OCR Psychology A-level

Issues in Mental Health

Notes

Part 1 — Definitions of Abnormality

- Statistical infrequency = Implies that a disorder is abnormal if its frequency is more than two standard deviations away from the mean incidence rates represented on a normally-distributed bell curve.

+ Statistical infrequency is almost always used in the clinical diagnoses of mental health disorders as a comparison with a baseline or 'normal' value. This is used to assess the severity of the disorder e.g. the idea that Schizophrenia only affects 1% of the general population, but subtypes are even less frequent (such as hebephrenic or paranoid Schizophrenia).

— Statistical infrequency makes the assumption that any abnormal characteristics are automatically negative, whereas this is not always the case. For example, displaying abnormal levels of empathy (and thus qualifying as a Highly Sensitive Person) or having an IQ score above 130 (and thus being a genius) would rarely be looked down upon as negative characteristics which require treatment.

- The failure to function adequately definition of abnormality was proposed by Rosenhan and Seligman (1989) and suggests that if a person's current mental state is preventing them from leading a 'normal' life, alongside the associated normal levels of motivation and obedience to social norms, then such individuals may be considered as abnormal. This occurs when the patient does not obey social and interpersonal rules (e.g. standing precariously close to others), are in distress or are distressing, and their behaviour has become dangerous (not limited to themselves, but may also pose a danger to others).

+ A major strength of this definition of abnormality is that it takes into account the patient's perspective, and so the final diagnosis will be comprised of the patient's (subjective) self-reported symptoms and the psychiatrist's objective opinion. This may lead to more accurate diagnoses of mental health disorders because such diagnoses are not constrained by statistical limits, as is the case with statistical infrequency.

— A major weakness of using this definition of abnormality is the idea that it may lead to the labelling of some patients as 'strange' or 'crazy', which does little to challenge traditional negative stereotypes about mental health disorders. Not everyone with a mental health disorder requires a diagnosis, especially if they have a high quality of life and their illness has little impact upon themselves or others. Instead, such labelling could lead to discrimination or prejudice faced against them by employers and acquaintances.

- The deviation from social norms definition of abnormality suggests that 'abnormal' behaviour is based upon straying away from the social norms specific to a certain culture. There are general norms, applicable to the vast majority of cultures, as well as culture-specific norms. For example, an individual would be diagnosed with antisocial personality disorder (APD) if they behave aggressively towards strangers (breaching a general social norm) and if they experience certain hallucinations (which breaches the social norms of multiple cultures also, whereas other cultures may encourage this as a sign of spirituality).

— The fact that mental health diagnoses based on this definition vary so significantly between different cultures has historically led to discrimination, as a mechanism for social control. For example, in the nineteenth century within Great Britain, 'nymphomania' described the mental health disorder suffered by women who demonstrated sexual attractions towards working-class men. In reality, this diagnosis was simply made to prevent infidelity, cement the differences between social classes and further discriminate against women, thus being a reflection of a patriarchal society.

— Due to its reliance on subjective social norms, this explanation also suffers from cultural relativism. One such example would be the hearing of voices which have no basis in reality, or 'hallucinations'. Some African and Asian cultures in particular would look upon this symptom positively, viewing it as a sign of spirituality and a strong connection with ancestors, as opposed to a symptom of Schizophrenia. This therefore suggests that the use of this definition of abnormality may lead to some discrepancies in the diagnoses of mental health disorders, between cultures.

- Deviation from ideal mental health is the fourth definition of abnormality, and was proposed by Jahoda (1958). Instead of focusing on abnormality, Jahoda looked at what would comprise the ideal mental state of an individual. The criteria include being able to self-actualise (fulfill one's potential, in line with humanism!), having an accurate perception of ourselves, not being distressed, being able to maintain normal levels of motivation to carry out day-to-day tasks and displaying high self-esteem.

— The main issue with this definition of abnormality is that Jahoda may have had an unrealistic expectation of ideal mental health, with the vast majority of people being unable to acquire, let

alone maintain, all of the criteria listed. This means that the majority of the population would be considered abnormal, even if they have missed a single criteria e.g. being able to rationally cope with stress (which most people would agree does not merit a diagnosis). Therefore, deviation from ideal mental health may be considered a very limited method of diagnosing mental health disorders.

— This definition, just like deviation from social norms, suffers from cultural relativism. For example, the concept of self-actualisation, which suggests that we must each put ourselves first in order to achieve our full potential, may be viewed as selfish in collectivist cultures (e.g. China) where the needs of the group are valued more than the needs of the individual. On the other hand, self-actualisation may be a more popular concept in individualist cultures (e.g. the UK), where personal achievement is celebrated and the needs of the individual are greater than the needs of the group. This suggests that deviation from ideal mental health would only be accepted as a definition for abnormality in some (individualist) cultures.

Part 2 — Characteristics of Phobias

- The behavioural characteristics of phobias are panic, avoidance and endurance.
- Panic — the patient suffers from heightened physiological arousal upon exposure to the phobic stimulus, caused by the hypothalamus triggering increased levels of activity in the sympathetic branch of the autonomic nervous system.
- Avoidance — avoidance behaviour is negatively reinforced (in classical conditioning terms) because it is carried out to avoid the unpleasant consequence of exposure to the phobic stimulus. Therefore, avoidance severely impacts the patient's ability to continue with their day to day lives.
- Endurance — this occurs when the patient remains exposed to the phobic stimulus for an extended period of time, but also experiences heightened levels of anxiety during this time.
- The main emotional characteristics of phobias are anxiety (the emotional consequence of the physiological response of panic) and an unawareness that the anxiety experienced towards the phobic stimulus is irrational (from an evolutionary perspective, the phobic anxiety is not proportionate to the threat posed by the stimulus).
- The cognitive characteristics of phobias are selective attention to the phobic stimulus, irrational beliefs and cognitive distortions.
- Selective attention — this means that the patient remains focused on the phobic stimulus, even when it is causing them severe anxiety. This may be the result of irrational beliefs or cognitive distortions.
- Irrational beliefs — this may be the cause of unreasonable responses of anxiety towards the phobic stimulus, due to the patient's incorrect perception as to what the danger posed actually is.
- Cognitive distortions — the patient does not perceive the phobic stimulus accurately. Therefore, it may often appear grossly distorted or irrational e.g. mycophobia (a phobia of mushrooms) and rectaphobia (a phobia of bottoms).

Part 3 — Characteristics of Depression

- The behavioural characteristics of depression include changed activity levels (may result in psychomotor agitation or, on the other end of the spectrum, an inability to wake up and get out of bed in the morning), aggression (towards oneself and towards others, which may be verbal or physical) and changed in patterns of sleeping and eating (insomnia and obesity on one end of the spectrum, whilst constant lethargia and anorexia may appear on the other).
- The emotional characteristics of depression include lowered self-esteem, constant poor mood (lasting for months at a time and high in severity, therefore not simply 'feeling down') and high levels of anger (towards oneself and towards others).

