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Learning Objectives

By the end of this chapter you should appreciate that:

- abnormal psychology (or psychopathology) deals with sets of behaviours or symptoms that produce a functional impairment in people's lives;
- psychological disorders (e.g. schizophrenia) have been documented across time and culture;
- throughout history, the causes of abnormal behaviour have been construed from a number of different perspectives;
- biological/genetic models focus on brain defects, biochemical imbalances and genetic predispositions as causes of psychopathology;
- Freudian, contemporary psychodynamic and attachment models focus on the effects of early parent-child experiences;
- behavioural models focus on the learning experiences that result in psychopathology;
- cognitive models focus on the effect of distorted thought processes;
- the diathesis-stress perspective suggests that the factors identified by each of the other models may work in accordance with one another;
- the developmental psychopathology perspective provides a framework for understanding how psychopathology develops from childhood to adulthood;
- these perspectives can help us understand the numerous disorders documented in classification systems such as the DSM-IV and the ICD-10;
- there are several major forms of psychopathology, including schizophrenia, mood disorders, substance abuse, eating disorders and personality disorders.

INTRODUCTION

Abnormal psychology is the study of mental disorders (also called mental illness, psychological disorders or psychopathology) – what they look like (symptoms), why they occur (etiology), how they are maintained, and what effect they have on people's lives.

Mental disorders are surprisingly common. For example, a study conducted by the World Health

Organization examined the prevalence, or frequency, of mental disorders in people visiting medical doctors in primary care settings in 14 countries. As figure 15.1 shows, the study revealed that 24 per cent of these people had diagnosable mental disorders and another 10 per cent had severe symptoms of mental disorders (Üstün & Sartorius, 1995).

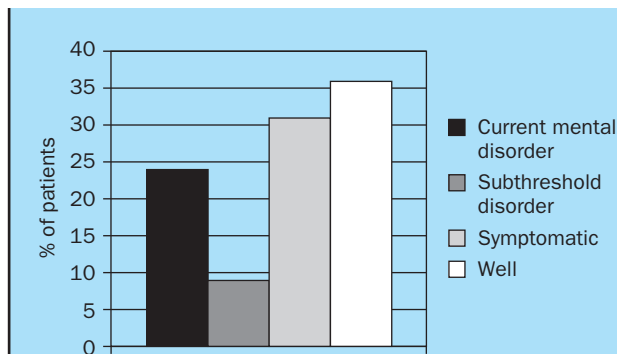


Figure 15.1

Rates of current mental disorder in patients presenting to primary care facilities across the world.
Source: Adapted from Üstün and Sartorius (1995).

Psychopathology can happen to anyone and affects many people around them – there is no age, race or group that is immune. Furthermore, many people experience more than one disorder at the same time (see figure 15.2).

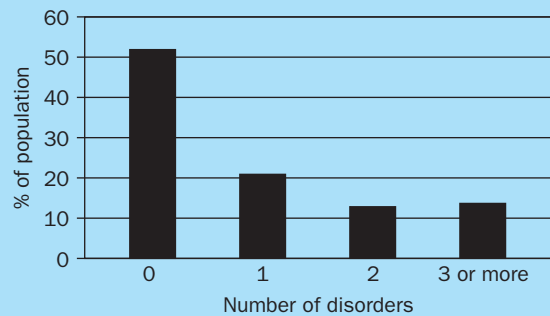


Figure 15.2

Comorbidity in lifetime rates of mental disorders. Comorbidity is the occurrence of two or more disorders at the same time. Of the 48 per cent of people reporting lifetime history of disorder in this study, over half reported two or more simultaneous disorders. These data come from a survey in the US, but similar rates of comorbidity have been found in countries around the world.
Source: Kessler et al. (1994).

The frequency and widespread suffering caused by mental disorders makes our understanding of them critical.

WHAT DOES 'ABNORMAL' MEAN?

Defining abnormality is deceptively difficult. When asked to describe abnormal behaviour, people typically say that it occurs infrequently, is odd or strange, is characterized by suffering, or is dangerous. All of these are reasonable answers for some types of abnormal behaviour, but none of them is sufficient in itself, and making them all necessary results in too strict a definition.

One parsimonious and practical way to define abnormal behaviour is to ask whether the behaviour causes *impairment* in the person's life. The more a behaviour gets in the way of successful functioning in an important domain of life (including the psychological, interpersonal and achievement/performance domains),

impairment extent to which a behaviour or set of behaviours gets in the way of successful functioning in an important domain of the individual's life

the more likely it is to be considered a sign of abnormality. When several such behaviours or symptoms occur together, they may constitute a psychological disorder.

Psychological disorders are formally defined in widely used classification systems, or nosologies: the International Classification of Diseases – 10th edition (ICD-10; World Health Organization, 1992) and the Diagnostic and Statistical Manual of Mental Disorders – 4th edition (DSM-IV; APA, 1994). Although they differ from one another in format, these two systems cover the same disorders and define them in a similar manner.

Importantly, both the ICD-10 and the DSM-IV require that the level of impairment a person is experiencing be taken into account when deciding whether they meet criteria for any mental disorder. For example, the DSM-IV diagnostic criteria for depression specify that: 'The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning' (p. 327). The ICD-10 description of depression also states: 'The extent of ordinary social and work activities is often a useful general guide to the likely degree of severity of the episode' (p. 121).

Finally, it is important to be sensitive to how contextual factors affect judgements about abnormality, so as not to over- or under-pathologize groups or individuals. Such factors include ethnicity and culture, gender, age and socio-political values. For example, homosexuality was once listed as a disorder in the DSM, but, as socio-political values changed to become somewhat more liberal and accepting, it was deleted.

WHAT CAUSES ABNORMAL BEHAVIOUR?

With a basis for understanding how to define abnormal behaviour, we can focus on its causes. Abnormal behaviour is construed from a number of different perspectives. Each of the following models tells us something about different aspects of a multi-faceted group of mental disorders.

BIOLOGY AND GENETICS

Biological and genetic models assert that mental disorders are diseases, and symptoms of mental disorders are caused by factors

biochemical imbalance complex neurotransmitter dysregulation process involving the various neurotransmitters in the brain

genetic predisposition likelihood of showing condition or characteristic carried by genetic material

such as brain defects (abnormalities in the structures of the brain), *biochemical imbalances* (complex dysregulation processes involving various neurotransmitters) and *genetic predispositions* (risk for psychopathology carried via our genetic material).

By and large, the evidence for brain defects and biochemical imbalances as

causes of mental disorders is correlational, which means that, although we know that such biological problems occur among people with mental disorders, we don't know whether they actually cause the disorder. Because the brain is a fairly malleable organ, our behaviour and experiences can also affect our brain functioning, suggesting that the association between biology and abnormal behaviour may be reciprocal rather than unidirectional.

Genetic models of mental disorder suggest that psychopathology is inherited from parents, and there is certainly evidence for

familial transmission genetic transmission of disorders

the *familial transmission* of many disorders. For example, monozygotic (identical) twins should be more likely than dizygotic (fraternal) twins to

have the same disorder because they share 100 per cent of their genetic material, whereas dizygotic twins share only 50 per cent. For many disorders, this is exactly what research shows.

But given that monozygotic twins share 100 per cent of their genetic material, you might expect them to have the same disorders 100 per cent of the time. But in fact they have the same disorders only about 50 per cent of the time. These findings have led researchers to conclude that, rather than being deterministic, genetics contributes about 50 per cent of the risk for mental illness. Such findings show that it doesn't make sense to question whether mental illness is a function of nature *or* nurture. Instead we need to focus on how the two interact.

PSYCHODYNAMICS AND THE PARENT–CHILD RELATIONSHIP

Freud emphasized the role of the early parent–child relationship in the development of mental illness. According to Freud, to the extent that the child did not successfully negotiate the psychosexual stages (see chapter 14), mental illness would develop.

But Freud didn't focus on what actually occurred in the parent–child relationship (e.g. whether parents were actually poor caretakers). Instead, his focus was on the unconscious internal desires and motivations of the child (e.g. sexual and aggressive impulses)

and how the child negotiated them as s/he progressed through the early relationship with the child's parents. For example, if an adult male found himself unable to deal with authority figures, this might be interpreted as unresolved aggressive impulses towards his father. Whether his father behaved as a harsh authority figure or not would be considered less relevant. So, according to Freud, mental illness is due to intrapsychic (i.e. within the mind) conflict. This means a person may have very little insight into the 'true' causes of their symptoms, as these are thought to be occurring at an unconscious level of processing.

Many of Freud's ideas have gone unsupported by research, but a number of them have proven to be fairly accurate. For example, there is ample evidence that people experience and process things at a non-conscious level (see Westen, 1998; also chapter 14) and that early interpersonal experiences affect later outcomes. In fact, this latter hypothesis became central to contemporary psychodynamic models of abnormal behaviour.

Contemporary psychodynamic models (e.g., Kohut, 1977; Kernberg, 1976; Mitchell, 1988) also suggest that the early parent–child relationship is the original source of mental illness, and that what goes on in the mind of the child (and the adult) is important.

But these models differ from Freud's in that they focus more on interpersonal relationships than on intrapsychic conflict. These later models suggest that the early relationship between the child and the primary caregiver is crucial to the development of the self-concept, concepts of others, and the quality of relationships throughout life. The idea is that this early caregiver–child relationship is internalized by children, so that they learn about themselves and others from the manner in which the caregiver treats them. According to this framework, the nature of this internalized relationship and its resulting impact on the sense of self and the sense of others is what can create vulnerability to psychological problems.

ATTACHMENT AND SECURITY

The attachment model of psychopathology, developed by Bowlby (1969; 1973; 1980; see also chapter 9) resembles the contemporary psychodynamic models in that it also emphasizes the early parent–child relationship and how the resulting models of self and others guide development. But rather than being interested in people's perceptions of their early experience, Bowlby was interested in the actual characteristics of the relationship. He relied on observational studies of parents and children to build his theory, rather than on retrospective reports of adults. The theory therefore has a strong empirical foundation.

