression treated?](" \l "Session5_Transcript1)

End of Media Content

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?

End of Question

*Provide your answer...*

End of Activity

## 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.

Start of Table

**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).

End of Table

Start of Box

**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.

End of Box

## 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.

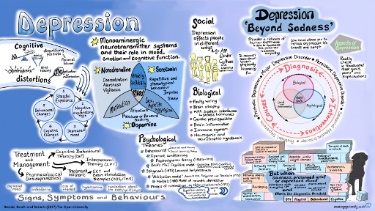
Nonpharmacological (somatic) treatments aimed at the underlying neurobiology for medication-resistant forms of severe depression include electroconvulsive therapy (ECT), brain stimulation techniques such as deep brain stimulation (DBS) and transcranial magnetic stimulation (rTMS). These procedures are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

Start of Figure



**Figure 4** Visual Summary ‘Depression’

End of Figure

We have provided you with a [larger version of this image in PDF format](http://www.open.edu/openlearn/ocw/mod/oucontent/olinkremote.php?website=S826_P2&targetdoc=Visual%20Summary%20Depression).

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](http://www.open.edu/openlearn/science-maths-technology/exploring-the-relationship-between-anxiety-and-depression/content-section-0), 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](https://www.open.edu/openlearn/health-sports-psychology/mental-health/introduction-mental-health-science) article page.

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## Further reading

Find out more about this topic on OpenLearn:

[Understanding depression and anxiety](http://www.open.edu/openlearn/health-sports-psychology/health/understanding-depression-and-anxiety/content-section-0)

[Emotions and emotional disorders](http://www.open.edu/openlearn/health-sports-psychology/health/emotions-and-emotional-disorders/content-section-0)

[Challenging ideas in mental health](http://www.open.edu/openlearn/health-sports-psychology/health/challenging-ideas-mental-health/content-section-0)

[Work and mental health](http://www.open.edu/openlearn/health-sports-psychology/health/work-and-mental-health/content-section-0)

[Exercise and mental health](https://www.open.edu/openlearn/health-sports-psychology/sport-fitness/exercise-and-mental-health/content-section-0?active-tab=description-tab)

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We gratefully acknowledge the following sources:

BBC World Service ‘Discovery’ − Depression (2012)

Oxford University Podcasts. Dr Danny Penman and Professor Mark Williams from the Department of Experimental Psychology discuss ‘What is depression?’, as part of a series on the ‘New Psychology of Depression’ produced in 2011. Available under Creative Commons Licence CC-BY-NC-SA (Creative Commons Attribution - Non-commercial - Share Alike 2.0)

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## Solutions

## Activity 1 The experience of depression

#### 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".’

[Back to - Activity 1 The experience of depression](" \l "Session1_Activity1)

## Activity 2 Understanding depression: key issues

### Part

#### 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.

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### Part

#### 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 interpersonal 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’.

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# TED Talk: Andrew Solomon – The Secret We Share

## Transcript

**ANDREW SOLOMON:** 'I felt a funeral in my brain, and mourners to and fro kept treading, treading, till I felt that sense was breaking through and when they all were seated, a service, like a drum kept beating, beating, till I felt my mind was going numb and then I heard them lift a box, and creak across my soul with those same boots of lead again. Then space began to toll, as if the heavens were a bell, and being were an ear, and I, and silence, some strange race wrecked, solitary here. Just then, a plank in reason broke, and I fell down, and down and hit a world at every plunge, and finished knowing then.'

We know depression through metaphors. Emily Dickinson was able to convey it in language. Goya, in an image. Half the purpose of art is to describe such iconic states. As for me, I had always thought myself tough. One of the people who could survive if I had been sent to a concentration camp. In 1991, I had a series of losses. My mother died. A relationship I'd been in ended. I moved back to the United States from some years abroad. And I got through all of those experiences intact. But in 1994, three years later, I found myself losing interest in almost everything. I didn't want to do any of the things I had previously wanted to do, and I didn't know why.

The opposite of depression is not happiness, but vitality, and it was vitality that seemed to seep away from me in that moment. Everything there was to do seemed like too much work. I would come home, and I would see the red light flashing on my answering machine, and instead of being thrilled to hear from my friends, I would think, what a lot of people that is to have to call back. Or I would decide I should have lunch, and then I would think that I'd have to get the food out and put it on a plate and cut it up and chew it and swallow it. And it felt to me like the stations of the cross.

And one of the things that often gets lost in discussions of depression is that you know it's ridiculous. You know it's ridiculous while you're experiencing it. You know that most people manage to listen to their messages and eat lunch and organise themselves to take a shower and go out the front door, and that it's not a big deal. And yet, you are nonetheless in its grip, and you are unable to figure out any way around it. And so, I began to feel myself doing less and thinking less and feeling less. It was a kind of nullity. And then the anxiety set in.

If you told me that I have to be depressed for the next month, I would say, as long as I know it'll be over in November, I can do it. But if you said to me, you have to have acute anxiety for the next month, I would rather slit my wrists than go through it. It was the feeling all the time like that feeling you have if you're walking and you slip or trip and the ground is rushing up at you, but instead of lasting half a second the way that does, it lasted for six months. It's the sensation of being afraid all the time, but not even knowing what it is that you are afraid of. And it was at that point that I began to think that it was just too painful to be alive, and that the only reason not to kill oneself was so as not to hurt other people.

And finally one day I woke up and I thought perhaps I'd had a stroke because I lay in bed, completely frozen, looking at the telephone, thinking something is wrong and I should call for help. And I couldn't reach out my arm and pick up the phone and dial. And finally, after four full hours of my lying and staring at it, the phone rang, and somehow I managed to pick it up. And it was my father. And I said, 'I'm in serious trouble. We need to do something.'

The next day, I started with the medications and the therapy. And I also started reckoning with this terrible question. If I'm not the tough person who could have made it through a concentration camp, then who am I? And if I have to take medication, is that medication making me more fully myself, or is it making me someone else? And how do I feel about it, if it's making me someone else?

I had two advantages as I went into the fight. The first is that I knew that, objectively speaking, I had a nice life, and that if I could only get well, there was something at the other end that was worth living for. And the other was that I had access to good treatment. But I nonetheless emerged and relapsed, and emerged and relapsed, and emerged and relapsed, and finally understood I would have to be on medication and in therapy forever. And I thought, but is it a chemical problem or a psychological problem? And does it need a chemical cure or a philosophical cure? And I couldn't figure out which it was.

And then I understood that actually, we aren't advanced enough in either area for it to explain things fully. The chemical cure and the psychological cure both have a role to play. And I also figured out that depression was something that was braided so deep into us that there was no separating it from our character and personality. I want to say that the treatments we have for depression are appalling. They're not very effective. They're extremely costly. They come with innumerable side effects. They're a disaster. But I am so grateful that I live now and not 50 years ago, when there would have been almost nothing to be done. I hope that 50 years hence, people will hear about my treatments and be appalled that anyone endured such primitive science.

Depression is the flaw in love. If you were married to someone and thought 'Well, if my wife dies, I'll find another one', it wouldn't be love as we know it. There is no such thing as love without the anticipation of loss. And that spectre of despair can be the engine of intimacy.

There are three things people tend to confuse: depression, grief and sadness. Grief is explicitly reactive. If you have a loss, and you feel incredibly unhappy, and then, six months later, you're still deeply sad, but you're functioning a little better, it's probably grief. And it will probably, ultimately, resolve itself in some measure.

If you experience a catastrophic loss, and you feel terrible and, six months later, you can barely function at all, then it's probably a depression that was triggered by the catastrophic circumstances. The trajectory tells us a great deal. People think of depression as being just sadness. It's much, much too much sadness, much too much grief at far too slight a cause.

As I set out to understand depression and to interview people who had experienced it, I found that there were people who seemed, on the surface, to have what sounded like relatively mild depression, who were nonetheless utterly disabled by it. And there were other people who had what sounded, as they described it, like terribly severe depression, who nonetheless had good lives in the interstices between their depressive episodes. And I set out to find out what it is that causes some people to be more resilient than other people. What are the mechanisms that allow people to survive? And I went out and I interviewed person after person who was suffering with depression.

One of the first people I interviewed described depression ‘as a slower way of being dead’. And that was a good thing for me to hear early on because it reminded me that that slow way of being dead can lead to actual deadness, that this is a serious business. It's the leading disability worldwide, and people die of it every day.

One of the people I talked to when I was trying to understand this was a beloved friend who I had known for many years, and who had had a psychotic episode in her freshman year of college and then plummeted into a horrific depression. She had bipolar illness, or manic depression, as it was then known. And then she did very well, for many years, on lithium. And then eventually she was taken off her lithium to see how she would do without it, and she had another psychosis and then plunged into the worst depression that I had ever seen, in which she sat in her parents' apartment more or less catatonic, essentially without moving, day after day after day.

And when I interviewed her about that experience some years later, she's a poet and psychotherapist named Maggie Robbins. When I interviewed her, she said 'I was singing "where have all the flowers gone" over and over to occupy my mind. I was singing to blot out the things my mind was saying, which were, "you are, you are nothing. You are nobody. You don't even deserve to live". And that was when I really started thinking about killing myself.'

You don't think in depression that you've put on a grey veil and are seeing the world through the haze of a bad mood. You think that the veil has been taken away, the veil of happiness, and that now you're seeing truly. It's easier to help schizophrenics who perceive that there is something foreign inside of them that needs to be exorcised. But it's difficult with depressives because we believe we are seeing the truth.

But the truth lies. I became obsessed with that sentence ‘But the truth lies’. And I discovered, as I talked to depressive people, that they have many delusional perceptions. People will say 'No one loves me'. And you say, 'I love you. Your wife loves you. Your mother loves you.' You can answer that one pretty readily, at least for most people. But people who are depressed will also say 'No matter what we do, we're all just going to die in the end'. Or they'll say 'There can be no true communion between two human beings. Each of us is trapped in his own body.' To which you have to say 'That's true, but I think we should focus right now on what to have for breakfast'.

[LAUGHING]

A lot of the time what they are expressing is not illness, but insight. And one comes to think what's really extraordinary, is that most of us know about those existential questions, and they don't distract us very much.

There was a study I particularly liked in which a group of depressed and a group of non-depressed people were asked to play a video game for an hour. And at the end of the hour, they were asked how many little monsters they thought they had killed. The depressive group was usually accurate to within about 10%. And the non-depressed people guessed between 15 and 20 times as many little monsters

[LAUGHING]

as they had actually killed.

A lot of people said, when I chose to write about my depression, that it must be very difficult to be out of that closet to have people know. They said 'Do people talk to you differently?' And I said 'Yes, people talk to me differently. They talk to me differently insofar as they start telling me about their experience, or their sister's experience, or their friend's experience.' Things are different because now I know that depression is a family secret that everyone has.

