Your course requires that you study two mental disorders: one being schizophrenia, and the other your choice from a list provided by the board. The latter disorder is covered in the next section, and for this series of notes the chosen disorder is unipolar depression, although other choices are available. This document covers the compulsory disorder, schizophrenia and what you have to know: key features, key symptoms, two explanations and two treatments (both from different approaches to psychology).

Symptoms and features of schizophrenia

Schizophrenia is a chronic mental health condition that causes a range of different psychological symptoms. Common symptoms include:

- **hallucinations** – hearing and seeing things that do not exist
- **delusions** – believing things that are not true

and so schizophrenia is therefore a psychosis, not a neurosis, as the patient does not realise they have a condition.

The exact cause of schizophrenia is unknown as of yet, although there are a number of possible explanations. Two are covered in this unit. The NHS suggests that as many as 1 of every 100 people will suffer at least one episode of acute schizophrenia in their lifetime, although Jablensky (2000) suggested that the disorder is found in about 1.4 to 4.6 people in every one thousand for any nation, on average. Men and women appear equally affected by schizophrenia, although in men it is typically active between the ages of 15 and 30, whereas in women it usually becomes active between 20 and 30.

It is commonly thought that schizophrenics have a split personality, acting perfectly normal one minute, and then irrationally the next. However, this is not the case, and though ‘schizophrenia’ derives from Greek and German words meaning ‘split mind’, it is not a split personality disorder. Sufferers are said to suffer episodes of dysfunction, however.

Most studies confirm that there is some link between schizophrenia and violence, although the media tends to exaggerate this. In reality, a sufferer is actually more likely to be the victim of violence than the initiator of a violent attack. Experts at the RCP estimate that less than 1% of UK violent crime is committed by schizophrenics.

When discussing mental disorders, symptoms are what characterise the disorder, describing ways in which sufferers think, feel and behave. Symptoms of schizophrenia are broken down into positive symptoms and negative symptoms. Positive symptoms are additions or changes to the behaviour of a patient, whereas negative symptoms are removals from the character or personality of the patient:

- **first-rank symptoms** include hearing voices and believing that others are guiding you, for example – these are positive symptoms such as hallucinations and delusions, they are added to the patient’s behaviour as they were not there before the disorder became active
- **second-rank symptoms** include flattened emotions and other negative symptoms, such as lack of energy, sex drive, etc (so these are all removals from the personality) – these often start well before the positive symptoms and any diagnosis, in what is known as the prodromal period

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<tr>
<th>POSITIVE SYMPTOMS</th>
<th>NEGATIVE SYMPTOMS</th>
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<tr>
<td>delusions – firmly held erroneous beliefs due to distortions or exaggerations of reasoning</td>
<td>affective flattening – a reduction in range and intensity of emotional expression, including eye contact, voice tone, body language and facial expression</td>
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<td>delusions of grandeur – one specific type of delusion, where the patient believes they are more powerful than they are</td>
<td>alogia – (lack of speech) lessening of speech fluency</td>
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<td>hallucinations – auditory and visual distortions, the hearing voices can be critical, providing a commentary on what the person is doing, controlling or telling them what to do</td>
<td>avolition – (lack of energy) reduction or difficulty to undertake normal daily tasks (sometimes mistaken for lack of interest – apathy)</td>
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<td>disorganised thinking/speech – or ‘thought disorder’ this is usually noticed through confusing speech and incoherence</td>
<td>anhedonia – (lack of pleasure) inability to experience pleasure from previously-enjoyable activities/experiences, including hobbies, social interaction, exercise and sex</td>
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<td>grossly disorganised behaviour – may include unpredictable agitation, social disinhibition and other bizarre behaviour</td>
<td>social withdrawal – avoidance of interaction with others</td>
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The features of a disorder are aspects about how a disorder develops and consider the disorder on a wider scale, often include facts and statistics. The key features of schizophrenia include:

