Edexcel Psychology A-level

Topic 5: Clinical Psychology

Notes

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, 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.
- 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.

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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.

[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

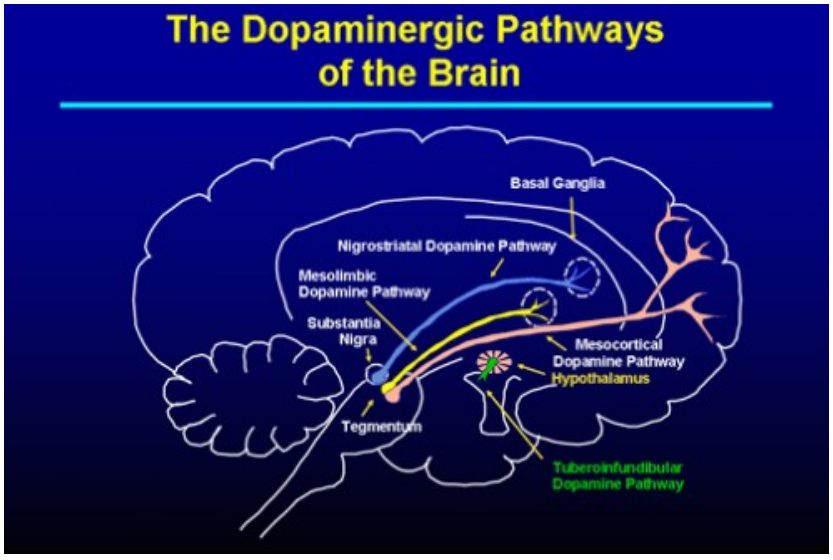
Particularly in the case of depression, this suggests that if schizophrenia is so frequently diagnosed with other psychiatric disorders, then these two disorders may actually be the same, and so a more accurate and valid method of diagnosis would be to combine these two. Therefore, there are issues of 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 [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.



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

- 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 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

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[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/

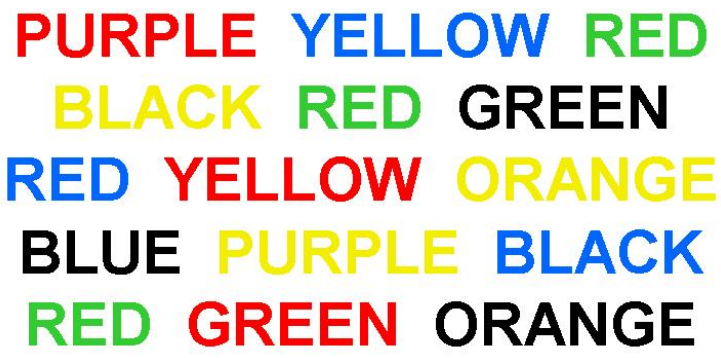
[6] Neural correlates of the misattribution of speech in schizophrenia
PAUL ALLEN, EDSON AMARO, CYNTHIA H. Y. FU, STEVEN C. R. WILLIAMS, MICHAEL J.BRAMMER, LOUISE C. JOHNS, PHILIP K. McGUIRE *The British Journal of Psychiatry* Feb 2007, 190 (2) 162-169; **DOI:** 10.1192/bjp.bp.106.025700

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, 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 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.
- 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 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 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.

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

- 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 — Biological Explanations for Anorexia Nervosa:

Anorexia Nervosa — A type of eating disorder in which an individual, despite being seriously underweight, fears that she or he might become obese and therefore engages in self starvation to prevent this happening. The DSM-IV criteria for Anorexia Nervosa are A. Weight loss leading to a maintenance of body weight less than 85% of that expected, B. Intense far of gaining weight or becoming fat and C. Disturbance in the way in which one's body weight or shape is experienced, alongside denial of the seriousness of the current low body weight.

