Abnormal psychology is the branch of psychology that deals with studying, explaining and treating ‘abnormal’ behaviour. Although there is obviously a great deal of behaviour that could be considered abnormal, this branch of psychology deals mostly with that which is addressed in a clinical context. In effect, this means a range of behaviours, emotions and thinking that tend to result in an individual seeing a mental health professional, such as a psychiatrist or a clinical psychologist.

Abnormal psychology attracts researchers who investigate the causes of abnormal behaviour and try to find the most effective treatments for them, whether these involve medication or a talking cure or a combination. There are also practitioners, psychologists who use their knowledge of theory and research to deliver treatment to people in a therapeutic setting.

A large number of conditions occur commonly enough to be categorized systematically within various cultures and, in some cases, across the world. The IB Psychology syllabus deals with only three groups:

- anxiety disorders
- affective disorders
- eating disorders.

Defining these groups of disorders is straightforward because of the diagnostic systems available, but there is considerable disagreement about the validity of the distinctions between normal and abnormal behaviour.
5.2 Concepts and diagnosis

Learning outcomes
• Examine the concepts of normality and abnormality.
• Discuss validity and reliability of diagnosis.
• Discuss cultural and ethical considerations in diagnosis.

Normality and abnormality
The word normal usually refers to conformity to standard or regular patterns of behaviour. The concept of abnormality is essentially a label applied to behaviour that does not conform. Unfortunately, this explanation is not very precise and it remains difficult for mental health professionals to agree on who is abnormal enough to require or deserve treatment.

Looking at different common interpretations of abnormality highlights this problem.

Statistical abnormality
An interpretation of normality that depends on literal meaning assumes there is such a thing as average behaviour, or behaviour that most frequently occurs in particular situations. Thus, behaviour that does not occur very often in a given context can be considered to be abnormal. This is a particularly useful approach when dealing with numbers, such as IQ scores for measuring intelligence, or scores on personality measures like extroversion. When using numbers, we can obtain means; the majority of people scoring around the middle of the scale, with very few people scoring extremes.

It is much harder to be sure about what the average is when we are not dealing with numbers. For example, how much hunger is normal or abnormal? Hunger is not often expressed numerically, and it is very difficult to compare such a subjective experience between two people.

It may be better to consider what is normal for the individual in a particular situation, rather than what others would do. Research into helping behaviour among humans, for example, suggests that those working in helping professions (e.g. nurses and doctors) are more likely than others to help a stranger who seems to be in need. Also, those who have previously helped a stranger are more likely to intervene. With this in mind, it seems that we need to know quite a lot about a person before we can begin to claim that their behaviour is normal or abnormal based on comparison with the behaviour of ‘the average person’.

Statistically, unusual behaviour is often attributed to mental illness, perhaps because assuming that people are suffering from some sort of psychiatric condition helps us understand the strangeness of their behaviour. However, it must be noted that not all statistically infrequent behaviour is considered a sign of madness or mental incompetence. For example, low intelligence is frequently labelled in various ways and education programmes attempt to help individuals at the extreme low end of the intelligence scale, but same is not often true at the other end of the scale, where high intelligence is usually valued, and even if it is considered abnormally high, it is not stigmatized or compensated for in the same way. There are no special schools that try to reduce genius IQ.
**Deviation from social norms**

Social norms are not necessarily related to statistical norms. Instead of referring to what is frequent in a particular context, the idea of deviation from social norms assumes that in any situation there is an expected behaviour. The expected behaviour is that which the rules of society and culture dictate is appropriate for that context. Using this definition, we can say that although it is acceptable to talk loudly to friends in a noisy café, it is abnormal to talk at the same volume in a library or a cinema. Many teachers might also find it abnormal for a student to be asleep during class, not simply because it is an infrequent event, but because it seems to be socially inappropriate.

When people violate such social rules, we have a tendency to assume there is something wrong with them, and it is easy to attribute this to some kind of madness. This approach to defining abnormality has been with us for a long time, but there are three key problems with it.

- **First**, social norms vary enormously across cultures and social situation. For example, the appropriate response to dealing with a running nose ranges from using a tissue to blow your nose, spitting the mucus out from the mouth or, if you are a sportsman, blowing your nose without a tissue. Any of these options would seem strange if carried out in the wrong situation.
- **The second key problem is historical variation.** In *Models of Madness* (2004), John Read details a number of examples of behaviour that were considered symptomatic of mental illness in the past but would now be acceptable in many cultures and social situations. Examples include sexual interest in people of the same gender and a slave’s desire to run away.
- **The third key problem is controversial.** In many cases, what is considered socially acceptable or unacceptable has been established by groups with social power. Judging normality according to conformity to expectation has led to cruel treatment of many individuals who engage in behaviours that threaten the interests of the powerful group(s). One of the most pervasive examples of this has been the existence of double-standards for men and women: in many cultures, men have reserved certain activities for themselves. Smoking and drinking are typical examples of this, and it is often still considered more abnormal for a woman to drink too much alcohol than it is for a man. In some cultures, for a woman to have more than one sexual partner can result in legal execution. In Western culture, it was previously considered wrong for a married woman to protest at her husband forcing her to have sexual intercourse and abnormal for a woman to live an unmarried life.

It is, therefore extremely important for mental health practitioners to understand the diversity of behaviour across cultures, times, and even genders, if they are to treat their clients well.

**Maladaptiveness and adequate functioning**

Another way to decide what is abnormal is to assume that all humans perform behaviours that are good for them in their particular environmental context. We might expect people to develop an understanding of social expectations, regardless of whether they agree with them or not, and to be able to function within their social group. Behaviours that threaten one’s ability to function well within that social context can be considered maladaptive.

This approach works well when we consider such conditions as alcoholism and anorexia, where it is clear that a person’s health is in danger because of the way he or she is behaving. People who spend so much time on the internet that they lose contact with their peers might also be seen to be engaging in maladaptive behaviour — internet addiction is a fairly recent addition to the range of abnormal behaviour practitioners might deal with.
The most frequently cited problem with this approach is that people whose motivation is clearly not indicative of a serious disorder will sometimes engage in maladaptive or dangerous behaviour. Examples include extreme sports people and political protestors such as Guillermo Farinas, who went on a hunger strike in Cuba to protest at internet censorship.

**Suffering and distress**

If it is insufficient to define behaviour that is bad for a person as an indicator of abnormality, perhaps it is better to ask whether the person is suffering or not, or if they are experiencing distress. This conveniently ignores the problems of most of the other criteria above and can allow those who feel that they need some kind of medical or psychological attention to gain it. However, it carries with it the assumption that individuals engaging in this kind of abnormal behaviour will have enough insight to experience distress. This is not always the case.

For example, irritability is a common symptom of depression among men, but it is not the kind of symptom that would encourage a man to seek help. Another example occurs when a person begins drinking a lot of alcohol – their behaviour may be unhealthy for them, but until there is damage to their physical health or changes in relationships with family or friends, there may be no suffering. Users of illegal drugs may be unaware of the problems they are causing themselves or they simply may not experience any significant distress – but this does not mean they don’t need help. In addition, some degree of suffering and distress must be considered a normal response to challenging life events such as the death of a loved one or divorce, and nobody should consider this a sign of mental illness – it is rather a sign of mental health.

**Jahoda’s positive mental health**

Marie Jahoda (1958) took a different approach in that instead of defining what is abnormal, she tried to define what is normal. If a definition of normal in a mental health context could be established, it should be logical and straightforward to consider behaviour that deviates from the ideal to be indicative of the kind of abnormality that mental health practitioners might feel needs treating. Jahoda identified six components of ideal mental health based on a review of literature:

- positive attitude toward own self
- growth, development, and self-actualization
- integration
- autonomy
- accurate perception of reality
- environmental mastery.

Her approach suggests that the state of ideal mental health is achieved when a person has a realistic and positive acceptance of who they are, and is able to resist stress (so they maintain a lack of symptoms of psychological disorders) while they act voluntarily in the interests of their own growth in the physical and social environments they inhabit.

Internet addiction can lead to isolation.
While this is an attractive approach in many ways, it appears that the more intensely one analyses it, the fewer people we might be able to say are in such a state of ideal mental health. Some research, for example, has found that those with depression are more accurate in their perception of reality, and that for most of us, functioning adequately requires an element of self-delusion (Taylor and Brown, 1988). But unreasonable optimism seems to be beneficial for many people.

Thus, it is extremely difficult to explain exactly what abnormal behaviour in a population is. But perhaps it is a goal that is unnecessary to achieve. There are diagnostic symptoms available to mental health practitioners that attempt to remove some of the problems of subjectivity and help us deal with the large numbers of exceptions to all of the criteria outlined above.

**Exercise**

1. Summarize the main approaches to defining abnormality and use bullet points to identify problems with each one. Try to think of your own examples of behaviour that is abnormal or normal and cannot be accounted for by each of the approaches detailed above.

**Diagnostic systems and the validity and reliability of diagnosis**

Worldwide, there are several major systems of diagnosis.

The *Diagnostic and Statistical Manual of Mental Disorders* (DSM)

This is publication by the American Psychiatric Association is probably the most famous system of diagnosis. It was first published in 1952 and has been revised several times; it is now in its revised fourth edition (DSM-IV-TR), and the fifth edition is scheduled for release in 2013. Revisions are intended to make the diagnosis of mental disorders a more reliable process and a more valid reflection of general wisdom at the time. For example, the manual attempts to describe any disorder in such terms that two clinicians referring to the system would probably agree with the diagnosis it suggests, Disorders are added and removed as time goes on. Homosexuality was removed from the DSM as a disorder in 1980, and alterations have been made to the class of eating disorders several times since then.

The DSM groups disorders into categories and then offers specific guidance to psychiatrists by listing the symptoms required for a diagnosis to be given. An improvement made in 1987 was the creation of a multiaxial approach, whereby a diagnosing clinician should consider the individual under investigation not only in terms of whether they qualify for diagnosis, but also whether they have medical conditions, psychosocial and environmental problems, and how well they are functioning generally. This approach has encouraged psychiatrists using the system to take a more holistic approach to understanding the person who has presented with some problems. This reflects a widely held belief among mental health practitioners today that the origin of each person’s problems should be analysed according to a bio-psycho-social framework.

Modern diagnosis is often able to take advantage of medical breakthroughs. For example, Alzheimer’s disease and attention-deficit-hyperactivity disorder (ADHD) are disorders that can sometimes be diagnosed using brain-imaging technology thanks to our understanding of changes in brain structure or functioning.
The International Classification of Diseases (ICD)

The ICD is now in its tenth edition (ICD-10) and is more commonly used internationally than the DSM (Mezzich, 2002). It was originally intended by the World Health Organization to be a means of standardizing recording of cause of death. It therefore covers a wide range of diseases and conditions for the sake of classification rather than diagnosis. One chapter categorizes mental disorders and it looks very similar to the DSM-IV system because the authoring teams worked in consultation with each other. With each revision, differences between the ICD and the DSM are becoming fewer.

The Chinese Classification of Mental Disorders (CCMD)

This system has not yet generalized far outside Chinese territory. It is currently in its third edition (CCMD-3). It has not attempted to capture the full range of human diagnoses but maintains a focus on issues that are of interest in Chinese culture. For this reason, some disorders identified in the ICD-10 and DSM-IV-TR that are not common in China are left out, and others are included that appear to be culture-bound disorders found only within Chinese culture. Examples include koro – a kind of anxiety or depression, and mental disorder due to Qigong – a form of meditative exercise – which is also now included in the DSM. Ego-dystonic homosexuality is also included in the CCMD; this disorder is characterized by homosexual urges that are unwanted. Although at first glance, some of these disorders may appear strange, it is interesting to consider how unusual some of the Western disorders we take for granted are. Some researchers consider anorexia nervosa to be a culture-bound disorder, for example. This is discussed in more detail later.