- schizophrenia appears to be a universal disorder (the condition can affect anyone in any place, males and females)
- the profile of sufferers seems to be as follows:
  - ¼ of patients will have one episode of schizophrenia and then fully recover
  - ¼ of patients will suffer chronic schizophrenia, having continuous schizophrenic episodes
  - ½ of patients will have occasional schizophrenic episodes but do not have a chronic disorder
- positive symptoms (those added to the sufferer’s being) can be overcome (e.g. hallucinations, delusions), whereas the negative symptoms (those lacking in sufferers) tend to remain
- male sufferers tend to experience a more severe course of the disorder than females (Goldstein, 1988)
- there are five categories which psychiatrists and diagnosticians use to describe different types of schizophrenia

Types of schizophrenia

One feature of the disorder is that there are different types. The five types are described below:

- **paranoid schizophrenia** is when the patient is suspicious of others (delusions of being watched or followed are common, and thinking that messages in TV and radio programmes or newspapers are aimed directly at them) and common symptoms are delusions of grandeur and hallucinations
- **disorganised schizophrenia** is when the patient’s speech is hard to follow and behaviour may be bizarre, acting with inappropriate moods for certain situations, although there are no delusions or hallucinations
- **catatonic schizophrenia** is when the patient becomes very withdrawn and isolated and has little physical movement, they appear to be in a trance-like state, with very little speech or activity
- **residual schizophrenia** is when there are low-level positive symptoms but there are psychotic symptoms present
- **undifferentiated schizophrenia** is when the patient does not fit any of the four above types

The dopamine hypothesis: a biological explanation

The dopamine hypothesis is a theory that argues a biological explanation of schizophrenia. It suggests that the unusual behaviour and experiences of schizophrenia can be explained by changes in dopamine function in the brain. Dopamine is one of many chemical neurotransmitters which send messages of neuronal synapses.

The hypothesis suggests that there are an excess number of dopamine receptors at the post-synaptic membrane (receiving end of the dopamine) of neurones in schizophrenic patients.

This is partly based on a number of studies that look at the effects of drugs on dopamine receptors and behaviour. It is also suggested that positive symptoms of schizophrenia might be due to an increase in dopamine in one part of the brain, and negative symptoms due to an increase in another part. Both PET scanning and animal studies have been used to look at excess dopamine receptors.

One suggestion is that development of receptors in one area of the brain might lead to the inhibition of their development in another area, so explaining why there are different numbers of dopamine receptors in different parts of the brain. There also seems to be links between damage to the prefrontal cortex and schizophrenia. This area finishes developing in adolescence which is when the onset of schizophrenia may be observed. Much support for the dopamine hypothesis comes from drugs studies, in particular those involving amphetamines (speed), which are dopamine agonists and so prevent the breakdown of dopamine, leading to high levels. When amphetamines are given in large quantities, they lead to delusions and hallucinations, similar to those in schizophrenic patients, and symptoms get even worse when given to patients of schizophrenia.
The table below shows pieces of evidence for and against the dopamine hypothesis. These also serve as an evaluation of the dopamine hypothesis for schizophrenia.

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<tr>
<th>Evidence for the dopamine hypothesis</th>
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<tr>
<td><strong>Amphetamines give similar symptoms</strong></td>
<td><strong>Amphetamines only produce positive symptoms</strong></td>
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<td>When given amphetamines, which in effect increase the levels of dopamine, delusions and hallucinations are common effects, which suggest that excess dopamine neurotransmitter action produces positive symptoms</td>
<td>The effects of amphetamines are only those similar to positive symptoms of schizophrenia, which suggests that the dopamine hypothesis is not a completed explanation</td>
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<td><strong>Schizophrenics are more sensitive to dopamine uptake</strong></td>
<td><strong>Other neurotransmitters might be involved</strong></td>
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<td>PET scanning has shown that when given amphetamines, schizophrenics show greater release of dopamine than non-schizophrenics, suggesting those with schizophrenia are more sensitive to excess dopamine</td>
<td>Whilst this research shows schizophrenics to be more sensitive to dopamine, other chemicals such as glutamate have been shown to have similar psychotic effects, so perhaps dopamine is not the full explanation</td>
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<td><strong>Biological factors influence dopamine sensitivity</strong></td>
<td><strong>Social and environmental factors are also involved</strong></td>
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<td>Brain differences have an effect, as the brains of those with schizophrenia seem to be different, such as differences in volume of grey matter, and such changes at an early age are linked with sensitivity to dopamine</td>
<td>It cannot be said that the explanation is exclusively biological, as environmental and social factors seem also to be able to trigger schizophrenia – perhaps stressful events trigger excess dopamine production</td>
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<td><strong>Blocking dopamine receptors reduces symptoms</strong></td>
<td><strong>Blocking receptors does not take immediate effect</strong></td>
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<td>When given phenothiazines, drugs which block dopamine receptors, the positive symptoms of schizophrenic patients are alleviated, and since less dopamine is being taken up, this seems to support the hypothesis</td>
<td>However, it is interesting that whilst a patient who takes anti-psychotic drugs has their dopamine receptor blocked immediately, the reduction in positive symptoms is not effective for at least a couple of days</td>
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Much of the evidence for the dopamine hypothesis comes from animal studies and PET scanning, but there is also incidental evidence from cases of those on drug treatment for conditions such as Parkinson’s disease and the use of recreational drugs on behaviour. All of these research methods give the methodology triangulation which improves validity, another strength of the hypothesis. The methodology can also be said to be reliable, as there have been many repeated studies into dopamine levels and its effects.