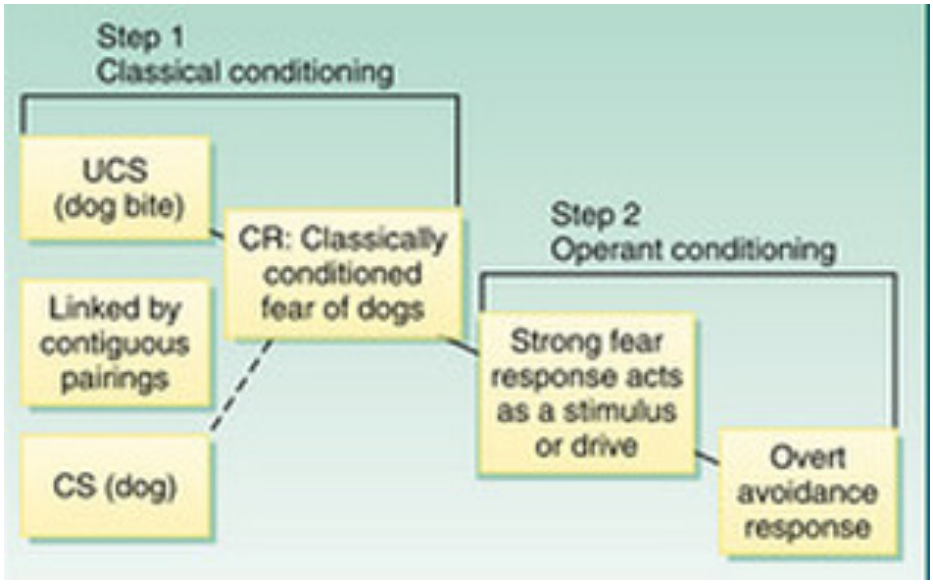
- The cognitive characteristics of depression include absolutist thinking (jumping to irrational conclusions e.g. “I am unable to visit my mother today and so I am a failure of a son”), selective attention towards negative events (patients with depression often recall only negative events in their lives, as opposed to positive) and poor concentration (the consequent disruptions to school and work add to the feelings of worthlessness and anger).

Part 4 — Obsessive-Compulsive Disorder (OCD)

- The main behavioural characteristics of OCD are compulsions (repetitive and intrusive thoughts focused around the stimulus which reduce anxiety through being a method of acting upon obsessive thoughts) and avoidance behaviour. This avoidance behaviour is once again negatively reinforced (in terms of classical conditioning) because an individual who avoids the specific stimulus will avoid the anxiety associated with having to carry out compulsive behaviours and suffer from obsessive thoughts.
- The emotional characteristics of OCD are guilt and disgust, depression (due to the constant compulsion to carry out compulsive/repetitive behaviours, which often interfere with day to day functioning and relationships) and anxiety (associated with the acknowledgement that the obsessive thoughts are irrational, but despair at the fact that they will always lead to compulsive behaviours).
- The cognitive characteristics of OCD include the patient’s acknowledgement that their anxiety is excessive and irrational (a hallmark of OCD), the development of cognitive strategies to deal with obsessions (such as always carrying multiple bottles of hand sanitiser) and obsessive thoughts (these are repetitive, focus on the stimulus, are intrusive, cause excessive amounts of anxiety and lead to compulsive behaviours).

Part 5 — The Behavioural Approach to Explaining Phobias

Mowrer suggested that phobias are acquired through classical conditioning and then maintained through operant conditioning. Watson and Rayner demonstrated how Little Albert associated the fear caused by a loud bang with a white rat. He was exposed to a white rat (NS), producing no response. When paired with the loud bang (UCS), this produced the UCR of fear. Through several repetitions, Albert made the association between the rat (CS) and fear (CR). This conditioning then generalised to other objects e.g. white fluffy Santa Claus hats. Operant conditioning takes place when a behaviour is rewarded or punished. For example, phobics practice avoidance behaviours, meaning that they avoid the phobic stimulus. By avoiding this phobic stimulus, they avoid the associated fear. By avoiding such an unpleasant consequence, the avoidance behaviour is negatively reinforced and likely to be repeated again, hence maintaining the phobia.



+ - Good explanatory power - The main advantage of this theory is that it can explain the mechanism behind the acquisition and maintenance of phobias, which classical or operant conditioning alone cannot do. This translates to practical benefits in systematic desensitisation and flooding. Mowrer emphasises the importance of exposing the patient to the phobic stimulus because this prevents the negative reinforcement of avoidance behaviour. The patient realises that the phobic stimulus is harmless and that their responses are irrational/disproportionate, thus translating into a successful therapy.

— = Alternative explanation for avoidance behaviour (Buck) - Buck suggested that safety is a greater motivator for avoidance behaviour, rather than simply avoiding the anxiety associated with the phobic stimulus. For example, he uses the example of social anxiety phobias - such sufferers can venture out into public but only with a trusted friend, despite still being exposed to hundreds of strangers which would usually trigger their anxiety. This means that Mowrer's explanation of phobias may be incomplete and only suited for some.

— = Alternative explanation for the acquisition of phobias - Seligman suggested that we are more likely to develop phobias towards 'prepared' stimuli. These are stimuli which would have posed a threat to our evolutionary ancestors, such as fire or deep water, and so running away from such a stimulus increases the likelihood of survival and reproduction, and so this behaviour has a selective evolutionary advantage. This means that alternative theories can explain why some phobias (i.e. towards prepared stimuli) are much more frequent than other phobias (i.e. towards unprepared stimuli).

Part 6: The Behavioural Approach to Treating Phobias

Systematic desensitisation is a behavioural therapy designed to reduce phobic anxiety through gradual exposure to the phobic stimulus. It relies upon the principle of counterconditioning i.e. learning a new response to the phobic stimulus i.e. one of relaxation rather than panic. This works due to reciprocal inhibition i.e. it's impossible to be both relaxed and anxious at the same time. Firstly, the patient and therapist draw up an anxiety hierarchy together, made up of situations involving the phobic stimulus, ordered from least to most nerve-wrecking. The therapist then teaches the patient relaxation techniques e.g. breathing techniques and meditation, to be used at each of these anxiety levels. The patient works their way up through the hierarchy, only progressing to the next level when they have remained calm in the present level. The phobia is cured when the patient can remain calm at the highest anxiety level.

+ Supporting evidence = Gilroy et al. followed up 42 patients treated in three sessions of systematic desensitisation for a spider phobia. Their progress was compared to a control group of 50 patients who learnt only relaxation techniques. The extent of such phobias was measured using the Spider Questionnaire and through observation. At both 3 and 33 months, the systematic desensitisation group showed a reduction in their symptoms as compared to the control group, and so has been used as evidence supporting the effectiveness of flooding.

+ Systematic desensitisation is suitable for many patients, including those with learning difficulties = Anxiety disorders are often accompanied with learning disabilities meaning that such patients may not be able to make the full cognitive commitment associated with cognitive behavioural therapy, or have the ability to evaluate their own thoughts. Therefore, systematic desensitisation would be a particularly suitable alternative for them.

+ More acceptable to patients, as shown by low refusal and attrition rates. = This idea also has economical implications because it increases the likelihood that the patient will agree to start and continue with the therapy, as opposed to getting 'cold feet' and wasting the time and effort of the therapist!