Biological explanations — An approach to explaining behaviour which suggests that a full understanding of thoughts, emotions and behaviour must include an understanding of their biological basis, i.e. the role of genetics, neural correlates and hormones.

Genetic explanations — These explanations suggest that the likelihood of behaving in a particular way is determined by a persons's genetic makeup, i.e. it is inherited from parents. In the case of AN, this includes the roles of candidate genes, such as Ephx2.

Neural explanations — Explanations which involves areas of the brain and nervous system and the action of chemical messengers in the brain known as neurotransmitters as a means of controlling and predicting behaviour.

Genetic explanations

- **Family studies** have shown that eating disorders such as AN run in families. First degree relatives of individuals with AN have a 10 times greater chance of developing AN than relatives of unaffected individuals (Strober et al, 2000). It therefore follows that people may inherit a vulnerability towards developing eating disorders (Tozzi et al, 2005).
- Holland et al. found concordance rates of 56% for monozygotic twins, compared to only 5% of dizygotic twins, out of a sample of 45 pairs of female twins. Since MZ twins share 100% of their genes with each other, whilst DZ twins share only 50%, these concordance rates are highly suggestive of a genetic basis to AN.
- **Twin studies** suggest a high heritability for AN, varying from 28% to 74%. For example, Wade et al (2000) interviewed over 2163 female MZ and DZ twins, evaluating them on the DSM-IV criteria for AN. The researchers found a heritability estimate of 58%, alongside reaching the conclusion that "the comorbidity between anorexia nervosa and major depression is likely to be due to genetic factors that influence the risk for both disorders". Since these concordance rates were over 50%, such twin studies suggests that there may be candidate genes which lead to a genetic predisposition of some individuals towards AN.
- Conclusions drawn from twin studies are difficult to make reliably as MZ twins share the same genes and environment, making it hard to separate the influences of nature and nurture. **Adoption studies** avoid this because each pair of twins do not share the same environment. For example, Klump et al (2009) studied 123 adopted sibling pairs and 56 biological sibling pairs, finding heritability estimates ranging from 59% - 82% for the different aspects of disordered eating.

Evaluation of Genetic explanations:

— Problems with the genetic explanation = Fairburn et al (1999) found that although many studies have been conducted, the actual heritability estimate is still unknown. The researchers point out that results have been inconsistent and the studies not always replicated, making it difficult to draw reliable conclusions. Many studies violate the equal environments assumption ([8]'that identical and fraternal twin pairs experience equivalent trait-relevant environmental exposures'). Twin researchers also assume than MZ and DZ twins raised together have equal environments. However, this may not be the case as it has been shown that MZ twins tend to be treated more similarly than DZ twins (Loehlin and Nicols, 1976), and so their similar environmental factors may have a greater influence on the development of AN compared to DZ twins.

— The genetic explanation ignores the role of media in AN = The media's portrayal of ultra-thin models is an important risk factor for eating disorders. However, Builk (2004) suggests that genetically vulnerable individuals might seek out images themselves of thin role models. Vaughn et Fouts conducted a longitudinal study which found that adolescent girls anorexia symptom severity increased over a period of 16 months when they read more fashion magazines during this time. This suggests that social learning theory, through the use of mediational processes and vicarious reinforcement, may be an explanation for why adolescent girls identify with ultra-thin models role models, and hence progress the development of AN.

—In the US, treatment for AN is restricted under many insurance plans as it is not considered to have a biological basis = In 2014, the US tried unsuccessfully to get eating disorders included in