I went, a few years ago, to a conference, and, on Friday of the three-day conference, one of the participants took me aside and she said 'I suffer from depression and I'm a little embarrassed about it, but I've been taking this medication and I just wanted to ask you what you think'. And so I did my best to give her such advice as I could. And then she said 'You know, my husband would never understand this. He's really the kind of guy to whom this wouldn't make any sense, so it's just between us.' And I said 'Yes, that's fine'.

On Sunday at the same conference, her husband took me aside, and he said 'My wife wouldn't think that I was really much of a guy if she knew this, but I've been dealing with this depression, and I'm taking some medication and I wondered what you think?' They were hiding the same medication in two different places in the same bedroom. And I said that I thought communication within the marriage might be triggering some of their problems.

[LAUGHTER]

But I was also struck by the burdensome nature of such mutual secrecy. Depression is so exhausting. It takes up so much of your time and energy, and silence about it really does make the depression worse. And then I began thinking about all the ways people make themselves better. I'd started off as a medical conservative. I thought there were a few kinds of therapy that worked. It was clear what they were there. There was medication; there were certain psychotherapy; there was possibly electroconvulsive treatment and that everything else was nonsense.

But then I discovered something. If you have brain cancer, and you say that standing on your head for 20 minutes every morning makes you feel better, it may make you feel better, but you still have brain cancer and you'll still probably die from it. But if you say that you have depression and standing on your head for 20 minutes every day makes you feel better, then it's worked, because depression is an illness of how you feel. And if you feel better, then you are effectively not depressed anymore. So I became much more tolerant of the vast world of alternative treatments.

And I get letters. I get hundreds of letters from people writing to tell me about what's worked for them. Someone was asking me backstage today about meditation. My favourite of the letters that I got was the one that came from a woman who wrote and said that she had tried therapy. She had tried medication. She had tried pretty much everything, and she had found a solution and hoped I would tell the world. And that was making little things from yarn.

[LAUGHING]

She sent me some of them, and I'm not wearing them right now. I suggested to her that she also should look up obsessive compulsive disorder in the DSM.

And yet, when I went to look at alternative treatments, I also gained perspective on other treatments. I went through a tribal exorcism in Senegal that involved a great deal of ram's blood and that I am not going to detail right now. But a few years afterwards, I was in Rwanda working on a different project, and I happened to describe my experience to someone, and he said 'Well, you know, that's West Africa and we're in East Africa, and our rituals are in some ways very different, but we do have some rituals that have something in common with what you're describing'. And I said, 'Oh'. And he said 'Yes, but we've had a lot of trouble with Western mental health workers, especially the ones who came right after the genocide'. And I said 'What kind of trouble did you have?' And he said '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.'

[LAUGHING]

He said 'We had to ask them to leave the country'.

[LAUGHING]

Now, at the other end of alternative treatments, let me tell you about Frank Russakoff. Frank Russakoff had the worst depression, perhaps, that I've ever seen in a man. He was constantly depressed. He was, when I met him, at a point at which every month he would have electroshock treatment. Then he would feel sort of disoriented for a week. Then he would feel OK for a week. Then he would have a week of going downhill. And then he would have another electroshock treatment. And he said to me when I met him 'It's unbearable to go through my weeks this way. I can't go on this way. And I figured out how I'm going to end it if I don't get better.' But, he said to me 'I heard about a protocol at Mass General for a procedure called a cingulotomy, which is a brain surgery, and I think I'm going to give that a try.

And I remember being amazed at that point to think that someone who clearly had so many bad experiences with so many different treatments still had, buried in him somewhere, enough optimism to reach out for one more. And he had the cingulotomy, and it was incredibly successful. He's now a friend of mine. He has a lovely wife and two beautiful children. He wrote me a letter the Christmas after the surgery, and he said 'My father sent me two presents this year. First, a motorised CD rack from the Sharper Image that I didn't really need, but I knew he was giving it to me to celebrate the fact that I'm living on my own and have a job I seem to love. And the other present was a photo of my grandmother, who committed suicide. As I unwrapped it, I began to cry, and my mother came over and said "Are you crying because of the relatives you never knew?" And I said "She had the same disease I have". I'm crying now as I write to you. It's not that I'm so sad, but I get overwhelmed, I think, because I could have killed myself, but my parents kept me going and so did the doctors, and I had the surgery. I'm alive and grateful we live in the right time, even if it doesn't always feel like it.'

I was struck by the fact that depression is broadly perceived to be a modern, Western, middle-class thing, and I went to look at how it operated in a variety of other contexts. And one of the things I was most interested in was depression among the indigent, and so I went out to try to look at what was being done for poor people with depression. And what I discovered is that poor people are mostly not being treated for depression. 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.

And yet it turns out that if you have a really lovely life but feel miserable all the time, you think 'Why do I feel like this? I must have depression', and you set out to find treatment for it. But if you have a perfectly awful life and you feel miserable all the time, the way you feel is commensurate with your life, and it doesn't occur to you to think, maybe this is treatable.

And so we have an epidemic in this country of depression among impoverished people that's not being picked up, and it's not being treated, and it's not being addressed. And it's a tragedy of a grand order. And so I found an academic who was doing a research project in slums outside of DC, where she picked up women who had come in for other health problems and diagnosed them with depression and then provided six months of the experimental protocol.

One of them, Lolly, came in, and this is what she said the day she came in. And she was a woman, by the way, who had seven children. She said 'I used to have a job, but I had to give it up because I couldn't go out of the house. I have nothing to say to my children. In the morning, I can't wait for them to leave, and then I climb in bed and pull the covers over my head. And 3 o'clock, when they come home, it just comes so fast.' She said 'I've been taking a lot of Tylenol, anything I can take so that I can sleep more. My husband has been telling me I'm stupid. I'm ugly. I wish I could stop the pain.'

Well, she was brought into this experimental protocol, and when I interviewed her six months later, she had taken a job working in child care for the US Navy. She had left the abusive husband, and she said to me 'My kids are so much happier now'. She said, 'There's one room in my new place for the boys, and one room for the girls, but at night, they're just all up on my bed and we're doing homework all together and everything. One of them wants to be a preacher. One of them wants to be a firefighter. And one of the girls says she's going to be a lawyer. They don't cry like they used to, and they don't fight like they did. That's all I need now is my kids. Things keep on changing, the way I dress, the way I feel, the way I act. I can go outside not being afraid anymore, and I don't think those bad feelings are coming back. And if it weren't for Dr. Miranda and that, I would still be at home with the covers pulled over my head, if I were still alive at all. I asked the Lord to send me an angel, and he heard my prayers.'

I was really moved by these experiences. And I decided that I wanted to write about them not only in a book I was working on, but also in an article. And so I got a commission from The New York Times Magazine to write about depression among the indigent, and I turned in my story and my editor called me and said 'We really can't publish this'. And I said 'Why not?' And she said 'It just is too far-fetched, these people who are at the very bottom rung of society, and then they get a few months of treatment and they're virtually ready to run Morgan Stanley. It's just too implausible.' She said 'I've never even heard of anything like it'. And I said 'The fact that you've never heard of it is an indication that it is "news”'.

[LAUGHING]

[APPLAUSE]

'And you are a news magazine'. So after a certain amount of negotiation, they agreed to it. But I think a lot of what they said was connected, in some strange way, to this distaste that people still have for the idea of treatment. The notion that, somehow, if we went out and treated a lot of people in indigent communities, that would be an exploitative thing to do because we would be changing them. There's this false moral imperative that seems to be all around us that treatment of depression, the medications and so on, are an artifice, and that it's not natural. And I think that's very misguided. It would be natural for people's teeth to fall out, but there is nobody militating against toothpaste. At least not in my circles.

And people then say, 'Well, but 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?' To which I would say 'Mood is adaptive. Being able to have sadness and fear and joy and pleasure and all of the other moods that we have,that's incredibly valuable. And major depression is something that happens when that system gets broken. It's maladaptive.' People come to me and say 'I think, though, if I just stick it out for another year, I think I can just get through this'. And I always say to them 'You may get through it, but you'll never be 37 again. Life is short, and that's a whole year you're talking about giving up.' Think it through. It's a strange poverty of the English language and, indeed, of many other languages, that we use the same word, ‘depression’, to describe how a kid feels when it rains on his birthday, and to describe how somebody feels the minute before they commit suicide.

People say to me 'Well, is it continuous with normal sadness?' And I think, in a way, it's continuous with normal sadness. There is a certain amount of continuity. But it's the same way there is continuity between having an iron fence outside your house that gets a little rust spot that you have to sort of sand off and maybe do a little repainting, and what happens if you leave the house for 100 years and it rusts through until it's only a pile of orange dust. And it's that orange dust spot, that orange dust problem, that's the one we're setting out to address.

So now, people say 'You take these happy pills and do you feel happy?' And I don't. But I don't feel sad about having to eat lunch, and I don't feel sad about my answering machine, and I don't feel sad about taking a shower. I feel more, in fact, I think because I can feel sadness without nullity. I feel sad about professional disappointments, about damaged relationships, about global warming. Those are the things that I feel sad about now. And I said to myself 'Well, what is the conclusion? How did those people who have better lives even with bigger depression manage to get through? What is the mechanism of resilience?'

And what I came up with over time was that the people who deny their experience, the ones who say 'I was depressed a long time ago, and I never want to think about it again, and I'm not going to look at it, and I'm just going to get on with my life',ironically, those are the people who are most enslaved by what they have. Shutting out the depression strengthens it. While you hide from it, it grows. And the people who do better are the ones who are able to tolerate the fact that they have this condition. Those who can tolerate their depression are the ones who achieve resilience.

So Frank Russakoff said to me 'If I had it to do over, I suppose I wouldn't do it this way. But in a strange way I'm grateful for what I've experienced. I'm glad to have been in the hospital 40 times. It taught me so much about love, and my relationship with my parents and my doctors has been so precious to me and will be always.'

And Maggie Robbins said 'I used to volunteer in an AIDS clinic, and I would just talk and talk and talk. And the people I was dealing with weren't very responsive, and I thought, that's not very friendly or helpful of them. And then I realised. I realised that they weren't going to do more than make those first few minutes of small talk. It was simply going to be an occasion where I didn't have AIDS, and I wasn't dying, but could tolerate the fact that they did, and they were. Our needs are our greatest assets. It turns out I've learned to give all the things I need.'

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 very meaningful. It's of seeking that meaning, and thinking, when it comes again 'This will be hellish, but I will learn something from it'.

I had learned, in my own depression, how big an emotion can be. How it can be more real than facts. And I have found that that experience has allowed me to experience positive emotion in a more intense and more focused way. The opposite of depression is not happiness, but vitality. And these days, my life is vital, even on the days when I'm sad. I felt that funeral in my brain, and I sat next to the colossus at the edge of the world, and I have discovered something inside of myself that I would have to call a soul, that I had never formulated until that day, 20 years ago, when hell came to pay me a surprise visit.