Flooding is a behavioural therapy designed to reduce phobic anxiety in one session, through immediate exposure to the phobic stimulus. This occurs in a secure environment from which the patient cannot escape - without the option of practising avoidance behaviour, such behaviour is not reinforced and so the phobia is not maintained. Thus, in the case of a spider phobia, the patient will instantly be exposed to a room full of large spiders, which can crawl over them. This relies on the principle that it is physically impossible to maintain a state of heightened anxiety for a prolonged period, meaning tat eventually, the patient will learn that the phobic stimulus is harmless.

+ = Cost-effective - Ougrin compared flooding to cognitive therapies and found it to be cheaper. This is because the patient's phobia will typically be cured in one session, thus freeing them of their symptoms and allowing them to continue living a normal life.

— = Less effective for complex phobias. Social phobias involve both anxiety and a cognitive aspect i.e. thinking unpleasant thoughts about a situation. Thus, in such cases, cognitive therapy may be more appropriate because this therapy can target the distal causes of the phobia, as opposed to the mere proximal (indirect) causes. This suggests that alternatives may be more effective.

Part 7: The Cognitive Approach to Explaining Depression

Becks proposed that one has a cognitive vulnerability towards developing depression, through faulty information processing, negative self-schemas and the cognitive triad of automatic negative thoughts. Through faulty information processing, the patient blows small problems out of proportion, attending to and dwelling on the negative, whilst thinking in 'black and white' terms. Through negative self-schemas, the patient interprets all information about themselves from the world in a negative light, further lowering their self-confidence. Through the cognitive triad, the patient suffers from negative automatic thoughts about the self, the future and the world.

+ = Supporting research evidence - Grazioli and Terry's evaluation of 65 pregnant women for cognitive vulnerability and depression before and after birth. The researchers found a positive correlation between an increased cognitive vulnerability and an increased likelihood of acquiring depression after birth. This supports the link between faulty cognition and depression, which is in line with the predictions made by Beck's cognitive theory, thus increasing the validity of this theory.
+ = An increased understanding of the cognitive basis of depression translates to more effective treatments i.e. elements of the cognitive triad can be easily identified by a therapist and challenged as irrational thoughts on the patient's part. Thus, it translates well into a successful therapy and the consequent effectiveness of CBT (as discussed later on) is merit to the accuracy of Beck's cognitive theory as an explanation for depression.

Ellis proposed that an activating event (A), leads to an irrational belief (B), which results in an emotional consequence (C) in the form of depression. The key here is the specific interpretation of the irrational belief, which is why some people have depression, whilst others don't, according to the ABC model.

— = Ellis' ABC model cannot explain all types of depression, apart from those which clearly have an activating event i.e. reactive depression. However, many suffer from depression without an apparent cause, and may feel frustrated that their concerns/experiences are not reflected in this theory. Therefore, this suggests that the ABC model is limited at best.

+ = The ABC model shares the same advantage as Beck's cognitive theory in that it provides a practical application in CBT. The effectiveness of CBT suggests that identifying and challenging irrational beliefs are at the core of 'curing' depression, which in turn supports the theoretical basis of the ABC model, through a specific focus on the role of faulty cognitions in the development of depression and specifically, in the interpretation of an activating event.

— = Both the ABC model and Beck's cognitive theory of depression share the same disadvantage in that they cannot explain all aspects of depression e.g. hallucinations, anger, Cotard Syndrome. This poses a particularly difficult practical issue in that patients may become frustrated that their symptoms cannot be explained according to this theory and therefore cannot be addressed in therapy.

Part 8: The Cognitive Approach to Treating Depression

CBT aims to identify and challenge irrational thoughts, replacing them with more productive behaviours, and thus treating depression. Beck's CBT aims on identifying the patient's thoughts

and challenging them as irrational. This can be guided by the cognitive triad of automatic negative thoughts (negative thoughts about the self, the future and the world), faulty information processing and negative self-schemas. Cognitive therapy also aims for patients to test the reality of their beliefs. For example, a patient may record each time someone was nice to them for the past week. Next time they say that everyone hates them, the therapist can point towards the journal as counter-evidence, thus proving the patient's beliefs as irrational. This demonstrates the idea of 'patient as scientist'. Ellis's rational emotive behaviour therapy aims to identify the patient's thoughts and challenge them as irrational, leading to a vigorous argument. This may be a logical argument (i.e. the belief doesn't follow on logically from the facts) or an empirical argument (there is no evidence to support the irrational belief). Thus, this aims to change the irrational belief and to break the link between negative life events and depression. Through behavioural activation, patients are encouraged to engage in enjoyable activities, to provide further counter-evidence for their irrational beliefs.

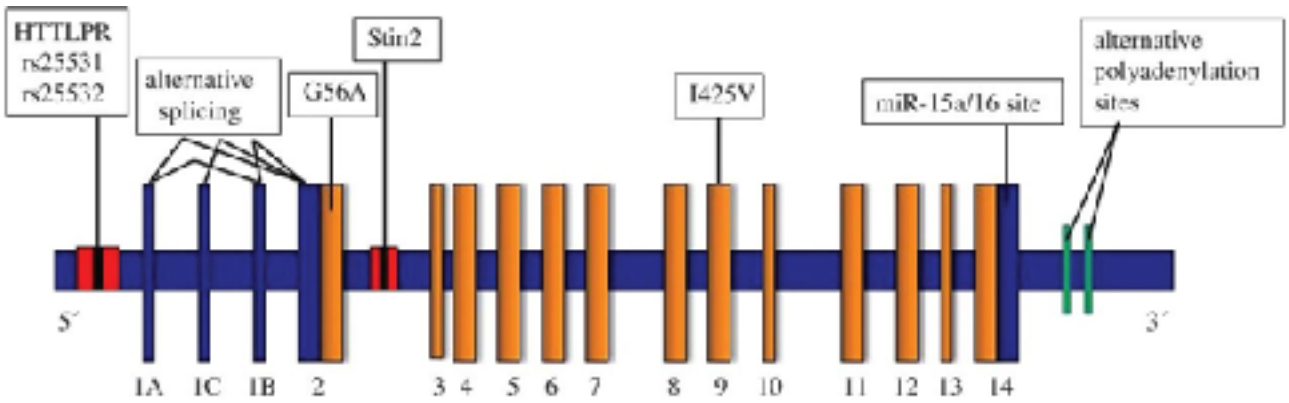
+ = Supporting research evidence - March followed a group of 327 adolescents with a main diagnosis of depression. After 36 weeks - 81%, 81% and 86% were the respective improvement rates for each of the three experimental conditions (CBT, antidepressants, CBT+ antidepressants). Therefore, this is compelling evidence for the idea that antidepressants are effective in treating depression and are based on accurate biological explanations of depression i.e. linked to the role of serotonin and noradrenaline in the development of depression.