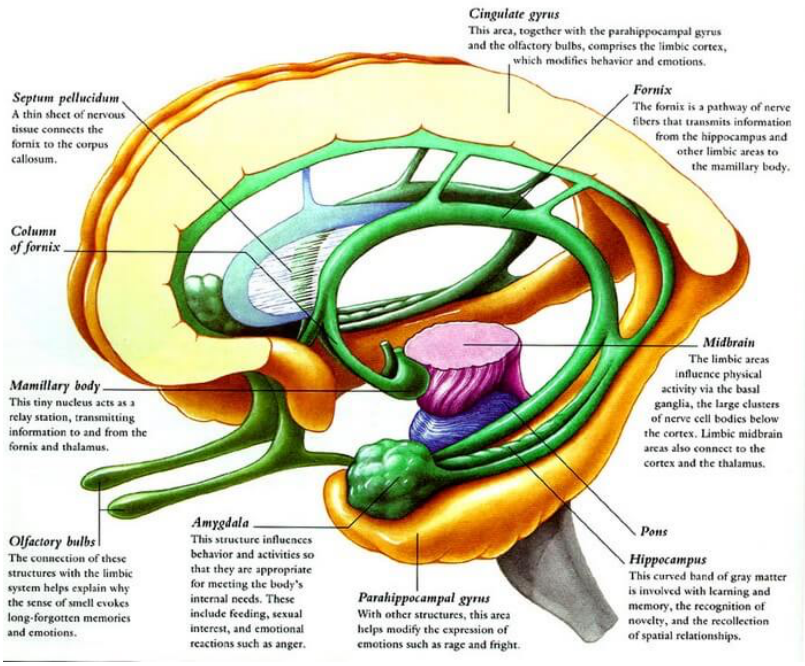
[8] Fosse R, Joseph J, Richardson K. A Critical Assessment of the Equal-Environment Assumption of the Twin Method for Schizophrenia. *Frontiers in Psychiatry*. 2015;6:62. doi:10.3389/fpsyt.2015.00062.

essential health benefits through legislation. However, research creates a case allowing AN to be considered in the same way as schizophrenia as they are both considered to be biologically based e.g. [9]AN seems to affect between 1 and 4% of schizophrenia patients, and so there is a high prevalence of comorbidity between schizophrenia and eating disorders.

Neural explanations

- **Serotonin:** Disturbances in the levels of the neurotransmitter serotonin may be responsible for producing characteristic symptoms of AN, such as appetite suppression and obsessiveness. For example, Bailer et al (2011) measured serotonin activity in women recovering from either restrictive type anorexia or binge eating and found higher levels of serotonin activity. In addition, the researchers found that the women with the highest levels of serotonin had the highest level of anxiety, suggesting that persistent disruption of serotonin levels may lead to increased anxiety which could trigger AN. Those suffering from AN were found to have low levels of metabolites, such as 5-HIAA, which are produced by the breakdown of serotonin. These low levels suggest that AN sufferers have abnormally functioning serotonin systems, where the neurotransmitter is now always broken down.

- **Dopamine:** Kaye et al (1991) used a PET scan to compare dopamine activity in the brains of 10 women recovering from AN and 12 healthy women. They found overactivity in the dopamine receptors of the women with AN in the basal ganglia, where dopamine plays a part in the interpretation of harm and pleasure. Increased dopamine activity in the basal ganglia leads people to interpret rewards differently. This matches the fact that individuals with AN find it difficult to associate good feelings with things, such as food, which people find pleasurable.



- Secondly, Bailer et al (2012) found that AN sufferers injected with amphetamines, causing an increase in dopamine levels, experienced anxiety whilst healthy control patients experienced euphoria. Therefore, restricting food intake may be a way of controlling anxiety through reducing bodily dopamine levels.

- **Limbic system dysfunction:** Lipsman et al (2012) suggests that the neural roots of AN appear to be related to a dysfunction in the limbic system. Researchers claim that dysfunction in these

[9] Youssef Kouidrat, Ali Amad, Jean-Daniel Lalau, and Gwenole Loas, "Eating Disorders in Schizophrenia: Implications for Research and Management," Schizophrenia Research and Treatment, vol. 2014, Article ID 791573, 7 pages, 2014. doi:10.1155/2014/791573

areas whose normal functions is to regulate emotion, can lead to deficits in emotional processing, which may lead to pathological thoughts and behaviours that are typical of AN.

