**Schizophrenia (PSYCHOTIC)**

Schizophrenia is a disorder which affects thinking, feeling and behaviour. It usually starts between the ages of 15 to 35 and affects about one in every 100 people during their lifetime.

**Symptoms**

Those that represent a change in behaviour or thoughts are called ‘**positive**’ symptoms.

- **Delusions** - believing something completely even though others find your ideas strange and can't work out how you've come to believe them.
- **Difficulty thinking** – finding it hard to concentrate, drifting from one idea to another. Other people may find it hard to understand you.
- **Feeling controlled** – feeling as though your thoughts are vanishing, that they're not your own, or that your body is being taken over and controlled by someone else.
- **Hallucinations** - hearing, smelling, feeling or seeing something that isn't there. Hearing voices is the most common problem. The voices can seem utterly real. Although they may be pleasant, they are more often rude, critical, abusive or annoying.

Those that represent the reduction or total lack of thoughts or functions that you would usually expect to see in a healthy person are called '**negative**' symptoms.

- **Loss of interest, energy and emotions.** You don't bother to get up or go out of the house. You don't get round to routine jobs like washing, tidying, or looking after your clothes. You feel uncomfortable with other people.
- **Some people hear voices** without negative symptoms. Others have delusions but few other problems. If someone has only muddled thinking and negative symptoms, the problem may not be recognised for years.

**Types and Symptoms**

<table>
<thead>
<tr>
<th>Simple</th>
<th>Hebephrenic (Disorganised)</th>
<th>Catatonic</th>
<th>Paranoid (paraphrenic)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Often appears during <strong>late adolescence</strong></td>
<td>Only diagnosed in <strong>adolescents or young adults</strong>, probably conforms most to people’s idea of “crazy”</td>
<td>Range between <strong>hyperactivity</strong> (hyperkinesis) and <strong>stupor</strong> (decrease in responsiveness)</td>
<td>Relatively <strong>stable</strong>, often <strong>paranoid delusions</strong></td>
</tr>
<tr>
<td><strong>Difficulty making friends</strong></td>
<td><strong>Mood</strong> is shallow and <strong>inappropriate</strong></td>
<td><strong>Automatic obedience</strong> (“command autonomism) and <strong>negativity</strong></td>
<td>(especially <strong>auditory</strong>) <strong>hallucinations</strong></td>
</tr>
<tr>
<td><strong>Aimlessness</strong></td>
<td><strong>Thought disorganised</strong></td>
<td><strong>Episodes of violent excitement</strong></td>
<td><strong>Few other symptoms</strong></td>
</tr>
<tr>
<td><strong>Loss of drive</strong></td>
<td><strong>Speech incoherent</strong></td>
<td><strong>Dreamlike state with vivid hallucinations</strong></td>
<td><strong>Most homogenous type</strong> – more alike to one another than other types</td>
</tr>
<tr>
<td><strong>Decline of</strong> occupational or academic <strong>performance</strong></td>
<td><strong>Delusions and hallucinations</strong> brief and fragmented</td>
<td></td>
<td><strong>Undifferentiated (atypical)</strong></td>
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<td></td>
<td><strong>Behaviour is unpredictable, silly, childish</strong> or naughty</td>
<td></td>
<td>Meant to accommodate patients not easily placed elsewhere</td>
</tr>
</tbody>
</table>

**Treatment**

**Drugs work best for treatment, e.g. Antipsychotic drugs**

This helps to weaken any delusions and hallucinations, and helps you to think more clearly and look after yourself better. It can control (but not cure) the symptoms in around four out of five people, and works best when taken regularly, even if you've felt better for some time.