— = CBT may not be an appropriate treatment for all cases of depression, and particularly the most severe cases. This idea could also have been reflected in the evidence provided by March et al, where a combination of CBT and antidepressants is the most effective combination. This is because those with severe depression may not be able to attend the regular CBT sessions, due to a lack of motivation/ an inability to get out of bed in the morning, and also may feel completely hopeless i.e. that they are beyond help. This means that CBT cannot be used to address all cases of depression, and arguably is not suitable for cases which need help the most!

— = The focus of the cognitive approach is on present life and the present challenges which life presents. It is then assumed that the patient's current circumstances are responsible for their depression. However, a considerable number of patients may be aware of specific past events which may be responsible for their depression, such as a traumatic life event or the death of a loved one. Therefore, since CBT therapists are unwilling to 'dwell on the past', patients may become frustrated that they have such little input or say into how their therapy is brought about.

Part 9: The Biological Approach to Explaining OCD

The genetic explanation, through the diathesis-stress model, suggests that some have a genetic vulnerability towards developing depression. For example, Lewis et al. found that of his OCD patients, 37% had parents with OCD and 21% had siblings with OCD. OCD is polygenic, meaning that up to 230 different genes are involved in its development (Taylor). These are often associated with the functioning of neurotransmitters, such as dopamine and serotonin, both associated with regulating mood. Researchers have identified candidate genes which increase a person's



vulnerability towards developing OCD. One of these is 5HT1-D beta, which is implicated in the efficiency of serotonin transport across synapses. OCD is also aetiologically heterogenous, meaning that its origin has many different causes. For example, it has been suggested that hoarding disorder is caused by a particular genetic variation.

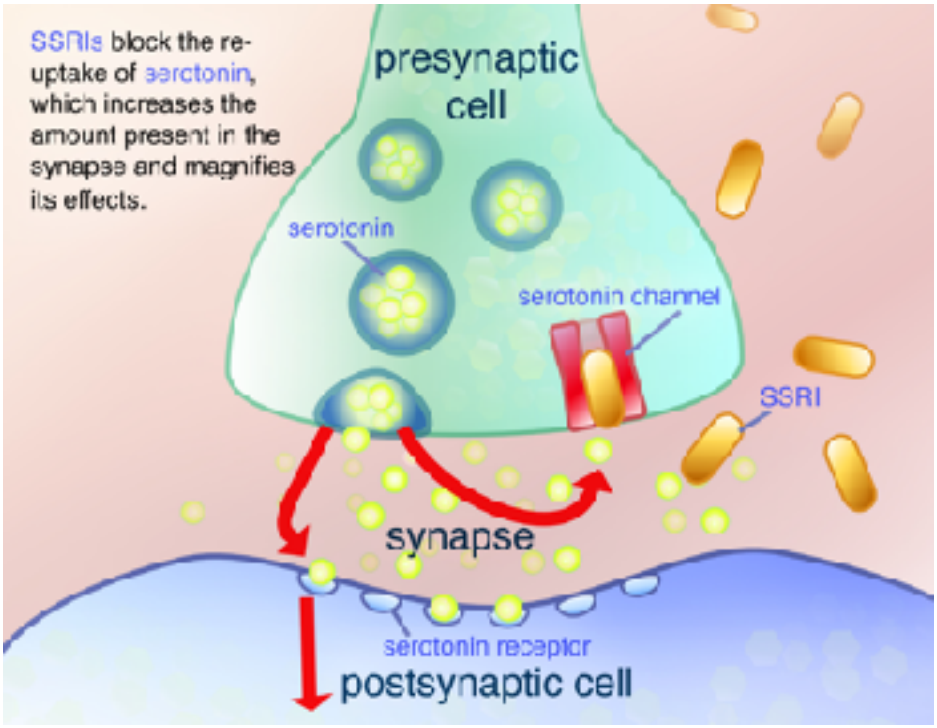
+ = Supporting evidence - Nestadt et al. reviewed previous twin studies of OCD and found that 68% of identical twins, compared to 31% of non-identical twins, share OCD. This strongly suggests that there is a genetic basis for this disease because identical twins share 100% of their genes with each other, whilst dizygotic twins only share 50% of genes with each other. However, it is important not to be deterministic - just because an individual has a particular combination of candidate genes does not mean that the individual is 'doomed' to develop OCD, but rather that this genetic vulnerability must be paired with an environmental stressor to result in OCD, as dictated through the diathesis-stress model.

— = Too many candidate genes = With over 230 candidate genes each individually coding for an increased risk of OCD, then this poses a practical issue in that it is difficult to assess which candidate genes have the greatest influence and so which genes drug treatments should target. Thus, such an explanation is likely to have little predictive value in the future.

— = Ignores environmental factors = Cromer et al. found that of his OCD patients, over half had experienced a trauma in their lives, and that there was a positive correlation between an increasing number of traumas and the increasing severity of the OCD which patients suffered from.

Part 10: The Biological Approach to Treating OCD

- Selective serotonin reuptake inhibitors (SSRIs) act on the serotonin system by preventing the reuptake and breaking down of serotonin by the presynaptic neuron. Thus, the concentration of serotonin within the synapse increases, causing the post-synaptic neuron to be continually stimulated.
- Tricyclics have a similar effect, but are reserved for those who do not respond well to SSRIs. Selective noradrenaline-reputake inhibitors (SNRIs) increase the concentration of the noradrenaline neurotransmitter in the brain.



— A limitation of drug therapy are the serious side effects. For example, for those taking Clomipramine, more than one in 10 suffer from erection problems, weight gain and tremors. More than 1 in 100 suffer from increased heart rate and aggressiveness. These side effects can have serious implications on how the patient can go about their everyday lives.

+ Increased knowledge about the effectiveness of certain drug treatments for OCD and cognitive treatments can reduce the time people take off work through sick days, thus increasing the productivity of the workforce and ensuring that more people are working. This means that more

people will be paying taxes. Research into the cost-effectiveness of treatments for OCD (and other psychiatric disorders) can be the basis of public health services choosing which treatments they use, which can help organisations like the NHS save money.

— A strength of such drug use is that it's cost-effective and non-disruptive. They are cheap compared to psychological treatments, and so prove to be good value for public health organizations like the NHS. They are also non-disruptive. Patients can discretely take the drugs to manage their symptoms and lead a relatively normal life, as compared to life in hospital.