I think that, while I hated being depressed, and would hate to be depressed again, I found a way to love my depression. I love it because it has forced me to find and cling to joy. I love it because, each day, I decide, sometimes gamely, and sometimes against the moment's reason, to cleave to the reasons for living. And that, I think, is a highly privileged rapture. Thank you.

[APPLAUSE]

Thank you.

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# BBC World Service ‘Discovery’ − Depression part 1

## Transcript

**Pauline:**

There were a lot of stresses everywhere. I was trying to get back in to work. Financially we were struggling. I had three children, and I were on my own.

**Aaron:**

I used to have a business which we ended up having to close, and that resulted in losing my employment, I lost my income and ended up losing my accommodation.

**Geoff Watts:**

Aaron and before him Pauline. Two people with problems, but while Pauline suffered from depression,Aaron did not. At times, not surprisingly,Aaron’s mood has become low, but outright depression, no. Some researchers think that one way of helping Pauline would be to understand Aaron.

I’m Geoff Watts and in this, and the next edition of Discovery from the BBC World ServiceI’ll be asking two different but related questions. First,the obvious one. Why do any of us get depressed? Why hasn’t natural selection rid us of this unpleasant and apparently unhelpful state of mind? Why haven’t our brains evolved in such a way that we don’t experience this extra burden of misery when confronted with real difficulties like Pauline’s? I’ll also be turning the question round. As life events able to trigger depression are so common, how come a majority of us don’t get depressed? What is the resilience that people like Aaron seem to possess? And if we could understand it, could we exploit it? But those are issues for next time. It’s the question of why depression exists that I’ll be tackling in this programme. We need to know, not least and on account of the number of people affected by it.

**Professor Bill Deakin:**

We think that about a fifth of the population will suffer from diagnosable symptoms of depression at some point in their lifetime.

**Geoff Watts:**

Bill Deakin is Professor of Psychiatry at the University of Manchester.

**Professor Bill Deakin:**

It seems to be increasing in frequency, and so if you look at the prevalence of depression in the population and the age of onset, the prevalence is increasing and the age of onset seems to be getting less over succeeding decades. You know, we don’t quite know why that is. I mean there’s more diagnosis of depression, and general practitioners,which is where most depression presents, are much more sensitive to detecting it. And, you know, understand much better than they used to that depression can be a manifestation, manifested in different ways and people turning up with physical symptoms, for example, in aches and pains that won’t go away and feeling generally unwell, when the real underlying problem is depression.

**Pauline:**

I think with each time that I’ve suffered from depression, it’s the isolation and emotionally I was very detached. I would come in and sit by my bed and cry. And when it got so bad that I didn’t want to speak to my children, that’s when I went to the doctor’s.

**Geoff Watts:**

Pauline reminding us how debilitating depression can be. So debilitating,in fact,that it leaves you wondering why it still exists. Why hasn’t evolution by natural selection removed this irksome trait and left us happy, happy, happy all the time? Surely we’d then be better adapted to deal with life’s difficulties, or could it be that depression has an upside, some virtue, some benefit of which we’re unaware. And could treating it, in some cases at least, have a downside? One man who studies disease, and its origins from an evolutionary point of view, indeed he practically invented the field, is Professor Randolph Nesse of the University of Michigan.

**Professor Randolph Nesse:**

You know,I started off interested in psychiatry in particular, and quickly realised that I was missing important things that were there in the rest of medicine. And that led me to,really, 20 years, trying to develop the field of evolutionary medicine. It turns out there are new questions that haven’t been systematically addressed. Mainly, ‘Why isn’t the body better designed?’ I mean, why do we have an appendix? Why do we all have wisdom teeth that are so problematic? Why is the birth canal so narrow? Why do we all have so much excessive pain? Why do we get so sweaty when in hot environments? Now these are questions that have two kinds of answers. One kind of answer we call approximate or mechanistic answer, how the body works, that’s most of medical research. But the perspective George Williams and I took on these questions is to try to ask, why on earth didn’t natural selection make the body better? Some things in the body are so great,like the eye and the knee joint and the heart valves, so why leave these other parts really apparently poorly done indeed?

**Geoff Watts:**

It also seems to show why disease is not just arbitrary failure. It makes more sense, I guess, if you look at it in evolutionary terms doesn’t it?

**Professor Randolph Nesse:**

It certainly does. I mean,there are aspects of disease that probably are just arbitrary failure. Cystic fibrosisresults from a mutation and that’s that. And maybe it has some adaptive significance, but I rather doubt it. And so there are different explanations for different kinds of diseases, but for every disease we really need to ask ‘What variations are there?’ and ‘Why do we have bodies that have variations that leave us vulnerable to disease?’

**Geoff Watts:**

When it comes to depression, evolutionary theorists have several ways of explaining its persistence. Not all of them agree with Randolph Nesse, but as we’ll hear, his views do have the advantage of being backed by some experimental evidence. I asked him why he thinks that a capacity to get depressed might be beneficial, might be adaptive. It turns out this was not a good idea.

**Professor Randolph Nesse:**

That’s not the right question in my mind. The question is why we have a capacity for mood.And so many people assume that all depression is abnormal, and a great majority of it is that it gets very confusing if you try to talk about the adaptive benefits of depression per se. I think the question is ‘Why on earth have mood?’ Some people don’t have much mood. They don’t get especially happy. They never get especially sad. They just go on and on, pretty even-keeled. But most of us do have uptimes and downtimes and the question is, why?

**Geoff Watts:**

And the answer is?

**Professor Randolph Nesse:**

There are times in life when investing a lot and taking a lot of risks pays off handsomely. When the crops are ripe, and a thunderstorm is brewing, you had better get out there and gather everything as fast as you can. When you have a big opportunity to be in front of your group and say things that might be useful or interesting you’d better be there and do it well. 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. For some of my ancestors in Norway in the middle of the winter, when there wasn’t any game to get and any time you went out you were liable to die, the best thing to do was?Nothing. And there are times like that, fewer of them in modern societies, but there certainly were times like that.

**Pauline:**

Having the goal of wanting a secure job, and a partner and buying a house and all that and I,just, that bout of depression really made me think,‘Well this job’s making me unhealthy and this relationship is unhealthy for me’. At the time, I thought the best thing to do was to just get out. Yes, it was.

**Professor Randolph Nesse:**

We’re all trying to do things throughout our lives and usually things are working. We’re gathering food and getting friends and helping our neighbours and doing things that are working. Unfortunately there are some things that we’re trying to do that just aren’t working and the more effort we put in the worse it gets. For instance, I saw a patient recently who was desperately depressed, and it turned out the problem was that this person had dedicated her life to helping her child stay off drugs and now he was addicted to heroin. And worse yet,the more she called and the more she tried to help the more he pushed her away. She was dedicating her entire life to this goal, and not making any progress whatsoever. You can readily understand why a mother couldn’t give up on a goal like this. But nonetheless it was plunging her in to a deep depression.

**Geoff Watts:**

So, much of natural selection is about helping us strive for evolutionary goals for survival and,as Pauline’s experience shows, the goals in our lives today shaped by our desires and the society we live in, are still powerful drivers of behaviour and emotion. Randolph Nesse’s view is that an inability to fulfil these goals is what triggers low mood. Surprising as it might seem, perhaps mild depression is in itself a way of building some sort of resilience within sufferers to help mitigate future bouts of low mood.

**Professor Randolph Nesse:**

Much of medicine is using medications to block normal, aversive, defensive responses such as pain, nausea, fever, vomiting and anxiety and low mood. And in most cases it’s perfectly safe, because natural selection has shaped these responses to be greater than they really need to be in the individual circumstance. This is best described as something Williams and I have talked about as the ‘smoke detector principle’. When we all put up with lots of false alarms on our smoke detectors, because we want to be absolutely sure the smoke detector goes off when there’s a fire, even though there is a constant mild annoyance when the smoke detector goes off, when we boil tea or make a piece of toast. Now, likewise, natural selection has shaped the regulation for things like fever and nausea, vomiting, pain and,I think,low mood and anxiety according to the smoke detector principle. These mechanisms are very sensitive. They go off a lot of times when they’re not needed but this is still perfectly normal, because the system has to ensure that they go off in situations when they are needed.

**Geoff Watts:**

So the smoke alarm principle may be one explanation for the increasing prevalence of depression. It’s 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 and Carsten Wrosch hypothesise that low mood helps us to recognise when our ambitions are too lofty. If they’re right about this, depression is delivering a message we do well not to ignore.

**Pauline:**

It’s a part of me. And while the episodes have been quite negative, I do think that I’ve gained a lot of strength from it. I think it has helped me to look at the stress within my life and to seek strategies for dealing with it. So I do think it has been a great benefit to me and the balance that I’ve found just lately in my life has helped a lot. And I don’t think that would have come about so soon in my life if I hadn’t had the depression.

**Geoff Watts:**

So even Pauline, someone who suffered the real misery of depression,can see how it may be helpful in spurring the adoption of new goals.

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# BBC World Service ‘Discovery’ − Depression part 2

## Transcript

**Geoff Watts:**

Last time we heard how low mood in the form of mild depression can actually be helpful. When life in one way or another is thwarting us the experience of depression can make us rethink our goals, can encourage us to scale back our ambitions or try following a different path. This isn’t always possible of course, or at least that’s how it seems. Either way, depression can become chronic and severe and of no benefit at all. All of us face setbacks that can disrupt our lives so you might begin to wonder why we don’t all get depressed. Psychologists like Dr Rebecca Elliott of the University of Manchester speak of people who don’t succumb to these events as having ‘resilience’.

**Dr Rebecca Elliott:**

In the context of mental health we think of resilience as how well somebody is able to adapt and function in the face of stressful or traumatic experiences.

**Geoff Watts:**

Two people who face a marriage break up, one person might get depressed, the other person might not and you’d say the person who didn’t was ‘resilient’.

**Dr Rebecca Elliott:**

Resilient to that particular stressful event. That’s right.

**Geoff Watts:**

I mean it’s a very useful thing to have. Is it widely distributed in the population? I mean, are most people resilient to some degree?

**Dr Rebecca Elliott:**

My instinct says it’s probably one of those occasions where we’re looking at a continuum where at one end of the continuum you have people who are very vulnerable, 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. Whereas at the other end of the continuum you have people who, 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. And my instinct says most of us 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.

**Geoff Watts:**

If resilience sounds more like a description than an explanation that’s exactly right. And that’s why Rebecca Elliott and her colleagues at the university are running a research project designed to find out what makes some people more able to cope than others. In essence, they’re comparing the brains of people with and without resilience. Although it so far has aroused only limited interest among depression researchers, resilience has already called the attention of those who work on post-traumatic stress disorder. And some of their insights, according to Bill Deakin, Professor of Psychiatry at Manchester University, are relevant to both conditions.