There are strong arguments that such a system is not reliable and, more fundamentally, that it is not valid to take such a medical approach to the treatment of psychological problems. In addition, many people have expressed fundamental objections to the way in which the system has been used. In many countries where ego-dystonic homosexuality is considered a disorder, treatments are available to remove the source of the problem. In Soviet Russia, the diagnosis of schizophrenia was applied much more liberally than in the US at the time. It lead to the incarceration and compulsory treatment of many individuals who might have been better understood under a more narrowly applied diagnostic system.

However, while examples like these may appear to lend ethical superiority to the ICD and DSM systems, it is by no means certain that ethnic minorities and women are treated differently from others in terms of their likelihood of receiving a diagnosis, level of understanding offered by practitioners, and the type of treatment offered.

Reliability issues

Receiving a diagnosis can be a difficult experience for some and a huge relief for others. In either case, it seems important to ask whether the systems used are in fact reliable. Two key forms of reliability, as outlined on page 3, are relevant here: inter-rater reliability and test–retest reliability. Inter-rater reliability can be assessed by asking more than one practitioner to observe the same person and, using the same diagnostic system, attempt to make a diagnosis. If practitioners make the same decision, the system is reliable. Test–retest reliability is concerned with whether the same person will receive the same diagnosis if they are assessed more than once (e.g. on two different days).

One of the most commonly cited studies of inter-rater reliability across several diagnostic systems was carried out by Nicholls et al. (2000) and asked two practitioners to use either DSM-IV, ICD-10 or the Great Ormond Street Hospital’s own diagnostic system (GOS) to diagnose 81 children who had come to a specialist clinic with eating problems. This was a correlational study in the sense that reliability was measured based on rates of agreement.
between the two practitioners, expressed as correlations. When the practitioners used DSM-IV, inter-rater reliability was only 0.36, compared to 0.636 for ICD-10 and 0.879 for the GOS system. This shows quite clearly that for some reason the GOS system is more reliable than either of the other two systems. There was also a rather awkward problem in that less than half of the children assigned to be diagnosed according to DSM-IV could be diagnosed with a classified eating disorder. With more children, we might expect agreement to increase. The researchers suggest that the success of the GOS system is because it is specifically designed for use with young children.

Mary Seeman (2007) completed a literature review examining evidence relating to the reliability of diagnosis over time. She found that initial diagnoses of schizophrenia, especially in women, were susceptible to change as clinicians found out more information about their patients. It was common for a number of other conditions to cause the symptoms for which women were receiving the diagnosis of schizophrenia. This indicates the problem of test–retest reliability with schizophrenia diagnoses.

**Validity issues**

Several forms of validity are discussed on page 4. The key concern for diagnostic systems is whether they correctly diagnose people who really have particular disorders and do not give a diagnosis to people who do not. Unfortunately, there is a circular logic involved here – it is difficult to establish whether a person truly has a disorder without using a diagnostic system. This means that the only people we can be fairly sure about are those who have already been diagnosed, although many will argue this is also insufficient.

Thomas Szasz and R.D. Laing are two famous critics of the biomedical approach using diagnostic systems.

**R.D. Laing**

Laing’s work suggests that although diagnosis is made within a medical model, the diagnosis is more of a social fact than a medical one. Psychiatrists do not diagnose because of a set of biological facts; this is because (as you will see), there are no reliable biological tests for diagnosing for most psychological disorders, only guidance about categorizing behaviour, thoughts and emotions. The process of diagnosing is full of financial, political and legal implications. It is also frequently a bureaucratic step of secondary importance to treatment, which (within the medical model), often consists of medication, hospitalization, and limited attempts to see the presenting symptoms as an understandable reaction to life difficulties.

The pharmaceutical industry has been heavily criticized for its influence over the medical profession and it is of interest to note that a large number of those serving on advisory panels for revisions in the fourth edition of the DSM had financial ties to the pharmaceutical industry, mostly in terms of research funding (Cosgrove et al., 2006).

**Thomas Szasz**

Thomas Szasz also suggested that it is wrong to use a mental illness metaphor to describe behaviour that does not conform to our expectations. It is clear that there are biological correlates of behaviour (page 15), but it is reductionist to assume that conditions like depression and schizophrenia are diseases like any other, especially as biological causes have not yet been found for most psychological disorders. In particular, it is important to note that the terms ‘depression’ and ‘schizophrenia’ are essentially labels given to a set of behaviours, emotions, or thoughts. Can a person really have depression in the same way that they can have influenza? It is somehow more attractive for many of us to imagine that an underlying condition called depression is the cause of a person’s severe unhappiness, but
there is no underlying condition tested for. The depression is the unhappiness (along with other symptoms), rather than the cause of it.

The DSM symptoms for major depressive disorder have been reviewed several times, and a concession that sometimes life events (rather than an underlying medical condition) might make a person feel bad was included. The specific allowance in DSM-IV-TR is for the death of a loved one although, as you will see (page 166), symptoms should only continue for two months.

Wakefield et al. (2007) conducted a study which suggests that a wide range of other life events can account for symptoms of depression and therefore the exclusion is inappropriately narrow. This lack of clarity about when the symptoms of depression really indicate a medical condition and when they indicate an understandable response to life events is only one example of the problems with validity.

**Caetano and Rosenhan**
The studies below demonstrate some other problems.

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**EMPIRICAL RESEARCH**

(Caetano, 1973)

Caetano conducted an experiment in which two he videoed a male psychiatrist carrying out separate, standardized interviews with a paid university student and with a hospitalized mental patient. Two groups of people were shown these interviews – a group of 77 students of psychology and a group of 36 psychiatrists attending a meeting. They were asked to diagnose the interviewees and rate their degree of mental illness. Within each sample of viewers, there was random assignment to two different groups each of which received different information about the interviewees: either that both were volunteers who were paid to participate, or that both were patients in a state mental hospital.

Caetano acknowledged that it was possible to argue that the student selected could have had an undiagnosed psychiatric disorder and that the patient selected was close to normal (with the appearance, manner and attitudes of a hippie whose drug use was explainable). The results indicated that psychiatrists with clinical experience were more likely to be persuaded by the information given about the two interviewees and label them both as mentally ill (if they were described as patients) or both not mentally ill (if they were described as volunteers).

This study demonstrates labelling theory: the theory that the behaviour of the person being diagnosed is not the most important component of diagnosis and, in the ambiguous situation of a diagnostic interview, any suggestion that the subject is or has been mentally ill will be a powerful influence on any decision.

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**EMPIRICAL RESEARCH**

(Rosenhan et al., 1973)

Rosenhan and a group of colleagues and acquaintances (including a housewife, a painter and a student) presented themselves at 12 different hospitals across the USA complaining of hearing voices, but otherwise presenting their life history and present state as normal. All but one were admitted with a diagnosis of schizophrenia.

On admission to the hospital, their instructions were to cease complaining of any symptoms (although they were generally rather nervous because of the situation). Their goal was to get out. They did all achieve this, but with a diagnosis of schizophrenia in remission. Thus, their (apparent) normality was never detected although descriptions from nursing staff show that there was no further evidence of difficult or psychotic behaviour.

continued
In fact, 35 of the 118 patients in the wards to which the Rosenhan’s pseudopatients were admitted expressed some kind of concern about their presence, assuming in many cases that they had an alternative purpose such as journalism or checking on the hospital in some way. It took between 7 and 52 days for the pseudopatients to be released, with an average of 19 days. This time was used by the pseudopatients to conduct a participant observation of life in the hospital, which involved making notes about the interactions between staff and patients. Although their writing was noticed by staff in some cases, this was seen as a symptom of their illness, perhaps because it is not something that patients normally do. They were not asked what they were writing about. It is clear then, that with some significant symptoms, an apparently normal person can receive a diagnosis.

Conversely, Rosenhan found that ‘abnormal’ people can be mistakenly assumed to be healthy. He followed his first study with a second, in which staff at a hospital who had claimed that they would not have been fooled by Rosenhan’s pseudopatients were invited to estimate how many times Rosenhan attempted to trick them in this way during a 3-month period. With great confidence, at least one staff member estimated that 41 out of the 193 people who were admitted during the test period were pseudopatients. This time, Rosenhan had sent none: all were genuine patients. Which people should really have been admitted and which ones are actually normal? Of the patients admitted in this time, 19 were thought to be normal by a psychiatrist and at least one other staff member. Although this study was conducted in 1973, it provides some important evidence for a general inability to tell the difference between normal and abnormal behaviour.

Criterion-related validity

Criterion-related validity is a form of validity based on whether a new system agrees with existing measures of the phenomenon in question. Gavin Andrews has published research on using DSM and ICD-10 systems, particularly in the diagnosis of anxiety disorders. He has generally found only moderate agreement between them. When a person has been diagnosed according to one system but cannot be diagnosed according to another system by the same practitioner or group of practitioners, this indicates poor validity.

In one study, Peters, Slade and Andrews (1999) found only moderate agreement between the two systems because the DSM-IV requires the presence of distress or impairment to functioning in the person being diagnosed. Andrews has been working on constructing more systematic diagnostic interviews and refinements to the criteria from both systems to help them converge. This should help improve their criterion-related validity, even if we risk over-diagnosis of people who in fact are functioning quite well.

It is still the case that certain groups of people are more likely to receive a diagnosis, and it is very difficult to remove the subjectivity of practitioners from the diagnostic process. Some of these problems are outlined in the following section.

Culture, gender and ethical considerations in diagnosis and treatment

Labelling theory (described above and demonstrated by the studies of Caetano and Rosenhan) indicates that once a diagnosis has been made, it tends to stick. The two key problems with this are:
we are not convinced that such diagnosis is reliable or valid, even if this situation is improving
there are significant negative effects of such a diagnosis on a person's subsequent treatment by other people.

Many employers require job applicants to declare any mental illnesses or medication they are taking when they apply for a job, and although discrimination is often prevented by law, fear of being treated differently can be a significant cause of discouragement among those who have been diagnosed. Recent research suggests that 92% of people in the UK would be afraid of admitting they had a mental illness because it would damage their career. More than half of those surveyed would rather not hire someone if they knew they had a mental illness.

Read (2007) summarized a large amount of research relating to stigma. The findings showed that attitudes towards those diagnosed in a medical context tend to be characterized by fears, especially regarding dangerousness and unpredictability; also that knowing someone has a diagnosis of mental illness increases reluctance to enter into romantic relationships with them. Sato et al. (2006) discuss how schizophrenia (formerly, seishin bunretsu byo, split-mind disease) has been renamed in Japan because there was such stigma attached to it that less than 40% of patients who had been diagnosed with it had actually been informed of the diagnosis.

There is also a concern that treatment after diagnosis may worsen or create symptoms. Iatrogenesis is the phenomenon whereby treatment for a condition causes other complications. It has been used to describe the process by which adaptation to life in an institution causes mental patients to develop new behaviours which are then considered symptoms of their condition. Rosenhan’s (1973) study (page 151) gives an indication of how this can occur, particularly in the sense that a lot of the social interactions observed by the pseudopatients were lacking in care and concern.

Similar concerns have been raised about life in prisons, where conditions can be cruel and dehumanizing in a way that makes a return to the community difficult. Additionally, diagnoses can be part of a self-fulfilling prophecy. An example might be a person diagnosed with depression who takes time off work, finds it difficult to return and ends up losing their job. The consequential unemployment can increase the person's sense of isolation and feelings of worthlessness, and compound the symptoms that they originally presented with.