Part 11 — Schizophrenia:

- Schizophrenia is defined as [1]"a psychotic disorder marked by severely impaired thinking, emotions, and behaviours. Schizophrenic patients are typically unable to filter sensory stimuli and may have enhanced perceptions of sounds, colours, and other features of their environment".
- Sufferers may experience either or both positive and negative symptoms.
- Positive symptoms enhance the typical experience of sufferers, and occur in addition to their normal experiences e.g. hallucinations and delusions.
- Negative symptoms take away from the typical experience of sufferers, and so represents a 'loss' of experience e.g. speech poverty and avolition.
- Hallucinations = A positive symptom of schizophrenia, which is characterised by a distorted view/ perception of real stimuli or perceptions of stimuli which have no basis in reality. Auditory hallucinations may involve hallucinating the voices of loved ones or the deceased and are, for example, thought to be caused by an excess of dopamine receptors in Broca's area (a neural correlate).
- Delusions = A positive symptom of schizophrenia and are a set of beliefs with no basis in reality at all e.g. the sufferer may be paranoid that they are being stalked by the Royal Family. Different types of delusions include persecutory, delusions of grandeur, delusional jealousy, erotonomania and somatic delusional disorders.
- Speech poverty = A negative symptom of schizophrenia which occurs when there is an abnormally low level of the frequency and quality of speech. A common type of speech poverty is 'derailment', which is thought to be caused by dysfunctions in central control (Frith et al, 1992) and so the sufferer cannot suppress the automatic associations that come with each new word or idea.
- Avolition = [2]"A subjective reduction in interests, desires and goals and a behavioural reduction of self-initiated and purposeful acts, including motivational deficits". Therefore, avolition means the inability to cope with the normal pressures and motivations associated with everyday living and day-to-day tasks.
- There are two types of classification systems for mental disorders: The Diagnostic and Statistical Manual (currently the DSM-V) and the International Classification of Disease (currently the ICD-10). These two systems have different requirements for the diagnosis of schizophrenia. Despite both requiring persistence of symptoms for at least 1 month, the DSM-V has more specific diagnostic criteria and so requires at least 2 or more of delusions, hallucinations, disorganized speech and catononic behaviour, whereas the ICD-10 takes a broader approach to diagnosis, simply stating that "the clinical picture is dominated by relatively stable, often paranoid delusions, usually accompanied by hallucinations".
- Therefore, the main differences between the DSM and the ICD is in terms of what organisations produces them (the WHO or the American Psychiatric Association), the number of symptoms and specificity of symptoms required for diagnosis, as well as the recognition of different subtypes of schizophrenia.

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[1] Schizophrenia. (n.d.) *Gale Encyclopedia of Medicine*. (2008). Retrieved August 8 2017 from http://medical-dictionary.thefreedictionary.com/schizophrenia

[2] Messinger JW, Trémeau F, Antonius D, et al. Avolition and expressive deficits capture negative symptom phenomenology: Implications for DSM-5 and schizophrenia research. *Clinical psychology review*. 2011;31(1):161-168. doi:10.1016/j.cpr.2010.09.002.

- There are different subtypes of schizophrenia. For example, [3]positive schizophrenia is seen as having the symptoms of prominent delusions, hallucinations and positive formal thought disorders. On the other hand, in mixed schizophrenia, the prominent symptoms are either both negative and positive, or neither is prominent. Subtypes are currently recognised in the ICD-10 only, whereas previous editions of the DSM also made these distinctions.

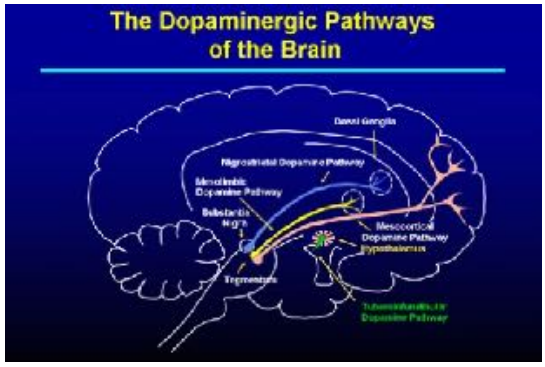
— There is a significant co-morbidity (high frequency of diagnosis of two disorders together) between schizophrenia and other mental health disorders, such as OCD and post-traumatic stress disorder, as suggested by Buckley et al (2009). These researchers found that 29% of their SZ patients suffered from post-traumatic stress disorder, whilst 50% suffered depression. Particularly in the case of depression, this suggests that if schizophrenia is so frequently diagnosed with other psychiatric disorders, then these two disorders may actually be the same, and so a more accurate and valid method of diagnosis would be to combine these two. Therefore, there are issues of validity in the diagnosis of SZ and attempting to differentiate its symptoms from that of other disorders.

— There may be gender bias in the diagnosis of schizophrenia, as suggested by Longenecker et al (2010), who could not find an explanation for the sudden increase in the number of male SZ diagnoses made after 1980s. Cotton et al (2009) suggests that because there are no differences in genetic susceptibility for men and women in terms of SZ, then gender bias must be to blame. Dispositional traits of most women, such as high interpersonal functioning and being able to work even when suffering, means that such traits may mask the symptoms of schizophrenia or distort their severity so that they are not serious enough to call for a diagnosis. This means that the current system of the diagnosis of SZ does not account for these biases or differences in functioning between men and women, increasing the likelihood of inaccurate diagnoses.

— A second type of bias which may reduce the validity of the diagnosis of SZ is the problem of gender bias, as suggested by Escobar et al (2012). For example, African Americans are far more likely to be diagnosed with SZ compared to patients belonging to Western cultures, due to their increased openness about admitting to certain SZ symptoms which may appear normal in their respective cultures. For example, the phenomenon of hearing voices may be considered a desirable sign of increased spirituality and connectedness with ancestors, and so may even be encouraged. However, both classification systems would view this as a hallmark characteristic of SZ and, combined with the potential distrust in African Americans that white psychiatrists may have, could increase the likelihood of false diagnoses.

Part 12 — Biological Explanations For Schizophrenia:

- There is evidence that schizophrenia runs in families, and so appears to have a genetic basis. Gottesman (1991) demonstrated a positive correlation between the increasing genetic similarity of family members and their increased risk of developing schizophrenia. The concordance rates are as follows = Monozygotic twins (48%), dizygotic twins (17%), siblings (9%) and parents (6%). This, particularly due to monozygotic twins sharing 100% of their genes, strongly suggests a genetic basis and the existence of candidate genes for schizophrenia. However, it is important to note that there are no 100% concordance rates, therefore demonstrating that there are environmental influences acting on the development of SZ e.g. the schizophrenogenic mother and dysfunctional thought processing.
- As suggested above, candidate genes have been identified for SZ. For example, Ripke et al (2013), conducted a genome-wide study of 5,001 cases of Swedish



[3] Andreasen NC, Olsen S. Negative v Positive SchizophreniaDefinition and Validation. *Arch Gen Psychiatry.*1982;39(7):789–794. doi:10.1001/archpsyc.1982.04290070025006

nationals with SZ and compared them to 6243 healthy controls. The researchers found [4] "22 loci associated at genome-wide significance; 13 of these are new, and 1 was previously implicated in bipolar disorder", alongside 8300 separate candidate genes. Each candidate gene represents a genetic variation which marginally increases the risk of developing SZ. Therefore, SZ is a polygenic disorder i.e. has multiple, contributing candidate genes.