**ECT**
**Causes for SCHIZOPHRENIA**

Psychosis – whole personality is affected; person loses contact with reality

1) **Biological causes**

   a) Genetics (twin and family studies)

   **Twin studies**
   - Most famous twin study by Gottesman 1991: twins were compared, they looked at their concordance rate (e.g., 100 twins have S, they find that 48 of their corresponding wins have S, too) – 48% was found in identical twins (monozygotic MZ) and 17% concordance rate in non-identical twins (dizygotic DZ). HOWEVER, there must be something else than genetics because not all corresponding twins have S! BUT if it was NOT at all due to genetics, the percentage would have to be lower!
   - Children born to 2 parents with S have a 46% chance of developing S.
   - 1% of the population have S – suggests the involvement of genetics

   **Adoption study**
   Heston 1966. He followed up on children of 77 mothers who had S and compared them to 50 children of “normal” mothers. They found that the children with schizophrenic mothers were 5 times more likely to be hospitalised with S.

   **Link with INFECTION**
   - **Viral study Crow 1984**: He proposes that a retro-virus becomes incorporated in DNA and causes S → this explains why S can appear in a family where it has never been there before
   - Recent research has proposed that the *influenza A virus* has the potential to cause S
   - Torry et al. 1988 found a correlation between flu epidemics and the onset of schizophrenia (doesn’t show cause and effect, only relationship)
   - A cluster of genes associated with S was found on chromosome 5 which may make people susceptible
   - There also seems to be a gender link; i.e., recessive gene on X chromosome so men show it more often because they don’t have a dominant X chromosome

   b) **Biochemical causes**

   It’s assumed that S syndromes are caused by an excess of dopamine
   - **Iverson 1979**: found in post-mortems of P with S high levels of dopamine in their limbic system *(this is concerned with emotions)*
   - Post mortems and brain scans show that schizophrenics have more dopamine receptors and they are more active
   - Amphetamines and drugs used to treat Parkinson’s which increase dopamine can cause S-symptoms (e.g., LSD)

   **Problems:**
   - Don’t know whether it’s the cause or effect of S
   - Dopamine levels go right down but symptoms are still there for some more weeks after taking medicine – contradictory!

   c) **Neuroanatomy**

   - **Brown et al. 1986**: Post mortems have shown that people with S have smaller brains. Their ventricles in the brain are bigger, so the brain tissue is smaller and the brain is thus (6%) lighter.
   - **Castner 1908**: Subjected monkeys to brain-damaging x-rays while in the mother’s womb. No defects were shown during childhood but during puberty, they developed S! (CGEM: unethical, can’t be generalised)
2) Psychological causes

a) Psychoanalytic (Freud)

S results from an ego that has difficulty distinguishing between the self and the external world OR A regression to an infertile stage, i.e. narcissism (in oral stage, self-loving): When EGO is overwhelmed by
- Demands of ID (ME ME ME)
- Guilt from Super EGO
The person goes back into pre-ego state (baby state in oral stage, narcissism, self-loving) → S!
Supported by some symptoms of schizophrenics, e.g. childish behaviour
BUT psychoanalytic therapies (sitting on coach...) don't work on schizophrenics!

b) Cognitive

- S is characterised by disturbances in thought, attention and language – the cognitive model uses these as CAUSES rather than effects of S. E.g. bizarre language (“word salad”) is a result of faulty info-processing.
- Faraone 1999: found people with S have impairments in their auditory attention
- It has been discovered that schizophrenics respond to environmental cues inappropriately (respond to sound not meaning of a word)

c) Learning

S is a result of faulty learning: once you’re institutionalised you can watch other’s behaviour and pick it up (social learning, modelling); get attention for behaviour (reinforcement)
(Family-evidence) Bateson 1956: If you have a dysfunctional family, S is a response to impossible demands being made on a child
- Can’t avoid them (e.g. abuse)
- Or can’t satisfy them (too much pressure)

3) Environmental Causes

- Stress aggravates S; BUT Rabkin 1980 found that schizophrenics don’t report more stressful episodes before S-onset.
- Social Class: Evidence for more schizophrenics in lower social classes; people with S are disadvantaged educationally (less intelligent), of lower working class; BUT don’t know whether it’s cause or effect (social drift hypothesis – see before)
- Fromm-Reichman 1948: S is more likely to come from families with these characteristics:
  o High number of secrets
  o High emotional tension
  o Conspireness
- Immigration: per 100 000 people who immigrate to GB, 1 000 Irish men and 290 Afro-Caribbean men will develop S compared to 87 British men (living in GB)
# Depression (unipolar, AFFECTIVE, i.e. MOOD)

Depression is a mental health disorder that can affect the way you eat, sleep, you feel about yourself, you think about things. A depressive disorder is more than a passing mood. It involves body, mood, thoughts.