This is in line with the argument that psychiatry functions to exclude those who are perceived as different or difficult in some way, or at least uses diagnosis and institutionalization instead of attempting to understand differences. This is reflected in rates of diagnosis for conditions like schizophrenia and depression, which are wildly different between ethnic groups and between men and women. Types of treatment offered are also quite different.

For example, Morgan et al. (2006) found that in the UK, the incidence of schizophrenia is nine times higher for Afro-Caribbeans and six times higher for those of black African descent, than for white British people. The researchers argue that genetic differences cannot account for this and it is more likely that diagnostic biases account for it. Migrants and...
ethnic minorities in many European countries and the Anglo-American world are over-represented in mental hospital populations (Read et al., 2004). Women are more likely to be diagnosed with depression. While one vein of research investigates the unique biological reasons why this might be the case, another argument suggests that diagnostic criteria for depression are a description of normal female responses to social pressures, and as such should not be pathologized but be understood better and treated on a social rather than individual level.

The reverse side of this is the potential denial of treatment to people who need it. The possibility that an individual with suicidal thoughts might go through a diagnostic interview, not be diagnosed with depression and offered treatment, and then carry out a suicide attempt, is a very powerful incentive for a clinician to make a diagnosis as a precaution. The immediate safety of the interviewee is a higher priority than any potential long-term negative effects of stigma related to the diagnosis. In many cases, medication and institutionalization are favoured options as they are frequently very successful in preventing harm to the patient or to other people. It could be argued that medical diagnosis is not the most efficient way to achieve this, but there is a lack of well-funded alternatives and the public has a high level of respect for and trust in the medical establishment.

**Behaviour and interpretation across cultures**

In Chapter 4 (page 139), you considered emic and etic approaches to mental health research. You might wish to review this material before reading on. **Culture-bound syndromes** were referred to earlier (page 149). Different parts of the world and different ethnic groups have different ways of explaining the kind of strange behaviour that is dealt with by psychology and psychiatry in the Western world.

**EXERCISE**

3 Conduct further research about two of culture-bound syndromes found on weblink 5.5, looking for causes and treatments and write a paragraph to explain both.

Where there is such a huge variety of labels for behaviour that shares as its basis either dangerousness or violation of social norms, it must be asked whether the DSM criteria are valid beyond the culture they were created in. In particular, it is a problem that the existence of labels for disorders may affect the likelihood of a person developing the symptoms for one of those disorders, and there are significant implications for treatment.

An interesting example of the possible role of culture was noted in a statistical analysis of data from two cities in the United States by Levav et al. (1997). Rates of alcoholism and depression were compared across various religious groups and it was found that Jewish males were much more likely to have a diagnosis of depression, and less likely to have a diagnosis of alcoholism.

One implication of this is that there is some kind of underlying issue that manifests itself differently depending on cultural traditions and expectations. This becomes very important in a clinical setting where there are relatively few practitioners are members of ethnic minorities and might be able to offer greater insight into patients’ problems.

A second possibility is that the diagnosing clinician’s cultural stereotypes influence his or her clinical judgement, for example in terms of what constitutes a symptom. Read et al. (2004) note that in New Zealand, where Maori people are over-represented in psychiatric institutions, psychiatrists do not feel it is inappropriate to use European diagnostic systems with non-European patients. This may lead to misunderstandings and misdiagnosis.
Kirov and Murray (1999) studied a group of patients taking lithium prophylaxis (lithium is a drug sometimes used for depression and bipolar disorder). They found that there were clear differences in the symptoms and diagnoses that had resulted in patients being medicated: black patients were less likely than white patients to have suicidal ideas or to have attempted suicide and generally had more manic symptoms, resulting in a diagnosis of bipolar disorder. The authors suggest that because of this difference in the manifestation of the underlying problem, many black patients in the UK may be diagnosed with schizophrenia rather than with an affective disorder. This may contribute to the finding by Riordan et al. (2004) that compulsory hospitalization orders are more likely to be applied to black than to white patients.

From these examples, we can see that there are a number of important cultural and ethical considerations when diagnosing psychiatric and psychological disorders. Further investigation into this area will show you how political and full of controversy the literature is.

**Culture and mental health in the media**

*Shankar Vedantam, writing online on the Washington Post website, Tuesday 28 June 2005*

Roberto Lewis–Fernandez was a young doctor in training in Massachusetts when he encountered a patient who was 49 and suicidal at Cambridge Hospital. The Puerto Rican woman begged for help in resolving a conflict with her son, but the Harvard University-affiliated psychiatrists focused on one set of symptoms – she was hearing voices, seeing darting shadows and sensing invisible presences.

They diagnosed her as depressed and psychotic, or out-of-touch with reality, and medicated her. She was discharged. Soon after, the woman had an argument with her son and nearly killed herself by overdosing on the medication.

For Lewis–Fernandez, who is Puerto Rican, the suicide attempt confirmed his fears that his superiors had misjudged the situation. For months, as top psychiatrists ordered him to keep increasing the potency of her drugs, he had told himself that hearing voices, seeing shadows and sensing presences is considered normal in some Latino communities. But he dared not challenge the wisdom of the medical model.

‘I wasn’t sure if she was psychotic, but I treated her as if she was,’ he said about the case, which he wrote up in a medical journal. ‘I gave her the medicines.’

When the hospital’s outpatient unit evaluated the woman anew, doctors there came up with a different diagnosis. They concluded that her symptoms were not abnormal in the context of her culture – they were expressions of distress, not illness. Lewis–Fernandez helped her reconcile with her son. She still heard voices and saw shadows, but as before, they did not bother her.

I think it would be a huge mistake to suggest that there is no such thing as psychosis among Latinos from the Caribbean. Rather, the point of the story is that by focusing only on her symptoms, doctors were misled. The same symptoms, can mean different things depending on the context. The goal of people like Dr Lewis–Fernandez is to have doctors focus on the context as well as the symptoms; to ask, for instance, how the patient interprets the symptoms, and to ask what the culture from which the patient comes thinks about such symptoms. Many advocates of cultural competence talk about how the culture influences ‘idioms of distress’ – the ways in which patients express symptoms. Arthur Kleinman, the Harvard psychiatrist, told me that during his research in China after the Cultural Revolution, patients with what we would now call depression mainly complained about dizziness, exhaustion and sleeplessness – symptoms associated with what was then known as neurasthenia. ‘Depression was a
highly stigmatized mental illness, neuresthenia was thought of as a physical condition and didn’t have any disgrace or humiliation,’ Kleinman told me. But as China has globalized, this has changed. Now depression is a very common diagnosis.

5.3 Psychological disorders

Learning outcomes

- Describe symptoms and prevalence of one disorder from two of the following groups:
  - anxiety disorders (page 156)
  - affective disorders (page 165)
  - eating disorders (page 174).
- Analyse etiologies (in terms of biological, cognitive and/or sociocultural factors) of one disorder from two of the above groups.
- Discuss cultural and gender variations in prevalence of disorders.
- Examine biomedical, individual and group approaches to treatment.
- Evaluate the use of biomedical, individual and group approaches to the treatment of one disorder.
- Discuss the use of eclectic approaches in treatment.

The abnormal psychology option in the IB Psychology exam requires you to be familiar with at least two specific disorders from two different groups of disorder. You need to describe the disorder, analyse the range of possible causes, and discuss treatment for one disorder in more depth. You also need to be aware of the prevalence of the disorders you choose, to discuss them in general and to evaluate research relating to them.

The prevalence of a disorder refers to the number of people in the population who have the disorder (as opposed to the incidence rate, which is the number of new diagnoses each year). It is impossible to know how many people actually have a specific phobia, for example, because many people who have a phobia will be undiagnosed, so the figures are estimates. Prevalence rates are sometimes also expressed as lifetime risk estimates, indicating the percentage of the population who are likely to be diagnosed with a disorder.

Anxiety disorders
Definitions and diagnosis

This category of disorders includes a range of conditions characterized by the experience of anxiety or fear. In ICD-10 they are referred to as neurotic, stress-related and somatoform disorders. This is a broader category and includes phobias, post-traumatic stress disorder, panic disorder and obsessive–compulsive disorder, as well as conditions where there appear to be physical symptoms (such as intense pain) because of the anxiety. You only need to know about one of these categories. Here we consider specific phobia.

Diagnostic criteria for specific phobia

American Psychiatric Association

A Marked and persistent fear that is excessive or unreasonable, cued by the presence or anticipation of a specific object or situation (e.g. flying, heights, animals, receiving an injection, seeing blood).
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Definitions and diagnosis

Anxiety disorders

The prevalence of a disorder refers to the number of people in the population who have it. In epidemiological studies, the prevalence is usually determined by surveys and is given as the percentage of people in the population who have met the diagnostic criteria for the disorder at some point in their lives (the prevalence lifetime) or at some point during a specific time period (the prevalence 12-month). It is impossible to know how many people actually have a specific phobia, for example, because many people who have a phobia will be undiagnosed, so the figures each year). It is impossible to know how many people actually have a specific phobia, for example, because many people who have a phobia will be undiagnosed, so the figures each year). It is impossible to know how many people actually have a specific phobia, for example, because many people who have a phobia will be undiagnosed, so the figures each year). It is impossible to know how many people actually have a specific phobia, for example, because many people who have a phobia will be undiagnosed, so the figures each year).

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The abnormal psychology option in the IB Psychology exam requires you to be familiar with at least two specific disorders from two different groups of disorder. You need to choose, to discuss them in general and to evaluate research relating to them.

We can break the symptoms described in the DSM-IV definition into four types:

- affective (related to mood)
- behavioural
- cognitive
- somatic (related to the body).

Although the experience of anxiety is different for everybody, it is typical for people to experience feelings of terror or dread and a loss of control, the persistent and salient thought that they are going to faint, vomit or even die, to feel dizzy, to have trouble breathing and to start sweating. The behavioural symptoms of phobias are concentrated around avoiding the thing that the person is afraid of. It is this urge to avoid the fear stimulus that causes the problems of daily function that might bring a person to the attention of a psychologist or psychiatrist. For many people, avoiding something they are afraid of does not require daily effort. For example, many people are afraid of flying in aeroplanes, but rarely need to do so. Others have a specific phobia of stairs, which are experienced more often. Others have a phobia of loud noises, which could occur at any time and are not within the person’s control – it is clearly more difficult to find strategies to avoid this.

Types:

- animal type
- natural environment type (e.g. heights, storms, water)
- blood–injection–injury type
- situational type (e.g., airplanes, elevators, enclosed places)
- other type (e.g., phobic avoidance of situations that may lead to choking, vomiting, or contracting an illness; in children, avoidance of loud sounds or costumed characters)

Note: In children, the anxiety may be expressed by crying, tantrums, freezing, or clinging.

B Exposure to the phobic stimulus almost invariably provokes an immediate anxiety response, which may take the form of a situationally bound or situationally predisposed panic attack.

Note: In children, the anxiety may be expressed by crying, tantrums, freezing, or clinging.

C The person recognizes that the fear is excessive or unreasonable.

Note: In children, this feature may be absent.

D The phobic situation is avoided or else is endured with intense anxiety or distress.

E The avoidance, anxious anticipation, or distress in the feared situation interferes significantly with the person’s normal routine, occupational (or academic) functioning, or social activities or relationships, or there is marked distress about having the phobia.

F In individuals under age 18 years, the duration is at least 6 months.