Attachment theory suggests that when parental behaviour fails to make children feel safe, secure, and able to turn to and trust the parent in times of need, then children will be unable to regulate their emotions and needs adaptively and will develop negative, 'insecure' views of themselves and others. This would put children at risk for developing psychological disorders. Research supports this hypothesis, as 'insecure' children and adults show more psychopathology than 'secure' children and adults (see Dozier, Stovall & Albus, 1999; Greenberg, 1999).

Research close-up 1

Reconciling the roles of genetics and the environment in risk for major depression

The research issue

As you have read in this chapter, genetic and environmental models make very different assumptions about the causes of depression. Kendler et al. (1995) conducted a study in an effort to determine the extent to which genetic and environmental factors contribute to depression. In their study, Kendler and his colleagues examined two important questions: (1) do genetic factors and stressful life events make unique contributions to risk for depression in women? and (2) do genetic factors and stressful life events interact to create risk for depression? In particular, the researchers wondered whether the association between stressful life events and risk for depression would be greater among people at high genetic risk compared to people at low genetic risk.

Design and procedure

To examine these questions, Kendler and colleagues studied four groups of women: (1) women with a depressed monozygotic (MZ) twin, (2) women with a depressed dizygotic (DZ) twin, (3) women with a non-depressed MZ twin, and (4) women with a non-depressed DZ twin. Women with a depressed MZ twin are at the highest genetic risk for depression, and women with a non-depressed MZ twin are at the lowest genetic risk for depression. For each woman, they assessed whether the person in question had experienced a depressive episode in any given month over the course of approximately one year, and they recorded whether any severe life events occurred during each month over this one year time period.

Results and implications

Both stressful life events and genetic factors made unique contributions to depression. Regardless of genetic risk, stressful life events were associated with depression, and regardless of life stress, genetic risk was associated with depression.

- However, the impact of stressful life events on risk for depression was greater among women at high genetic risk than it was for women at low genetic risk (see figure 15.3).
- Interestingly, the stressful life events that were found to be most strongly associated with depression were mainly interpersonal in nature (death of a close relative, serious marital problems, divorce/break-up, and assault), highlighting the importance of relationship factors in risk for depression.

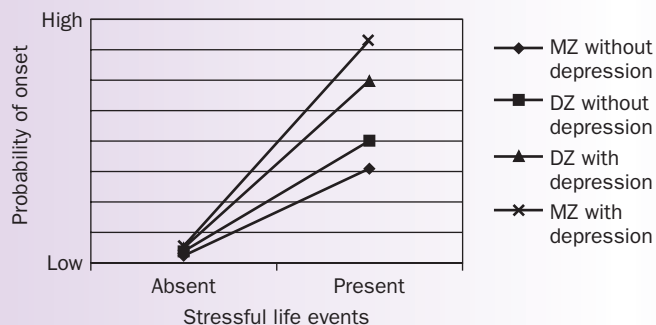


Figure 15.3

Risk of onset of major depression as a function of genetic risk and stressful life events. Source: Kendler et al. (1995).

The findings suggest that both genetic risk and stressful life events are important factors in understanding women's risk for depression. Moreover, consistent with a diathesis-stress model (see p. 319), women at high genetic risk who experience a stressful life event in the interpersonal domain of their lives are at greatest risk of all. Therefore, to understand risk for depression among women best, we must consider both genetic factors and environmental factors.

Kendler, K.S., Kessler, R.C., Walters, E.E. et al., 1995, 'Stressful life events, genetic liability, and onset of an episode of major depression in women', *American Journal of Psychiatry*, 152, 833–42.

LEARNED BEHAVIOUR

Behavioural models suggest that all behaviour, abnormal included, is a product of learning – mainly learning by association (see chapter 4).

For example, according to the classical conditioning model of learning (e.g. Pavlov, 1928), if a man experiences chest pains which result in anxiety while shopping in a department store, he may develop a fear of department stores and begin to avoid them because he associates them with anxiety. There is nothing inherently frightening about department stores, but this man fears them because of the association that he has formed with his earlier anxiety about having a possible heart attack. Here is another example which instead uses the operant model of learning (e.g. Skinner, 1953): if a young normal weight woman begins to lose weight and her friends and family praise her for doing so, she may continue to lose weight, even if it means starving herself. Her restricted eating behaviour will continue because she now associates a reduction in her diet with the praise and acceptance of others.

There is a third type of learning that does not rely on personal experiences to establish associations. In observational learning, behaviour is learned simply by watching someone else do something and observing what happens to them (Bandura, 1969). For example, a young boy may learn to be aggressive after watching his peers act aggressively.

Each of these learning models was built on a solid foundation of empirical research, and there is a great deal of evidence that each of the three learning processes plays an important role in abnormal behaviour.

DISTORTED THINKING

Cognitive models of abnormal behaviour focus on the way people think about themselves, others and the world (e.g. Beck et al., 1979). Distorted cognitive processes – such as selectively attending to some information and ignoring other information, exaggerating negative feelings, expecting the worst, or making inaccurate attributions about events (see chapters 14 and 17) – have been shown to play an important role in various types of psychological disorders.

For example, suppose a woman has a bad day at work. If she says to herself, ‘Oh well, tomorrow will be better’, she will probably feel fine. But if she says to herself, ‘Oh, I’m just a horrible person with no future’ (i.e. if she exaggerates her negative feelings), she may become depressed. Or suppose a young man loses at a game of cards. If he thinks, ‘I sure had bad luck with the cards today’, he will feel fine. But if he thinks, ‘My rotten friends purposely cheated me!’ he may become hostile and aggressive.

INTEGRATIVE MODELS

The models of abnormal behaviour described above are quite different from one another, and each is more or less well suited to particular disorders. As most disorders are quite complex, no

single model can provide a full explanation of their onset and course over time. Instead, each model can help us to understand a different aspect of each disorder. This is where integrative models are useful.

You may have noticed that only some of the models above explicitly focus on childhood factors that may contribute to the development of abnormal behaviour, whereas the others only do so at an implicit level. For example, behavioural models suggest that abnormal behaviour is the product of ‘earlier’ learning experiences, but they don’t elaborate on exactly what those experiences are. By contrast, *developmental psychopathology* provides a more rigorous framework for understanding how psychopathology develops from childhood to adulthood. It is also likely that mental illness results only when particular combinations of factors are present. This notion is at the heart of the *diathesis–stress model*.

developmental psychopathology a perspective suggesting that risk for psychopathology depends on success at negotiating and mastering important developmental tasks

diathesis–stress model suggests that some people possess an enduring vulnerability factor (diathesis), which, when coupled with a proximal stressor, results in psychological symptoms

The diathesis–stress model

The diathesis–stress model of mental illness (figure 15.4) suggests that some people possess an enduring vulnerability factor (a diathesis) which, when coupled with a proximal (recent) stressor, results in psychological disorder. Neither the diathesis nor the stressor alone is enough to lead to symptoms – both must be present.

Diatheses and stressors can be defined broadly. For example, a genetic or biological predisposition to mental illness might be the diathesis, and a troubled parent–child relationship could be the stressor; or a dysfunctional pattern of thinking about the world can be the diathesis, and a major life event the stressor.

		Diathesis?	
		Yes	No
Stress?	Yes	Disorder? Yes	Disorder? No
	No	Disorder? No	Disorder? No

Figure 15.4

The diathesis–stress model of psychopathology. According to this model, psychopathology is most likely to result when a person with a diathesis (vulnerability) experiences a stressor. This model helps us to understand why only some people with vulnerabilities, such as a genetic predisposition, develop psychopathology.

As you read through the following sections on the various disorders, you might want to consider how a diathesis–stress process could describe how each comes about.

Developmental psychopathology

According to this model, psychopathology is best understood using a lifespan development approach. It considers how the negotiation and attainment of earlier developmental tasks affects people's capacities to manage later tasks (e.g. Cicchetti, Rogosch & Toth, 1994).

In other words, people may travel down one of many paths; their success or failure at various junctures along the way determines the subsequent path that they follow. So earlier deficits in functioning may leave us unprepared to successfully negotiate subsequent related situations, putting us at even greater risk for psychopathology.

For example, a young girl who is harshly and chronically criticized by her parents may develop low self-esteem and the expectation that people will not like her, which puts her at risk of becoming depressed. She may then have difficulty making friends in school because she is afraid of rejection. She may feel lonely and undesirable, her withdrawal leading to actual rejection by her peers, continuing her risk for depression. But if this young girl has a teacher who treats her with warmth and care and helps her learn how to make friends, her risk for depression might be reduced. This is because she is acquiring important skills that have the potential to change the course of her subsequent development.

DISORDERS – SYMPTOMS AND CAUSES

For each disorder we will look at its symptoms and the course it takes. Then we will consider its causes, both biological/genetic and psychosocial, and the factors that affect its course. Prevalence rates (i.e. the cross-sectional proportion of occurrences of the disorder in the population) for various disorders are shown in figure 15.5.

SCHIZOPHRENIA – A LIVING NIGHTMARE

Images of schizophrenia are easy to conjure – a dishevelled person, alone, talking to himself or yelling at someone else that only he seems to see. This is a frightening image, for the symptoms it portrays are extremely odd and disconcerting. Indeed, schizophrenia can be a frightening disorder to deal with, not only for those involved with schizophrenic people, but for the sufferers themselves.

Schizophrenia is a severe mental disorder, experienced by many sufferers as a living nightmare, a fact highlighted by the high rate of suicide among schizophrenics (Caldwell & Gottesman, 1992; see table 15.1).