**Professor Bill Deakin:**

The three general ideas that we are interested in, are that firstly, some people may inherently be more sensitive to rewards in the environment. So, you know, experience in pleasurable events puts us in a good mood. And some people may need less of that to be in a good mood so they’re kind of more sensitive to rewarding or reinforcing events that occur. So that’s one aspect.

And then the second is that some people seem to be more flexible in how they solve problems, you know, we call is cognitive flexibility. So some people just seem to be more flexible and adaptable and creative about how they get out of their difficulties.

And then the third aspect is the emotional part of the brain. So we know that parts of the brain, particularly the middle parts of the brain, which seems to be a sort of central station for controlling anxiety and our responses to threat, and also responses to loss actually. That some people may be more sensitive, and what sort of loss will trigger off a big emotional response.

We’re trying to find people who appear to be very resilient to adversities of life and others who have been rather sensitive, and to see the mix of those three mechanisms, pleasure sensitivity, emotional sensitivity and cognitive flexibility. How that comes together and how it might relate to genes and, you know, whether it can be changed or not as well.

**Geoff Watts:**

So the characteristics of a brain which is resilient might be thought of in terms of their chemistry or in terms of their wiring and those things would possibly reflect their early experiences and their genetic inheritance?

**Professor Bill Deakin:**

That’s right. The two continuously interact and I think most genetic influences in depression are going to be like that.

**Geoff Watts:**

The Manchester researchers are working on the assumption that different states of mind, including vulnerability to depression, should correlate with different patterns of activity inside the brain. To explore them they’re using the scanning technique known as functional magnetic resonance imaging or fMRI. When volunteer subjects are slid in to the vast circular electromagnets on which these machines rely, it’s possible to image the structure of their brains. And, crucially, see which areas are functioning while they perform certain tasks. As I peered through the window that separates the control room from the instrument suite where the subjects are tested, Rebecca Elliott told me what they have to do.

**Dr Rebecca Elliott:**

They’re doing three different tasks while they’re in the scanner. One is an emotional memory task. We give them pictures to look at which are emotionally charged. So some are happy, positive pictures, some are sad and more negative pictures, and some are neutral pictures. And firstly, we ask them to look at them and try to remember them while their brain is being scanned. And then a little later we show them some of the same pictures again and ask them whether or not they’re pictures they’ve seen before. That’s probing emotional memory, how well people remember material that has an emotional component to it.

The second task we give them is what we call an emotional ‘go/no-go’ task. And in this people see a series of words flashing on the screen and they have to either ‘go’ (i.e. press a button), or ‘no-go’ (don’t press a button), depending on the emotional content of that word. So we have some blocks where they have to go to happy words and not go to sad words. Some blocks where we reverse that.

And then the final task we do is a task looking at their responses to rewards and punishments. And we actually use a task where they either win or lose money. And we look at how the brain responds when either they gain something, they win some money or when they lose some money.

**Geoff Watts:**

And in each case the hypothesis that you have is that people who have higher levels of resilience will show different pictures of activity from people who have low resilience?

**Dr Rebecca Elliott:**

That’s right. So at one end of our continuum, the ‘vulnerable’ people, we expect to show one pattern. The ‘resilient’ people at the other end, another pattern. And both of these will differ from, if you like, the average pattern that we see from people in the middle.

**Aaron:**

I’m generally a happy person, a busy person. I mean everybody has sort of stressful moments, I suppose, in their life, but day-to-day I would class myself as fairly happy. There’d be something wrong with you if you were happy all the time. But no, I certainly wouldn’t consider myself as ever having an episode of depression.

**Geoff Watts:**

Aaron, one of the people who volunteered to take part in the Manchester study. As you’ll have guessed he’s among those chosen for their resilience.

**Aaron:**

I suppose from my childhood when I first realised that I was gay, and lived with that for a long time. That taught me to sort of build up, I suppose, a sort of strong defence mechanism. And I didn’t actually come out to my parents or friends until I was much older. And so I lived with that for a long time. I suppose if you’ve gone through that situation whereby something is always in the background that is sort of fairly personal and stressful, I think perhaps that has made me deal with situations in a better way later on in life. I would say that my life can get fairly stressful at times, but it doesn’t seem to affect me in the way that it perhaps affects other people. I think it’s perhaps to do with my outlook on life. I do tend to think that if there is a problem that there’s always a solution, and I, kind of, think my way out of any problems. So it’s not that I don’t sort of think about any issues that might be going on in my life, but I always think that you can find a solution to any problems.

**Professor Bill Deakin:**

Well, I think that’s one aspect of being resilient. Certainly being optimistic, cheerful people. Think of your friends, you know, some of them are more cheerful and optimistic. There are some that don’t turn things over. Others turn things over a lot and ruminate about things. So using those sorts of aspects of personality I think we automatically think of some people as being resilient and tough, and others as being sort of sensitive, perhaps more liable to depression.

**Geoff Watts:**

Most of us do manage most of the time to stave off depression. So to understand resilience you also need a better understanding of ‘normality’, hence Bill Deakin and Rebecca Elliott’s attempts to peer inside the brain, looking for differences between subjects who do show resilience to depression and those who don’t.

**Dr Rebecca Elliott:**

Two particular regions that previous work have suggested may be important are a region of the brain called the amygdala, which we believe is important in processing emotional information. So we know, for example, 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. Another part of the brain we’re interested in is a part of the prefrontal cortex. And the prefrontal cortex is important in our cognitive performance, and to some extent in controlling our emotions. So regions of the prefrontal cortex will tend to inhibit the functions of regions like the amygdala. And we see this imbalance in depression, and in other emotional disturbances, and our hypothesis is that we’ll see, if anything, the opposite pattern in people who are resilient. Our early data suggests that people who are more resilient are more likely to recognise happy faces and less likely to recognise sad or fearful faces. We’ve also shown that people, exactly in line with what we hypothesise, the more resilient somebody is, the better they remember positive words and positive pictures.

**Geoff Watts:**

In the last few decades more and more hopes have been pinned on neuroscience as the means by which we’ll get to grips with mental health problems. But there are those who urge caution in this approach. One such is Professor Randolph Nesse of the University of Michigan. He tries to make sense of illness including depression by studying its evolutionary origins. It was he who explained in our last programme why mild depression may be beneficial. So what does he think of a ‘neuroscientific’ approach?

**Professor Randolph Nesse:**

A full understanding of anything in biology requires knowing every detail of the mechanism, and also how that mechanism came to be, and how it might be useful. And I have grand hopes along with most everybody else in psychiatry who is sensible that we will find specific brain abnormalities, or I should say differences, that account for why some people are more vulnerable to depression than other people who are less vulnerable to depression. However, I do see some neuroscientists acting as if the entire solution is going to be found in finding specific brain abnormalities. And it’s been very discouraging to actually look at what we’ve discovered over the past 20 years. Is there anything that can distinguish a brain of someone who has depression from a brain of someone who does not have depression? No. Not the brain scan, not the brain cutting, not the microscopic examination, not the hormones, not the neurotransmitters. There are some differences which are good clues, important clues. But our hope that we would find something specific like we find with multiple sclerosis or something has been completely dashed. 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.

**Geoff Watts:**

The hope of the Manchester team is, of course, that whatever insights they glean about the nature of resilience will offer ways of improving or better targeting existing treatments to prevent depression from taking hold. They’re aiming to build on what’s already done. Professor Deakin.

**Professor Bill Deakin:**

I mean, we do try and promote the resilience against recurrence of depression. And a lot of psychological therapies are based around that. So there’s quite good evidence that cognitive behavioural therapy, different psychological approaches, interpersonal psychotherapy, for example, a powerful technique, is a good way of reducing the chance of having a further episode if you’ve had depression. So by having depression you’re, kind of, shown to be perhaps a vulnerable person, not a resilient person.

Better psychological approaches could be devised, and enhanced by new kinds of medication that promote learning. And the combination may be particularly powerful in preventing relapse from depression. Because once you’ve had depressive illness the chance of having another one is very substantially increased over the general population. So it’s an important issue. And so that’s where we move out of society and in to the clinical situation. People who have had depression trying to promote resilience mechanisms to stop it happening again because we know it’s a big risk.

You might be able to in the future make a sort of a neuroscientific diagnosis so you get a readout of perhaps a simplified form of a scan or some EEG. Or you might get a sort of neuroscientific profile of what the particular problem is. So, in this individual, this individual got depressed because their reward mechanisms were completely fused, and actually from the emotional processing point of view and from the cognitive flexibility point of view they’re fine. So the target of therapy has to be on sort of promoting ability to respond to reward and the target might be something else. So that would really tailor make the therapy that you would have to reduce the chance of you having a second episode of depression.

**Geoff Watts:**

While cautious about the concept of resilience and the methods being used to study it, he applauds the intention and in particular the attempt to compare people who do get depressed with those who don’t.

**Professor Randolph Nesse:**

This is one of the wonderful things that’s come from positive psychology is studying not just people who have severe problems, but the people who don’t have problems despite being exposed to very dire circumstances either early in life or in adulthood. And this is very important research. We need to know how people differ and why some people just can get through things that other people cannot.

The difficulty though, is assuming that resilience is always good. Resilience is wonderful and especially in modern life where low mood, I think, is probably less useful than it used to be. The more resilient you are, up to a point at least, the better. But I really think it’s terribly important to try to, you know, be sympathetic towards people with depression not just as people with diseases who are somehow less able, and less fit than others but with people who have advantages as well as disadvantages.

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# Oxford University Podcast − What is depression?

## Transcript

**Dr Danny Penman:**

Hello and welcome to the ‘New Psychology of Depression’, a series of programmes with me, Dr Danny Penman, and Professor Mark Williams of Oxford University.

We live in a world filled with material wealth. We have never had so much political and economic freedom. We live longer and healthier lives, and yet anxiety, stress, unhappiness and depression have never been more common. Why is this, or, perhaps more importantly, what can we do to stem the rising tides of these mental health problems?

Mark, what exactly is depression?

**Professor Mark Williams:**

Well, depression is a syndrome. It’s a combination of symptoms that occur at the same time. So most people know what sadness is, most people know sometimes how difficult it is to get out of bed in the morning, this sort of thing. Depression is different from that. It’s low mood, feeling hopeless, feeling very sad and listless but also it can be lack of energy and enthusiasm for things that you used to actually quite enjoy. And those are the core symptoms of depression, but even then if you get those for a couple of weeks and they go on and on and on, that’s not enough to get you diagnosed with depression.