G The anxiety, panic attacks, or phobic avoidance associated with the specific object or situation are not better accounted for by another mental disorder, such as obsessive–compulsive disorder (e.g. fear of dirt in someone with an obsession about contamination), post-traumatic stress disorder (e.g. avoidance of stimuli associated with a severe stressor), separation anxiety disorder (e.g. avoidance of school), social phobia (e.g. avoidance of social situations because of fear of embarrassment), panic disorder with agoraphobia, or agoraphobia without history of panic disorder.

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Common phobias include arachnophobia, social phobia and brontophobia.
Specific phobia is the most commonly diagnosed of the anxiety disorders in the USA. It is overall third among all disorders, behind major depression and substance abuse disorders, with a lifetime prevalence of 12.5% (Kessler and Merikangas, 2004). Prevalence rates are lower in other countries; as little as 0.63% reported in Florence, Italy (Faravelli et al., 1989). Women usually make up a far greater part of these statistics, and symptoms usually begin during childhood, around the age of 7 (Kessler and Merikangas, 2004). When looking at reasons for the different prevalence of phobias in men and women, it is important to remember that diagnosis requires significant impairment of functioning. Because it is possible to manage one’s life around avoiding the situation, animal or item that causes anxiety, it is clear that some types of phobia are more likely than others to be diagnosed, even if they are not the most common phobias. Situational phobias appear to be dealt with in a clinical context more than others, and a further reason for this might be that a person’s expectation of a positive outcome from therapy is higher.

**Etiology: Biological level of analysis**

According to the biological level of analysis, specific phobias should be explainable in terms of evolutionary adaptation, genetic susceptibility, the action of neurotransmitters in specific regions of the brain, and the role of hormones. Recent research even suggests that there is a possibility that anxiety disorders are caused by breathing problems and an excessive intake of carbon dioxide (Wilhelm and Roth, 2001).

**The fight or flight response**

The fight or flight response (page 54) is generally considered to be the basis of the anxiety reaction. It seems to prepare the human body for action to deal with a threatening situation through activating the sympathetic nervous system. Researchers have investigated the hormone adrenaline, which is known to be involved in this system, and the neurotransmitter GABA, which is known to be involved in the parasympathetic nervous system which returns the body to its normal state when a threat has passed. The biological level of analysis predicts that overactivity in the sympathetic nervous system, or underactivity in the parasympathetic nervous system, is responsible for the phobia.

Merkelbach et al. (1996) indicate that increased levels of adrenocorticotrope hormone (ACTH) enhance the acquisition of new fears and that phobic individuals (both humans and primates) do tend to have elevated levels of this hormone. In addition, premenstrual women are reported to be more susceptible to fear-learning than other women. There is a great deal of research relating to the role of GABA in preventing the anxiety reaction, but this comes mostly from animal studies. Evidence from studies of treatment effectiveness show that anxiety can be successfully reduced in humans by increasing the availability of GABA in the brain (Cryan and Kaupmann, 2004).

While research into hormones and neurotransmitters assists in establishing the biological correlates of the fear reaction, they are not yet able to offer a causal explanation. Clearly, we are limited in what we can do experimentally with humans for ethical reasons.

**EXERCISE**

4. What ethical and practical problems are there in experimental research into specific phobias in humans? Write a short paragraph to describe them.

Figure 5.1 shows two structures deep in the centre of the brain (one in each temporal lobe) called the amygdalae (singular, amygdala). See also Figure 2.1 (page 48).
Åhs et al. (2009) studied the role of the amygdalae by exposing 16 female volunteers with either snake or spider phobias to pictures of both, so that reactions to the fear stimulus could be compared with reactions to the animal they were not afraid of. To ensure participants were genuinely afraid of the animals, the snake anxiety and spider anxiety questionnaires were used.

PET scans (page 42) were carried out to obtain images of brain activity during exposure. The PET scans were correlated with participants’ estimates of distress. The researchers found that there was a strong correlation between amygdala activity in the right hemisphere of the brain and ratings of distress. Their conclusion was that the amygdala is activated after object-recognition areas in the brain indicate that the object is threatening. The amygdala then works to activate motor regions of the brain to support the activation of a fight or flight response to the stimulus. They suggest that a process of classical conditioning has biologically predisposed individual humans to have this biological reaction to some stimuli but not to others.

The role of the amygdala in activating this response may be countered by a regulatory system in the prefrontal cortex, as Hermann et al. (2009) found that women with a spider phobia showed lower than normal prefrontal activity in an fMRI study when trying to manage their emotional reaction to pictures of spiders.

**Classical conditioning**

Other research has already suggested that classical conditioning is a biological process involving the unconscious strengthening of neural connections in the brain according to experience (e.g. Öhman et al., 1975). Öhman experimented on human participants, testing whether he could create fear reactions to pictures of prepared stimuli like snakes and unprepared stimuli like flowers. It was easier for him to create the fear response for the prepared items and these responses were more likely to last than others. These results suggest that humans are biologically predisposed through an evolutionary process to fear some objects more than others.

Bennett–Levy and Marteau (1984) come some way to explaining this in a correlational study that measured fear of animals and asked participants to rate them on certain
characteristics like ugliness and how suddenly they moved. These two characteristics showed strong associations with fear and the research suggests that we carry an innate tendency to fear characteristics of animals rather than the animals themselves.

More recent research by Davey et al. (1998) suggests that what has been selected for in human evolution is fear elicited by stimuli associated with disease rather than direct predatory attack. These researchers conducted a cross-cultural study in the USA, the UK, India, Japan, Hong Kong, Korea and the Netherlands. They found that ratings of fear were strongest for disgust-relevant animals (e.g. slugs and spiders) although India’s ratings were lowest for these and Japan’s were highest. The results suggest that animals which trigger a disgust reaction (an adaptive response to help humans avoid disease) will trigger the brain mechanisms that activate the sympathetic nervous system and thereby the fight or flight response. This study clearly offers directions for both cognitive and sociocultural explanations that are explored below. It also found gender differences, which was not surprising given that females generally have a stronger disgust reaction than males, again possibly an adaptation favoured by natural selection because a failure to recognize disease would threaten a female’s own life and the life of her children.

Twin studies
One of the problems with evolutionary explanations is that they lack concrete evidence, and it is the ultimate aim at a biological level of analysis to find a genuine genetic basis for specific phobias. Twin studies offer some support for a genetic cause. Skre et al. (2000) found that specific phobias were shared by identical twins much more often than by non-identical twins. Merckelbach et al. (1996) suggest that although twin studies usually show some support for genetic inheritance, what is inherited is not the specific phobia, but a general tendency for neurotic responses, and this usually manifests itself as specific phobias. The exception is a phobia of blood, which does seem to be inherited more specifically than other phobias.

Weaknesses
This highlights the strongest weakness of biological explanations. Although there is increasing understanding of the biological mechanisms involved (e.g. activation in some areas of the brain and reduced activity in others) there is a bias in research towards phobias of animals, whereas the phobias mostly dealt with in clinical practice are situational. The inheritance of a disgust reaction seems like a poor explanation of a fear of flying, for example, even if they share the cognitive component of thoughts of dying.

Etiology: Cognitive level of analysis
Bandura’s self-efficacy theory (page 121) has been used to account for the causes of phobias (Bandura, 1982), focusing on an individual’s expectations about their ability to perform a particular task relating to the source of their phobia. For example, many people might have a fear of flying, but a person who cannot imagine they will successfully get onto an aeroplane and survive the flight will suffer greater anxiety than others (Figure 5.2). However, there is limited support for this as a causal explanation of specific phobias, and the work of Beck and Emery appears to be more relevant (Armfield, 2006).

Cognitive schemas
The Beck and Emery model explains that cognitive schemas are responsible for an increased and maladaptive perception of threat, a misinterpretation of environmental stimuli that triggers the fear response. Typical cognitive patterns are also responsible for this (e.g. magnification and personalization), which can make the person focus too much on themselves and the likelihood that they are facing a serious threat. This sense
of vulnerability combines with low self-efficacy and appraisal theory, such that when a possible threat is encountered (primary appraisal), the person makes a secondary appraisal about whether they can cope with it. If either part of the appraisal is biased in a maladaptive direction, the chances of a strong anxiety reaction increase. Learned avoidance strategies are then employed to remove the person from the fear situation.

There is some empirical support for this idea, such as a study by Thorpe and Salkovskis (1995). However, it appears that thoughts specifically relating to suffering harm are relatively infrequent before people with phobias encounter the phobic stimulus. This suggests that phobias are held by normally rational people with relatively correct interpretations of the level of danger and their ability to deal with it, but that this rationality is overcome by the kind of thinking Beck and Emery discuss during the experience of phobia. This limits its value as a causal explanation.

Armfield (2006) lists a number of cognitive factors (e.g. negative self-focused attention, memory bias and attentional bias) that have been shown to be associated with anxiety generally and phobia specifically. However, he concludes that there is a deficit in cause–effect relationships in every case. It seems that we are left unsure if the phobia causes the problems in thinking, or if the problems in thinking cause the phobia. He focuses on perceived unpredictability of the stimulus and perceived uncontrollability of both stimulus and situation, such as when a person sees a dog and believes they cannot predict or control the behaviour of the animal or escape from it. This is supported by experimental evidence that when participants are able to turn off unpleasant stimuli being presented to them, they show a smaller physiological reaction to the stimuli than participants who cannot (Sartory and Daum, 1992). Further supporting evidence is that when asked about their beliefs about spiders, phobic individuals tend to refer more to unpredictability and lack of control over their behaviour than to negative thoughts such as dirtiness or disgust (Arntz et al., 1993).

Armfield’s (2006) conclusion is a cognitive model based on schema theory: a person with a specific phobia has a vulnerability schema for the phobic stimulus which combines uncontrollability, unpredictability, danger and disgust; this schema is automatically employed to interpret the situation when a person is presented with their phobic stimulus. Armfield assumes that individual personality traits and biological dispositions (e.g. genetic susceptibility) and previous experience are all contributors to the schema. The activation of the schema triggers evaluative processes leading to the somatic, behavioural and cognitive components of the specific phobia.

Cognitive models like this are adequate in explaining the mechanisms involved in triggering a phobia in response to a specific stimulus, and appear to fit quite comfortably next to the descriptions of the biological correlates of the phobic response. However, we are still lacking a full explanation of how such a schema develops, and we are still faced with the difficulty that experimental evidence of a cause–effect relationship is almost impossible to obtain.
Etiology: Sociocultural level of analysis

There is much less research available about sociocultural influences in the development of specific phobias, but there is research that indicates there are differences across cultures that must be accounted for somehow. Research by Davey et al. (1998) indicates, for example, that ratings of disgust and fear for some animals were much lower in India than Japan (page 160). In Japan, there is also a specific phobia related to the disgustingness of one’s own body that does not tend to be reported in Western cultures.

Iancu et al. (2007) investigated the prevalence of specific phobias among 850 Israeli youths of both genders recruited into schools for military medicine or mechanics. They found that phobic symptoms were more present among males, those in the school for mechanics, those who had not graduated from high school, those not in a romantic relationship and those with less than two good friends. The researchers suggest that Israeli youths live in a masculine and high-stress psychological atmosphere. Both these characteristics are suggested by Arrindell et al (2003) as causing a higher prevalence of agoraphobia among young people. This 2003 study found that the prevalence of agoraphobia was much higher in countries such as Japan and Hungary that scored high on Hofstede’s masculinity/femininity index (page 135) than in low scorers such as Sweden and Spain.