As you read this section, try to imagine what it might feel like to experience some of the things schizophrenic people experience. For example, many schizophrenic people hear voices. Have you ever heard someone call your name, only to find there was

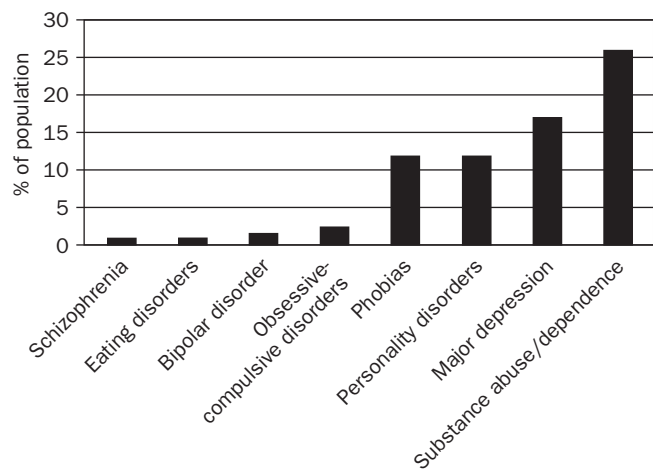


Figure 15.5

Lifetime prevalence rates for psychological disorders. These rates are from studies conducted in the US (Kessler et al., 1994), but cross-national studies indicate similar rates of disorders in other countries such as Switzerland, Germany and Puerto Rico. Source: Kessler et al. (1994).

Table 15.1 Suicide: a serious mental health and public health problem

Facts about suicide	Risk factors for suicide
<ul style="list-style-type: none"> ■ Suicide occurs across the world, but rates vary by culture ■ Self-inflicted injuries, including suicide, were the 12th leading cause of death in the world in 1998 ■ In all cultures, men are more likely than women are to complete suicide ■ Rates of suicide in children and adolescents are on the rise ■ People with mental disorders, especially depression, substance use disorders, schizophrenia, and borderline personality disorder, are at high risk for suicide 	<ul style="list-style-type: none"> ■ Past history of attempted suicide ■ Talking about committing suicide ■ A clear plan to commit suicide ■ Available means (e.g. firearms, drugs) ■ Depression ■ Substance abuse ■ Hopelessness ■ Impulsivity ■ Stressful life events ■ Lack of social support ■ Saying goodbye to people ■ Giving away personal items

no one there? How did that feel? Rather disconcerting, most likely. Now magnify your feelings about 100 times and you may start to sense how the schizophrenic person feels.

Symptoms

Schizophrenia is characterized by *psychosis*, or a break with reality. People who are

psychosis a break with reality, characteristic of schizophrenia

psychotic think and behave in ways that have little to do with reality, showing significant impairment in just about every important domain of functioning – perception, thought, language, memory, emotion and behaviour. People with schizophrenia may exhibit any of these symptoms:

- Perceiving things that are not there – these hallucinations are usually auditory (e.g. hearing voices), but visual and tactile hallucinations (e.g. seeing God or the devil, or feeling that insects are crawling under your skin) also occur relatively frequently.
- Believing things that are not true – *paranoid delusions* are particularly common. A schizophrenic woman may believe

paranoid delusions elaborate set of beliefs, commonly experienced by schizophrenics, characterized by significant distrust of others and feelings of persecution

that the government is plotting against her or that aliens plan to kill her. Everything will be interpreted in the context of the delusion, even things that are meant to help, so medication will be seen as poison. Delusions of grandeur

are also common: a schizophrenic person may believe that he is someone famous, such as Elvis or Jesus Christ, and may insist on behaving like and being treated as that person.

- Using odd or bizarre language, such as idiosyncratic meanings for common words or made-up words (neologisms) that only have meaning to them. They may also go off on tangents when they speak.
- Disturbances in affect – flat affect can result in a lack of facial expressions and emotionless, monotone speech, while inappropriate affect is characterized by laughing when nothing funny has happened, crying when nothing sad has happened or getting angry when nothing upsetting has happened.
- Behavioural disturbances in four important areas:
 1. odd mannerisms, such as repetitive movements or behaviours and odd facial expressions (e.g. grimaces);
 2. a significant lack of motivation, called avolition;
 3. difficulty taking care of themselves, including basic life skills such as paying bills, shopping, and dressing; and
 4. poor social skills, being socially withdrawn, and having disturbed relationships with others (the nature of interpersonal relationships plays a key role in the course of the disorder).

positive symptoms in schizophrenia, symptoms that indicate the presence of something unusual, such as hallucinations, delusions, odd speech and inappropriate affect

negative symptoms in schizophrenia, symptoms that indicate the absence of something normal, such as good social skills, appropriate affect, motivation and life skills

The symptoms of schizophrenia are grouped into two categories: positive and negative symptoms. *Positive symptoms* indicate the presence of something unusual (such as hallucinations, delusions, odd speech and inappropriate affect) and *negative symptoms* indicate the absence of something normal (such as good social skills, appropriate affect, motivation and life skills).

The course of schizophrenia

Schizophrenia is a chronic disorder. Although some people have brief episodes of schizophrenic-like behaviour (called brief reactive psychoses), most people with schizophrenia suffer from symptoms for their entire lives.

One common course of schizophrenia is a period of negative symptoms and odd behaviour during which the person's functioning slowly deteriorates (the prodromal phase), followed by a 'first break' – the first episode of positive symptoms. Some people experience an episode of positive symptoms with a few warning signs beforehand. The manifestation of symptoms can also take a number of different forms. For example, some people may be delusional but still be able to take basic care of themselves, carry on a conversation and succeed in school and work, whereas others may be completely debilitated by the disorder.

Schizophrenia typically has its onset in late adolescence or early adulthood. Although it can start in childhood, this is quite rare. Sufferers don't necessarily deteriorate over time, but they do have relapses into episodes of positive symptoms.

Causes of schizophrenia and factors affecting its course

1 Genetic and biological factors These account for our initial vulnerability to schizophrenia, although exactly how they do so is unclear.

What is clear is that schizophrenia tends to be inherited. For example, monozygotic twins have the highest *concordance rates* for schizophrenia (Gottesman, 1991), meaning that they are more likely to both have schizophrenia if one of them has it, compared to people who share less genetic material (such as dizygotic twins or siblings). Interestingly, schizophrenics

concordance rates the extent to which people show the same disorders

Pioneer

Emil Kraepelin (1856–1926), a German psychiatrist and one of the founding fathers of modern psychiatry, made three primary contributions to the field of mental illness. First, Kraepelin believed that mental illness was caused by biological factors. His work in this area helped define the field of biological psychiatry and research now supports a strong biological basis for some of the disorders in which Kraepelin was most interested (e.g. schizophrenia and bipolar disorder). Second, Kraepelin laid the foundation for modern classification systems used to diagnose mental disorders, which use patterns of symptoms rather than any one symptom in isolation. This led to his third contribution, which was the classification of and distinction between schizophrenia and bipolar disorder.

and their close family members show some similar types of neuropsychological functioning, even if these family members do not suffer from the illness itself. This suggests that it may be the biological risk factors for schizophrenia that are inherited from family members, rather than the disorder itself (Cannon et al., 1994).

Adoption studies also support the notion of genetic transmission of vulnerability to schizophrenia. Children born to a schizophrenic parent and adopted away to a non-schizophrenic parent are more likely to have schizophrenia, compared to children born to a non-schizophrenic parent (Kety et al., 1994). However, one study has shown that adopted-away children with a genetic predisposition to schizophrenia are more likely to become schizophrenic if they are also raised in a disturbed family environment. This supports a diathesis–stress model of schizophrenia (Tienari et al., 1987).

Several biological problems may cause schizophrenia, as various forms of brain dysfunction occur among sufferers, including enlarged ventricles, reduced blood flow to frontal brain regions and an excess of dopamine (Andreasen et al., 1992; Davis et al., 1991; Flaum et al., 1995). But we don't know for sure whether these problems are a cause or a consequence of the disorder.

A current debate focuses on the neurodegenerative hypothesis (that schizophrenia leads to progressively deteriorating brain functioning) versus the neurodevelopmental hypothesis (that brain deficits are present at birth, influence the onset of schizophrenia and remain fairly stable). Research supports the neurodevelopmental hypothesis, but there is also evidence that the brains of some schizophrenic people show greater deterioration over time than would otherwise be expected.

2 Psychosocial factors An early hypothesis of the 'schizophrenogenic mother' suggested that inadequate parenting causes schizophrenia (e.g. Arieti, 1955). There is absolutely no evidence of this, nor that any *psychosocial factors* cause schizophrenia. The evidence overwhelmingly points to

psychosocial factors psychological, environmental and social factors that may play a role in psychopathology

genetic and biological factors as providing the initial vulnerability. But there is evidence, consistent with a diathesis–stress model, that psychological and social factors influence the course of schizophrenia, such as the timing of onset and the likelihood of relapse.

expressed emotion (EE) specific set of feelings and behaviours directed at people with schizophrenia by their family members

The most well researched psychosocial predictor of the course of schizophrenia is a phenomenon called *expressed emotion (EE)*. This doesn't refer to the level of emotion the sufferer expresses, as the name implies, but to a specific set of feelings and behaviours directed at people with schizophrenia by their family members. A family that is high in EE tends to be critical and resentful of their schizophrenic relative and may be overprotective or over-involved in his life (Brown et al., 1962). A family that is low in EE tends to be more caring and accepting, and less enmeshed in the sufferer's life.

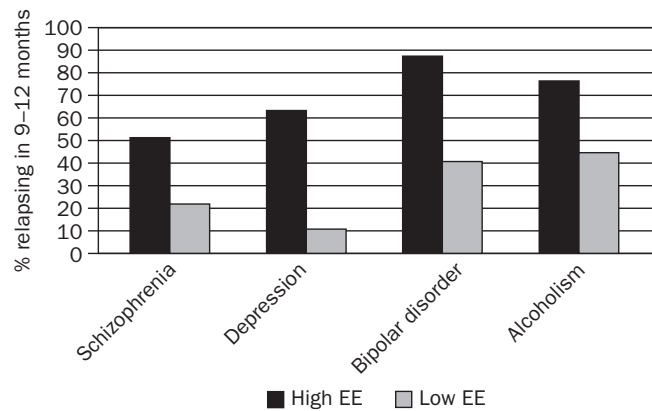


Figure 15.6

Expressed emotion and relapse. Across many disorders, people who have family members who are high on EE are more likely to relapse than are people who have family members who are low on EE. Source: Adapted from Butzlaff & Hooley (1998); O'Farrell et al. (1998).