However, because so many animal studies are used in this evidence, the methodology can be criticised somewhat as the findings of animal research might not always be generalisable to humans. Whilst we are fairly certain schizophrenia is a disorder unique to humans, however, animals share many similarities in terms of nervous systems and biological models, and so the research is most likely appropriate. Another possible weakness of the hypothesis is that it may be something else to do with schizophrenia which causes the difference in levels of dopamine receptors, rather than having too many dopamine receptors results in the development of schizophrenia.

**The environmental breeder hypothesis: a social explanation**

Statistics show that the majority of schizophrenics in the UK come from lower classes, or from groups such as immigrants. Research suggests that social class is either a cause of schizophrenia, or at least somehow involved in its development, according to a second possible, explanation: the environmental breeder hypothesis. This comes from the social approach. In one meta-analysis of 17 studies, Eaton et al. (1988) showed that statistically there are more lower class schizophrenics and the disorder is more common proportionately in lower classes.

Sufferers of a lower class also experience a different course of the illness and are treated differently. It was found that they are more likely to be taken by the police or social services for treatment than those of upper classes, and also more likely to become mandatorily committed or become long-term sufferers. Thus in the 1960s it was thought that being in a lower class was a causal factor of schizophrenia. This was known as the social causation hypothesis, or environmental breeder hypothesis. The ideas of social drift and social adversity offer two social explanations for schizophrenia, which are both outlined on the following page.
Social drift

It has been suggested that sufferers of schizophrenia actually become lower class because of the difficulties that arise from having the disorder. One study took a sample of schizophrenic men and their fathers, and compared their social classes. It was found that the sufferers were all in the lower classes, whereas their fathers typically were not, providing evidence for social selection theory, which suggests just that. The theory argues social class isn’t the causal factor but that schizophrenics drift downwards in terms of social class (this is known as social drift). Whilst social selection theory is now widely accepted by most, recently studies have suggested that there are actually environmental and social factors which can cause schizophrenia (or at least are involved in its development). Whilst social disadvantage may not be the main cause of such a disorder, it certainly appears to be a contributing factor to the development of schizophrenia.

Social adversity

Schizophrenia is certainly more common in urban communities (large towns and cities) rather than rural areas, so it is perhaps something about city life which leads to schizophrenia. In fact, schizophrenia tends to be almost exclusively found in cities, but there are many lower class people in rural places – so the social drift hypothesis doesn’t seem to fit here. Harrison et al. (2001) suggests being brought up in declining inner-city areas could lead to schizophrenia, as this is where the bulk of sufferers lie. Hjem et al. (2004) showed that social adversity (adverse being the opposite of favourable) in childhood has a correlation link with schizophrenia. The areas identified by Harrison et al. as where clusters of schizophrenics live support Hjem’s ideas, as the inner-city areas tend to be where the population groups of the lowest socioeconomic class are. It is therefore suggested that city life must have something which leads to development of schizophrenia. The sociogenic hypothesis (contrasting the social selection theory) suggests that it is stress factors which contribute to the disorder developing. These include the stress from poor education, unemployment, low rewards, low income and few opportunities, which have been suggested can lead to the disorder.