So there are other symptoms as well, and any combination−four of five of these other symptoms−are often considered necessary for a diagnosis of clinical depression. And they are things like changes in appetite, perhaps even weight loss or sometimes people eat too much and weight gain. Changes in sleep so that some people don’t get to sleep at night, or they can’t sleep in the middle of the night, or they wake up very early in the morning. Occasionally, especially with something called ‘seasonal affective disorder’ it’s sleeping too much and not, you know, feeling that you, you know, you want endless sleep. But generally, with depression it’s not sleeping, it’s insomnia.

And then there are things like feeling guilty, lack in concentration, feeling agitated or very slowed down. Feeling tired all the time, and even many people get suicidal ideas, ideas they’d rather be dead, that they’re just a burden to their family. Now what depression is, is these symptoms coming together most days for at least two weeks. In general, however, they go on for months. And that’s when you’d get a diagnosis of depression. At the point of which these things prevent you from living your life as you want to live. So it’s what might be called ‘functional impairment’. You can’t function and you can’t explain it in terms of illness, your physical illness. You can’t explain it in terms of a recent bereavement. This comes and stays and you don’t seem to be able to get rid of it.

**Dr Danny Penman:**

So how does it interrelate with other problems that we all suffer from, from time to time such as anxiety, stress and, you know, things like mental exhaustion?

**Professor Mark Williams:**

Well they’re very closely related so you very rarely get depression without having other things like high anxiety at the same time. Depression is often characterised by, you know, dwelling on the past a lot, but you hardly ever get that without people also worrying about the future and being anxious about the future.

Psychiatrists and psychologists usually put anxiety and depression in separate camps, but the new genetic evidence is suggesting they’re much more dimensional, they’re much more mixed and also the treatments that work for depression tend to work for anxiety as well. So there’s quite a lot of evidence that actually to make a too big a separation between anxiety, stress, depression, exhaustion isn’t quite what is going on in the world.

**Dr Danny Penman:**

Are things like anger, irritability, road rage, you know, typical explosions that you see, we all see every day. Are they related to depression at all?

**Professor Mark Williams:**

They can be. I mean, they’re more likely to be related to stress because you often get, in high chronic stress, you get people showing a lot of anger. But also, and it depends on the age groups, so, for example, in adolescent depression there can be quite a lot of anger, irritability and hostility, which is how often within that age group a lot of sadness is expressed. But you couldn’t get a diagnosis just from being angry all the time. You’d need some of these other things like weight loss or an appetite change, sleep change and that sort of thing.

**Dr Danny Penman:**

Depression is increasing worldwide. Is it increasing predominantly in the developed world or it is also increasing in the third world?

**Professor Mark Williams:**

Well, what we now know is that depression is rapidly becoming one of the biggest reasons for people to have, basically, lose years of their effective life through disability. So the World Health Organisation publishes data decade by decade over that and about two decades ago they recognised that depression is becoming a big problem. Well, it’s now arrived. They have a statistic of the years’ life lost to disability, and in high- and middle-income countries depression is the top of the list for that. So it’s higher than the disability caused by heart disease, for example, cerebrovascular disease, road traffic accidents, and even in low-income countries and very low-income countries it’s still in the top ten of years lost to disability.

**Dr Danny Penman:**

So depression actually exacts a bigger toll on society than cancer and heart disease and other things like osteoarthritis?

**Professor Mark Williams:**

Indeed, in terms of its global impact. Of course, there’s the suicide impact as well, nearly a million people die prematurely by suicide each year across the world. But also there’s the more hidden cost as well as the big cost of things like suicide. People feeling like they can’t function, feeling like staying in bed rather than getting up, which is not just laziness. This is depression, as it were, that’s doing this to them. It affects the ability to be a breadwinner for your family, the ability to look after your family, and that’s why it’s such a burden right across the globe.

**Dr Danny Penman:**

Can you give us some figures as to the prevalence of depression both in the developed world and the developing world?

**Professor Mark Williams:**

So, in high income countries, for example, depressive disorders, which is what we’ve been talking about, account for about 14% of the years’ life lost to disability. And putting that in proportion, if you look at Alzheimer’s and other dementias that’s about 5% of the years lost to disability. If you look at osteoarthritis it’s 4%. If you look at chronic obstructive pulmonary disease that’s about 3%. So 14% is huge compared with that.

And then if you look at low- and middle-income countries, then depression is about 10% of that and the next in line for the years’ life lost to disability are eye problems, what’s called ‘refractive errors’ and that’s about half that, about 4.7%. So again, in terms of the years' life lost to disability, it’s huge.

What epidemiologists seem to find wherever they look is about one in 20 of a population are depressed at any one time, and about 20% of the population at some point will get very depressed and that’s a major problem.

**Dr Danny Penman:**

Every time I hear these figures I’m just completely stunned. What’s the fundamental driving force behind the increase in depression?

**Professor Mark Williams:**

I think there’s always been a proportion of the population that has felt depressed. As far as we can go back in history you can look in the Psalms, in the Hebrew Old Testament, as it were, and you see people expressing, you know, sadness and depression and anger and irritability and this sort of thing. If you go back way in to the beginning of something like Buddhism, for example, 500 BC and you have people needing to learn to meditate, and to deal with the problems of the mind. The problem of the entanglement of the mind, as it were. So I think it’s been around a long time.

One of the things, however, that’s new is over the last 50 years in the Western world, the age of onset of depression has changed. It’s become earlier and earlier and earlier. And that’s one of the major discoveries. It started to emerge from epidemiology where big surveys started to pick up that people were beginning to report, from about people born in the 1950s onwards, when they were assessed towards the end of the 20th century, they started to report that their depression had started a bit younger than we had previously thought.

It was previously thought that depression was a bit of a late life problem. Late 50s, early 60s. And indeed the evidence that people born in the first part of the 20th century seemed to verify that. But decade by decade from about 1950 or 60 onwards the age of onset got younger and younger. And so by the 1980s and 90s, people were beginning to get depressed in their 20s. And there’s been a striking confirmation of that in the last few years.

One of the biggest clinical studies ever conducted was done in America. It was called the STAR\*D Trial and it was ‘ST’, the beginning of STAR\*D, it means ‘Sequenced Treatment’. So it was a big trial to look at what treatments provide, one treatment after the other, after the other. So they had 4,000 people who volunteered to take part, and as part of that trial they were asking them 'When did you first get depressed?' And they found that the mean age of onset in this sample was about 26, but actually the most common age in which people started to get depressed was between 13 and 15 years old. And that is an astonishing new development.

**Dr Danny Penman:**

Is the same true for, kind of, anxiety and stress and irritability and ...?

**Professor Mark Williams:**

We weren’t clear about that until about the year 2000, where somebody published a big paper in one of the big journals in America. They’d traced the anxiety level of children and young people over the 30 years from the early 1950s all the way through to the mid-1980s, and they found exactly the same thing. That it looked as if the anxiety pattern in children and young people had become, in a sense, the whole bell shaped curve had shifted towards greater anxiety. And by about one standard deviation which means that whole swathes of children and young people who hadn’t been anxious in the 1950s, as it were, if they were born in the 1980s, then they were likely to show anxiety which had been virtually at clinical levels 30 or 40 years ago.

**Dr Danny Penman:**

I think earlier you said that what we regard as ‘normal’ levels of anxiety and stress would have been regarded as a clinical level anxiety and stress 50 years ago. Is that true? Did I miss understand that, because that’s quite an astonishing figure?

**Professor Mark Williams:**

That’s true in anxiety. And now that we know the same is true of depression. That is that 50 years ago people would live out most of their life without getting these crashing depressions. But now that, you know, 35, 36% get depressed before the age of 18, then there’s a whole life ahead of them which is the biggest challenge. A whole life ahead of them where they might actually get another depression, because one of the things we now know is if you’ve been depressed once you tend to get depressed again, at least in half the cases. And once you’ve been depressed twice then the chances go up even further.

**Dr Danny Penman:**

So does this suggest that depression is really a problem of how we deal with the world, the way we think rather than a chemical imbalance in the way our brains are actually working?

**Professor Mark Williams:**

Well, one of the inferences we can make from these big changes being so rapid and recent is it can’t be genetic changes. It can’t be driven by our basic biological makeup. So there must be something else happening. And it must be environmental changes that have driven that. I mean, clearly it could be that people are just recognising depression that was always there, but now they’re recognising more. But the fact that both clinical studies and epidemiological studies show up the same thing argues that it’s not just that people are recognising it better, because you control for the sort of questions you can ask and check that the questions asked are the same over those decades.

But the other thing is that we know that after about 1977, you got increases in changing rates of suicide as well. Especially in young men, right the way over the Western world, you got increases in suicide rate which to some extent mimic this younger and younger depression hitting. Now, that’s stabilised over the last few years, but the fact that you get really confirmation from another area that we know is likely to have been affected by this change in pattern of depression I think shows that something is going on here. It’s very difficult to understand exactly what the causes are. Changing patterns of society, the increase in the gap between rich and poor, the fact that when economies develop rapidly often there seem to be some almost unavoidable changes in the gaps between rich and poor.

We know that in the countries that have the least gaps between rich and poor, then the levels of stress, the levels of trust even within the communities are higher. The levels of ‘hope’ are higher, and people tend to live longer in those societies. So there are little bits of evidence that people are beginning to put together to suggest what the changes might have been. But what we’re working on are treatments and approaches which can now deal with this depression epidemic.

**Dr Danny Penman:**

If you had to choose fundamental driving forces behind depression, what would they be?

**Professor Mark Williams:**

Well, at the individual level they’re the way we think about life. So where we feel we are, as it were, in relation to other people, in relation to our own standards, and the standards that other people set for us. So one of the ways in which society is changing, is the way in which it expects us to do things, the targets it sets for us and so on. And when society sets us targets and says 'Meet those or else!', then the best you can hope for from your work is relief when you’ve met your targets.

In other words if you could do a good job, and feel pleased that you’ve done a good job because of your work, you know, if you’re a heart surgeon and you’ve saved, you know, several lives this week, then that must be a really good sense of worth about what you’ve done. But what happens if you’ve got a target to save six lives this week. All you do is when you’ve saved six lives is think 'Ah,thank goodness that I’ve met my targets', you know?

So suddenly it turns the possibility of satisfaction with a job well done, into relief that you haven’t made a mistake. So you’ve turned potentially something really enjoyable about life into a thing that isn’t going to, as it were, give you the motivation. So gradually, that can work for a while, you know, but gradually it can eat away. I think. at you in ways that are a bit pernicious.

**Dr Danny Penman:**

So how could you, for example, you know, if you’re an omnipotent dictator, how could you change society or, you know, perhaps if you’re running a company how could you change the culture of that company or of that society to actually promote mental health and wellbeing?