Although Iancu et al. cannot show exactly how this engenders the development of specific phobias, some suggestions are offered by Chapman et al. (2007). These researchers offer explanations based on their findings when they compared African and Caucasian Americans with regard to the types of specific phobia experienced. They found that African Americans held more fears, with the greatest number grouped in the natural environment category (e.g. fear of deep water or storms) whereas Caucasian Americans tended to hold most fear over situations (e.g. public speaking or flying). Earlier studies finding similar results have been explained in terms of a generally higher level of anxiety among African Americans because of overt racism, but Chapman et al. prefer to explain that specific phobias are transmitted inter-generationally. This can also account for a larger number of phobias relating to spirits and demons in cultures where these are an accepted part of the culture. In addition, they note that African Americans tend to score lower on standardized measures of anxiety. This could be either because the tests fail to pick up some kind of culture-specific anxiety, or because there actually is less anxiety.

It is clear that, at each level of analysis, we are forced to consider the other levels of analysis. It appears that an interaction between environmental influences such as cultural climate and social expectations as well as individual personality and biological factors can contribute to the kind of cognitive and behavioural symptoms of specific phobias, and it is better to consider them in a more holistic way than to attempt to apply a blanket explanation for the etiology of this disorder.

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<th>EXERCISE</th>
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<tr>
<td>5 Write a table summarizing strengths and limitations of each level of analysis in explaining specific phobias.</td>
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Treatment for specific phobias

There is a range of successful treatments for specific phobias, and many psychologists and psychiatrists these days describe themselves as eclectic. This means they draw on many different theoretical approaches to guide their treatment. An eclectic approach has intuitive appeal but requires therapists to undergo training in a variety of areas.

Psychologists are unable to prescribe drugs but should be aware of how medication
affects patients. Psychiatrists can prescribe drugs, but they should also be able to perform several different therapeutic techniques to help solve a variety of problems. This approach acknowledges that the causes of problems are not always clear. Moreover, different therapeutic techniques will be appropriate according to the needs of clients in terms of class, status, gender, previous experience of therapy, expectations, thinking styles, the urgency of their situation and so on. Several examples of how therapies can work together are given below; you will need to refer to these to answer questions about eclectic treatment. More advice is given at the end of the chapter.

**Biomedical therapy**

Biomedical approaches to treating specific phobias focus on medication to alleviate anxiety symptoms and biofeedback training to help the individual manage their own physiological arousal. Neither of these tend to be used in isolation, but will be addressed separately here.

The use of medication is reviewed by Choy et al. (2007), and mostly consists of the use of benzodiazepines (e.g. alprazolam) although there is some strong evidence that paroxetine, a selective serotonin reuptake inhibitor (SSRI), is effective (Benjamin et al., 2000).

In a study into the use of medication by people with a fear of flying, less anxiety was reported during a flight by patients using alprazolam. However, as is typical of studies using medication, the researchers found that the effects did not last into another event a week later, and in fact symptoms were worse than in a group who had taken a placebo the week before.

For dental anxiety, similar results were found for midazolam (another benzodiazepine): after three months, symptoms were back to their original levels. This study compared the effectiveness of a single dose of the drug with a single individual psychotherapy session, and found longer-lasting benefits for the latter.

The use of benzodiazepines has been criticized for two main reasons.

- They can have significant side-effects and patients can easily develop physical tolerance to them, rendering the drug ineffective. Side-effects can include drowsiness and sexual difficulties, and sometimes an increase in aggression and irritability.
- They do not tackle the cause of the problem. They maximize the effectiveness of GABA, the key neurotransmitter involved in the parasympathetic nervous system that calms a person down, but fail to solve the problem of the initial reaction, whether this is a biologically, cognitively or socioculturally caused phenomenon, and individual psychotherapy is therefore usually favoured.
Sedatives like nitrous oxide have been found to be effective for dental phobia, perhaps because of the context in which the phobia manifests. Going to the dentist is not a daily activity for most people, and there is little risk of an unexpected encounter with a dentist’s chair, so the immediate calming effects in the presence of a trained professional are effective. Long-term studies show that even where there is not physical tolerance to the drug, the benefits of medication do not last.

**Individual therapy**

The two most successful treatments are behavioural and cognitive. Behavioural treatment is based on classical conditioning theory, which suggests that the fear is a learned response to a stimulus, and that this association can be broken through various different approaches in therapy.

Systematic desensitization is probably the best known of these approaches. It consists of the construction of a hierarchical set of fear situations relating to the phobic stimulus, training in muscle relaxation, and then exposure to the stimulus through imagination. Ideally, as the person undergoing desensitization imagines progressively worse situations as they learn to relax, they replace the response of fear and anxiety with a relaxation response. Initially, for example, a person might be asked to imagine being outside an airport; after they can relax at the thought of this, they then imagine going into the airport. Eventually, they will imagine getting on the plane. Ideally, by the time they try to do this in reality, their anxiety response will have been extinguished.

Choy et al. (2007) suggest that studies into systematic desensitization have shown that it is good at reducing anxiety levels, but not so good at reducing avoidance behaviours. Thus, people will feel less anxiety, but still have the problems of functioning that are likely to have brought them to the attention of practitioners initially. Choy et al. also show that when systematic desensitization has been beneficial, the effects are long lasting – up to 3.5 years in one study without further treatment. Individual therapy like this is preferable to medication because of its effectiveness, and also because it appears to be correcting the problem rather than alleviating the symptoms. It is a better long-term solution without side-effects.

In vivo exposure seems to be the most immediately effective individual treatment. This involves real life exposure to the phobic stimulus, such as a real flight on a plane, or actually touching a spider. This approach was founded on behaviourist principles and expects to extinguish the fear response by targeting the worst possible scenario the person can imagine. Generally, since by definition phobias involve fear disproportionate to the actual threat involved in the situation, this is not as dangerous as it sounds, and it is a very effective treatment for people who have problems with flying and heights in particular. With animal phobias it is unclear how effective the treatment is, but a study of dog phobics (Rentz et al., 2003) suggests that real exposure was no better than imagined exposure.

Virtual reality therapy is now used for the same purpose as in vivo exposure and has been shown to be quite effective but perhaps more expensive than necessary in some situations. Choy et al. (2007) give the example of treatment for spider phobia being something of a waste of money. It is obviously more sensible to apply this technology to more expensive situations like flying.
Cognitive therapies are also used to treat specific phobias. They attempt to correct some of the faulty thinking that is assumed to be causing the problem. This means that irrational or exaggerated thoughts need to be restructured, often with the presentation of factual evidence that challenges a person’s beliefs about the likelihood of danger. Booth and Rachman (1993) found this type of therapy to be successful by itself and when combined with in vivo exposure for claustrophobia. The effects tend to last longer, although this seems to be more the case for claustrophobia than dental phobia (Choy et al., 2007), possibly due to the genuine likelihood of discomfort and pain in a dental situation.

An eclectic approach can be used in individual therapy by combining medication with therapy or by choosing an appropriate psychotherapy from the range available. One of the most difficult problems a person faces when they have a phobia is the combination of anxiety and the desire to avoid therapy. If medication can help to reduce the effects of this, then it is possible that exposure therapies will be more successful.

Beutler (1991) gives guidance for psychotherapists interested in taking a truly eclectic approach by suggesting a number of questions that need to be addressed before therapy. In the specific example of a woman who has a phobia of automobiles, the initial questions are non-directive or non-confrontational to build rapport and relax the patient. He then begins behavioural therapy with homework exercises and later includes the husband to work on her interpersonal communication skills as well.

**Group therapy**

Group therapies are also used in treating specific phobias. Öst (1998) tested the effectiveness of treatment carried out in groups of eight people with spider phobia. One group received direct treatment with modelling from the therapist, each participant having their own set of four spiders of increasing sizes that they needed to learn to touch; a second group observed one person receiving this treatment; and a third group watched a video of this type of treatment. Anxiety levels were reduced much more in the first group, who all touched the spiders, than in either of the other groups. Öst explains that this is probably due to the increase in self-efficacy that having gone through such a procedure gives participants. Group therapy is cheaper and more efficient in terms of time than individual therapies. However, it can be difficult to predict how long it is going to take if individual outcomes are set, as in this case of group systematic desensitization where the aim was for all participants to complete their tasks.

Group cognitive behaviour therapy has also been used for children with a mix of different anxiety disorders and seems to have good long-term outcomes, partly because of the advantage of seeing peers develop new skills in dealing with their anxiety. In Lumpkin et al. (2002), all children showed improvement, except one. Although this child was not one of those with a specific phobia, it highlights one of the problems of group therapies: they are good for those who are showing improvement, but they can compound problems by reducing self-efficacy for those who are not showing improvement.

**Affective disorders**

**Definitions and diagnosis**

Affective disorders are disorders related to mood. There are several disorders included within this category, and again, for the purposes of the IB examination, you need to focus on only one of these. Here we consider major depressive disorder.

The DSM-IV-TR criteria for diagnosing this condition are outlined overleaf.
**Relevant criteria for major depressive episode**

*American Psychiatric Association*

**A** Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either:
- depressed mood
- loss of interest or pleasure.

**Note:** Do not include symptoms that are clearly due to a general medical condition, or mood-incongruent delusions or hallucinations.

1. Depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g. feels sad or empty) or observation made by others (e.g. appears tearful).
   **Note:** In children and adolescents, can be irritable mood.

2. Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated by either subjective account or observation made by others).

3. Significant weight loss when not dieting or weight gain (e.g. a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day.
   **Note:** In children, consider failure to make expected weight gains.

4. Insomnia or hypersomnia nearly every day.

5. Psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down).

6. Fatigue or loss of energy nearly every day.

7. Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick).

8. Diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others).

9. Recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

**B** The symptoms do not meet criteria for a mixed episode (including mania as well).

**C** The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

**D** The symptoms are not due to the direct physiological effects of a substance (e.g. a drug of abuse, a medication) or a general medical condition (e.g. hypothyroidism).

**E** The symptoms are not better accounted for by bereavement (i.e. after the loss of a loved one) the symptoms persist for longer than 2 months or are characterized by marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.

The United States National Institute of Mental Health claims that major depressive disorder is the leading cause of disability in the USA between the ages of 15 and 44 with a lifetime prevalence of 16.6% (Kessler and Merikangas, 2004). It affects women more than men. This difference appears to start around the age of 13, and results in up to three times more women than men having a diagnosis of depression. Several recent studies have compared the prevalence of depression in various countries, and there is significant variation. Polish men, for example, had a prevalence rate of 20.4%, and both Polish and Russian women were high at 32.9% and 33.7% respectively (Nicholson et al., 2008). However, this particular
study used self-report data and may reflect a reporting bias rather than genuinely higher rates of depression.

Differences can be difficult to account for as there is no experimental way to address the problem. Explanations include the tendency for psychiatrists to be male and from the dominant culture, which results in over-diagnosis for groups that they fail to understand or have stereotyped beliefs about (i.e. women and minorities). Otherwise, there may be genuine differences that are either biological or sociocultural in origin, as shown by Levav’s 1997 study indicating a higher prevalence of depression among Jewish males with relatively low alcoholism. This same study also offers the possibility that some groups are more likely than others to seek help for depression and, in some cultures, going to see a medical doctor when there are mood problems is not the expected path of action. A barman, a priest, a shaman or a friend might all be more appropriate. Etic and emic approaches to the study of depression are discussed on page 140.

**EXERCISE**

6 Under the headings Affective, Cognitive, Behavioural and Somatic, group together the symptoms of depression.

**Etiology: Biological level of analysis**

Hagen et al. (2004) offer an evolutionary perspective on major depressive disorder, suggesting that it is a psychological adaptation favoured by natural selection and serves two main purposes: to signal need and to elicit help from others in the social group. This is an interesting approach with powerful logical argument (including, for example, the point that most suicides by depressed individuals are preceded by the threat of suicide, which therefore signals a need for help and attention). However, like all evolutionary explanations, it is impossible to test experimentally. In addition, we would still need to find the genetic basis of this evolved behaviour.