Evaluation of Neural explanations:

— Problems with the serotonin explanation = SSRIs, which increase the levels of serotonin in the brain by reducing the breakdown at the presynaptic cleft, are ineffective when used with AN patients. Ferguson et al found no difference in symptom outcomes between patients taking SSRIs and patients of a similar age who were not taking an SSRI. A second problem with serotonin explanations is the effort to isolate serotonin as the single neurotransmitter responsible for AN. In reality, serotonin interacts with dopamine, where both are precurosors to noradrenaline, which may also interact with the neurotransmitter GABA. Therefore, the focus on serotonin is a very simplified explanation for AN.

+ Research has supported the role of dopamine = Food aversions, weight loss, and distorted body image cognition have been found to be related to an increased activity in dopamine pathways (Kaye 2008). Barbeto et al (2006) found that increased blink activity is indicative of higher levels of dopamine activity, with a correlation found between blink rate and the duration of AN, suggesting that [10]there is a significant positive correlation between blink rate and the duration of illness. Kaye et al (1999) found that recovering AN patients displayed decreasing HVA/ homovanillic acid levels (a metabolite of dopamine). This study is particularly useful because it removes the confounding variable of HVA levels decreasing during AN due to malnutrition or a lack of exercise.

+ Biological Explanations of AN reduce stigma = Such explanations also prevent the belief that it's "their fault" , alongside offering the possibility of treatment. For example, deep-brain stimulation/ DBS , particularly of the nucleus accumbens in acute chronic cases, may therefore be a potential treatment for AN (counteracting the abnormalities in the limbic system), where [11]Lipsman et al. found an average BMI of 3.5 points to 17.3 within the AN group, with 6 of the 16 patients achieving a normal BMI.

Part 7 - Family Systems Theory and Anorexia Nervosa:

Key words

Autonomy — The freedom to make decisions and determine actions without the constraints imposed by others, and the extent to which we are dependent from others.

Control — Means to direct or to exercise authoritative influence over events of behaviours. In the case of AN, sufferers exert control in an effort to reduce family dependence.

Family systems theory — A psychodynamic explanation which argues that in order to understand a family system, we must look at the family as a whole and not just at its individuals members.

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[10] Increased dopaminergic activity in restricting-type anorexia nervosa, Barbato, Giuseppe et al. *Psychiatry Research*, Volume 142 , Issue 2 , 253 - 255

[11] Nir Lipsman, Eileen Lam, Matthew Volpini, Kalam Sutandar, Richelle Twose, Peter Giacobbe, Devin J Sodums, Gwenn S Smith, D Blake Woodside, Andres M Lozano. **Deep brain stimulation of the subcallosal cingulate for treatment-refractory anorexia nervosa: 1 year follow-up of an open-label trial**. *The Lancet Psychiatry*, 2017; DOI: 10.1016/S2215-0366(17)30076-7

Minuchin et al (1978) developed the psychodynamic family theory, which suggests that a dysfunctional family was the prerequisite for the development of AN. The psychodynamic family was a necessary context for the development for AN, so treatment must aim to change the way in which the family functions.

Characteristics of the "psychosomatic" family

1. Enmeshment — Describes a family where the parents are excessively emotionally- involved with their children but may be dismissive of their emotional needs. This can make it hard for the child to develop an independent self concept. The self-identity of each family member overlaps with other members, due to a lack of boundaries and respect for each other's privacy. Enmeshed families are over involved with each other and there is a lack of boundaries. Barber and Beuhler contended that enmeshment stifles the development of children skills to deal with common social stressors and make the development of AN more likely. The assumption is made that each member understands and can speak for the other. This leads to the adolescent AN sufferer having the desire to distinguish themselves from other family members, and particularly the mother.