**Professor Mark Williams:**

Well. people talk about ‘work-life balance’ don’t they? And that’s really important. It’s also to recognise that productivity needs engagement, and engagement needs a sense of control, a sense of choices. So as you go down companies traditionally you get people having less and less choice about what they do. And if you can find a way to increase the choice then you naturally increase the creativity. We know that when a company’s run on just target lines and so on, in this stressful way, that actually people. in order to meet their targets. they feel very stressed. They put in more hours, but they aren’t necessarily more productive, because they’re not seeing the whole picture.

And if you want employers to see the whole picture, and if we want us in our family life to see the whole picture, then you have to learn to attend and you have to learn to see the whole picture by reducing stress. And there are things we can do about that. And mindfulness, which is what I’ve spent much of my life researching over the last 20 years. is one of the answers to that.

**Dr Danny Penman:**

Does depression inevitably return or, you know, is it possible to just have one episode of depression and, you know, that’s it for the rest of your life. You get over it, you dust yourself down and carry on with the rest of your life. Is that possible or does it tend to return?

**Professor Mark Williams:**

OK. Depression can be a one-off, so it’s not inevitable that it will return. So it’s a bit of a message of hope. If we’d said 'Oh it’s always going to return', then it’s a counsel of despair for many people. On the other hand, if you’ve been depressed once you do have a slightly increased threshold, and it rather depends why you got depressed. If you got depressed because of a big life event, for example, bereavement, unemployment, separation − the sort of reasons that would make any of us low − then so long as you don’t have a repeat of those sort of events then you don’t necessarily, you’re not necessarily going to get depressed again.

But the problem is that if you get depressed the threshold for you getting depressed again is slightly altered, and if then you get depressed for a second time, what we know is the triggers of a third depression are less. So, for example, you might need bereavement or unemployment the first time you get depressed, the second time it might be something slightly less of a stressor, but then the third, fourth time then you may not need a stressor at all. By the time actually you’ve had three, four, five depression it may be that you just wake up one morning feeling a bit low and by the end of the day you’re feeling very depressed.

So the statistics suggest that about 50% of people might have a one-off episode and then it doesn’t bother them again. But if they’ve been depressed twice, the chances they’ll get depressed again are much higher. Three times, the rate’s at about 70 to 80%. And in our studies we find, for example, if we follow people up for about twelve months without offering any treatment to them, then if they’ve had three depressions in the past before they come to see us, then between 60 and 80% of them will get depressed again in those next twelve months.

**Dr Danny Penman:**

People who become repeatedly depressed year after year, what proportion of their lives do they actually spend in that depressed state?

**Professor Mark Williams:**

Well, we didn’t know the answer to this question until fairly recently. And there’s some researchers in the States who’ve done a long-term follow-up of a study called the NIMH, that’s the National Institute for Mental Health. They started a study in 1975, and because they’re able to keep in touch with this large number of people that started then, they’ve been able to look at, you know, how much time do people actually spend depressed if they’ve been repeatedly depressed. And the figures are staggering. What they found was, and it’s just been published in the last couple of years, is that people on average spend 32% of their time in episode over a 20-year period. So on average four months a year are spent in episodes of depression which, I mean, considering the burden that we talked about earlier, it’s an incredible statistic.

**Dr Danny Penman:**

That’s a really disturbing statistic. What proportion of people then go on to begin self-harm or even commit suicide?

**Professor Mark Williams:**

There are changing rates of self-harm and suicidal behaviour, and the definitions vary, so self-harm sometimes mean people who harm themselves physically − cutting themselves and so on. Deliberate self-harm is sometimes broader than that. People that take overdoses and so on. And we know that the rates of suicide in people who’ve been seriously depressed are elevated compared with the general population.

Now, about 1% of the population die by suicide anyway. If you’ve been depressed in the past, that’s likely to be higher, about 4 or 5%. If you’ve been depressed and been an in-patient at some point in your life and been hospitalised for depression, it can be as high as 10 to 15%. So one in six, one in seven people die by suicide if they’ve been an in-patient, hospitalised for depression. But of course it’s not inevitable. Most people don’t die in this way, but it’s always a tragedy when it happens for the family left behind, and for the friends and colleagues of the person.

**Dr Danny Penman:**

Is suicide always linked with depression?

**Professor Mark Williams:**

It’s the most closely linked problem. You get elevated risk of suicide in other mental health problems like schizophrenia, for example, or bipolar disorder. But it’s the depressive aspect of schizophrenia, so many people can survive, as it were, having serious mental health problems in an amazingly courageous way. But depression takes away that hope. And it’s when hopelessness comes, which tends to come with depression, that people become at greater risk of suicide whatever the condition it is. It’s the occurrence of depression that accounts for that.

**Dr Danny Penman:**

Do people tend to kill themselves as they come out of a depression rather than when they actually have the kind of hopelessness and the lack of energy when they’re in the teeth of a depression?

**Professor Mark Williams:**

There’s quite a lot of clinical evidence for that. It’s never been proven by research. It’s a very difficult thing to prove, but many people have said that they thought that the person was actually feeling happier now, and the natural inference is exactly as you suggest. That people when they’re very, very depressed actually have very little energy and that it’s when the energy starts to come back, but the mood has not yet improved that is a very dangerous time.

It’s also true to say that some of the big studies that Lewis Appleby and others have done in the United Kingdom have found that the most vulnerable time is a time just after a discharge from hospital, for example. After a change in medication which reflected, very often reflected the fact that their physician thought they were feeling better. So that’s also indications that when people are on the mend and people around them think they can stand on their own two feet that, almost, that transition, is a very difficult time for people.

You were mentioning about the close links between depression and suicide, and asking about that. And somebody’s calculated actually how much of the suicide risk in the world could be eliminated if we could eliminate depression. And it turns out to be about 80%, because of the very close association. People don’t tend to be suicidal outside an episode of depression. Depression is the thing that, as it were, the final common pathway, depression and hopelessness.

And one of the interesting things that’s emerged in the last few years is that when you get repeated depressions, you can get depressed again and again, but different symptoms can be there each time. So that always those core symptoms seem to be there, like low mood and lack of interest. But the other things like weight loss, sleep loss, guilt, they may or may not be there. But our research in Oxford has found that of all the symptoms that recur, when depression recurs suicidal feelings are the most recurrent.

And of course that’s important clinically to realise because it means that often doctors will ask somebody who’s depressed whether they feel suicidal. Well, that’s an important question to ask but it’s more important to ask 'Did you feel suicidal when you were last depressed?' Because if people felt suicidal at their worst ever time, then there’s some chances that during this episode at some point they’re going to feel suicidal. So, clinically it’s important and research-wise it’s important to get to the bottom of what are the characteristics of recurrence so we can begin to help.

**Dr Danny Penman:**

So what exactly does a full-blown depression feel like?

**Professor Mark Williams:**

It’s a combination of experiences of a distortion as, you might say, in the way you think, the way you feel, the body and your impulses. So if you take each in turn. Your thoughts are dominated by ideas of helplessness, rejection, being a failure, not being good enough, not being worth your space in the world. You feel like the lowest of the low. And that nobody wants you, nobody likes you and that even if they do like you that’s because they haven’t found out the truth about you, you’re just a fraud and as soon as they find out what you’re really like, they’ll reject you. So you’re thoughts are dominated by that.

That becomes habit. So that although many of us might think like that for, you know, once or twice a day or a week or a month, in depression it just, like, comes all the time. I mean, many of us know what it’s like to wake up in the middle of the night, for example, and not be able to get back to sleep and our thoughts go round and round and round. We just ruminate and brood. Well, depression is like that sort of middle of the night thinking, but it happens during the day as well.

Secondly, your feelings get bombarded. There’s feelings of sadness, of hopelessness, of worthlessness and they’re very closely tied in with your thoughts. If you could imagine somebody standing behind you all day saying how useless you were, then sooner or later you’d feel sad, irritable, run-down, exhausted and a miserable failure. And that’s the way in which the feelings reflect those thoughts.

It’s not just a mental thing, your body slows down. You lack energy. Your body fails to work in an efficient way, so you don’t sleep well. You don’t eat well. And this itself feeds back in to your sense of fatigue and slowness, lack of energy. Either being in some cases very agitated, in some cases being very slowed down.

And if you look at the way people walk when they’re depressed, for example, their gait is very different. Not, as it were, walking upright, walking slouched and going from side to side instead of actually more steady on their feet.

And lastly your behaviour is affected. Either you feel suicidal, but also you feel like withdrawing from the world and that sense of withdrawal, of not wanting to see things. Now once again most of us have had times in our life when the phone rang and we said 'Oh no, do I have to answer that?' Or when we didn’t want to get up in the morning and anybody could have seen we were, you know, quite sort of withdrawn. But that goes on relentlessly. It feels like it goes on relentlessly in depression. And when you put these together, the thoughts, the feelings, these body changes and your impulse is just to act, or your behavioural tendency is to withdraw, then that is what drags you down. It’s not surprising then that people feel the burden, and can’t function when they’ve got all of this going on in their life.

**Dr Danny Penman:**

So is there any one thing that drives people or tips people over the edge from normal ‘run-of-the-mill’ sadnesses, or periods of rumination or reflection, into a period of full-blooded depression?

**Professor Mark Williams:**

There are, and actually I’d like to tackle that in greater detail in later episodes, because it’s exactly what tips people into other episodes that the research is most exciting over the last 20 or 30 years. And out of that comes the interest in mindfulness research. So that’s something that I think we’ll be able to go in to detail in future episodes.

**Dr Danny Penman:**

So that implies it is possible to stop a depression, or rather normal feelings of unhappiness that we all experience from day-to-day. It’s possible to stop that, and prevent it from tipping over the edge into a clinical depression?

**Professor Mark Williams:**

That’s the most exciting development in the last 20 years. And the way in which mindfulness is able to help people to notice when the tipping point is coming and allow you to deal with what you’ve got then without going down into the depths is,I think, one of the major things.

**Dr Danny Penman:**

And that must have huge clinical relevance?

**Professor Mark Williams:**

It’s got huge clinical relevance because if we could find and I’ll describe that in the later episodes. If we can find that this is actually as useful as antidepressants or as other treatments, it’s of global significance because it doesn’t depend on medications which in some contexts are just too expensive for people to purchase.

**Dr Danny Penman:**

Thanks very much for that, Mark. In this episode we were talking about the ‘New Psychology of Depression’ and what exactly depression is.

And in the next episode we’ll be talking about the major treatments for depression and how they’ve changed over the last century or so. For further information about the issues raised in this programme you can read ‘The Mindful Way through Depression’ by Professor Mark Williams and his co-workers, or you can read our book ‘Mindfulness: Finding Peace in a Frantic World’ by Mark Williams and me, Danny Penman. Or you could visit our website: franticworld.com

If you’d like to support further research in this area you could visit: oxfordmindfulness.org and follow the links to the development campaign.

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# Oxford University Podcast − How is depression treated?