The level of EE in the family plays an important role in what happens to the schizophrenic person. Schizophrenic people who have families that are high in EE are more likely to suffer a relapse of symptoms (figure 15.6). This association between EE and relapse is also true for a number of other disorders.

Caring for a schizophrenic family member is a stressful, tiring undertaking, which can, in itself, lead to high EE behaviours. Fortunately, psycho-educational programmes can help families and their schizophrenic relatives learn better ways of communicating. These programmes, in conjunction with appropriate medication for the sufferer, can lower relapse rates (e.g. Leff et al., 1982; McFarlane et al., 1995).

MOOD DISORDERS – DEPRESSION

Although mood disorders have some symptoms in common, they are very different in terms of their prevalence and causes.

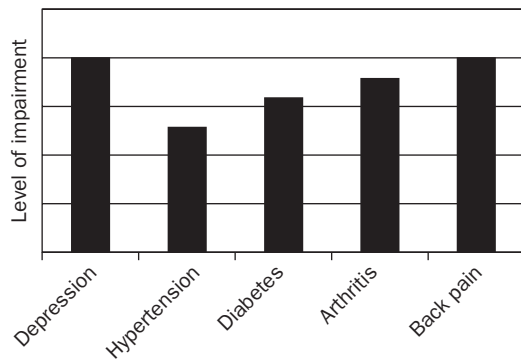
Major depressive disorder, also called unipolar depression, is one of the most common of these disorders, whereas bipolar disorder (also known as manic-depression), like schizophrenia, is less prevalent.

Both disorders often result in severe impairment. Figure 15.7 shows that depression results in as much impairment as common physical health problems, if not more.

Symptoms of major depressive disorder

The primary symptom of major depressive disorder is, not surprisingly, a depressed or sad mood. Almost everyone experiences a sad mood some time in their life, but major depressive disorder goes much further than simply feeling sad. Other symptoms include:

- Losing interest or pleasure in things that you usually enjoy – an experience called anhedonia.

**Figure 15.7**

Level of impairment caused by depression in comparison to physical disorders. Notice that depression is as impairing or more impairing than many common health problems. The impairment scale should be interpreted with regard to the relative impairment across disorders, rather than absolute impairment of each disorder. Source: Adapted from Üstün and Sartorius (1995).

- Changes in appetite – some find nothing appealing and have to force themselves to eat, resulting in significant weight loss, while others want to eat more and gain a lot of weight.
- Changes in sleep habits – depressed people may be unable to sleep or want to sleep all the time.
- A very low level of energy, extreme fatigue and poor concentration. Depressed people have no motivation to do anything, often find themselves unable to get out of bed and unable to complete school or work assignments. They may move through their lives very slowly, feeling that even simple activities require too much energy.
- Feeling very badly about themselves – low self-esteem, feeling worthless and blaming themselves for all that has gone wrong in their lives and the world. Depressed people tend to feel hopeless about the future and don't believe they will ever feel better.

Major depressive disorder has negative consequences not only for how people feel about themselves and their future, but also for their relationships. During a depressive episode people tend to withdraw socially, feel insecure in relationships, elicit rejection from others and experience high levels of interpersonal conflict and stress. Romantic, family and peer relationships all suffer.

Given their level of suffering, impairment and hopelessness, it is hardly surprising that depression is one of the biggest risk factors for suicide, with around 15 per cent of depressed people committing suicide (Clark & Goebel-Fabbri, 1999).

The course of the disorder

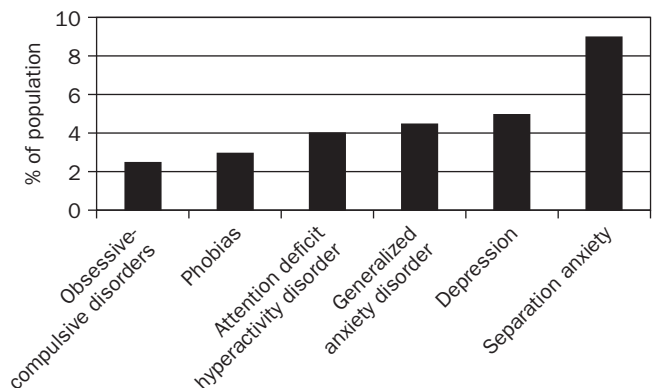
Major depressive disorder follows a recurrent course. Although some people have isolated episodes, most experience multiple episodes of depression that may become more severe over time (e.g. Lewinsohn, Zeiss & Duncan, 1989). Mild forms of depres-

**Figure 15.8**

A depressed person will tend to withdraw socially, feel insecure, elicit rejection from others and experience high levels of interpersonal conflict and stress.

sion with just a few symptoms rather than full-blown major depressive disorder can predict the onset of more serious depression later on (e.g. Pine et al., 1999).

Although depression was once thought to be a disorder of adulthood, we now know that it affects people of all ages, including children (figure 15.9). In fact, the age of onset of major

**Figure 15.9**

Children experience psychological disorders too. These rates are from studies in the US. Cross-national studies indicate that rates may differ somewhat in different countries because of differing cultural values that result in different definitions of abnormality and different symptom expression. However, children in all cultures suffer from various psychological disorders.

depressive disorder is decreasing, and the rates of major depressive disorder in childhood and adolescence are increasing rapidly. Early onset predicts a worse course of depression over time (e.g. Lewinsohn et al., 1994), so depression in childhood and adolescence is a serious problem that can lead to ongoing difficulties throughout life.

Causes of major depressive disorder and factors affecting its course

1 Genetic and biological factors Like schizophrenia, major depressive disorder can be genetically transmitted (e.g. McGuffin et al., 1996).

As for biological factors, the current view is that no single neurotransmitter is associated with major depressive disorder. Instead, it most likely involves dysregulation of the entire neurotransmitter system (Siever & Davis, 1991). Indeed, it may be the balance of various neurotransmitters that regulate mood.

Major depressive disorder may also involve neuroendocrine dysfunction. Depressed people tend to have elevated cortisol levels (e.g. Halbreich, Asnis & Shindledecker, 1985). Cortisol is involved in regulating the body's reaction to stress and becomes elevated under stress. This suggests that, physiologically, depressed people may be in a state of chronic stress and they are perhaps more reactive to stress than are non-depressed people (e.g. Gold, Goodwin & Chrousos, 1988). As we see in the next section, stress plays an important role in vulnerability to major depressive disorder.

2 Psychosocial factors Unlike schizophrenia, which almost certainly has a genetic and/or biological trigger, major depressive disorder can be caused by either genetic/biological or psychosocial factors.

One of the primary psychosocial factors is life stress, including significant negative life events and chronically stressful circumstances (e.g. Brown & Harris, 1989). Of course, many people experience stressful situations, but they don't all become depressed, suggesting that a diathesis-stress process might be occurring. Specifically, it may be the particular way we perceive and think about life stressors that leads to depression. Consistent with a cognitive model of psychopathology, people who think about life events in a pessimistic, dysfunctional way are more likely to get depressed than people who think about life events in an optimistic way (e.g. Metalsky, Halberstadt & Abramson, 1987).

cognitive distortions dysfunctional ways of thinking about the self, the world, other people and the future that can make people vulnerable to depression and other negative emotions

ing our positive qualities. When people engage in cognitive distortions, like those below, to explain their life circumstances, they put themselves at risk for experiencing negative moods like depression:

Beck (1967; Beck et al., 1979) describes pessimistic ways of thinking about ourselves, the world and the future as *cognitive distortions*. Examples are viewing things in a black and white manner, focusing on and exaggerating negative aspects and minimiz-

- All or nothing thinking – 'I'm a total loser!'
- Overgeneralization – 'I'm always going to be a total loser!'
- Catastrophizing – 'I'm so bad at my job that I'm sure to fail, then I'll get fired, I'll be totally humiliated, nobody will ever hire me again, and I'll be depressed forever!'
- Personalization – 'It's all my fault that my sister's boyfriend broke up with her – if I hadn't been so needy of her attention, she would have spent more time with him and they would have stayed together!'
- Emotional reasoning – 'I feel like an incompetent fool, therefore I must be one!'

Similarly, Seligman and colleagues (e.g. Abramson, Seligman & Teasdale, 1978) suggest that people who are vulnerable to depression tend to offer internal, global and stable causal explanations for negative events (see also chapter 14). For example, if a date goes badly, reactions might include:

- It's all because of me (internal);
- I always do the wrong thing (global); and
- I'll never have a proper boyfriend (stable).

Negative interpersonal circumstances are particularly likely to play a role in depression. Marital, family and peer relations are often troubled, and interpersonal forms of stress – such as relationships ending, conflicts and lack of supportive relationships – are consistently associated with depression (see Beach & Fincham, 1998; Davila, 2000; Hammen, 1991).

Interpersonal models of depression highlight how the disorder can be both a cause and a consequence of interpersonal problems. For example, Coyne (1976) suggested that depressed people engage in behaviours that elicit rejection from others, and this rejection leads to further depression. Similarly, Hammen (1991) proposed that depressed people generate interpersonal stress in their lives, which then makes them more depressed. It's possible that, for some people, depression has its roots in childhood experiences (Cicchetti et al., 1994). An insecure attachment in childhood may set the stage for depression by putting children at a disadvantage in four important areas:

1. Low self-esteem puts them at risk for a pessimistic way of viewing themselves and the world.
2. Inability to successfully regulate their negative emotions leaves them unable to fend off feelings of depression.
3. Never having learned to cope well with stress, they may employ inappropriate strategies (such as keeping problems hidden or ruminating on them).
4. Negative views of others and learned dysfunctional ways of interacting with others (e.g. excessive dependence on, or complete avoidance, of others) puts them at risk for depression through the negative effect it has on their interpersonal relationships.