Below is an evaluation of the environmental breeder hypothesis, suggesting social class is linked to schizophrenia:

- The ideas support the facts that there are more schizophrenics in inner-city areas and in lower classes, and both the social drift and social adversity ideas explain a possible link between the disorder and class
- Although not everyone who lives in environmental conditions suggested by these explanations develops schizophrenia, it is still highly likely that there are these environmental triggers (rather than strict causes), it is possible that there is a biological explanation also, which requires some environmental activation for the disorder to develop
- Since those in a lower socioeconomic group, with no jobs and living alone are more likely to be diagnosed, it suggests maybe a diagnosis problem, not an environmental problem
- It is hard to separate those factors which might be causing schizophrenia with those that are being caused by schizophrenia, it may be that lower social class, economic status and the lack of a job are all consequences of the disorder, not the other way around, as the social drift hypothesis suggests

Drug therapy: a biological treatment

Whilst there is no cure for schizophrenia, it is considered to be a highly treatable disorder. One treatment comes from the biological approach: drug therapy. Drug treatment, according to the NAMH of the USA, for schizophrenia is comparable to the success rate for drug treatment of heart disease. Drug therapy was an important step forward in the 1950s, allowing the movement of sufferers out of mental institutions by helping them to function normally.

One of the biological explanations for schizophrenia suggests that the symptoms, such as hallucinations and delusions, are due to excess levels of dopamine receptors, leading to increased dopamine activity in the brain. Therefore, it only makes sense that drug treatment uses drugs which act to reduce dopamine action by blocking its receptors. For example, drugs such as phenothiazines can be used (e.g. chlorpromazine) which removes excess dopamine by blocking its receptors. This came about from French neurosurgeon Henri Laborit, who tried using anti-histamines to relax his patients before surgery, and when he saw the calming effects chlorpromazine had on his patients, he thought they might be useful in calming schizophrenic patients too. The results was spectacularly successful, and later it was discovered that the reason for this was that chlorpromazine blocked dopamine receptors.
Drugs which are used to treat schizophrenia are called antipsychotic drugs. They work to suppress hallucinations and delusions, by correcting these chemical imbalances in the brain. Antipsychotic drugs are separated into two categories: typical drugs (which are older, and already well-established – these have been in use for a long time) and atypical drugs (which are much more modern and in less use, although they are more experimental they do have fewer side effects, but act differently to typical antipsychotics).

Common side effects of antipsychotic drugs include sleepiness and tiredness, shaking and muscle spasms, low blood pressure, problems with sex drive and weight gain. Other symptoms include dry mouth, constipation and blurred vision. A particularly nasty side effect, Tardive Dyskinesia affects older patients on the chemotherapy for schizophrenia, which causes involuntary spasms of the face and other areas of the body. These side effects are seen as a weakness.

Meltzer et al. (2004)
In 2004, Meltzer et al. conducted a study into the effectiveness of drug treatment for schizophrenia. He used 481 patients randomly assigned into groups, where some were given a placebo, some given a new investigative drug, and others an already established antipsychotic drug (haloperidol). They found that haloperidol and two of the new investigative drugs were effective at reducing the schizophrenics’ symptoms, more so than the placebo. This was seen as strong evidence that drug treatment works, at least to an extent.

- Drug therapy for schizophrenia is seen as much more ethical, and certainly more effective, than some of the pre-1950s treatments that there were, including lobotomies which would not be allowed nowadays
- The treatment is supported by strong evidence (such as Meltzer et al. 2004) and is based on a scientifically-sound explanation of the disorder
- Drug treatment is a fairly easy and quick treatment, the drugs are easily prescribed and all patients have access to it, and it is also far cheaper than a person-based therapy, such as psychodynamic therapy, and so is the NHS therapy of choice for schizophrenia
- The drugs have many possible side effects which can be a problem, including damaging effects on the central nervous system and synaptic junctions becoming blocked, which are serious issues
- It is often criticised for only masking the problem, as drug therapy is by no means a cure for the illness, it is just alleviating the symptoms, possibly for the doctors’ convenience more than anything else?
- Patients often forget or purposefully stop taking their medication (if the latter, probably because they are worried about the side effects), possibly due to their disorder leading to problems with functioning

Cognitive-behavioural therapy: a cognitive treatment
This is currently the most popular form of psychological therapy for schizophrenia (drug treatment is chemotherapy, not psychotherapy, but drug treatment is the most commonly used), and it comes from the cognitive approach. It provides coping mechanisms, as it does not directly alleviate the symptoms of the disorder themselves. Cognitive-behavioural therapy (CBT) involves accepting the patient’s view of reality, and using that to help them learn to cope.