## Transcript

**Dr Danny Penman:**

Hello and welcome to the ‘New Psychology of Depression’, a series of programmes with me, Dr Danny Penman and Professor Mark Williams of Oxford University. In this episode, we’ll be looking at the treatment of depression, and how it has changed over the last few decades or so.

Mark, at the moment, what are the main ways of treating depression?

**Professor Mark Williams:**

Well there’s two big approaches, physical treatments and psychological treatments. Those are the main two approaches, but there are many psychological treatments. I’ll come to those in a moment. Of the physical treatments, there are some that are fairly rarely used nowadays that used to be used a few decades ago like ECT ‘electro-convulsive therapy’ treatment. But the main physical treatment is antidepressant medication. They’re very, very widely used now all over the world, and they work pretty well.

**Dr Danny Penman:**

So, anti-depressant medication. They obviously affect brain chemistry. How in detail do they work?

**Professor Mark Williams:**

Well, in the brain there are many, many different receptor sites, for example, the way in which nerve cells work is an electrical impulse running down the nerve cell, but then it comes to a junction. And then the current, as it were, the impulse has to be transferred from one nerve cell to another and that’s called the synapse, the junction of these two things. And that’s the point where chemicals come in, because those neurotransmitters are released from one end and are picked up at the receptor sites of the other one, and then the transmission continues.

Now there are millions and millions of these in the brain, and so much of our functioning, our eating, our sleeping, our mood, everything is governed by the action of neurotransmitters in the brain. And what we know is that when they get unbalanced, then, depression can result.

There are many neurotransmitters. The ones that are most thoroughly studied are noradrenaline and serotonin. And what we know is that the antidepressants that work, are often antidepressants that effectively increase the availability of these substances in the brain. They usually do so by blocking reuptake. What that means is that usually when the neurotransmitter is released, if that synapse is going to be effective it can’t linger for very long, so there’s got to be a mechanism for as it were, ‘gobbling up’ the neurotransmitter. So when the next impulse comes, you know, it can release. So that’s called ‘reuptake’.

Now if you have a reuptake blocker that means that when a neurotransmitter has been released it stays around for longer. That’s one of the ways in which it works. Now we know that it takes some time, it takes a few days for most antidepressants to, as it were, kick in and affect your mood in a noticed and significant way that you actually notice it. Although they start working straightaway the fact that it takes a little time suggests that it’s not just more neurotransmitter that’s the critical thing, but it’s actually changing the balance. The sort of change in the balance of sensitivity in the cells at the, sort of, sending and receiving sites.

**Dr Danny Penman:**

So is depression caused by low levels of these chemicals in the brain such things as serotonin, or is it more complex than that?

**Professor Mark Williams:**

It’s probably more complex than that because if it was, you could just give things to increase the levels and people would be instantly happier. That doesn’t happen. So what people think is that there might be something in some of the receptor sites. For example, they might be just insensitive. Now if you get a receptor site insensitive, just increasing the amount of available neurotransmitter isn’t going to work instantly, so that might be something.

We also know that the transporter gene, that’s the genetic, as it were, messenger, mechanics of getting the stuff available to the brain, that might also be effective. And there’s quite a lot of interesting work now at the frontier of this science looking at serotonin transporter genes, for example.

**Dr Danny Penman:**

How effective are these drugs? Are they are all similarly effective?

**Professor Mark Williams:**

Well, they seem to be. I mean the drugs that were introduced in the 1950s and 60s−what are called ‘tricyclic’ antidepressants−are pretty effective. The new generation of antidepressants are about as effective. The major aspect of them is they’re less toxic in overdose, for example. They’re safer, they don’t have so many side effects. Of course, side-effects are always an individual interaction between the profile, the biological profile of the individual taking them and the biological profile of the medication. And therefore it’s impossible to predict exactly what side-effects come with each antidepressant.

So, very often a physician will try a person on one antidepressant, and then say 'Come back and tell me if this suits you' and 'Try another one of the same or a different class', if it doesn’t. So there is a part of this which is experimental and it’s always of discovery for the physician and the person themselves in discussion, to decide what works for them. But if you ask whether they work? Well, it’s pretty controversial. It looks as if about 60% of people in most studies, you know, if you go on for a year you’ll get remission or recovery. That’s depression really going down to bearable levels. and maybe there’s only one or two symptoms around in any one week instead of these crushing, weighted depression. About 60% will recover.

Later data from things like the big STAR\*D Trial I was referring to in episode one, that suggests that after twelve weeks on one form of medication, then about 30% will respond to that. If you then change to another antidepressant, a further 19% will respond to that. If you then change to another in those who haven’t actually responded to either of those first two, then another 14% will respond to a third.

Now that cumulative rate of about 47% over one year is slightly more pessimistic than many would have hoped. And there’s new evidence all the time on the effectiveness of antidepressants, and some of the big meta-analyses, that’s when people get all the data, all the studies together, are beginning to suggest that from ‘mild’ depression, or perhaps even some ‘moderate’ depression, antidepressants don’t work much better than a placebo, a sort of sugar pill. But for ‘severe’ depression, which cause the greatest amount of problem, then you do see clear blue water between the antidepressant and a placebo.

Now, having said all that, of course, your doctor, if you go to the doctor, will never give you a sugar pill, as it were, if you’re not part of the research trial where you’ve got consent to do that. And therefore the fact that you get both a placebo effect and a therapeutic effect can be for many people a life saver, as it were. It does change lives. It does change moods. So nothing about the psychology or the ‘new psychology’ of depression means that these are now irrelevant, or should be substituted for psychology. For many people they’re a life saver, but probably the best estimate is 50 to 60% of people responding.

And, of course, one of the things that emerges from that is, well, what about the other 40%? And also what about the people who don’t respond to one or the other? And, of course, some people don’t want to stay on antidepressants for a long time. The major problem with antidepressants is this, that when you stop taking them, let’s say you take them for six to nine months or a year, and it deals with the depression and then you feel stable. If you then come off antidepressants then your risk of getting depressed again returns to the risk that you had before you started taking them.

That means if you’ve had three episodes of depression, and your risk was 60 to 70% you can deal with that with antidepressants for, say, a year, two years. When you come off them your risk goes back to 60 to 70%. If you’ve had five depressions in the past and your risk is 90%, when you come off antidepressants your risk goes back. Now you might not become depressed immediately, but a recurrence is, of course, a very, very damaging and disappointing thing to happen when you thought you’d coped with it.

And therefore many physicians are saying 'Well, why not go on antidepressants and stay on them?' If they suit you, treat it like diabetes, you know, like it’s a permanent lifetime thing you’ve got, treatment of the diabetes. But the trouble is that many people don’t want to be on drugs for the rest of their life. Some people naturally come off.

So in that long-term study from what I was telling you that started in the 1970s, virtually everybody after a year had spontaneously come off their antidepressants. We know from recent data that 30% of people don’t even go back for their repeat prescription. So there’s a large, sort of, weight of evidence that people naturally come off despite their doctor’s advice, they come off antidepressants. And then of course they are at risk of getting depressed again.

**Dr Danny Penman:**

Is that risk a withdrawal symptom or is it just they’re returning back to normality and, you know, the world hasn’t changed?

**Professor Mark Williams:**

It’s a good question. If it was just a withdrawal system then you’d expect the depression to come back quite quickly and that does indeed happen when you come off your antidepressants, say, within three months, four months, five months, six months. And when you think about it imagine that a depression was going to last four, five, six months naturally. We know that depression naturally gets better if you don’t do anything with it. It tends to be episodic. That means it comes, it’s crushing when it happens, but then you get over it.

Now if you take antidepressants for an episode of depression, then if you take your antidepressants for a few weeks, the depression,as it were, is still there underneath, the biological process is still at work. If you come off prematurely the depression comes back pretty quickly. That’s what’s called a ‘relapse’. And that’s where you get this sort of kick-back effect. However, if you’ve taken antidepressants for six, nine months, a year, that has covered that episode of depression. And when you come off, you’ll be OK for a while probably. But then there’s what we call a ‘recurrence’ as opposed to ‘relapse’, which is that within a year or two the depression tends to start again.

So it’s probably not just a kick-back effect, a withdrawal, although some of antidepressants are harder to come off than others and some, I mean and you always of course need your doctor’s advice. If you’re thinking of coming off antidepressants you always go and see the physician who has given you them or has prescribed them and ask their advice and they’ll help you. Because some are easy to come off, others are much more difficult and you can get a lot of other symptoms, a lot of flu-like symptoms from some, for example. And you need to have a doctor’s reassurance that these are recognisable and to help you get through that, so you can get out to the other side.

**Dr Danny Penman:**

So does this mean the only long-term solution to depression is a psychological approach?

**Professor Mark Williams:**

Well, yes, in sense it is. Now if it’s true that just by carrying on taking your antidepressants you can reduce the risk of depression, that will work for some people. And for those who’ve been advised by their doctor that’s what to do and if they’re happy with that, and it doesn’t cause great side-effects, then there’s no reason why they should suddenly stop their antidepressants.

Of course, there’s still a bit of a risk of relapse coming, even if you carry on with the antidepressants and that’s where you can supplement them with psychological skills and psychological therapies as well. But we do know, and this is in a sense turning to our second major strategy, that there are psychological approaches that help, and that when they help they actually also protect against relapse and recurrence.

**Dr Danny Penman:**

So let’s come to psychological treatments. What are the main psychological treatments at the moment?

**Professor Mark Williams:**

There are five or six psychological treatments that work well in dealing with acute episodes of depression. The one that’s got most evidence for it is cognitive therapy. So I’ll come back to that. But it’s true to say that there’s something called ‘behavioural activation’, which is getting people moving as it were, in their behaviour. There’s something called ‘interpersonal psychotherapy’, which is as effective as cognitive therapy in trials which deal with things that happen in your interpersonal life. I mean, it’s an individual therapy that deals with things like guilt, with role transitions, with interpersonal effectiveness, these sort of things.

In the early days people thought that ‘psychodynamic psychotherapy’, sort of Freudian and more analytic psychotherapies would be good. And there wasn’t much evidence for that, but then there wasn’t much evidence collected so there weren’t many scientific studies. And indeed, as people began to make those approaches, more psychoanalytic approaches,more structured and shorter, then, for example, there’s something called ‘psychodynamic interpersonal psychotherapy’ which they got it down to 15 sessions just like other psychotherapies and they found that it worked pretty well.

There’s ‘problem solving therapy’ which works pretty well, there’s, for milder depression certainly, and exercise. We know that exercise works pretty well for milder depressions.

So there’s a whole range of things that we can do to deal with acute depression, but probably the main one is, and the one with the biggest evidence base is cognitive therapy. And the history of cognitive therapy is really interesting.