- The original dopamine hypothesis suggests that hyperdopaminergia (abnormally high dopamine levels) in the subcortex is responsible for SZ, whereas the revised dopamine hypothesis suggests that hypodopaminergeria (abnormally low dopamine levels) in the cortex is more likely to be responsible for SZ. The modern understanding is that both hyper- and hypodopaminergia in different areas of the brain contribute to the development of SZ.
- For example, hyperdopaminergia in the frontal lobe, and specifically Broca's area which may have an excess of D2 receptors, may be responsible for the positive SZ symptom of auditory hallucinations, due to the overactivity of neurotransmission in the auditory areas of the brain.
- In addition, Goldman Rakic et al (2004) suggested that hypodopaminergia in the prefrontal cortex may be responsible for negative symptoms of SZ, such as speech poverty and avolition. This is because the prefrontal cortex is associated with logical thinking, so abnormally low dopamine levels in this area may impair an individual's ability to construct grammatical sentences that are focused upon one topic (speech poverty) or the ability to make decisions about how to function in day to day living (avolition).
- The dopamine hypothesis has particularly important implications for the development of drug treatments for SZ, such as antipsychotics/dopamine antagonists.
- Neural correlates = Specific patterns of cortical activity or neural structures which coincide with specific psychological symptoms, and so are assumed to contribute towards those symptoms.
- Juckel et al (2006) suggested that abnormally low levels of activation in the ventral striatum, when compared to healthy neurotypical controls, may be associated with the negative symptom of avolition. This is because the ventral striatum is associated with [5]evaluating reward values, predictability and risks. Therefore, low levels of activation and neurotransmission may mean that individuals cannot accurately assess the reward of having enough motivation to carry out normal day-to-day tasks, and so are therefore unable to cope with 'normal' life.
- Allen et al (2007) concluded that [6]"the mis-identification of self-generated speech in patients with auditory verbal hallucinations is associated with functional abnormalities in the anterior cingulate and left temporal cortex", as SZ patients' brain activity was recorded using fMRI during auditory hallucinations, and compared to a control group who identified pre-recorded words as their own or not. Therefore, this suggests that speech poverty (a positive symptom) may be associated with this neural correlate, as shown by the SZ group also making more mistakes compared to the control group.

+ There is evidence supporting the biological and genetic basis of schizophrenia. For example, Brown et al (2002) found that the risk of having offspring with SZ increased by over 1.3% if the father was over 50 years old, compared to if the father was under the age of 25. Therefore, this suggests that mutations in the sections of DNA containing the candidate genes, such as those coding for serotonin and dopamine production specifically, means that SZ is likely to have a strong heritability

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[4] Ripke et al (2013), Genome-wide association analysis identifies 13 new risk loci for schizophrenia, *Nature Genetics 45*, pp.1150-1159

[5] Haber SN. Neuroanatomy of Reward: A View from the Ventral Striatum. In: Gottfried JA, editor. *Neurobiology of Sensation and Reward*. Boca Raton (FL): CRC Press/Taylor & Francis; 2011. Chapter 11. Available from: https://www.ncbi.nlm.nih.gov/books/NBK92777/

[6] Neural correlates of the misattribution of speech in schizophrenia
PAUL ALLEN, EDSON AMARO, CYNTHIA H. Y. FU, STEVEN C. R. WILLIAMS, MICHAEL J.BRAMMER, LOUISE C. JOHNS, PHILIP K. McGUIRE *The British Journal of Psychiatry* Feb 2007, 190 (2) 162-169; **DOI:** 10.1192/bjp.bp.106.025700

coefficient and biological basis. This supports the use of family studies and neural correlates as ways of studying and explaining incidence rates of SZ.

— The evidence for the dopamine hypothesis can be best described as 'mixed'. On the one hand, support comes from Tauscher et al (2014) who found that antipsychotics, which act as dopamine antagonists and so reduce dopamine activity by binding to complementary receptors on the post-synaptic membrane, alleviated the symptoms of SZ, suggesting that dopamine has a key role in its development, in line with the predictions of the dopamine hypothesis. On the other hand, some researchers such as Moghaddam and Javitt (2012) have criticised the dopamine hypothesis and biological explanations of SZ as emphasising the role of dopamine too far. For example, the neurotransmitters glutamate and serotonin may also play a key role, as evidenced by the antipsychotic Clozapine acting upon both of these substances and being more effective than other atypical antipsychotics in reducing SZ symptoms, as suggested by Meltzer (2012).

— The main issue associated with the use of neural correlates as a means of explaining schizophrenia is that such evidence is correlational and so does not take into account the 'third variable problem', whereby a third unstudied factor could be affecting both outcomes. Taking the example of the link between lower levels of activation in the superior temporal gyrus and anterior cingulate gyrus, and the experience of auditory hallucinations, one explanation would be the lowered activation levels causing the hallucinations, or the hallucinations themselves causing the lowered activation levels. A third possible explanation would be the third variable problem. Therefore, this demonstrates that correlational research cannot be used to reliably demonstrate a 'cause and effect' relationship between two variables.

Part 13 — Psychological Explanations For Schizophrenia:

- Psychological explanations suggest that the development of schizophrenia is due to abnormal family communication styles created by the schizophrenogenic mother, mixed messages according to double-bind theory, and the stress caused by high levels of expressed emotion. Despite none of these three factors explicitly causing schizophrenia, they are involved in its development and maintenance as contributory factors.
- Fromm-Reichmann suggested that there is a classic, schziophrenogenic mother who is characterised as being cold and rejecting. This means that the family climate is tense and lacking honesty, which leads to the development of paranoia and anxiety. These feelings manifest themselves in the (positive) schizophrenic symptom of paranoid delusions.
- Double-bind theory (Bateson) suggests that within a family, the child receives mixed messages from both parents about what is right or wrong. The tense atmosphere or controlling parenting style means that the child is unable to clarify these messages or voice their opinions about the unfairness of conflicting messages. When the child makes a mistake, as they often do, they are punished through a withdrawal of love. This means that the child sees the world as unfair and confusing due to this confliction, as reflected in the schizophrenic symptoms of disorganised thinking and paranoid delusions.
- Expressed emotion describes the level and type of emotion shown towards the patient by their carer, and is often a significant source of stress for the patient. This means that they are less likely to take their medication or comply to cognitive therapies provided by their hospital or institution, hence being a leading cause for relapse.
  Examples of high levels of negative expressed emotions include verbal criticism of the patient, needless 'sacrifices' for the patient and violence with hostility.
- Frith et al (1992) suggested that dysfunctional thought processes (abnormally-functioning thought processes which lead to unpleasant/ undesirable outcomes), including metarepresentation and central control,

contribute to the development of schizophrenia.

- Metarepresentation is the cognitive ability to differentiate between our own actions and the actions of others, allowing us insight into the intentions and emotions as others, as well as maintaining a realistic/functional view of our own goals and intentions. Dysfunctions in metarepresentation have been associated with auditory hallucinations, and specifically thought insertion, due to the inability to differentiate between our own thoughts and that of others. This may lead to paranoid delusions due to the contents of inserting others' thoughts into the mind of the patient.
- Central control is the cognitive ability to carry out a deliberate action whilst suppressing an automatic response, and is often measured using the Stroop Test. This test involves identifying the colour of each word, where there is often a discrepancy e.g. the word 'brown' written in a yellow font. Therefore, the automatic response of reading the word must be suppressed, to allow for identification of the font colour. People with SZ often have dsyfunctional central control abilities, and so often suffer from derailment because they cannot suppress the automatic associations that each new word in a sentence brings, and so begin to talk off-topic.