2. Control/Overprotectiveness— The psychosomatic family is characterised by overprotective control, where there is a high degree of concern for each other's welfare. This can lead the individual to try and gain back some control by refusing to eat. Particularly important, according to Palazzoli (1974), is the role of the mother: she sees her actions as an example of self-sacrifice for her daughter, and so will automatically blame the daughter whenever any of her 'decisions' are bad. This in turn reduces the independence of the anorexic daughter. The mother plays a particularly important role by being intrusive and domineering.

3. Rigidity — Rigid families show an inflexibility in their adaptation to new situations. In the face of stress, they increase the rigidity of their patterns of behaviour and resist any exploration of alternatives. Any attempts at 'self-differentiation', such as the daughter trying to assert more independence, is quickly extinguished by the family, who cannot and will not adapt to internal or external threats to their circumstances. This may be reflected in the child starving themselves, in an attempt to 'control their own destiny' and achieving autonomy by acting against the wishes of her parents (Bruch, 1978).

4. Lack of conflict resolution/ Conflict Avoidance— Munichin believes that AN families were in a state of constant but unresolved conflict, with conflicts being abandoned rather than solved. In families with AN, patients typically present a strong façade of togetherness in order to avoid conflict (Latzer et al, 2002). An excerpt from their (Latzer et al) report reads: [12] "The families of eating disorder patients were found to be less cohesive, expressive, and encouraging of personal growth than were controls … this may reflect family difficulties in supporting the child during the process of separation individuation, and exploration of the outside world".

Within the family where child is involved in conflict, Munichin identified 3 characteristics of patterns of conflict-related behaviour:

A. Triangulation — Involves a pair of family members either incorporating or rejecting a third family member.

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[12] Yael Latzer, Zipora Hochdorf, Eitan Bachar, Attachment Style and Family Functioning as Discriminating Factors in Eating Disorders, *Contemporary Family Therapy*, 2002, Volume 24, Number 4, Page 581.

B. Parent-child coalition — The child now moves into a more stable coalition with one parent against the other. The coalition parent will respond to the child with excessive concern (enmeshment) whilst the other parent withdraws.

C. Detouring — When parents are unable to solve conflicts by themselves they direct focus away from themselves and onto the child. The child consequently becomes the scapegoat because their wellbeing is sacrificed for avoidance of marital conflict.

Evaluation of Family Systems Theory:

+ Research support for enmeshment = Manzi et al (2009) found that cohesion between family members was linked to positive outcomes and psychological wellbeing amongst adolescents, whereas enmeshment had the opposite effect. Family cohesion was indicative of supportive family interactions, whereas enmeshment was rooted in manipulation and control. This supports the idea that enmeshment may be a trigger for AN because starvation may be an attempt of the child to differentiate themselves from other family members and maintain a degree of control/ autonomy, which increases the thinner they become.

— Problems with the psychosomatic family model = Research has tried to establish characteristics that are specific to families in which a member has AN. However, this has produced inconsistent findings. For example, Kog et Vandereycken failed to find the characteristics provided by the psychosomatic family model and instead proposed that this model can be simplified to [13]3 components: the intensity of intrafamilial boundaries, the degree of the family's adaptability, and the family's ways of handling conflicts. There is also growing evidence that families in which someone has an eating disorder are a diverse group in terms of the nature of family relations. Therefore, this suggests that the psychosomatic model is an over-simplified explanation for AN, and can only be applied to some families, hence not being a universal explanation.

— Inconclusive support from family-based therapy = The success of family focused therapy is a key part of recovery. Carr et al (2000) concluded that there is compelling evidence for the effectiveness of family intervention, citing Shadish and Baldwin (2003), who found that over 71% of families experienced improved family dynamics, compared to a control group, after familial interventions. However, Grange et Eisler (1987) pointed out that whilst there is some evidence that family therapy is accompanied by changes these may not happen in all families, and therefore has limited practical applications, further questioning the validity of the psychosomatic family model explanations.