One of the puzzles about the psychology of depression was that in the 1970s, people didn’t actually think that depression was treatable with psychological means. They thought perhaps the new antidepressants work one way because it was a biological thing, and there were people with the analytic side talking about psychoanalysis, but without much evidence. But certainly there was accumulating evidence that depression could be treated with antidepressant medication. And that although in the 1950s and 60s people had many psychological treatments for anxiety and phobias, nobody thought that depression was the sort of thing that you could treat with this approach.

**Dr Danny Penman:**

So did people once think that you could treat depression by correcting irrational ways of thinking?

**Professor Mark Williams:**

Well it seems obvious now, but actually no they didn’t. I mean, obviously, in history, throughout history people had a go at that. It’s obvious that irrational thoughts and negative thinking is a very important part of depression. But people thought that it was a symptom of depression, that you had to cure the underlying thing. If you think about what antidepressants are doing you’re treating the underlying biological problem, and you expect the thoughts to clear up by themselves.

In psychodynamic psychology, you’re in a sense doing a similar thing. You’re going for the underlying, say, intrapsychic conflict, whatever it is, and hoping that the thoughts themselves would sort themselves out when you got to the underlying problem. Both biological and dynamic models have this in common. Go for the underlying source of the problem and negative thinking, and so on, would naturally just dissolve.

It wasn’t until two major things happened in the late 60s and early 70s that we changed our mind as psychologists about this. First of all, there was a big emphasis within animal learning theory on what is called ‘learned helplessness’. There is a professor in the United States called Martin Seligman who found almost by accident, that when he had animals in experiments that had been subject to uncontrollable stresses, electric shocks, noise, this sort of thing, that if they were then put, used in another experiment in the days when you did this sort of animal experimentation, in another experiment where they could actually escape the stress they were going to be put under. He found that two-thirds of them didn’t escape. It was almost they’d learnt to be, as he described it, helpless. That they learned there was nothing they can do. He called it ‘response outcome independence’, and they would learn that, well there’s nothing I can do. And he said, you know, this could be a model for depression.

And he started to look at human laboratory experiments and invited ordinary people, well students anyway, to come to the laboratory and to be subject to stress that they couldn’t control or anagrams they couldn’t do. Or little problem solving they couldn’t do. And then he began to wonder if they can’t do one, what’s their effect on the next thing they do?

That’s where I started my PhD studies. I mean my interest in depression started in 1975, 1976 when I started to research why do some people who fail then expect to fail on the next task, whereas other people who expect to fail, it galvanises them for the next task? And that'd been what Seligman was interested in. It was what my colleague, John Teasdale who was my PhD supervisor, originally was interested in. And so that’s what I worked on for my doctoral studies.

Now the interesting thing about learned helplessness is that it works right through the animal kingdom, and it suggested that when you have human depression you have to discover what it is that makes humans begin to, as it were, learn from one event something which may not be true of the next event. You know, 'I’ve just failed, does it mean I’m going to be a failure in the future?' In as much as you believe that, then you’re going to have problems. You know, if you think 'I’m not going to enjoy this party, therefore there’s no point in going', you’re making an inference about the next event based on your experience. And therefore you begin to reduce what you do in your life.

But Seligman didn’t have any treatment. He had a great theory which then started to be picked up by undergraduates and graduate students all over the world. Taught in medical schools all over the world, a very famous theory of depression, but no treatment. So people started to come out of medical school, come out of psychology, come out of clinical psychology training saying 'Well we’ve got a theory, but what can we actually do and offer to our patients'. And then there was somebody called Tim Beck, Aaron Beck who had been working in Pennsylvania on something that he called ‘cognitive therapy’.

Now this, of course, elicits a great deal of interest, because cognitive psychology was the Zeitgeist within experimental psychology. The term had been coined by Dick Neisser in 1967. By 1970,72 the world was abuzz in psychology about cognition, about thinking, about memory, about attention, how it’s deployed. He was a psychiatrist, Tim Beck, trained as a psychoanalyst. He’d worked with Korean veterans coming back from the Korean War. He’d noticed how they were often depressed as a result of the trauma they’d been through, and how often thoughts about worthlessness, helplessness, about not being good enough, how much their thoughts were going round and round and round. As an analyst he’d thought 'Right I need to get underneath this to find out what it’s about'.

As a biological psychiatrist, because he was a psychiatrist, he could treat it with antidepressants. But his amazing insight was to say 'What would it be like, what would a theory look like if the thinking that’s going on in their mind was actually part of the cause of their depression? And if not the cause, part of what was maintaining the depression?'

And that one insight led him to start asking his patients 'Why don’t every time you feel down, just catch what went through your mind just the moment before?' And he started to notice that people began to be able to catch their negative thinking. That there was often a flash. It might be very fast, it might be slow, of a negative thought. And he started to get people to write them down and then say 'Look, what if this is the depression speaking? What about seeing whether the depression is telling the truth or not? Let’s take these ideas of how useless you are, how much you’ve always failed and so on. Let’s write them down, and let’s actually be a bit of a scientist here. Let’s look at them, look at the evidence for, the evidence against.'

And soon he began to find that this was liberating huge swathes of his patients without having to do long psychoanalysis, and without having to do biological treatments. And in 1977 he and his colleagues, Gary Emery, Brian Shaw, John Rush and him, published the first randomised clinical trial where they compared antidepressants for properly diagnosed depressed patients with this new what called ‘cognitive therapy.’ And it was astonishing. Not only did it do as well, there was some suggestion that he could do better, with this ability to test out your thoughts and change your behaviour in these interesting ways. And that was the major, major change in depression in the last 50 years, I think.

**Dr Danny Penman:**

Presumably this insight was revolutionary?

**Professor Mark Williams:**

It was and like most revolutionary insights, after the fact, it just seemed so obvious. I remember in the mid-70s when I started to read Beck’s work thinking that it was trivial and I don’t really even need to read very much because it’s so obvious that when people are depressed they get negative thoughts. We need to go underneath the problem. Just to deal with negative thoughts was a trivial way of approaching a really hard problem. But now when you look at the work by Chris Padesky and Dennis Greenberger their book, Mind Over Mood is an incredible best-seller for obvious reasons, because people can do this by themselves. David Burns, The Feeling Good: The New Mood Therapy. Work that Beck himself has continued to do and his colleagues, has revolutionised the study of depression.

I mean, one of the other indications is that my colleague John Teasdale, when he was a psychologist and due to move to Oxford to start off one of the first laboratory programmes in the Psychology of Depression anywhere in the world in 1975, that he was advised by his colleagues there that this would be a waste of time. Everybody knew, they said, that depression was a biological problem or needed some long-term analysis. But as a biological problem, what’s the point in dealing with psychology. Psychology is ‘epiphenomenon’. Psychology is the epiphenomenon. You can’t treat depression by dealing with something which is just a surface epiphenomenon. And how wrong they were, because Teasdale’s work has turned out to be the most influential work together with Tim Beck, I think,in the last 40 or 50 years.

**Dr Danny Penman:**

And you were his student.

**Professor Mark Williams:**

And I was his student. I was very lucky to be his student.

**Dr Danny Penman:**

Just a point of terminology really. What’s the difference between CBT ‘cognitive behavioural therapy’, and CT ‘cognitive therapy’?

**Professor Mark Williams:**

There’s actually no difference at all. CT tends to be used in the United States, cognitive therapy. But there was a long history of behaviour therapy in Britain and Europe, before cognitive therapy came along and therefore cognitive therapy was sort of seen as an add-on to behaviour therapy. And therefore cognitive behaviour therapy is the word coined in Britain and Europe. But actually it refers to the same set of procedures.

**Dr Danny Penman:**

So is it possible to tell which patients are going to respond most favourably to the likes of anti-depressant medication or CT?

**Professor Mark Williams:**

It’s really interesting that you ask that question because one of the earliest studies asked exactly that question. And what they did was they gave people a scale which they called ‘learned resourcefulness scale’, and it measured basically how much you tend to take an active approach to your problem-solving or how much you prefer a passive approach where people do something for you and to you rather than you taking an active approach.

And what these early studies found was that people that tend to like to take an active approach to their problem really responded to cognitive therapy, but don’t do too well with antidepressants. Whereas people that tend to be passive do pretty well with antidepressants, but actually they don’t do too well with cognitive therapy.

Now how can we check that out? Well, one way of checking it out is giving people, like, say, if somebody comes for cognitive therapy. It’s been found that if you give people a little leaflet, six page long, at the first session and you say to people, in fact this is one of the first things that John Teasdale did with my colleague Melanie Fennell who still works with us here in Oxford. And then you say 'Part of your homework for this week is to read this and see whether you like it or not, you know, tell me what you think of it. This is what we’re going to be doing together and I’d be interested in your response.'

Well, they came back in Session 2, and 'How did you get on with this leaflet?' And some people said, 'Ah. It was just describing me. I felt I was the person referred to there, and it was exactly what I think I need. 'And other people said “'Oh you know, I didn’t get on with all this business about taking an active approach, you know. I’ve got a biological depression. I’m not sure about this at all.'

Now if you transcribe that and put it on a seven-point scale from plus 3, through 0, to minus 3 they found that that scale predicted outcomes after twelve weeks just about as well as anything else. And the people who took to it in those early days actually worked hard, did the homework that they had to do, they got a lot of benefit. Those people that were saying' Ah, I don’t know', they didn’t make very much progress in the first few weeks. They didn’t make very much progress in the last few weeks either.

So there are ways of telling. It’s a matching process between what people think is plausible for them, you know, what they think the model of their illness is. And if you get a meshing between people’s own feelings about what’s going wrong and what you’re offering to help do about it, then you get that sort of gelling. You get that enthusiasm, and you get a lot of progress.

**Dr Danny Penman:**

Are these underlying character traits that determine this or is it a character of the depression that they’re suffering from?

**Professor Mark Williams:**

It’s quite possibly both. Actually the truth is we don’t know. So that there are underlying traits in how passive or active people are. Whether it’s a character trait, as it were, like, there from birth we don’t know. It may just be your experiences over life-time, your learning history, as it were, has set you up to be passive or to be active. So it’s not necessarily a character trait in a sense of, you know, biologically determined.

However, you’re quite right that there are different sorts of depressions as well. And that some people do respond, some sort of depressions do respond better than others. And when we come later, in later episodes to talk about mindfulness and mindfulness-based cognitive therapy we’ll be able to describe ways in which we can begin to learn who’s responsive to what aspect of treatment, because that turns out to be very important.

**Dr Danny Penman:**

Well thank you very much for that. In this programme we were looking at the treatment of depression. And in the next episode we’ll be looking at why people relapse despite their best efforts and the best drugs available.

For further information about the issues raised in this programme you can read The Mindful Way through Depression by Professor Mark Williams and his co-workers or you can read our book Mindfulness: Finding Peace in a Frantic World by Mark Williams and me, Danny Penman or you can visit our website: franticworld.com

If you’d like to support further research in this area you could visit:oxfordmindfulness.org and follow the links to the development campaign.

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