Symptoms of bipolar disorder

In bipolar disorder, depression alternates with periods of mania, which is virtually the polar opposite of depression. During a

Research close-up 2

Identifying neural correlates of vulnerability to psychopathology

The research issue

Affective neuroscience is a rapidly growing field in psychology that has the potential to help us understand how brain functioning is related to risk for emotional disorders. By using brain imaging techniques (e.g. Positron Emission Tomography, PET; Functional Magnetic Resonance Imaging, fMRI) researchers may be able to identify areas of the brain that are associated with different emotions and motivational states.

Liotti and his colleagues (2002) attempted to do just that in order to determine whether there are any disease markers in brain functioning for unipolar depression. Specifically, they were interested not just in whether depression was associated with certain aspects of brain functioning, but whether there are brain changes that may play a causal role in the onset of unipolar depression.

Design and procedure

In order to test their question, Liotti et al. compared three groups of participants:

- remitted depressed individuals – people who were diagnosed with major depressive disorder, but who were no longer symptomatic;
- currently depressed individuals – people diagnosed with major depressive disorder; and
- healthy comparisons – people with no personal or family history of depression or any other psychiatric disorder.

Each participant underwent PET scans during (1) a resting state and (2) a sad-mood induction in which he or she thought about personal events that induced sadness. Then the researchers compared changes in brain functioning from the resting state to the sad state in the three groups.

Results and implications

Comparisons of brain functioning changes across the three groups resulted in two important findings:

1. Of most importance, the brain functioning of the currently depressed was very similar to that of the remitted groups, but they were both different from the healthy comparisons. Therefore, when remitted depressed people (who are not currently depressed) are made to feel sad, their brains function more like those of people who are currently depressed than like those of people who are not currently depressed. So these common aspects of brain functioning may be markers of risk for unipolar depression. Of course, this research design does not tell us for sure whether the observed changes are a marker of vulnerability to depression or a reflection of brain changes that follow from having the disorder.
2. Some aspects of changes in brain functioning were similar across all three groups. This suggests that some types of brain functioning may reflect processes that are common to mood regulation among all people.

In conclusion, the results of this study provide intriguing evidence that there may be neural markers of risk for psychopathology. You can be certain that this will be an exciting area of research for future generations.

Liotti, M., Mayberg, H.S., McGinnis, S., Brannan, S.L., & Jerabek, P., 2002, 'Unmasking disease-specific cerebral blood flow abnormalities: Mood challenge in patients with remitted unipolar depression', *American Journal of Psychiatry*, 159, 1830–40.

manic period, people feel euphoric or elated. And just as major depression isn't the same as simply feeling sad, mania is not the same as simply feeling happy. Mania is characterized by these symptoms:

- An excessively euphoric mood typically associated with a sense of grandiosity. Manic people feel unbelievably good

about themselves, to the extent that they often believe they can do anything. And they frequently try to!

- Engaging in many more activities than usual. This increase in activity often becomes excessive to the point of being dangerous. For example, manic people may go on shopping sprees, spend amounts of money that go well beyond their means, and incur enormous levels of debt. They may take

off on a trip in their car, driving recklessly and leaving responsibilities behind. They may engage in frequent sexual indiscretions, putting themselves at risk for sexually transmitted diseases, pregnancy and relationship conflict.

- A decreased need for sleep – even staying awake for days at a time.
- High distractibility and poor concentration as the mind races with a million thoughts.
- Speaking very quickly – others can barely get a word in during conversations.

The course of the disorder

The most common onset for bipolar disorder is in early adulthood, but, like major depressive disorder, it can occur earlier. Bipolar disorder is a lifelong, recurrent disorder that can take a variable course. Although some people regularly alternate between mania and depression, the number of episodes, their timing and their order can vary widely.

Bipolar disorder can be seriously debilitating, but with appropriate medication many sufferers live highly productive, normal lives between episodes.

Causes of bipolar disorder and factors affecting its course

1 Genetic and biological factors There is even more evidence of genetic transmission for bipolar disorder than for major depressive disorder (Gershon, 1995).

There is also evidence of dysfunction of various neurotransmitters, including serotonin, dopamine and norepinephrine, although it may not be the levels of neurotransmitters themselves that are problematic, but the pattern of neuronal firing. Sodium ions are critical in proper neuronal firing (see chapter 1), and lithium, which is used to treat bipolar disorder, is chemically similar to sodium, so lithium may work by regulating dysfunctional neuronal firing (e.g. Goodwin & Jamison, 1990).

Pioneer

Kay Redfield Jamison (1946–), Professor of Psychiatry at Johns Hopkins University School of Medicine, is an award-winning psychologist and expert in the field of bipolar disorder, a condition from which she suffers. With Frederick K. Goodwin, she wrote one of the classic texts on bipolar disorder, *Manic-depressive Illness*. Her autobiography, *An Unquiet Mind*, has made a lasting impact because of the candid and caring manner in which she describes life with bipolar disorder. She has also produced three television programmes about bipolar disorder. Jamison has served on the first National Advisory Council for Human Genome Research, and is clinical director for the Dana Consortium on the Genetic Basis of Manic-Depressive Illness.

2 Psychosocial factors Like schizophrenia, there is no evidence that psychosocial factors are the initial cause of bipolar disorder. But they do influence the course of the disorder. Stressful life events, particularly those that disrupt social and biological regularities (e.g. birth of a child, change in work hours, travel), may lead to relapse (see Johnson & Roberts, 1995).

Negative social relations may also lead to relapse. In particular, sufferers with high EE families are more likely to relapse (Miklowitz et al., 1988; see figure 15.6).

ANXIETY DISORDERS – WHEN FEAR TAKES OVER

Anxiety is a set of symptoms:

- emotional (e.g. fear, worry)
- physical (e.g. shortness of breath, heart pounding, sweating, upset stomach)
- cognitive (e.g. fear of dying, losing control, going crazy).

When someone experiences this cluster of symptoms, it is often called a panic attack.

Like depressed mood, anxiety is a common experience – almost everyone has felt some level of anxiety in their lives. In many circumstances, it is a normal adaptive experience, physiologically preparing our bodies to respond when we sense danger. Our autonomic nervous system (see chapter 3) gets us ready for fight or flight and then, when the danger has passed, calms us back down again so that we can go back to normal functioning.

So how do we differentiate ‘normal’ fear from an anxiety disorder? In addition to the level of impairment caused by the anxiety, a disorder often involves fear and anxiety in response to something that is not inherently frightening or dangerous. For example, it is normal to feel anxiety in response to poisonous snakes, but it is less normal to feel anxiety in response to pictures of snakes.

Anxiety disorders have four things in common:

1. each is defined by a specific target of fear (the thing the person is afraid of);
2. anxiety or panic attacks are experienced in response to the target of fear;
3. the target of fear is avoided by the sufferer; and
4. anxiety disorders tend to be chronic – they tend to persist rather than come in episodes.

Symptoms and course of anxiety disorders

1 Specific phobias The most common and straightforward of the anxiety disorders are specific phobias – fear and avoidance of a particular object or situation (e.g. dogs, heights, flying). This anxiety may be very circumscribed, occurring only in response to the target of fear, and may result in impairment in only a very specific domain.

For example, someone who is afraid of flying may lead a very normal, productive life but simply isn’t able to fly. This may impair their work if they are expected to travel for business, or

Pioneer

David Barlow (1942–), Professor of Psychology and Director of the Center for Anxiety and Related Disorders at Boston University, is an expert on the nature and treatment of anxiety and related disorders (e.g. sexual dysfunction). In his classic text, *Anxiety and its Disorders*, he describes the predominant cognitive-behavioural approach to understanding anxiety disorders. He has developed a series of empirically supported cognitive-behavioural treatments for various anxiety disorders and is particularly well known for his panic control treatment.



Figure 15.10

A specific phobia will involve fear and avoidance of a particular object or situation.

their relationships if, for example, they can't take a vacation with their partner. But it won't usually affect other areas of their life.

2 Social phobia Social phobia tends to be more impairing because it often results in significant social isolation. You might think that people with social phobia are afraid of people or of social situations – but this isn't the case. They are actually afraid

of negative evaluation and rejection by others and will attempt to avoid it at all costs.

Social phobia ranges from relatively mild (e.g. fearing and avoiding public speaking only) to extremely pervasive (e.g. fearing and avoiding all social interaction except with family members).

3 Panic disorder Panic disorder can also be quite debilitating, especially when it is coupled with *agoraphobia*.

Literally 'fear of the marketplace', agoraphobia is often thought of as fear of leaving the house. More accurately, it is fear of situations in which escape would be difficult or there would be no one to help should panic occur.

Panic disorder begins with sudden panic attacks that occur out of the blue. The disorder develops when people worry about having another panic attack and subsequently begin to avoid places and situations they associate with it. For example, if you had a panic attack while driving, you might avoid driving again.

When someone avoids so many places and situations that they are finally unable to leave their home, they are said to have agoraphobia.

4 Obsessive-compulsive disorder You won't be surprised to find that obsessive-compulsive disorder (OCD) is characterized by *obsessions* (unwanted, persistent, intrusive, repetitive thoughts) and *compulsions* (ritualistic, repetitive behaviours).

When someone with OCD experiences obsessions, such as fear of contamination, anxiety is generated. To reduce this anxiety, she might engage in compulsions, such as repetitive hand-washing. The compulsions reduce anxiety briefly, but the obsessions soon return, and the sufferer becomes caught in a vicious cycle.

Sometimes OCD is fairly circumscribed, but often it begins to dominate people's lives, causing significant impairment. Typical obsessions involve religion, contamination, fear of hurting someone, fear of losing something important, and fear of saying or doing something inappropriate or dangerous. Typical compulsions are hand-washing, checking, counting and hoarding.