+ There is evidence supporting the idea that dysfunctional thought processes are implicated in the development of schizophrenia, and that faulty central control skills may be responsible for some SZ symptoms, as demonstrated by Stirling et al (2006). The researchers found that SZ sufferers made significantly more mistakes and twice as long to complete the task, compared to a healthy neurotypical control group. However, it should be emphasised that dysfunctional thought processing can only offer explanations for the indirect, proximal causes of SZ, and not the distal causes, meaning that such theories can explain the symptoms but not the origin of SZ. This limits the utility of psychological explanations for schizophrenia.

— A comparison can be made between biological and psychological explanations for SZ. A significant weakness of psychological explanations for SZ is that they do not accommodate for biological factors. Since such biological factors can explain the distal origins of schizophrenia (i.e. in terms of dopamine levels in the brain, candidate genes and patterns of activity coinciding with symptoms/neural correlates), this suggests that psychological explanations would best be reserved for the proximal causes of SZ, as these causes are more likely to be most affected by psychological factors.

— A second major weakness would be the lack of support for family-based explanations. The idea of the schizophrenogenic mother was based upon historical observations of families with SZ members, where observers would be searching for 'crazy-making characteristics' (Harrington, 2012) which is hardly an objective and reliable indicator of the likelihood of developing SZ. Psychological explanations also place an increasing amount of blame on the families and caregivers of patients with SZ, as opposed to accommodating for the possibility of a genetic/biological predisposition. For example, caregivers/ parents are further hurt when they are forced to accept responsibility for their patient's schizophrenia, which is likely to have already upset family life and relationships through the development of severe and intrusive negative and positive symptoms. This may explain the sudden popularity of community care in the 1980s, which could have marked parents refusing to take responsibility for their child's condition, seeing as they are so dedicated to their care.

Part 14 — Biological Therapies For Schizophrenia - Drug Therapy:

- There are two types of antipsychotics which are used to treat SZ - typical and atypical.
- Antipsychotics are dopamine antagonists because they bind to complementary dopamine receptors on the postsynaptic membrane, thus preventing dopamine molecules from binding to these sites. The result is an inhibitory effect, where there is a lower rate of action potential generation in the postsynaptic membranes, and so returns neurotransmission (e.g. in the prefrontal cortex and subcortices) to a normal level.
- Typical antipsychotics are described as 'first generation' because these were the drugs historically prescribed to treat SZ patients. The main example of a typical antipsychotic would be Chlorpromazine. It is particularly favoured in psychiatric institutions due to its calming and sedative effects, due to acting upon histamine receptors in addition to dopamine receptors.

- Atypical antipsychotics are described as 'second generation' because they were developed to add to the effectiveness of first generation medications, and also alleviate the serious side effects associated with such drugs.
- Atypical antipsychotics work in the same way as typical antipsychotics, but also target other neurotransmitter receptors on postsynaptic membranes, in line with more modern research. For example, Clozapine targets serotonin and glutamate receptors, whilst Risperidone acts on dopamine and serotonin receptors.
- The key advantage of Clozapine is the improvements in cognitive functioning and mood which patients experience when taking it. This is particularly useful considering that SZ has a 50% comorbidity rate with depression - however, these benefits may be offset by the serious side effect of agranulocyotis, which is a severe and dangerous leukopenia which has caused several deaths in the past.
- On the other hand, the key advantage of Risperidone is that smaller doses are required because it acts more strongly on dopamine receptors compared to Clozapine, and so would be particularly suited to patients who do not suffer from depression but have a previous history of blood-related illnesses.

— The development of antipsychotics was mainly based upon the dopamine hypothesis, and so their use depends on this theory too. For example, if antipsychotics appear to alleviate symptoms by reducing the action of dopamine, this makes sense considering the original dopamine hypothesis i.e. hyperdopaminergia in the subcortex. However, this action is not in line with the revised version of the dopamine hypothesis, which suggests that abnormally low levels of dopamine in the cortex are responsible for symptoms. Therefore, a further reduction in dopamine levels should make symptoms worse, and not better. This paradox has caused some to question the validity of the use of antipsychotics, as well as the accuracy of the dopamine hypothesis as an explanation for schizophrenia.

— A serious consideration which must be made when using antipsychotics is thinking about the side effects. The short-term side effects of typical antipsychotics are relatively mild (e.g. agitation and weight gain), whilst the long-term risk include tardive dyskinesia (characterised by involuntary contraction and relaxation of the facial muscles) and neuroleptic malignant syndrome (NMS). [7]NMS is characterised by fever, altered mental states, muscle rigidity and autonomic dysfunction and is thought to be caused by dopamine receptor blockage or central nervous system infections. These side effects are not offset by atypical antipsychotics, where agranulocyotis remains a serious concern for those taking Clozapine, whose state must be continually monitored using blood tests. Therefore, a cost-benefit analysis should be carried out to consider whether the benefit of symptom reduction outweighs the cost of side effects for each specific patient.

— Despite there being a range of evidence supporting the use of typical (Thornley et al, 2003) and atypical antipsychotics (Meltzer, 2012), these studies still suffer from problems of validity, as suggested by Healy (2012). For example, since Chlorpromazine has particularly powerful sedative effects, then this suggests that studies reviewing the effectiveness of antipsychotics in terms of symptom reduction may actually be measuring how calm and functional the patient appears to be - simply suppressing the symptoms is not a way of controlling them, and so such studies may lack validity due to not accurately assessing the actual effectiveness of antipsychotics in treating the proximal cause of SZ. Secondly, drug companies are selective about what type of information they publish: many focus on the short-term benefits as opposed to the long-term risks, and use inappropriate control groups, such as patients suffering from withdrawal symptoms as they have stopped taking their medication. This distorted focus brings into question, yet again, the validity of research into the effectiveness of antipsychotics.

Part 15 — Psychological Therapies For Schizophrenia:

[7] Berman BD. Neuroleptic Malignant Syndrome: A Review for Neurohospitalists. *The Neurohospitalist*. 2011;1(1):41-47. doi:10.1177/1941875210386491.

- There are 3 main psychological therapies for SZ. These include cognitive behaviour therapy (CBT), family therapy and token economy systems.
- CBT involves an initial assessment of the patient by a therapist, where the patient's symptoms and problems are clarified. CBT emphasises the importance of understanding - although this treatment cannot directly 'cure' SZ, many patients find it comforting to understand the causes of their symptoms, especially if they are suffering from upsetting hallucinations or paranoid delusions. This understanding reassures patients that they are not 'crazy', reducing the intrusive effects of their symptoms and increasing their self-awareness, as proposed by Turkington et al (2004), who found that CBT could be used to challenge a patient's paranoid beliefs about being targeted by the Mafia.
- More effective behaviours are put into place by questioning the reality of the patient's beliefs and considering other, more reasonable alternatives. For example, beliefs about the Mafia can be rationalised as simply being based upon a single day where an individual was staring at the patient for an extended period of time, perhaps lost in thought.
- Family therapy aims to reduce the stress of living together as a family, with a schizophrenic mother. In particular, the levels of expressed emotion are lowered through improving the families' beliefs and attitudes towards schizophrenia, reducing stress, increasing feelings of self-efficacy and being trained to look for signs which may precede a schizophrenic episode. Therefore, the stress upon the SZ patient lessens, reducing the likelihood of relapse because they are more likely to be cooperative with medical advice and diligently take their medication.
- Token economy systems are based upon behaviourist principles and are frequently used in psychiatric institutions. Target, desirable behaviours are identified by the staff. Every time a patient displays one of these behaviours, they are rewarded with a token (which acts as a secondary reinforcer) which can then be exchanged for a reward or privilege (which acts as a primary reinforcer). Therefore, patients are motivated by the primary reinforcer to carry out the desirable behaviours, and their frequency of doing so increases as they are positively reinforced. Rewards may include extra TV time, exercise taken outside of the grounds of the hospitals and favourite magazines.