The search for genes for depression has involved a large number of twin studies and, more recently, molecular genetic research has attempted to identify genes with a role in depression. A very large Swedish twin study with over 42 000 participants used telephone interviews to diagnose depression on the basis of (a) the presence of most of the DSM-IV symptoms or (b) having had a prescription for antidepressants. The researchers found concordance rates among monozygotic twins of 0.44% for female and 0.31% for male, compared with 0.16% and 0.11% for female and male dizygotic twins respectively. If the disorder were purely genetic, we might expect the monozygotic concordance rates to be much higher. But the difference between monozygotic and dizygotic concordance rates is enough to indicate a strong genetic component (Kendler et al., 2006).

**EXERCISE**

7 What ethical and methodological problems are there with a study like this?

Some convincing research suggests that short alleles of a gene known as 5-HTT affect the transmission and reuptake of serotonin to increase the chances of a person suffering from depression. People with the long allele seem less likely to suffer depression.

Drugs that prevent serotonin reuptake (e.g. Prozac) are known to improve the symptoms of depression but Levinson (2005) notes that the short allele on 5-HTT acts in a similar way; it hinders reuptake of serotonin. This means that a hypothesized genetic cause of depression and a medical treatment for depression have the same physiological effect. This confusing
picture needs further research. Levinson (2005) also notes that rather than causing depression, the gene is more likely to make individuals more sensitive to stressful life events.

The catecholamine theory of schizophrenia concentrates on neurochemistry to explain depression. It was proposed in 1965 after researchers found that reserpine, a drug used for hypertension, was apparently able to cause a depression-like state, and that iproniazid, a drug used to treat tuberculosis, was able to improve mood (Rivas-Vazquez and Blais, 1997).

These drugs are no longer in use but they affected the release and break-down of catecholamine neurotransmitters, which include noradrenaline, dopamine and serotonin. This led to the rather reductionist theory that depression is caused by deficiencies in the availability of these neurotransmitters, with serotonin becoming the primary target of research and theory in more recent times. This theory was supported by the large amount of research demonstrating the effectiveness of the new drug fluoxetine (marketed as Prozac), timed around its introduction in 1988.

Problems with the serotonin hypothesis are summarized by Lacasse and Leo (2005). The central problem is a lack of evidence that any depressed person has low levels of serotonin. In fact, there is no baseline balanced level of serotonin to measure against. The authors cite a common analogy for what is wrong with using the effectiveness of a treatment as support for a causal explanation: headaches are successfully treated by aspirin, but we do not have any theory suggesting that headaches are caused by a lack of aspirin. In addition, tianeptine, a drug commonly used in Europe and South America to treat depression, has precisely the opposite function to fluoxetine (Sarek, 2006). Its effectiveness lies in the prevention of neuronal damage due to stress, which brings us to the role of hormones in depression.

The hormone that has received the most attention from researchers in depression in recent times is cortisol. It is seen as particularly relevant because of its role in stress. A meta-analysis of studies connecting this hormone with depression found that there seems to be a difference in reactivity to stress between depressed and non-depressed people: when non-depressed people are put under stress, cortisol levels rise and fall rapidly; depressed people have a more blunt reaction and remain under stress for longer (Burke et al., 2005).

A study of homeless children between the ages of 4 and 7 found a significant correlation between high levels of cortisol and a history of many negative life events (Cutuli et al., 2010). Higher levels of cortisol were found in children whose families were unable to participate in a poverty alleviation programme in Mexico. The most important group differences within the sample were between children whose mothers were depressed – the results indicated that participating in the programme, which included advice about good mothering, helped to reduce stress levels in children who might otherwise have been likely to also develop depression (Fernald and Gunnar, 2009).

Unfortunately, we are still left without a clear understanding of how depression might develop biologically, despite a number of physiological correlates of the disorder having been identified. At best, it seems that this information indicates a need for more research about the interactions between stressful life events, genes, hormones and neurotransmitters.

**Etiology: Cognitive level of analysis**

Cognitive psychologists have suggested a cognitive vulnerability model for depression. According to this model, people who have certain cognitive characteristics are more likely to become depressed. This is not because depression is caused by these characteristics, but that they make an individual more vulnerable.

Aaron Beck (1976) suggests that a cognitive triad underlies the information-processing style of depressed individuals. The cognitive triad is a cluster of negative thoughts grouped into three categories: the self, the world, and the future. A person develops and maintains
these negative core beliefs through a set of cognitive biases such as overgeneralization (e.g. I always fail tests), selective abstraction (usually focusing on the negative parts of something), and polar reasoning (not being able to appreciate ambiguity in interpretations of life). These thinking styles combine to give the person a negative self schema – a fundamentally pessimistic attitude about themself which can be contributed to by parents or peers early on in life, and makes it very, very difficult for a person to see anything positive in life. One of the problems with Beck’s work is that, although it is descriptively very powerful, it remains unclear whether this information processing is really a cause of depression, or if these models are simply a good description of disordered thinking.

Hankin and Abramson (2001) extend the model to try and correct for this weakness and also to improve its ability to explain gender differences in the prevalence of depression. Core to their extension is the occurrence of a negative life event that creates negative effect (e.g. a sad or angry mood) before the thinking styles described by Beck come into play. Their model helps explain how traumatic experiences like sexual abuse or separation from close relatives can contribute to the construction of negative self schemata and provides targets for therapy.

Another well-known theorist, Albert Ellis (1962), offers a similar explanation, focusing on negative cognitive style as the basis of depression. Specifically, irrational and self-defeating beliefs affect an individual’s interpretation of antecedent or activating events, leading to negative emotional consequences. For example, if all members of a class received a low score for a test, some would be content and others would be upset. The activating event was precisely the same, but the consequences are different, and it is underlying beliefs that are responsible for this, beliefs like ‘I am stupid’ or ‘the teacher hates me’ being possible culprits.

These explanations resonate with most people, and there is some empirical support suggesting that depressed people do indeed have negative thinking styles (Robins and Block, 1989). However, you should bear in mind the work of Taylor and Brown (1988) which suggests that depressed people are actually more realistic in their interpretations of activating events (page 148).

Etiology: Sociocultural level of analysis

At this level of analysis, we focus on social and cultural factors that seem to increase risk or vulnerability. There is a large amount of research on this area, much of it sparked by the work of Brown and Harris (1978), who provided a vulnerability model based on the interaction of vulnerability factors and provoking agents. Their original vulnerability factors were:

- losing one’s mother at an early age
- lack of a confiding relationship
- more than three young children at home
- unemployment.

To access Additional information 5.1 on how underlying beliefs can affect a person’s interpretation of the world and cause depression, please visit www.pearsonbacconline.com and follow the on-screen instructions.
Unemployment and poverty, in particular, are factors that have been associated with depression, but in themselves seem unlikely to be responsible for creating feelings of extreme sadness. They must be understood within the context of cultures where work and material wealth provide meaning, status and identity to people’s lives. In the same way, some cultures place more social value than others on the existence of an intimate relationship with one other person, and family members have very different roles in terms of social support.

Nicholson et al. (2008) found that men in the most socially disadvantaged groups in Poland, Russia and the Czech Republic were five times more likely to report depressive symptoms than their compatriates in higher socioeconomic groups. In the USA, there appears to be lower prevalence of depression in Hispanic communities (Wu and Anthony, 2000), supposedly because levels of social support are higher and act as a preventative against depression. Similarly, Gabilondo et al. (2010) found that depression occurs less frequently in Spain than in northern European countries, and that there is a lower rate of suicide. Stronger traditional roles of family and higher religiosity were proposed as sociocultural variables that might explain lower prevalence in this study.

Deeper analysis suggests that in countries and historical periods where social inequalities are more pronounced, rates of depression seem to be higher (Cohen, 2002). This is possibly because of feelings of powerlessness and worthlessness that, in some cases, have a physical basis through the experience of physical stress or undernourishment. In other cases, these feelings are a more subjective product of socialization within individualist, materialist cultures; such feelings lead to perceptions of inequality, unfairness and inability to participate in the ‘ideal’ society enjoyed and advertised by those in higher socio-economic groups.

Various commentators have suggested that depression did not exist in cultures outside the West, but others have found that with increasing Westernization, rates of depression increase. This may not simply reflect the influence of new social pressures or some sort of cultural channelling of unhappiness into a specific set of symptoms; it may reflect changes in diagnostic patterns as mental health practitioners become more Western in their methods. Researchers have noted in Africa and Asia, for example, that depression is accompanied by somatic symptoms, but certain core symptoms are shared across cultures; these core symptoms should be the focus of attention (Okulate et al., 2004). The core symptom most often shared is the affective component of depression, with somatic symptoms secondary in Africa, while suicidal thoughts and guilt are more common secondary symptoms in European patients (Binitie, 1975).
A final set of explanations for depression at a sociocultural level of analysis comes from studies employing Hofstede’s cultural dimensions (page 135). Arrindell (2003) found a high correlation between prevalence of depression and scores on the Masculinity–Femininity Index in a sample of European countries. Individualism has also been found to be associated with high rates of depression. In a recent study that integrated biological and sociocultural levels of analysis, Chiao and Blizinsky (2010) found that depression was associated with individualism and that this dimension had a negative correlation with the frequency of the short allele relating to serotonin transporters (page 167). Rather than suggest that a lack of social support causes depression, the researchers suggest that cultural norms, such as increased social support, have developed to protect the more biologically vulnerable groups; thus, collectivism is evidence of biological vulnerability. More research will be needed to confirm these findings as the data required are not readily available for many of the collectivist countries around the world.

Treatments for major depressive disorder

Biomedical therapy

Drugs are widely used to treat depression because we are aware of some of the neurochemical activity associated with the disorder. The serotonin hypothesis suggests that there is an inadequate amount of serotonin available in the synaptic gap between neurons for effective transmission to occur. Many medications aim to increase the amount of serotonin available. Most of these work by preventing the reuptake of serotonin, making it stay in the synaptic gap longer, and thereby increasing the efficiency of the serotonin already present. Such drugs are called selective serotonin reuptake inhibitors (SSRIs), and include fluoxetine (first marketed as Prozac). It is now produced by many drug companies under different brand names. The two main criticisms of fluoxetine are that it treats the symptoms but does not cure the disorder, and that there are significant side-effects. Side effects include sexual problems, dry mouth, insomnia, and even an increase in suicidal thoughts. For many people, these side-effects outweigh the benefits of the medication, and it seems that the drug is more helpful for more serious cases of depression. Because only the symptoms are treated and because depressive episodes usually recur, it is necessary for patients to continue taking the medication. Unless the medication is used with therapy, it is unlikely that the disorder will disappear permanently.

We can look at two main tests of how well these drugs work. One way is to compare the drug with a placebo when prescribed to patients with depression, and the other is to compare it with other forms of therapy. One difficulty in looking at this research is that many of the studies which show that medication is no better than placebos have gone unpublished. A large meta-analysis by Kirsch et al. (2008) showed that there is at best only a small difference in efficacy between placebo and medication.

The European Committee for Medicinal Products for Human Use has addressed these concerns and claims that the design of many outcome studies is flawed: instead of measuring only change in severity of symptoms, an absolute criterion should be set, and the proportion of patients on the drug who reach this criterion should form an additional...
Antidepressants do better in this kind of research. The debate is a very political one, and the potential consequences of regulatory bodies accepting Kirsch’s conclusion that antidepressants are not effective would include a massive loss of income for a number of pharmaceutical companies, so it is not taken lightly. There is also an ethical problem with research, in that lying to patients about the kind of treatment they receive is not only deceptive, but also possibly dangerous if the patient is having frequent suicidal thoughts.