5 Post-traumatic stress disorder Experiencing a traumatic event can lead to post-traumatic stress disorder (PTSD). It was first documented among war veterans who had been exposed to wartime atrocities, but we now know that it can occur in response to many types of event, including natural disasters, accidents, rape and physical abuse. And it isn't just the victim who is vulnerable to the disorder. Someone who observes severe physical abuse, for example, is also at risk.

PTSD has a paradoxical set of symptoms. The target of fear is the trauma itself, which creates tremendous anxiety, so the sufferer will desperately try to avoid anything associated with the trauma. They may even lose their memory for the event. On the

agoraphobia fear of situations in which escape would be difficult or help is not available should panic or anxiety occur

obsessions unwanted, persistent, intrusive, repetitive thoughts

compulsions ritualistic, repetitive behaviours that a person feels compelled to engage in

other hand, people with PTSD might be plagued with unwanted and intrusive thoughts about the event, such as flashbacks and nightmares.

Sufferers also tend to become psychologically numb. Their emotions shut down, and they can't derive pleasure from things or even envision the future. But again, paradoxically, they may also experience symptoms of hyper-arousal. They are usually hyper-vigilant to their environment, they startle easily, can't sleep or concentrate, and are irritable and easily angered. This complex set of symptoms makes PTSD a very debilitating disorder.

6 Generalized anxiety disorder In some ways, generalized anxiety disorder is the simplest, and in other ways the most complex, anxiety disorder. It is characterized by an extended period – say, six months or more – of chronic, uncontrollable worry about numerous things. This sounds simple. Sufferers spend their lives worried and tense all the time, they are easily irritated, and they have trouble sleeping and concentrating.

On the other hand, it isn't entirely clear what people are attempting to avoid and what function their worry serves. Some theorists have suggested that people with this disorder fear that they will not be able to control their lives or themselves, and worry is a way to exert control (Borkovec, 1985). It doesn't work, of course, but sufferers may feel completely out of control if they stop worrying. They may have no other coping strategies to rely on.

Causes of anxiety disorders and factors affecting their course

1 Genetic and biological factors The extent of heritability varies across disorders. For example, panic disorder shows relatively high rates of heritability, whereas generalized anxiety disorder shows lower rates (e.g. Hettema et al., 2001; Kendler et al., 1992). There is also evidence that people who are prone to

behavioural inhibition shyness, quietness, fearfulness, social avoidance, and high levels of physiological arousal and stress reactivity in young children

anxiety disorders are born with something called *behavioural inhibition* (see Kagan & Snidman, 1991). Children who are behaviourally inhibited are shy, quiet, fearful, socially avoidant and have

high levels of physiological arousal (i.e. they are aroused easily and are very reactive to stimulation and stress). These children are more likely to develop an anxiety disorder (e.g. Hirschfeld et al., 1992).

Regarding biological factors, there a number of pathways in the limbic system that are hypothesized to produce various types of anxiety reactions (e.g. Gray, 1982). In addition, people with anxiety disorders show low levels of the neurotransmitter gamma aminobutyric acid (GABA). This is a central nervous system inhibitor that works to lower physiological arousal and keep us calm and relaxed. Low levels of GABA can therefore lead to increased neuronal firing, which may in turn lead to high levels of physiological arousal and, consequently, anxiety.

Although some forms of biological dysfunction may be associated with anxiety in general, each anxiety disorder may also have

unique biological causes. For example, some research indicates there is a specific brain circuit that, when over-activated (e.g. in times of stress), results in repetitive patterns characteristic of OCD (e.g. Rapoport, 1989).

Research also suggests that vulnerability to panic disorder may be the result of a biological sensitivity to physical sensations (e.g., Klein, 1993). Our bodies may have an 'alarm system' that is hypersensitive to certain sensations (e.g. lack of oxygen). When the alarm sounds, we may experience a panic attack. This is an interesting model, but it doesn't indicate how exactly this process leads to panic disorder (i.e. how fear and avoidance of panic attacks develop).

2 Psychosocial factors Cognitive, behavioural and life stress factors all affect risk for anxiety disorders. In fact, stress is, by definition, the cause of PTSD.

When fear is generated by life experiences, be they actual experiences, things we see or even things we are told about, this can serve as a powerful conditioning experience. But, like depression, the way we view a frightening event affects whether it results in an anxiety disorder. Anxiety is associated with viewing the world as dangerous and uncontrollable and viewing the self as helpless (e.g. Beck & Emery, 1985).

The development of panic disorder is a good example of how various causal factors may interact. Imagine you are biologically sensitive to physiological changes in your body. Suppose one day you suddenly feel short of breath for no identifiable reason. You assume the shortness of breath means something terrible is about to happen ('I'm going to die!', 'I'm going to lose control of myself!'), and so you experience more anxiety, likely resulting in a full-blown panic attack. Because this frightening event is made even more so by your catastrophic interpretation, you develop a fear of the panic attack (Clark, 1986; see also figure 15.11). If the panic attack occurs while you are driving, you might also develop a fear of driving and begin to avoid it. This avoidance is reinforced, because it reduces the likelihood of further panic attacks.

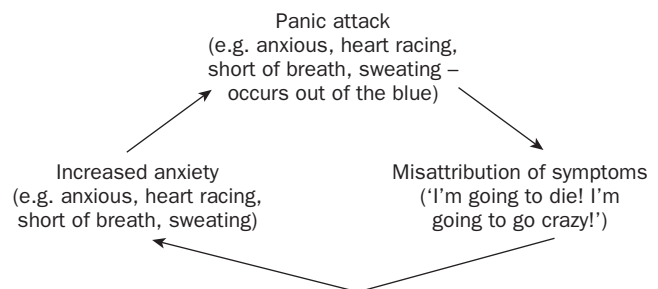


Figure 15.11

Cognitive model of panic disorder. According to this model, people develop panic disorder after misinterpreting a set of physical symptoms, such as pounding heart or shortness of breath. The misinterpretation serves to make them more anxious and increase the likelihood of a panic attack. This results in a vicious cycle of physical symptoms, misinterpretation and panic attacks.

EATING DISORDERS – BULIMIA AND ANOREXIA

Eating disorders have attracted a great deal of attention in recent years, particularly in university settings where they tend to be prominent. Yet despite greater public awareness, certain misconceptions still exist. For example, many people think eating disorders are brought about by vanity. This couldn't be further from the truth. Rather than being vain, people with eating disorders struggle with issues about who they are, what they are worth, whether they will be able to take care of themselves and how to negotiate relationships. Eating disorders are complex and difficult to overcome.

There are currently two eating disorders included in the ICD-10 and DSM-IV – bulimia nervosa and anorexia nervosa. Although they differ in important ways, they have four things in common:

1. a distorted body image (inaccurate assessment about shape and weight);
2. an intense fear of being fat;
3. a sense of self that revolves around the individual's body and weight; and
4. eating that is regulated by psychological rather than physiological processes, although the form of eating regulation is quite different for the two disorders.

Symptoms

People with bulimia tend to be of normal weight and are sometimes even overweight. Bulimia nervosa is characterized by recurrent episodes of binge eating and purging. During a binge, bulimic people consume an enormous number of calories in a brief period of time and feel an overwhelming loss of control as they are doing so. The binge is then followed by purging behaviour – usually vomiting, taking laxatives, taking diuretics or using enemas, and sometimes fasting or excessive exercise.

Other symptoms may include:

- somewhat chaotic lives;
- a tendency to be impulsive, emotionally labile, sensitive to rejection and in need of attention;
- depression and/or substance abuse.

Anorexia nervosa is characterized by a refusal to maintain normal body weight. People with anorexia restrict their food intake through diet and typically engage in excessive exercise. Their weight often becomes so low that their bodies stop functioning normally (e.g. females stop menstruating), and they often appear emaciated.

Anorexics also tend to:

- be perfectionist, rule-bound and hard-working;
- have a strong need to please others, but never feel special themselves;
- be high-achievers, but also feel uncertain of their capacity to be independent.



Figure 15.12

People with anorexia nervosa refuse to maintain normal body weight; they restrict their food intake and tend to engage in excessive exercise.

Some people with anorexia also engage in bingeing and purging and have other features of their personalities and lives in common with bulimics.

The course of eating disorders

Both bulimia and anorexia typically begin in adolescence and can become chronic. For example, about one third of people with anorexia will have a lifelong disorder.

Both anorexia and bulimia pose significant health risks. This is particularly true for anorexia, in which almost 5 per cent of people die from malnutrition and other related complications.

Causes of eating disorders and factors affecting their course

1 Genetic and biological factors Research supports genetic transmission, but some suggest that it may not be the disorder itself that is inherited. They believe that a set of personality traits – such as obsessiveness, rigidity, emotional restraint in the case of anorexia and impulsivity and emotional instability in the case of bulimia – might increase the likelihood of poor coping. The eating disorder is seen as a maladaptive way of coping with stress (e.g. Strober, 1995).

Biological models focus on dysfunction in the hypothalamus (the part of the brain related to eating behaviour; see chapter 5)

and on serotonin dysregulation (e.g. Wolfe, Metzger & Jimerson, 1997; see Ferguson & Pigott, 2000). There is presently no evidence that these dysfunctions actually cause eating disorders, but they may affect their course.

2 Psychosocial factors One of the primary sets of psychosocial factors in the development and course of eating disorders are social pressures and cultural forces.

In cultures where thinness is the ideal of beauty, eating disorders are most prevalent. There are expectations of thinness everywhere – in the media, in the family, and in society at large. Adolescents, particularly young women, often internalize these expectations, and their entire sense of self-worth may become dependent on being thin. Furthermore, they are usually socially reinforced for being thin. Think how often you have heard someone say, or have even said yourself, ‘Oh, you’ve lost weight – you look great!’ For vulnerable young people, a seemingly benign comment like this reinforces the belief that they must be thin in order to be worthy of attention.