## Symptoms

<table>
<thead>
<tr>
<th>Type</th>
<th>Emotional</th>
<th>Physical</th>
<th>Social</th>
<th>Cognitive</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>• Feelings of hopelessness and helplessness.</td>
<td>• Slowed movement or speech.</td>
<td>• Feeling anxious or worried.</td>
<td>• Forgetfulness</td>
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<td>• Low self-esteem.</td>
<td>• Change in appetite or weight (usually decreased, but sometimes increased).</td>
<td>• Not performing well at work.</td>
<td>• Finding it difficult to concentrate and think straight</td>
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<td>• Tearfulness.</td>
<td>• Constipation.</td>
<td>• Taking part in fewer social activities, avoiding contact.</td>
<td>• Finding it harder than usual to make decisions</td>
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<td>• Feelings of guilt.</td>
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<td>• Feeling isolated.</td>
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<td>• Feeling irritable and intolerant of others.</td>
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<td>• Reduced hobbies and interests, and difficulties in home and family life.</td>
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<td>• Lack of motivation and enjoyment, less interest, difficulty in making decisions.</td>
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<td>• Suicidal thoughts or thoughts of harming someone else.</td>
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<td>• Feeling anxious or worried.</td>
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<td>• Reduced sex drive.</td>
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<td></td>
<td>• Emotional</td>
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<td>• Lack of energy or lack of interest in sex.</td>
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## Types

**Major Depression**
- Persistent
- Enormous effect on a person’s thoughts, moods and functioning
- People feel sad, hopeless, unhappy about their lives and future
- They may cry a lot and feel numb to the world
- Symptoms are usually intense in that they prevent a person from functioning normally

**Atypical Depression**
- Slightly different from major D
- Sometimes the sufferer is able to experience happiness and moments of elation
- Symptoms include fatigue, oversleeping, overeating, weight gain
- People believe that outside events control their mood (i.e. success, attention, praise)
- Episodes may last for months or forever

**Psychotic Depression**
See and hear imaginary things (sounds, voices, visuals that don’t exist) = Hallucinations (more common in schizophrenics); no “positive” hallucinations like they are with a manic depressive – he imagines frightening and negative things

**Dysthymia**
- People who just seem depressed – sad, blue, melancholic; have been like this all life long
- People are not even aware of their disorder and just live with it
- They feel unimportant, dissatisfied, or simply don’t enjoy their lives

**Manic Depression (Bipolar Disorder)**
- Emotional disorder characterised by changing mood shifts from depression to mania
- High rate of suicide

**Postpartum Depression**
- Can occur to women who have recently given birth; typically occurs in the first few months after delivery
- Symptoms: like major depressive episode
- Often this disorder interferes with the mother’s ability to bond with her newborn

## Treatment
- Anti anxiety drugs, ECT
- With a psychoanalytic approach to some extent
- Behavioural Therapies
- Beck’s Cognitive Restructuring Therapy
Description & Ethologies (causes) of Dysfunctional Behaviours

1) Biological

   a) Genetics

   Twin studies
   Allen 1976: He found that MZ’s concordance rate is 40% and DZ’s 11% for uni-polar depression (just being down). He also looked at Bipolar and found 72% concordance rate in MZ despite it's not 100% which would mean it's totally due to genetics, this shows that there is a genetic link!