A meta-analysis of studies comparing the effectiveness of various treatments for depression (Cuijpers, 2009) found that psychotherapy groups do significantly better than control groups (which sometimes include discussions as a placebo). Medication was found to be more effective than psychotherapy in improving symptoms, especially when SSRIs were used. But the best results were found in studies that used a combination of medication and psychotherapy. The authors of this study note that although psychotherapy appears to be effective in alleviating symptoms and may have good long-term effects, this is more true for patients with milder forms of depression.

One further biomedical treatment should be considered here: electroconvulsive therapy (ECT). This is a very controversial treatment that has significant restrictions on its use in most countries. It is generally claimed that this form of treatment is only appropriate for individuals for whom other forms of treatment have failed. Its use is in decline, but it is interesting to note that almost half of the people who receive ECT are over the age of 65, and by far the majority are female, up to 76% in Finland (Read et al., 2004).

Individual therapy

The most well-known individual therapy for depression is cognitive-behavioural therapy (CBT). It was initiated by several psychologists in the 1950s and 1960s, the most famous among them being Aaron Beck. The therapy consists of identifying the automatic, negative thoughts assumed to underlie the depression and helping the depressed person see and understand the connection between these thoughts and their emotional state. By addressing these thoughts together, and through individual homework exercises like keeping a mood diary, the person in therapy and the therapist can gradually change the negative self-schema and find more positive ways to interpret life events. This clearly accounts for the cognitive name in this approach to therapy, but there is also a behavioural side. This involves identifying behaviours that are rewarding for the individual and encouraging him or her to engage in them. One of the key symptoms of depression is a loss of interest in activities that used to give pleasure, and it is an aim of therapy to regain these levels of interest. At first, this may be a difficult and effortful process, but it should provide enough positive reinforcement for the client to continue participating in the world in a positive way.

Another form of therapy emerged accidentally when it was found repeatedly that tests of efficacy of psychotherapy using a control group saw improvements in the control group as well as the patient group. The control groups often consisted of a sympathetic person discussing past experiences but without any theoretical guidance, and yet this proved to be
Interpersonal therapy (IPT) was developed based on this finding. It concentrates on helping the client develop and use any positive social support networks they have in their life with improved communication skills. At times, IPT adjusts clients’ expectations to be more realistic.

A review of the effectiveness of CBT and IPT indicates that IPT alone is not as quick as medication in relieving symptoms, but does provide substantial improvement at a slightly later point (Parker et al. 2006). The review highlighted an interesting finding: patients who receive medication in addition to IPT do better than patients who receive IPT in addition to medication. This is perhaps a reflection of patient expectation in terms of a preference for medication to solve problems. Studies comparing the effectiveness of IPT and CBT by assigning participants randomly to treatment groups have found no significant differences between IPT and CBT treatments, but Parker et al. (2006) suggest that this may be because psychotherapies are not necessarily delivered according to a pure theoretical format.

Butler et al. (2006) reviewed several meta-analyses of efficacy studies for CBT and concluded that CBT is extremely effective for depression, although the effect is not usually greater than medication alone, and outcomes are usually better when CBT is combined with medication.

Although individual therapies like this might appear to be inappropriate for use in other cultures with more collectivist tendencies, an interesting review by Hodges and Oei (2007) considers the likely applicability of CBT to Chinese culture. Their conclusion is that because of the power distance between therapist and client, CBT may actually be more effective in Chinese culture because clients are likely to accept the therapist’s interpretations and advice about faulty information-processing. An expected disadvantage would be that for many clients, successful CBT requires an element of argument, and the therapist needs to be persuasive. Thus, although for Chinese clients the therapist’s word would be taken, without the process of argument and justification, the true nature of the thoughts at the heart of depression might never be identified.

The most common form of eclecticism in treatment for major depressive disorder combines the use of medication with psychotherapy. In many cases, a person has come to the attention of the health system because they have become a danger to themselves or others. In these circumstances, medication is often the fastest way to see the kind of results that mean psychotherapy can begin. It is sometimes considered irresponsible to use either medication without therapy or therapy without medication. In the former case, this is because it makes the person dependent on the medication and they will likely relapse if they stop taking it. In the latter, because when a person’s thinking is disordered, appealing to them on a rational basis can be very difficult. When CBT is successful, it teaches people the kind of skills they need in order to function without further use of medication.

Group therapy
Group therapy is another option for people with depression. The rationale here is that people who may not hear or share when they are alone with a therapist may be encouraged to participate in discussion when they are surrounded by others. There is a chance that they can learn vicariously through the experience of others and become more optimistic about their own chances for recovery if they meet others who have improved.

Group CBT is commonly carried out and has been shown to be effective in several countries, including South Korea, where Hyun et al. (2005) randomly assigned depressed adolescents at a shelter for runaways to group CBT or a group receiving no treatment. They found group CBT to be extremely effective at relieving symptoms of depression. This is not an isolated result. Meta-analyses by McDermut et al. (2001) and Toseland and Siporin (1986) indicated that group therapy is at least as useful for patients as individual therapy.
Truax (2001) considers group therapy to be well validated empirically as a treatment for depression but qualifies this by saying that most of the studies included in meta-analyses excluded the more severely depressed patients. This means that we do not know if group CBT is effective for all depressed people.

Clearly one of the disadvantages of group therapy is that dissatisfaction with the group or any of its members might lead to drop-out, and Truax (2001) cites this as the main reason why people drop out from studies like this. Clearly if optimism is to be improved among individuals suffering from depression, it could be counter-productive to populate the group with severely depressed people, especially if they have been undergoing treatment longer.

### Eating disorders

#### Definitions and diagnosis

Eating disorders are known to affect females much more than males: only an estimated 5–15% of people with anorexia or bulimia are male. The National Institute of Mental Health in the United States suggests that females with anorexia have a death rate 12 times higher than the general female population. This clearly contributes to the status of anorexia as the psychiatric disorder with the highest mortality rate (Van Kuyck et al., 2009). The disorder affects those from households with above-average income to a greater extent, and affects around 0.3% of the population (Zandian et al., 2007). It usually begins between the ages of 14 and 19. Symptoms can include performing rituals at mealtimes like cutting food into small pieces, and excessive exercise.

The condition is far more common in Western and individualist cultures, and it is not clear why this is so. Possible explanations include a greater focus on dieting since the 20th century in Europe and Anglo-American societies, exposure to unreasonably thin models in television, film and magazines, and social pressures to conform to a particular body weight, all of which appear to affect females more than males. While a biological explanation for this might be possible, it may be that males with body-image issues tend to perform different behaviours rather than self-starving.

It is interesting to compare the prevalence of eating-related behaviour in different cultures. Although eating disorders appear to be more uncommon in less industrialized countries and countries where there are more limitations on women’s behaviour, it is also possible that cases of anorexia are more concealed in some cultures and therefore never diagnosed. Within the USA, Roland (1970) found that class and ethnicity were both important contributors – the vast majority of anorexia patients were Caucasian, with people of Italian and Jewish origin being over-represented in the statistics. Rates of anorexia seem to have increased during more affluent periods and in cultures where food is valued and in abundance.

The DSM-IV diagnostic criteria for anorexia nervosa are given below.

#### Diagnostic criteria for anorexia nervosa

*American Psychiatric Association*

A. Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g. weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).

B. Intense fear of gaining weight or becoming fat, even though underweight.

C. Disturbance in the way in which one’s body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
In postmenarcheal females, amenorrhea (i.e. the absence of at least three consecutive menstrual cycles). (A woman is considered to have amenorrhea if her periods occur only following hormone (e.g. estrogen) administration.)

**Specify type:**
- restricting type: during the current episode of anorexia nervosa, the person has not regularly engaged in binge-eating or purging behavior (i.e. self-induced vomiting or the misuse of laxatives, diuretics, or enemas)
- binge-eating/purging type: during the current episode of anorexia nervosa, the person has regularly engaged in binge-eating or purging behavior (i.e. self-induced vomiting or the misuse of laxatives, diuretics, or enemas)

**EXERCISE**

8 Under the headings Affective, Cognitive, Behavioural and Somatic, group together the symptoms of depression.

**Etiology: Biological level of analysis**

An evolutionary explanation for anorexia nervosa has been offered by Surbey (1987). Based on the findings that the weight loss usually comes after the amenorrhea (cessation of menstruation) and that anorexia often occurs in girls who are maturing early, the reproductive suppression model suggests that starvation is an adaptive response to stress that deliberately delays the onset of reproductive capabilities until a more appropriate time. The obvious weakness of this idea is that it excludes males; this is a weakness even if males are a very small sub-group of anorexics. However, the model does offer a possibility for longitudinal research in that it predicts that females who delay the onset of menstruation (or halt it after it has started) should enjoy greater reproductive success later. It also helps explain the obsession with food that many anorexic patients have; adaptive behaviour in times of food shortage and starvation is to shift attention to the acquisition of food.

Zandian et al. (2007) suggest that anorexia is an expression of an underlying obsessive–compulsive disorder (OCD, an anxiety disorder) as these disorders frequently precede anorexia. There is an hypothesized genetic basis to the disorder: in males, the OCD does not usually manifest itself as anorexia, but female biology interacts with the OCD to transform it into an eating disorder. Twin studies have provided varying estimates of how heritable anorexia is. A Swedish study that considered shared environment as a confounding variable and attempted to correct for it gave a heritability estimate of 56%, suggesting that there is a strong genetic component (Bulik et al., 2006). Striegel–Moore and Bulik (2007) indicate that molecular genetic studies have isolated potential genes, particularly related to the development of serotonin receptors, that may be responsible for mood issues among anorexic patients. However, the relationship between these genes and the disorder remains unclear.

Serotonin levels have been found to be low in many anorexic patients, and this has therefore been proposed as a possible cause of the disorder. However Zandian et al. (2007) point out that the studies which measure serotonin levels are not usually longitudinal – they do not have a ‘before’ measure as this is almost impossible to obtain. When an ‘after’ measure is included, it appears that the resumption of normal eating habits and return to healthy weight are accompanied by a return to normal serotonin levels. Given that serotonin is known to inhibit eating, it seems likely that decreased serotonin levels do not cause anorexia, but instead are a consequence of reduced food intake (Zandian et al., 2007).

Van Kuyck et al. (2007) summarize brain-imaging studies with anorexic patients, and show that the parietal cortex is frequently underactive. Decreased activity in this part of the brain could relate to anorexic patients’ symptoms of overestimating their own weight and shape, and...
and to a kind of anosognosia – lack of knowledge and insight into their disorder. This part of the brain is sexually dimorphic (i.e. it is different in size in males and females) and this may explain the large difference in prevalence of the disorder between the sexes. Again, however, there is a problem of identifying cause here. Differences in neurocircuitry may be responsible for the development of the disorder, but it is also possible that poor diet changes the brain in these areas.

A model that integrates much of our knowledge about hormones, neurotransmitters and the brain is offered by Zandian et al. (2007). According to this model, there are two risk factors for the development of anorexia: reduced food intake and physical activity. In combination, these factors encourage the release of corticotrophin-releasing factor and cortisol, which in turn activate the dopamine reward pathways in the limbic system. This gives anorexic patients a reward when they begin dieting and increases the chances of repeated diet-and-exercise behaviour, thus locking the patient into a cycle of addiction-like behaviour.