But if everyone in a culture that values thinness grows up facing the same pressures, why do some develop eating disorders and some not? Apart from possible genetic or biological vulnerabilities, the way people think about themselves and the world may make them vulnerable. Cognitive distortions such as, ‘If I eat one cookie, I will be a fat, horrible person’ or ‘Being thin will

make all the problems in my life go away,’ may increase vulnerability to eating disorders (e.g. Butow, Beumont & Touyz, 1993).

People who come from certain types of families may also be more vulnerable to particular types of eating disorders (Bruch, 1978; Minuchin, Rosman & Baker, 1978). For example, anorexia is thought to develop when families are very concerned about external appearances and prefer to maintain an impression of harmony at the expense of open communication and emotional expression. These families tend to be enmeshed (family members are unaware of or unable to maintain personal boundaries), over-protective, rigid and conflict-avoidant. Anorexia might be a rebellion or an assertion of independence and autonomy, or it may serve to mask the real problems in the family. Other risk factors include families who diet, or parents who are critical of their child’s weight or appearance (e.g. Pike & Rodin, 1991).

A recent perspective, which fits with family and genetic/personality models, suggests that eating disorders are the product of maladaptive emotion regulation processes (e.g. Wiser & Telch, 1999). So food is used to help regulate emotions (typically negative ones) when the person has not developed more adaptive strategies. Attachment theorists take a similar position, suggesting that people with certain forms of insecure attachment (e.g. avoidant) may distract themselves from upsetting, attachment-related concerns (e.g. fear of intimacy, low self-worth) by focusing on food and weight (Cole-Detke & Kobak 1996).

Everyday Psychology

Eating disorders

As discussed in chapter 5, obesity is one of the so-called ‘diseases of affluence’ that beset many contemporary Western societies. But modern developed societies also manifest a range of disorders at the other end of the weight spectrum, known as eating disorders (i.e. anorexia nervosa and bulimia nervosa). These disorders appear to develop as outward signs of inner emotional or psychological distress or problems. For the sufferer, they seem to be a way of coping with difficulties in their life. Eating, or not eating, can be used to block out painful feelings. Without appropriate help and treatment, eating problems may persist throughout the sufferer’s life.

Anyone can develop an eating disorder regardless of age, race, gender or background, but young women seem to be most vulnerable, particularly between the ages of 15 and 25. This may well relate to the changes and challenges occurring in young women’s lives at around this period of personal development.

Biological research suggests that genetic make-up may make someone more or less likely to develop an eating disorder. Within the psychosocial domain, a key person or people (for example, parents or relatives) may adversely influence other family members through their attitudes to food. Or someone might focus on food and eating as a way of coping with the stresses of high academic expectations or other forms of social and/or family pressure.

Traumatic events can also trigger anorexia or bulimia nervosa. These events may be especially prominent during the teenage and young adulthood periods, such as the death of a parent or other close relative, being bullied or abused at school or at home, upheaval in the family environment (such as divorce) or concerns over sexuality.

Eating disorders are complex illnesses with critical psychological elements requiring treatment as well as the physical aspects (such as the disturbed eating pattern and its biological consequences). A regular eating pattern, including a balanced diet, is needed to restore balanced nutrition. And helping someone to come to terms with the fundamental emotional issues underlying their eating disorder will enable them to cope in their future lives with personal difficulties in a way that is not harmful to them.

Striegel-Moore, R.H., Seeley, J.R., & Lewinsohn, P.M., 2003, ‘Psychosocial adjustment in young adulthood of women who experienced an eating disorder during adolescence’, *Journal of the American Academy of Child and Adolescent Psychiatry*, 42, 587–93.

SUBSTANCE USE DISORDERS – ABUSE AND DEPENDENCE

Regardless of what people may experience as positive effects of drugs and alcohol, they both have negative effects on our health and ability to function, especially when used repeatedly. This recurrent use may result in a substance use disorder.

Symptoms

There are two substance use disorders – abuse and dependence. Substance abuse is defined entirely on the criterion of impairment. If someone's repeated use of a substance causes significant impairment in even one area of life, he can be described as a substance abuser. Common impairments include:

- failure to fulfil major role obligations – e.g. constantly late to or absent from work;
- recurrent use in dangerous situations – e.g. while driving;
- frequent substance-related legal problems – e.g. arrests for disorderly conduct; and
- social and interpersonal problems – e.g. conflict with partner or other family members.

Substance dependence is indicated by physical or psychological dependence or addiction. Physical dependence includes:

- tolerance – the need for increased amounts of the substance or diminished effect with same amount; and
- withdrawal – the experience of physical symptoms when the substance is stopped, or turning to another substance to relieve or avoid those symptoms.

Psychological dependence is indicated by:

- taking substances in larger amounts or over longer periods of time than intended;
- a persistent desire to use or unsuccessful efforts to cut down or control use;
- spending a great deal of time trying to obtain, use or recover from the substance;
- giving up important activities; and
- continued use, despite knowledge of a problem that is exacerbated by the substance.

The course of substance use disorders

Substance abuse and dependence can be chronic, progressive, degenerative problems with severe negative outcomes. But the course they take varies, depending on the substance being used. Alcoholism in particular can have tragic outcomes, including health problems, interpersonal problems and early death. People who use substances frequently will often use more than one kind of substance.

Substance disorders can begin at any age and are becoming more prevalent, particularly among adolescents. Although



Figure 15.13

Substance use disorders are becoming more prevalent, particularly among adolescents.

most adult substance abusers began using in adolescence, most adolescents who try drugs don't progress to severe abuse. So experimentation doesn't necessarily lead to lifelong addiction or adverse consequences.

Some people with substance use disorders show remission, especially late in life, but relapse is frequent, particularly in response to high-risk situations, such as negative emotional states, social pressure and interpersonal conflict. Unfortunately, because of the high relapse rates, few people fully recover from substance disorders.

Causes of substance use disorders and factors affecting their course

1 Genetic and biological factors Most research into substance abuse involves alcohol, as alcohol is legal and very widely available. So evidence of genetic transmission comes primarily from alcoholism research. This research supports the role of heredity, particularly among men (e.g. Goodwin, 1979).

Consistent with a biological approach, substance use disorders are considered by some to be diseases (e.g. Jellinek, 1960). Some theorists have suggested that alcoholics may be biologically sensitive to alcohol, which may lead to progressive and irreversible alcoholism (e.g. Pollock, 1992). The body's ability to metabolize alcohol is another possible explanation. The liver produces an enzyme called aldehyde dehydrogenase, which breaks down alcohol in the body. If alcohol isn't broken down, it can build up and lead to illness. In some groups of East Asians, this enzyme is absent or reduced – a possible reason for the relatively lower rates of alcoholism in these groups (e.g. Higuchi et al., 1992).

We know very little about other biological causes of substance abuse, but researchers continue to study the effects of neurotransmitter functioning, brain-wave functioning and biological sensitivities to substances in order to elucidate relevant mechanisms and relationships.

2 Psychosocial factors Numerous psychosocial factors have been implicated in the onset and course of substance problems. Reinforcement certainly plays an important role. Consistent with

tension-reduction hypothesis the notion that people use substances in order to reduce tension and negative affect

the *tension-reduction hypothesis*, continued substance use is reinforced because substances often lead to positive feelings and help people escape negative feelings through use of these substances (Conger,

1956). Substance users are said to engage in 'self-medication', using substances to help relieve tension or temporarily eliminate feelings of anxiety or depression. People also learn to use substances through observation. Those whose families or peers use substances are at high risk for substance use disorders (e.g. Jessor & Jessor, 1975). Learned associations also affect the course of substance use. If someone comes to associate particular people, places or circumstances with substance use, they are more likely to use the substance in similar circumstances (Collins & Marlatt, 1981). That is why people who get treatment for substance problems often relapse when they return to their former environment and social group.

Cognitive factors also play a role in the development and course of substance problems, at least in the case of alcoholism. People who expect positive results from using alcohol (e.g. they think it will make them feel good or improve their social standing) are more likely to use it and to develop alcohol problems (Marlatt, 1987; Smith, 1980).

abstinence violation effect a more severe relapse resulting from a minor violation of substance use abstinence (e.g. one forbidden drink leading to more)

In addition, people who fall prey to the *abstinence violation effect* are more likely to relapse than are others (Marlatt, 1978). This effect occurs when a minor relapse (a violation of abstinence) leads to guilt, which then

leads to a more severe relapse. So if an abstinent alcoholic has one drink, she may feel guilty and decide that, having already failed at abstinence, she may as well drink more. She ends up having a full-blown relapse instead of a momentary one.

The notion of an 'addictive' personality has been suggested. This is a controversial topic. So far, there is no evidence for its existence, but research does indicate that some aspects of personality may contribute to substance problems. A disinhibited personality style that includes impulsivity and antisocial traits may be the best personality predictor of substance problems (e.g. Shedler & Block, 1990). Consistent with the tension-reduction hypothesis discussed earlier, it is also possible that substance problems are masking some other form of psychopathology. But the research on this topic is mixed and suggests that problems such as depression are as likely to follow from substance problems as they are to precede them (e.g. Schuckit, 1994).

There are also broader environmental factors that may contribute to substance abuse, such as the extent to which substance use is condoned by a particular culture (e.g. Westermeyer, 1999; Yeung & Greenwald, 1992). For example, groups whose religious values prohibit or limit the use of alcohol (e.g.

Muslims, Mormons, Orthodox Jews) show relatively low rates of alcoholism.

Perhaps the best way to approach substance use disorders is from a multiple risk factor perspective, which suggests that the more risk factors someone experiences, the more likely he is to develop a problem (e.g. Bry, McKeon & Pandina, 1982). In addition to the risk factors already discussed, many others for substance abuse have been identified, including low socio-economic status, family dysfunction, peer rejection, behaviour problems, academic failure and availability of substances. Of course, because of the nature of the research in this field, some of these factors may be consequences of substance abuse instead of (or as well as) being risk factors.