   Nurnberger and Gershon: MZ 65%; DZ 14%

Family
   - Wender et al 1986: His research showed that you are 8 times more likely to suffer from D if your biological relatives do (he also looked at adopted children whose parents suffered from D and they didn’t have D; so it’s not the social environment)
   - Egeland 1987 C investigated the Amish in Pennsylvania: Because they are an in-bred community, he found high incidence of depression. He found there’s a gene on chromosome 11 that was present in 100% of the depressed people studied. This is also the gene for noradrenalin (works like serotonin, keeps you up). However; no replication studies to support this! (no depression gene!)

   b) Biochemical

   - D is due to a lack of neurotransmitter (either serotonin or noradrenalin)
   - Wender & Klein 1984 found when animals were given drugs that diminish noradrenalin, they became sluggish and inactive (= symptoms of depression)
   - Kety showed that both neurotransmitters are low in D (blood samples). If serotonin is alone low, it’s mania. So the difference (imbalance) makes you manic – when noradrenalin fluctuates all over the place. The relationship affects the mood!
   - However, not clear whether the level of neurotransmitter is the cause or effect!

2) Psychological

   a) Psychoanalytic – Freud

   - D is repression of early trauma; Freud thinks hostile feelings towards parents can be redirected towards self (self-hatred), that feelings can come from lack of love and support or child abuse
   - Bowy 1950s (after war; controversial nature + biased because the government wanted him to prove that women should stay at home): Found that if people were separated or lost their mother in their early childhood, they became depressive in adulthood
   - Harlow 1950s, Monkey Love experiment: Monkeys were removed from their mothers, and offered a choice between 2 surrogate mothers, one made of cloth providing comfort, one of wire providing food. Even when the wire mother was the source of nourishment, the infant monkey spent a greater amount of time clinging to the cloth surrogate. Since they were unable to bond with the mother, they became depressive when they grew up (exhibited aggression, destroyed sex behaviour...)

   b) Cognitive

   Seligman 1975 proposed a cognitive theory suggesting that people become depressive when they believe that nothing will improve their situation, i.e. when there’s nothing they can do to make it better
   There are 3 different levels, when something goes wrong and you use the left side then those are the cognitive processes, thoughts, that lead to D:
   - e.g. when you’ve failed an exam:
     1) Internal (personal) External (environment) It’s my fault/the teacher’s fault
     2) Stable (always so) Unstable (just this time) I always fail/I just failed this time
     3) Global (everything) Specific (only one situation) I’ll fail all my exams/I won’t fail all my exams

Seligman 1974 found that if dogs are repeatedly subjected to unavoidable punishment – placed in a situation in which they cannot avoid receiving electric shocks – they fail to learn to escape when placed in a situation in which the electric shocks CAN be avoided = Learned Helplessness:
   - The dogs were kept in a pavlovian harness, receiving several shocks paired with a conditions stimulus (CC)
   - Then they were placed into a box where they could avoid shocks by jumping over a barrier (operant conditioning); most of the dogs failed to learn to avoid shock

Beck 1988: Depressives make errors in logic; they falsely magnify/minimise events, overgeneralise, have a negative (cognitive) triad
c) Learning

- Depressives may suffer from lack of positive reinforcement → leads to “sad” behaviour which may be further reinforced by the attention that it draws.
- However, depressives tend to be avoided in the long run → lack of further reinforcement and vicious circle.
- Reductionist! Depression is not about getting negative or positive rewards for behaviour!
- **Maier & Seligman 1976** tested this theory with humans → gave participants an inescapable noise and unsolvable problems and shocks → females showed depressive responses (= learned helplessness) but males got angry (men don’t admit to being depressive and helpless so easily).

Seligman argues that there are similarities between the symptoms of depression in humans and helplessness, for example:

<table>
<thead>
<tr>
<th>Symptom of depression</th>
<th>Corresponding symptom in learned helplessness</th>
</tr>
</thead>
<tbody>
<tr>
<td>decreased appetite leading to weight loss</td>
<td>helpless animals eat less and lose weight</td>
</tr>
<tr>
<td>feeling without energy</td>
<td>lack of response initiation</td>
</tr>
<tr>
<td>feelings of worthlessness and guilt</td>
<td>perception that individual cannot control their environment</td>
</tr>
</tbody>
</table>

3) Environmental

- Reactive: 1 event to cause disorder
- Endodginess: Need to be predisposed and then a trigger

Life-events: unemployment, child birth, low economic status, divorce... are triggers for depression

**Brown & Harris 1978 (Londoner housewives study):** Found that D is more common in women who are in the working class (house-bound with 3+ children) than in women in the middle class (This can be attributed to the working class mother having to leave home to work, having to leave her child alone. This interpersonal relation can cause excessive worry and guilt that the women is not being a good mother as compared to the middle class mom, who can afford to stay at home and take care of the children/her family).