— None of the three psychological therapies above actually treat the patient and 'cure' their schizophrenia. Instead, these therapies simply improve their quality of life through making the symptoms more manageable. For example, token economies increase the likelihood that the patients act in accordance with hospital rules and breaks disruptive patterns of behaviour, whilst family therapies reduce stress within a schizophrenic family and so increase the likelihood of the patient complying with their medical advice, whereas CBT improves the patient's understanding of their symptoms. This suggests that an interactionist approach towards treatment is best adopted: biological therapies can treat the distal causes of SZ, whilst psychological therapies can treat the proximal symptoms!

— There are serious ethical issues associated with the use of psychological therapies, and specifically concerning token economies. For example, some may argue that the 'privileges' that patients receive upon displaying appropriate behaviours are actually rights. Preventing patients from calling home or exercising outside may increase their stress and so aggravate their condition further. In addition, patients with the most severe SZ may find it near impossible to comply with these rules, and so will bear the most negative consequences. Similarly, CBT raises ethical issues because the therapist essentially has control over the patient's views. This means that by challenging the idea of a Mafia as a controlling government instead, for example, the therapy is infiltrating into the patient's personal beliefs. These changes can be anything, and not always beneficial.
+ There are alternative psychological therapies which may address the issues above, such as the use of art therapy, as suggested by the National Institute for Health and Clinical Excellence. This provides the patients with a creative outlet which reduces stress but does not require changing the patient's beliefs (as is the case with CBT) or discriminate against severely ill patients (as is the case with token economies). Thus, this all suggests that psychological therapies are not appropriate for all patients, but must be selected according to the type and severity of the patient's symptoms.

Part 16 — The Interactionist Approach To Schizophrenia:

- The interactionist approach suggests that we both biological and psychological explanations and therapies should be used in relation to SZ, to reflect both the biological and psychological aspects, as proposed by Turkington et al (2006). Central to the idea of an interactionist approach is the use of the diathesis-stress model.
- The original diathesis-stress model, as proposed by Meehl (1962), would now be considered outdated. He proposed that the diathesis is biological in origin (i.e. a single 'schizogene') which causes a schizotypic personality which in turn eventually manifests itself as schizophrenia. However, this only occurs when the diathesis is accompanied by a purely psychological stressor (excessive exposure to stress, particularly through the schizophrenogenic mother).
- The renewed, modern understanding of stress is that it need not be biological in origin but could also be psychological, such as in the form of childhood trauma, as suggested by Ingram and Luxton (2005). The idea of a single schizogene has also been refuted by Ripke's finding of over 108 candidate genes, whilst early childhood trauma causes dysfunction in the functioning of the HPA system (hypothalamic-pituitary-adrenal system), leading to a greater sensitivity to stressors in the future, and thus increasing the likelihood of developing SZ according to the diathesis-stress model.
- Our understanding of diathesis has changed too, and is not limited to psychological factors but could also be biological in nature, as long as it increases the risk of developing SZ, as according to Houston et al (2008). For example, cannabis use may be considered a lifestyle stress which, when companied with childhood trauma, a biological predisposition or chronic stress, increases the risk of developing SZ by 7-fold.
- If, according to the diathesis-stress model, both psychological and biological explanations apply to SZ, then it also follows that the same approach should be used in SZ treatment. This is particularly the case as biological treatments appear to address the (direct) distal causes of SZ, whilst psychological treatments appear to be more well-suited in treating the (indirect) proximal causes, as suggested by Turkington et al (2006). Such an approach is likely to be reflected in the use of antipsychotic medication with CBT, but less frequently used in the USA where there is still little overlap between biological and psychological approaches towards explaining and treating SZ.

+ The main evidence for the potential effectiveness of adopting an interactionist approach towards explaining SZ comes from Tienari et al's 2004 adoption study. The researchers used data from 19,000 Finnish mothers and adoptees who suffered from SZ and compared these findings to a neurotypical group of children adopted across the same period (1960 to 1979). The researchers found that "in adoptees at high genetic risk of schizophrenia, but not in those at low genetic risk, adoptive-family ratings were a significant predictor of schizophrenia-spectrum disorders in adoptees at long-term follow-up". Therefore, this provides strong support for the diathesis-stress model because the findings demonstrate that a single diathesis or stressor is not enough to trigger the development of SZ, but rather a combination of the two is required.

— The original diathesis-stress model can be considered as an over-simplified explanation of SZ and a reflection of the outdated understanding of that disorder in the mid-twentieth century. For example, Ripke et al (2014) demonstrated that there are over 108 candidate genes, each slightly increasing the risk of SZ, and so there is no single 'schizogene'. Stress can come in many forms apart from the schizophrenogenic mother, such as high levels of expressed emotion, childhood trauma (Read et al, 2001) and the excessive use of cannabis (Houston et al, 2008). Therefore, the diathesis is not exclusively biological, nor is the stressor exclusively psychological. Hence, this may also be considered a strength in the sense that our current understanding of SZ is far more accurate than the original perspective.

+ There is significant evidence supporting the use of a combination of treatments and the interactionist approach to treating SZ, as suggested by Tarrier et al (2004). These researchers studied 315 patients who were randomly allocated to one of three conditions, where the last control group received no treatment and the first two groups received a combination of psychological and biological

treatments. The researchers found that, after an 18 month follow-up, [8]"there were significant advantages for CBT and supportive counseling over TAU (treatment as usual) alone on symptom measures at 18 months but no group difference was seen for relapse or re-hospitalisation. Therefore, adjunctive psychological treatments can have a beneficial long-term effect on symptom reduction". This suggests that there is a therapeutic advantage in adopting an interactionist approach, further supporting the validity of the diathesis-stress model as an explanation for SZ.

[8] Cognitive-behavioural therapy in first-episode and early schizophrenia
Nicholas Tarrier, Shôn Lewis, Gillian Haddock, Richard Bentall, Richard Drake, Peter Kinderman, DavidKingdon, Ronald Siddle, Julie Everitt, Karen Leadley, Andy Benn, Katy Grazebrook, Cliff Haley, ShahidAkhtar, Linda Davies, Steve Palmer, Graham Dunn
The British Journal of Psychiatry Mar 2004, 184 (3) 231-239; **DOI:** 10.1192/bjp.184.3.231