— Gender bias in family systems = Gremillion claims that there is a gender bias in family systems because they tend to focus only on mother-daughter relationships, with enmeshment is nearly always being seen as maternal. Focus on therapy tends to focus on reforming mothers and that there is a lack of awareness that fathers too contribute to the enmeshment and that the father's overly controlling tendency is often overlooked. Therefore, this may lead to stereotyping of an 'excessively overprotective and rigid' mother and parent-blaming, whereas both parents in reality have a role to play.

+ Research support for lack of conflict resolution = Latzer and Gaber (1998) carried out an observational study of conflict resolution in 40 families with AN and 40 matched families without AN. Parents and daughters were told to choose two areas of disagreement and were asked to keep off the topic of food. The families with AN had greater difficulty choosing topics and

[13] Kog, E., Vandereycken, W. and Vertommen, H. (1985), The psychosomatic family model. A critical analysis of family interaction concepts. Journal of Family Therapy, 7: 31–44. doi:10.1046/j.. 1985.00663.x

remaining focused on them whilst moving towards resolution. In addition, [14]"the pattern of conflict avoidance is also reflected in a low level of measured criticism and hostility in relatives of AN patients". This supports Munichin's claim that there is an avoidance of conflict where one family member has AN, and the role of conflict resolution in the development and maintenance of AN.

Part 8 — Social Learning Theory and Anorexia Nervosa:

Key words

Media — The various means of communication such as radio, television, newspapers and the internet that reach or influence people widely.

Modelling — A form of social learning where individuals learn a particular behaviour by observing and imitating a role model performing that specific behaviour. This effect is greater if the role model is seen to be attractive, have high social status, being the same gender as the observer and of a similar age. The role model does not need to be physically present in the environment to exert influence.

Reinforcement — A term used in psychology to refer to anything that strengthens a response and increases the likelihood that it will occur again in the future. Vicarious reinforcement, according to social learning theory, increases the likelihood that an observed behaviour will be repeated if the model is seen to be rewarded.

Social learning — Learning through the observation and imitation of behaviours modeled by the role model, who has been identified by the observer. The modeled behaviors that are seen to be rewarded are more likely to be imitated by the observer, through the process of vicarious reinforcement. Learning occurs both directly (operant and classical conditioning) and indirectly (vicarious reinforcement).

Bandura believed that people learn by observing the behaviour of others as well as observing the outcomes of these behaviours. Social learning theory suggests that children pay attention to the role models in their lives and if they observe behaviour that is likely to produce a positive response from others, as judged by the role model being vicariously reinforced for displaying this behaviour, then they will imitate them in the belief that they will also receive the same positive feedback. Such learning depends upon the 4 mediational processes (cognitive processes which come between stimulus and response): Attention, retention, motivation and motor reproduction.

Maternal role models — Studies have found similarities between mother-and-daughter restrained eating and dieting behaviours amongst children as young as 10 years old, with such children also tending to overeat in a behavioural test of restraint (Hill et al, 1989). Smolak et al suggested that mothers who complain about their weight are more likely to have children who have their own weight concerns, with these influences being greater for daughters rather than sons.

Peer influence — Peer reinforcement is particularly important during adolescence, so they may be susceptible to peer influence over eating patterns. A US study found that dieting amongst friends was linked to unhealthy weight control behaviours, such as taking diet pills or purging (Eisenburg et al). Jones and Crawford (2006) found that overweight girls and underweight boys were more likely to be teased, suggesting that teasing serves to enforce gender based ideals.

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[14] Yael Latzer, Lee B. Gaber, Pathological Conflict Avoidance in Anorexia Nervosa: Family Perspectives, *Contemporary Family Therapy*, 1998, Volume 20, Number 4, Page 539

Their [15]results show that despite girls reporting more appearance conversations, boys perceived more appearance pressure and teasing. In addition, Keel et al (2012) investigated the influence of college peers, both male and female, on disorganised eating. The researchers found that [16]'for women, college roommate dieting significantly predicted Drive for Thinness, Bulimia scores, and purging at a 10-year follow-up'. This further demonstrates the strong impact of peer influences on disorganized eating, through the mechanism of social learning, particularly due to some college peers being identified as role models.