A full course of cognitive-behavioural therapy lasts over a series of regular sessions, around 50 minutes to an hour in length each, with between five and twenty weeks’ worth of sessions (usually at one per week). Progress will normally be assessed at this stage of twenty weeks to see if further treatment is needed. An agreed agenda is established with the patient and the therapist tries to uncover their core beliefs (this is very much a person-centred therapy). This means not telling the patient their beliefs are ‘wrong’, but suggesting alternative thought patterns. The therapy may help the patient to normalise their experience of the disorder, allowing them to understand that it’s not just them, and that others have hallucinations and delusions too.

The eventual aim is to improve the self-esteem of the patient and to develop their self-concept (getting them to see beyond their disorder, and see their positive features to focus on – for example a particular area they are interested in or talented in, which they can build upon).

Bradshaw (1998)
An example of cognitive-behavioural therapy for schizophrenia in use comes from Bradshaw (1998). The aim was to look at how the therapy was used to treat a woman with schizophrenia (this was a case study), including its effectiveness. Until the time of the patient’s therapy, drug therapy had been the preferred treatment – there was little done of this therapy.
The patient was Carol, who was educated up until her first year of college. She had a perfectly normal life and seemed otherwise healthy, when during her first year at college she had auditory hallucinations and delusions and felt she was “no good”. She began acting in a bizarre manner, and was subsequently hospitalised. There had been no family history of psychiatric illness, and Carol was diagnosed with undifferentiated schizophrenia.

The therapy began and it took about three months for a rapport to be built up. Self-disclosure was used to promote discussion of difficulties and to help build the rapport. Carol controlled a lot of the treatment: how often they met, how long the sessions were and roughly what they spoke about — a common feature of CBT. A further two months were used to get Carol to understand the therapy. The course of the therapy was as follows:

- the first phase lasted a year and focused on managing stress and anxiety
- behavioural activities were worked out during the second phase with further stress control training, with another sixteen months of building cognitive strategies to cope with stressful situations
- finally, an ending phase of around three months where thoughts about the end of the treatment were focused on and plans to maintain the treatment without the therapist’s input were developed (e.g. strategies written on cue cards)

It was shown that there was an improvement of psychosocial functioning after the treatment, as well as improvements in achievement of goals, and a reduction in symptoms and far fewer hospitalisations. Further analyses one year on showed more improvement as Carol’s social functioning improved yet again. There were considerable improvements in functioning in four measures after the three year course of therapy. It was therefore concluded that cognitive-behavioural therapy can be useful in the treatment of schizophrenia.

Below is an evaluation for cognitive-behavioural therapy as a treatment for schizophrenia:

- There is a wide range of evidence to support the therapy for the disorder, such as Pfammater (2006) who did a meta-analysis finding CBT often reduced positive symptoms; Gould et al. (2004) who found a large reduction in symptoms and fewer dropout rates of drug treatment when patients also had CBT; and a study by NICE (2004) who, backed by government funding, studied courses of CBT finding a reduction overall in positive symptoms and negative symptoms, and concluded it should be made available publically for all schizophrenic sufferers
- Turkington et al. (2002) found that CBT was effective in increasing patients’ insights and awareness of the importance of taking medication, but with insights into themselves, their rates of depression increased, suggesting a link between CBT and depression
- CBT seems to be mainly effective when used alongside drug treatment, as it does not directly tackle to symptoms of the disorder, so drug treatment is usually required as well as a psychotherapy such as CBT
- Certain studies have actually shown other person-centred therapies to be more successful than CBT, although the government uses CBT as its therapy of choice for patients, presumably because it is cheaper and has been in use longer, although this may not be the best for patients