**Etiology: Cognitive level of analysis**

At this level of analysis, the focus is on how disordered thinking or faulty perception might cause the disorder. Fairburn (1999) provides a detailed account of how low self-esteem and an extreme need for self-control are at the core of the disorder. Fairburn suggests that for most people with anorexia, the need for control is quite easily met through mastery in the eating domain in a way that is harder to achieve through work, school or sport.

The idea that dieting and control go together is a schema commonly built and encouraged in Western societies. The disordered eating patterns are maintained because the person’s sense of control is increased to the point where control over eating becomes a measure of self-worth. In addition, constant checking of body shape to obtain objective information about the success of dieting is made unreliable by distorted perception, caused by negative mood and the presence of thin women in media. In addition, there is an attentional bias towards negative information about body shape. This can compel some patients to stop looking, thus eliminating any chance they might have of finding out just how much weight they have lost (Fairburn, 1999).

Bruch’s body-image distortion hypotheses (1962) was an early recognition of this type of cognitive element to anorexia, suggesting that anorexic people overestimate their body size. However, this may be normal among women anyway, as a study by Rozin and Fallon (1988) found that when families were asked to compare their body shape to their ideal body shape, only the sons reported that their body shape was acceptable. Both mothers and daughters in the sample believe that men prefer thinner women than they actually do. Other psychologists have applied Beck’s ideas (page 160) to anorexia, and suggest that the same tendencies to overgeneralize, personalize and polarize thinking exist for anorexic people as well.

**Etiology: Sociocultural level of analysis**

It is often thought that eating disorders like anorexia and bulimia are culture-bound disorders restricted to the Western world and are encouraged by conceptions of beauty and attractiveness that tell females to be thin and males to be athletic. Lee et al. (1996) suggests that a social fat phobia may underlie anorexia, and that this phobia is beginning to thrive in many other parts of the world. Where anorexia has developed most intensely, there is considerable mass media influence, and large amounts of information about both dieting and eating disorders. Girls who become anorexic are more likely to accept the messages about beauty and thinness in the media. However, it is not clear whether the anorexia has caused this receptiveness, or if acceptance of the message led to anorexia, or if an alternative
explanation is better. Lee et al. also point out that the increase in anorexia in other countries could be an artefact of increased use of Western diagnostic systems that pathologize behaviours. Moreover, body weight is not necessarily indicative of psychiatric problems – an estimated 16% of healthy but slim Chinese women would meet Western diagnostic criteria for anorectic weight. In line with this idea, anorexia was four times more prevalent in Japan in 1998 than in 1993, probably reflecting changes in social support networks and moral values in Japan (Yasuhsara et al., 2002).

Strahan et al. (2007) offer the possibility that the influence of media is not so much that it causes women to believe that they are the wrong shape, as that it encourages them to think that everyone else accepts thin models and actresses as normal and attractive. It is then conformity to the perceived expectations of others that leads to excessive dieting. This influence may be playing a part in our development from a very young age for both males and females; research has shown that toy action figures are increasingly lean and muscular, yet the probability of finding a woman with Barbie’s body shape is less than 1 in 100 000 (Norton et al. 1996). Models for magazine centrefolds and Miss America beauty contestants have been becoming smaller at the same time as advertising for dieting and exercise products has increased (Sypeck et al., 2006).

As compelling as sociocultural arguments are, it is still something of a mystery that only some people are affected by these influences in a way that brings them to the attention of clinical staff. It is likely that an understanding of the interaction between biology, cognition, and the sociocultural context of a person’s life is needed in order to fully understand how anorexia develops.

Treatments for anorexia nervosa

**Biomedical therapy**

SSRIs are frequently used to treat anorexia, although there is limited evidence that this is effective on its own, even though some research has found these drugs can help prevent relapse (Holtkamp et al., 2005). A double-blind study by Kaye et al. (2001) showed that patients given a placebo over a one-year period were much more likely to drop out, indicating that there is some benefit. It seems likely, however, that this medication targets symptoms of anorexia that are not causing the disorder, and provides an argument against the idea that negative mood or depression play a causal role in the development of anorexia, even if they frequently occur together.
Instead of focusing on medication, the biomedical approach tends to ensure that weight gains are made in the first instance, which can mean attaching the patient to a drip. Following this, the patient needs to be encouraged to eat normally again, and this is more likely to be achieved through the use of individual therapies. It is sometimes suggested that anorexia is a form of anxiety disorder or depression, and for some individuals, ongoing use of medication can help prevent the kind of emotional state that precedes relapses. However, it is very uncommon for a person to be given purely biomedical treatment with no access to therapy; treatment for anorexia is almost always eclectic.

**Individual therapy**

Bowers (2002) details a form of CBT that is recommended for use in a multidisciplinary team, recognizing that neither a physician, a psychotherapist nor a dietician is likely to be able to deal with an anorexia patient alone. The ultimate aim of CBT here is to help the individual to understand that their thought processes and belief systems are causing problems, and to help change them. The CBT aims to change (a) negative self-statements like, ‘I’ll never be thin enough’ and (b) basic assumptions that are generally fixed and resistant to change, often in the form of high personal expectations. Changes are also needed to cognitive schemata to do with weight, food and control, so it is first essential to spend time talking with the patient to establish what the content of these schemata are. It may require some practice for patients to identify their own moods and thoughts about these areas. They can then be challenged to produce evidence for these ideas and encouraged to come up with logical thoughts as alternatives to what are generally negative or self-defeating but persistent thoughts. This type of therapy generally has good outcomes and as it attempts to address the thoughts at the core of the problem, relapse is relatively unusual.

There are also behaviourist treatments available for anorexia. Usually, an operant conditioning approach is taken, whereby certain target behaviours are to be reinforced with a reward personalized to the patient. Token economies are not uncommon in hospital wards dealing with anorexic inpatients, and these offer staff a way to observe and reward small improvements in behaviour. Rewards like watching television or spending time socializing with others can be made conditional on completion of a meal. Sometimes, patients are asked to eat a meal placed on a kitchen scale, so that they can see the weight of the food disappearing. The intention here is to form immediate feedback about success in learning new eating habits. While these behaviourist treatments are often quite successful at helping anorexic patients get back to a normal weight, relapse is more likely than with CBT, for two main reasons. First, the reasons behind the disorder have not been addressed, whether these are biological, cognitive, or sociocultural. Secondly, when the person leaves the hospital, they need to have internalized the reward process, or have strong support from family or friends, as the reward system is likely to be neglected.

It is for this reason that family therapy is often offered. This approach requires the whole family to become part of the therapeutic process and has been particularly successful in dealing with anorexia because of the importance of feedback to the anorexia patient from family members. Some models of causation assume that interactions, particularly between mother and daughter, are contributing factors to the development of the disorder, and learning more effective ways to communicate is beneficial for many family members, not just for the person with anorexia.
Harris and Kuba (1997), dealt specifically with the treatment of eating disorders among black women in the US. They noted that there are more individuals with eating problems than are being diagnosed, and that treatment for minority groups requires special attention. They recommend that therapy involve not just the individual patient and therapist, but family, community, and even other practitioners of more cultural relevance, such as a shaman.

**Group therapy**

Group therapy is very common for anorexia patients – both as inpatients, as part of treatment to help them get better, and as outpatients, to help prevent relapse. A study by Woodside and Kaplan (1994) put males and females together in group therapy that specifically targeted negative and destructive attitudes towards food and eating, using a CBT approach similar to that outlined above for individuals. Both males and females showed improvement on the eating attitudes test.

Group therapy is more cost-effective than individual therapy and offers the opportunity for group members to interact with others who are at different stages in dealing with the disorder. This provides hope for those in the early stages, and confirmation of progress for those who are successful, along with increased self-esteem as they have the opportunity to help others.

Polivy (1979) identifies two significant problems with group therapy, however. The first is that being in a group of other anorectic patients lends legitimacy to the development of a new identity based on group membership; the patient then requires individual therapy to help carve out an independent identity. The second problem is that members of the group often teach each other, not always intentionally, strategies to avoid weight gain or hide weight loss. This means group therapy can actually undermine individual progress.

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**Examiner’s hint**

Given that there is such a wide range of possible causes of anorexia and a similarly wide range of treatment options, an eclectic approach seems particularly appropriate for this disorder, so it is wise to use anorexia in an essay asking you to discuss eclectic approaches to treatment.
5.4 Treatment review

Learning outcomes

• Examine biomedical, individual and group approaches to treatment.
• Discuss the use of eclectic approaches to treatment.
• Discuss the relationship between etiology and therapeutic approach in relation to one disorder.

Evaluating treatment

The first learning outcome above requires you to look at treatment for more than one disorder. This chapter systematically deals with the treatments for each disorder under the discussions of each disorder. You therefore need to organise your own notes to bring together information on treatment options.

EXERCISE

9 Using the information in this chapter, make notes in bullet-point form under the headings Biomedical, Individual and Group on:

- types and examples of treatments used to treat disorders
- strengths and limitations
- gender and culture issues in treatment
- research: key examples and methodological problems.

Use these notes to construct an essay plan that describes the treatments offered and evaluates them, and uses research to back up any claims that you make.

The second of the learning outcomes listed above is concerned with eclectic approaches. In some countries, a general practitioner is able to prescribe medication, so sometimes a biomedical approach may be taken by itself; on the other hand, a patient might enter group or individual therapy without being medicated. However, it is rarely the case that a person who is suffering significant problems of living because of one of the disorders in this chapter, will be offered just one treatment. Usually, a multidisciplinary team involving at least one therapist and one medical professional (e.g. a psychiatrist, doctor or a prescribing nurse), will work together to combine their skills and aid the patient’s progress. This eclectic approach is referred to in this chapter several times and there is research demonstrating its effectiveness.

EXERCISE

10 Review the chapter and add to your notes from exercise 9 by noting references to combinations of treatment and record whether they are successful or not.

- Use these notes to construct an essay plan to answer the question Discuss the use of eclectic approaches to treatment.

You need to explain why treatments are not always used in isolation and what the benefits of an eclectic approach are. Remember to link your ideas to research from this chapter (make use of the examiner’s hint boxes indicating studies or information that will be useful).
The relationship between etiology and treatment

The final learning outcome listed above requires you to choose one disorder and consider the reasons why a particular approach is used.

Biomedical treatment usually focuses on the biological level of analysis, using medication to alleviate the symptoms of a disorder without necessarily attempting to cure the person. Individual psychotherapy focuses on the psychological and social features of the disorder and helps the client to develop strategies to function well on a daily basis. Group therapy is similar, but takes advantage of the benefits of being with other people to assist in building social competence as well as individual competence.

**EXERCISE**

11 Choose one disorder from this chapter and make notes under the headings **Biomedical**, **Individual**, and **Group** on:

- proposed cause
- possible treatment
- whether or not the treatment addresses the cause
- gender and culture issues
- research: key examples and methodological problems

Using this information, you can write an essay plan to answer the question *Discuss the relationship between etiology and therapeutic approach in relation to one disorder*. Remember, you need to consider the strengths and limitations of treatment for this disorder and present a balanced argument that includes both evaluation and reference to research.

**PRACTICE QUESTIONS**

1 Discuss how biological factors influence one psychological disorder.
2 Evaluate two studies related to abnormal psychology.
3 Evaluate the use of biomedical or individual or group approaches to treatment for one psychological disorder.

To access Revision notes 5.1 on the three disorders covered in this chapter, please visit www.pearsonbacconline.com and follow the on-screen instructions.

To access Worksheet 5.4 with additional practice questions and answer guidelines, please visit www.pearsonbacconline.com and follow the on-screen instructions.

To access Worksheet 5.4 with a full example answer to question X, please visit www.pearsonbacconline.com and follow the on-screen instructions.