PERSONALITY DISORDERS – A WAY OF BEING

So far, the disorders we have described have traditionally been considered syndromes, which – like physical illnesses – are not part of people's basic character structure. When treated appropriately, these syndromes usually remit and people return to normal functioning, at least for a while.

But personality disorders are different. They are disorders of people's basic character structure – so there is no 'normal functioning' to return to. The personality disorders themselves are people's 'normal' way of functioning, and appropriate treatment means learning entirely new ways of being.

Symptoms

All personality disorders have a number of things in common. They are:

- longstanding – i.e. begin at a relatively early age;
- chronic – i.e. continue over time; and
- pervasive – i.e. occur across most contexts.

The thoughts, feelings and behaviours that characterize personality disorders are:

- inflexible – i.e. they are applied rigidly and resistant to change; and
- maladaptive – i.e. they don't result in what the person hopes for.

People with personality disorders usually don't realize they have them. They experience themselves as normal and often feel that the people they interact *with* are the ones with the problems. The primary personality disorders and their key traits, as described in the DSM-IV (APA, 1994) are:

Cluster A – the odd and eccentric cluster

Paranoid – suspicious, distrustful, makes hostile attributions
Schizoid – interpersonally and emotionally cut off, unresponsive to others, a 'loner'

Schizotypal – odd thoughts, behaviours, experiences; poor interpersonal functioning

Cluster B – the dramatic and erratic cluster

Histrionic – dramatic, wants attention, emotionally shallow
 Narcissistic – inflated sense of self-importance, entitled, low empathy, hidden vulnerability
 Antisocial – behaviours that disregard laws, norms, rights of others; lacking in empathy
 Borderline – instability in thoughts, feelings, behaviour and sense of self

Cluster C – the fearful and avoidant cluster

Obsessive-compulsive – rigid, controlled, perfectionistic
 Avoidant – fears negative evaluation, rejection and abandonment
 Dependent – submissive, dependent on others for self-esteem, fears abandonment

As you can see, this organization of the personality disorders puts them into clusters. These clusters are thought to reflect disorders with common traits. Although the disorders within each cluster do show commonalities, it is also the case that there are high levels of comorbidity among disorders across clusters.

Borderline personality disorder and antisocial personality disorder (similar to what is often called psychopathy) have received more attention than the others, as they tend to have some of the most negative consequences, including suicide and violence.

Causes of personality disorders and factors affecting their course

1 Genetic and biological factors There is evidence of modest genetic transmission for some personality disorders, especially antisocial personality disorder, although environmental factors also play an important role (e.g. Cadoret et al., 1995).

There is also evidence that children are born with different temperaments, which may serve as vulnerability factors. For example, inhibition – which predisposes children towards shyness and anxiety – may put them at risk for personality disorders characterized by those traits. Disinhibited children are outgoing, talkative, impulsive and have low levels of physiological arousal. These children may be at risk for personality disorders characterized by impulsivity, erratic or aggressive behaviour, or lack of empathy.

Biological factors are also being explored as causes of some personality disorders, such as antisocial personality disorder. For example, research suggests that people with antisocial personality traits show low levels of physiological arousal, which may account for their ability to engage in behaviours that normally cause people to feel anxious (e.g. Raine, Venables & Williams, 1990).

2 Psychosocial factors Cognitive, psychodynamic and attachment theorists all suggest that negative early experiences in the family put people at risk for developing personality disorders. The assumption is that this happens, at least in part, through the cognitions that people develop.

Early experiences with people who fail to validate a child's self-worth may be internalized and result in a deep-seated set of severely rigid and dysfunctional thoughts about the self, others and the world, which then translate into rigid behavioural patterns. For example, if parents are not available to help a child cope with stress but are critical or abusive instead, the child will learn that she can't rely on her parents, even though she may desperately want to. She may learn to hide her feelings, to expect that she will be criticized and rejected by others, and so to avoid close interpersonal relationships, even if she secretly yearns for them. If this pattern continues to develop and becomes rigid as the child grows up, she may eventually develop an avoidant personality disorder.

Research is beginning to suggest that temperamental and psychosocial factors interact. Kochanska (1995) found that children of different temperaments show more adaptive moral development in response to different qualities of the parent-child relationship. For example, fearful children respond better to gentle discipline, whereas non-fearful children respond better when they are securely attached to a parent. This suggests that the closer the parenting style matches the needs associated with that particular child's temperament, the more adaptive their children will become. When a mismatch occurs, children may develop compensatory coping strategies, possibly leading to the rigid patterns that are associated with personality disorders.

Pioneer

Marsha Linehan (1943–), Professor of Psychology and Director of the Behavioral Research and Therapy Clinics at the University of Washington, is best known for her contributions to the understanding and treatment of suicidal behaviour and borderline personality disorder. Linehan proposed that borderline personality disorder can be best understood from a biopsychosocial approach, which bases the disorder in the interaction of an underlying biological dysfunction and an invalidating, non-accepting family environment. Linehan developed dialectical behaviour therapy (DBT) as a treatment for borderline personality disorder and suicidal behaviour. DBT is an empirically supported treatment, which combines traditional Western approaches with Eastern Zen approaches.

FINAL THOUGHTS

In this chapter you read about some of the major psychological disorders – schizophrenia, mood disorders, anxiety disorders, eating disorders, substance use disorders and personality disorders. Although these disorders (and their various subtypes) are among the most prevalent and impairing in the field of psychopathology, they by no means exhaust the number of psychological disorders that have been documented. Our goal has been to help you understand what these disorders look like, how they progress, and what causes them, highlighting potential biological and psychosocial causes.

In addition to getting a sense of what we know about these disorders, it should be clear that there is still a lot that we do not know. That is part of what makes the study of abnormal psychology so intriguing. There is so much left to learn, particularly with regard to how our biology and our environments interact to cause and affect psychological problems. As research on abnormal behaviour progresses, we can expect not only to better understand current disorders and their causes, but also to discover new ones.

Summary

- The field of abnormal psychology, or psychopathology, deals with sets of behaviours, or symptoms, that result in impairment in people's lives. These sets of symptoms constitute psychological disorders or mental illness.
- Although the definition of 'abnormal behaviour' is sensitive to a number of contextual factors, psychological disorders (e.g. schizophrenia) have been documented across time and culture.
- Throughout history, the causes of abnormal behaviour have been construed from a number of different perspectives, each of which tells us something unique about different aspects of psychological disorders.
- Biological/genetic models focus on brain defects, biochemical imbalances and genetic predispositions as causes of psychopathology.
- In contrast, Freudian, contemporary psychodynamic and attachment models focus on the effects of early parent-child experiences.
- Behavioural models focus on the learning experiences that result in psychopathology, whereas cognitive models focus on the effect of distorted thought processes.
- Other perspectives integrate various models. The diathesis-stress perspective suggests that the factors identified by each of the other models may work in accordance with one another, so that psychopathology only results when certain combinations of factors (e.g. genetic, environmental) are present.
- Similarly, the developmental psychopathology perspective provides a framework for understanding how psychopathology develops from childhood to adulthood.
- These perspectives can help us understand the numerous disorders documented in nosologies such as the DSM-IV and the ICD-10.

REVISION QUESTIONS

1. Do mental disorders reflect brain dysfunction and genetic abnormalities? If so, does this mean that biological interventions (e.g. drugs) would necessarily be the treatment of choice?
2. There are several models of abnormal behaviour. Discuss the extent to which they can be integrated. In particular, do psychodynamic models share any common ground with behavioural and cognitive models?
3. Do you think that the origins of mental disorders in adulthood can be found in childhood? What should we look for in childhood as precursors of adult abnormal behaviour?
4. Imagine you had the power to create a human being with a mental disorder. What disorder would you give them so that they were: (a) maximally dysfunctional; (b) minimally dysfunctional; (c) had the best chance of recovering from the disorder? Discuss why you made the choices that you did.
5. A friend hears that you are studying abnormal psychology and comes to ask you about some experiences she or he has had, wanting to know if they are signs of mental disorder. What would you do? What would you tell him or her?
6. Why are some disorders more common than others? What might be some of the cultural, societal, and biological reasons involved?
7. What kinds of things do the different disorders discussed in this chapter have in common?
8. Does it make sense to think about these disorders as exemplifying different categories of disorder? Or are there underlying problems that characterize all of these disorders?

FURTHER READING

Clarkin, J.F., & Lenzenweger, M.F. (1996). *Major Theories of Personality Disorder*. New York: Guilford Press.

A historical overview of many of the most prominent theories of personality disorders.

Hammen, C. (1997). *Depression*. Hove: Psychology Press/Lawrence Erlbaum and Associates.

Research-based information on causes and treatments of depression from various theoretical viewpoints.

Jamison, K.R. (1996). *An Unquiet Mind*. New York: Random House.

The author's personal account of her battle with bipolar disorder.

Leonard, K.E., & Blane, H. (1999). *Psychological Theories of Drinking and Alcoholism*. 2nd edn. New York: Guilford Press.

Recent reviews of all of the major psychological and genetic/biological theories of alcoholism.

Nasar, S. (1988). *A Beautiful Mind*. New York: Simon & Schuster.

Biography of John Forbes Nash, Jr, a mathematical genius whose career was cut short by schizophrenia and who miraculously recovered and was honoured with a Nobel Prize.

Rachman, S. (1998). *Anxiety*. Hove: Psychology Press/Lawrence Erlbaum and Associates.

Psychological approaches to the conceptualization and treatment of anxiety disorders, drawing on recent empirical work.

Zuckerman, M. (1999). *Vulnerability to Psychopathology: A Biosocial Model*. Washington, DC: American Psychological Association.

Describes how psychopathology can be understood from a diathesis–stress perspective, emphasizing interactions between biology/genetics, personality and stressful life events.

Contributing authors:
Joanne Davila & Frank D. Fincham