Media influence — The portrayal of thin models on TV and in fashion magazines is a contributory factor for body image concerns. The media does not influence everyone in the same way as individuals with low self esteem are more likely to compare themselves to idealised images. Low self esteem plays a crucial role in the development of eating disorders. Button et al (2005) found that girls with low self esteem aged 11-12 years old were at a significantly greater risk of developing an eating disorder aged 15-17. A report published by the BMA expressed concern for the use of thin models and reported that the degree of thinness exhibited was not only unachievable but also biologically inappropriate. These ideas were further supported by Dittmar et al (2006), who found that a significant proportion of the 162 British girls, aged 5-8 years old, involved in the study reported low self-esteem and increased body dissatisfaction when viewing the Barbie dolls, compared to a control group who'd viewed the Emme dolls (who have a more realistic body shape). This deep-rooted body dissatisfaction at a young age may manifest itself as AN in later life, particularly due to Barbie being such an attractive role model and possessing many characteristics (such as glamour, popularity and attractiveness) which girls in particular aspire to.

Evaluation of Social Learning Theory and Anorexia Nervosa:

— Maternal influence is more complex than social learning = Research on the role of mothers has not always produced consistent results. For example, Pike et Rodin (1991) found that there was no evidence for daughters imitating the weight concerns of their parents. Ogden and Steward (2000) found that although mothers and daughters were of a similar weight, there was no association for restrained eating. This suggests that although most daughters would identify with and view their mothers as role models, and so be expected to adopt similar eating habits through social learning theory, this is not always the case. This therefore casts doubts on the idea that SLT is a major explanation for AN.

+ Research support for peer influence = Costa-Fonta and Jofre Bonet (2009) investigated the effects of peer weight and developing an eating disorder. The researchers found that peers with a larger body mass had a smaller likelihood of developing AN, suggesting that having peers with an average or higher BMI protects individuals from eating disorders, through the mechanisms of

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[15] Jones, D.C. & Crawford, J.K. J Youth Adolescence (2006) 35: 243. https://doi.org/10.1007/s10964-005-9006-5

[16] Keel PK, Forney KJ, Brown TA, Heatherton TF. Influence of College Peers on Disordered Eating in Women and Men at 10-Year Follow-up. *Journal of Abnormal Psychology*. 2013;122(1):10.1037/a0030081. doi:10.1037/a0030081.

social learning theory. Secondly, [17]several opinions from peers of an individual's own body mass were found to decrease the likelihood of women being thin or extremely thin. These pieces of evidence further support the role of peer influence as an explanation for anorexia nervosa.

+ Research support for media influence = In a study conducted by Becker et al (2011), eating attitudes amongst Fijian children were studied following the introduction of TV in 1995. After exposure to TV, it was found that the children expressed a desire to lose weight, and this desire increases with the more friends the individual has who had access to TV, videos or DVDs. This increase in the will to lose weight may be due to the discussions made amongst friends about the ultra-thin models they have seen on TV, creating a mutual goal. However, other research has shown that intervention prior to media exposure can prevent the adverse effects. This suggests that media can be a powerful influence but can be prevented through education.

+ Explains gender differences = Some studies have found that reading magazines is a more consistent predictor of the development of eating disorders than watching TV. Harrison and Cantor (1997) found no correlation between watching TV and eating disorders but found a correlation between reading fitness magazine and attitudes towards food and dieting. As the researchers found, [18]'For women, media use predicted disordered-eating symptomatology, drive for thinness, body dissatisfaction, and ineffectiveness. For men, men, media use predicted endorsement of personal thinness and dieting'. This shows that media influences, through the mechanism of SLT, can explain gender differences in bodily attainment goals. Men suffer predominantly from bigorexia (Jones and Morgan, 2010) due to increased exposure to athletic and muscular role models, whereas females suffer increasingly from anorexia, due to increased exposure to Size Zero models in socially-desirable contexts.

