

AQA Psychology A-level

Option 2: Schizophrenia

Notes



Schizophrenia

Part 1 – 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, erotomania 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 catatonic 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.
- There are different subtypes of schizophrenia. For example, ³positive 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

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.

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



Schizophrenia

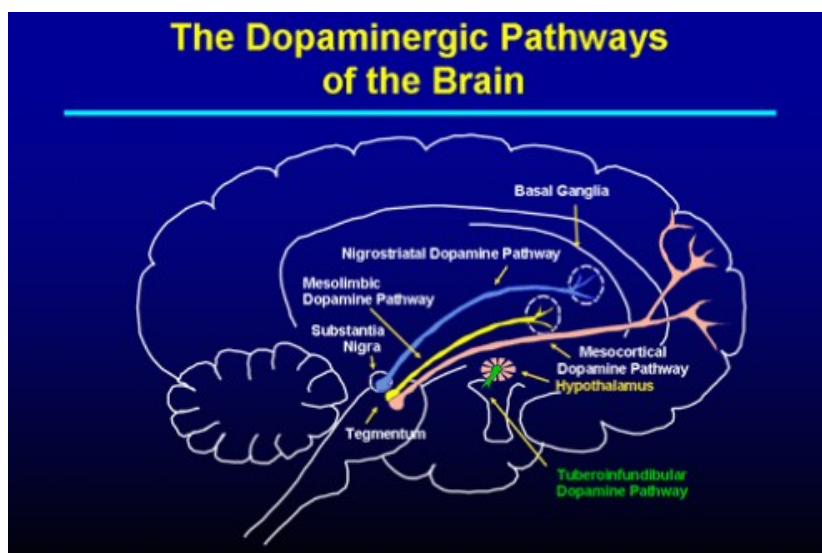
suggested by Buckley et al (2009). These researchers found that 29% of their SZ patients suffered from post-traumatic stress disorder, whilst 50% suffered depression. Particularly in the case of depression, this suggests that if schizophrenia is so frequently diagnosed with other psychiatric disorders, 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 2 – 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 nationals with SZ and compared them to 6243 healthy controls. The researchers found “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



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



Schizophrenia

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 hypodopaminergia (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 ⁵evaluating 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 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-

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



Schizophrenia

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 3 – 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, schizophrenogenic 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

PURPLE YELLOW RED
BLACK RED GREEN
RED YELLOW ORANGE
BLUE PURPLE BLACK
RED GREEN ORANGE



Schizophrenia

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 dysfunctional 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 4 – 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.



Schizophrenia

- 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 agranulocytosis, 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 risks 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 agranulocytosis 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 5 – Psychological Therapies For Schizophrenia:

- 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

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



Schizophrenia

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 6 – 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,



Schizophrenia

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 accompanied 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".

⁸ Cognitive-behavioural therapy in first-episode and early schizophrenia

Nicholas Tarrier, Shôn Lewis, Gillian Haddock, Richard Bentall, Richard Drake, Peter Kinderman, David Kingdon, Ronald Siddle, Julie Everitt, Karen Leadley, Andy Benn, Katy Grazebrook, Cliff Haley, Shahid Akhtar, Linda Davies, Steve Palmer, Graham Dunn

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Schizophrenia

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.