**Seasonal affective disorder:** thought to be caused by variation in daylight hours – get SAD in winter months when days are shorter (lack of melatonin; manufactured from serotonin that is chemically derived from Vitamin D which is obtained from sunlight).

**Cultural Studies**

- **WHO (1983)** identified common elements in four different countries: Iran, Japan, Canada and Switzerland. The symptoms were sad affect, loss of enjoyment, anxiety, tension, lack of energy, loss of interest, inability to concentrate, and ideas of insufficiency, inadequacy and worthlessness, but it did not come up with a clear-cut pattern of universals.
- **Prince 1968.** Earlier reports claimed that there was no depression in Africa and various regions of Asia, but this study found that rates of reported depression rise with Westernisation in the former colonial countries. The negative symptoms of depression (loss of appetite, insomnia, inability to experience sexual pleasure, fatigue) are present but in most non-Western cultures the experience of guilt is mostly absent.
- **Kleinman (1982)** showed that in China somatization served as a typical channel of expression and as a basic component of depressive experience.

**Gender Studies**

- **Williams and Hargreaves (1995)** argue that hormonal changes of the menstrual cycle may have an effect in change of mood even though it cannot be said to directly cause depression.
- **Cochrane (1995)** identifies a number of non-biological explanations of women’s greater susceptibility to depression:
  - Girls have a greater risk to experience sexual abuse
  - Learned helplessness as a result of general female gender role
  - Female-male difference in rate of depression highest between ages of 20-50. These are the years where females have a hard life being mothers, working etc.
  - The Social Creation of Mental Illness (Cochrane, 1983) proposes that depression might be a coping strategy for women.
**Phobias (ANXIETY)**

- Intense and persistent fear of an object/activity/person/thing. When the fear is beyond control and interferes with daily life, it can be diagnosed and dealt with.
- Phobias are the most common type of anxiety disorders (6-10% of population are affected) and a very common mental illness among women.
- It’s a lifetime problem in 10% of cases

**Symptoms**

- Excessive, unreasonable desire to avoid the feared thing
- Shortness of breath or smothering sensation
- Palpitations, pounding heart or accelerated heart rate
- Chest pain or discomfort
- Trembling or shaking
- Feeling of choking
- Sweating
- Nausea or stomach distress
- Feeling unsteady, dizzy, light-headed or faint
- Feelings of unreality or of being detached from yourself
- Hot or cold flashes

**Types**

- **Agoraphobia**
  Fear of open spaces/being in a situation that is difficult to escape from

- **Social Phobia**
  Fear of being in a situation and being scrutinised by others; humiliating or embarrassing

- **Specific phobias**
  Fear of a specific object, e.g. a spider – most common type of phobias but the least disruptive

**Treatment**

- Anti anxiety drugs, Antidepressant drugs
- Psychoanalytic Treatment
- Flooding, Systematic Desensitisation, BIO-Feedback, Observational-learning therapy
Cause for PHOBIA

1) Bio

   a) Genetics

   Twin studies
   - Greater similarities of MZ twins is assumed to indicate a genetic influence
     - Kendler et al 1992, found concordance rate of MZ was 25.9% and of DZ 11%
       (compared to depression and S not impressive) but as CR is more in MZ than DZ, this shows that genes must have
       some effect, genes play a part (as CR is lower than 100, environment influences too)
   - Problems with twin studies
     - Small samples
     - Unreliable zyority tests (can have DZ twins who look really similar – need to check – wasn’t possible in earlier years studies)
     - MZ twins tend to be treated more similar than DZ
     - DZ twins can be M/F, which may be important with sex-differentiated disorders (like simple phobia)

   Seligman 1970 – Biological preparedness
   - He suggested that species are genetically predisposed to learn certain phobias (survival instinct from survival gene)
   - Most phobias in humans are of dangerous animals etc – HOWEVER this doesn’t explain why some phobias are so irrational (e.g. of cotton wool)

   b) Neuroanatomy
   - The anxiety response in phobia involves the same structures and processes responsible for the stress response
   - Some researchers suspect that phobia involves abnormalities in these structures and processes

   NS Reactivity
   - Liebowitz et al 1985 gave social phobics and controls (ppl with no phobia) air with increased CO2-content to stress them; social phobics panicked significantly more often than controls
   - These and many similar results suggest that some types of phobia involve hyper-reactivity of the sympathetic NS

   Amygdala Abnormalities (part of the limbic system, involved in processing of fear and startle stimuli)
   - Pine 1999 compared amygdala responsiveness in social phobias and normal controls; found that social phobics had more responsive amygdala – CAUSE or EFFECT?
   - Suggests that phobias may be more susceptible for fear-evoking stimuli

   Hain et al 2002;
   - Compares phobics with different types of 5HT receptors binding site for a neurochemical;
   - Examined right amygdala responses to angry/fearful faces that p were presented with;
   - Abnormal receptor groups were significantly more responsive

2) Psychological Theories

   a) Psychodynamic
   - Disorders are a manifestation of repressed emotional problems (according to Freud, they stem from childhood)
   - Phobias are symbols for other fears that the conscious mind can’t face
   - Unconscious fears are displaced onto objects (e.g. Hans)
- Limited evidence from clinical case studies
- Methodological issues (bias, incomplete reporting, sampling...)
- Conceptual issues – reliance on interpreting symbols
- Competition from other theories

**b) Cognitive**

Beck 1963 suggested that **catastrophic thoughts** and **irrational beliefs** contribute to the development of phobias. Sufferers focus their intention internally, so they are more aware of their bodily sensations (“oh, my heart beat’s raising”) and they are more likely to misinterpret them as impending doom. This leads to anxiety which leads to a higher hear rate → vicious circle. It has been noticed that people with anxiety disorders are over-vigilant. – they are extremely sensitive to minor danger cues. Their expectation is that things go wrong.

**c) Learning**

- Phobias acquired through learning from the environment (**classical or operant conditioning, social learning**)
- 2 processes
  1) **Acquisition** of phobia
  2) **Maintenance** of phobic behaviour
     (both can involve learning theory)
- 50% of phobic can remember 1 significant experience that set off their phobia

1) **Classical conditioning (direct)**
   - Traumatic experience (e.g. bitten by dog)
   - False alarm (e.g. have a panic attack in a lift)

1) **Social learning (vicarious – not direct)**
   - Observe fear in someone else, e.g. parent
   - Hear about danger from someone else

   If phobias result from this association of traumatic experiences, why aren’t we afraid of cars?
   (you’re desensitised – can’t avoid cars, read in book about dangerous snakes etc)

2) **Operant conditioning (Maintenance)**
   - Approaching phobic object/situation elicits conditioned anxiety response
   - Retreat from phobic object reduces anxiety
   - This acts as a (negative) reinforcer, so the more the person avoid the phobic object/situation, the more likely they will continue to do so
   - Others may unwittingly reinforce avoidance

McGrath (classical conditioning) – case study Lucy (effective treatment for noise phobia)
10-year old girl had phobia of sudden noises (balloon popping, fireworks...). In therapy (systematic desensitisation), she made a hierarchy of noises and was taught breathing and imagery to relax. She had to pretend a fear thermometer and rate her level of fear from 1 – 10. After 4 sessions, she learnt to feel calm. After 10 sessions her fear thermometer was 3/10 from previous 7/10 for balloons popping. By the end of the sessions she was able to pop a balloon herself. The therapy worked because it gave her control over the fear response to say when and how the noises were made... Since the therapy worked, the explanation of phobia must be correct.