Part 9 — Cognitive Theory and Anorexia Nervosa:

Key words

Cognitive theory — When applied to disorders, this is any explanation about the way in which a person processes information that affects their feelings and their behaviour. The focus, according to the cognitive approach, is on how our mental processes (e.g. attention and perception) can affect our behaviour.

Distortions — Thinking that has a bias such that what is perceived by a person does not match reality. These may be a result from comparison with others, (i.e. thin models) leading to a misperception that the individual must be overweight, which in turn leads to feelings of self disgust and an attempt to lose weight. For example, Murphy et al (2010) believes that cognitive distortions are at the root of AN symptoms, particularly the tendency to overestimate personal weight and body size. Using the Body Image Assessment, Williamson et al (1993) supported this idea by showing how the (37) AN patients were significantly more likely to overestimate the size of their silhouette compared to the (95) healthy controls.

Irrational beliefs — Beliefs that are unhelpful, illogical and inconsistent with our social reality. In contrast, rational beliefs are helpful, logical and consistent with our social reality. Characteristic irrational beliefs of AN include absolutist thinking ("I have eaten an apple and so I have no self-control") and catastrophising ("I have gained half a kilogram and so I will be obese"). These irrational beliefs may stem from the anorexic's tendency towards 'never-ending' perfectionism -

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[17] Costa-Fonta and Jofre-Bonet., Body Image, Peer Effects and Food Disorders: Evidence from a Sample of European Women, *LSE Health*, November 2009, Working Paper No: 15/2009.

[18] Harrison, K. and Cantor, J. (1997), The relationship between media consumption and eating disorders. Journal of Communication, 47: 40–67. doi:10.1111/j.1460-2466.1997.tb02692.x

once they have reached their goals, the bar is set even higher, leading to a constant feeling of failure. For example, Hewitt et al (2003) argues that this façade of perfectionism is made up of [19]'perfectionistic self-promotion, non-display of imperfection and non-disclosure of imperfection'. Individuals may also develop self-harming habits due to faulty beliefs about themselves and the world around them. For example, a typical irrational belief of AN patients is that they must be thin for others to like them.

The Cognitive Behaviour Model — Garner and Bemis (1982) found that anorexia patients have many characteristics in common e.g. they are typically perfectionists and often full of self-doubt. These characteristics, coupled with an individual's exposure to cultural ideals of thinness, leads them to form extreme ideas about the importance of body weight and the irrational belief that losing weight will reduce their distress and make them more attractive to others.

Losing weight becomes self-reinforcing for the individual because of the positive comments from others. This can be explained by positive reinforcement according to behaviourism. Once the importance of thinness is established, anxiety about eating increases and this develops into a fear of food and weight gain, meaning that food avoidance becomes the norm. As the individual becomes more and more socially isolated, this reduces the chance of viewing their style of thinking as irrational.

The Trans-diagnostic Model — Fairburn et al (2003) suggested that we should see AN symptoms as manifestations of a more broadly defined eating disorder. According to this model, the underlying cause of all eating disorders is the same set of cognitive distortions. This involves the overestimation of body weight, appearance, and emphasis on self-control which Fairburn believes to be the central factor in AN. This, paired with a reduced capacity for 'set-shifting' , according to Treasure and Schmidt's interpersonal maintenance model of AN (2013), may lead to the development of AN. For those with AN ,their sense of self-esteem is determined by their weight and appearance, whereas for a healthy person it would be their achievements. The restriction of food intake that is a characteristic of AN is maintained by the mechanism below:

1) An enhanced sense of self control leads to increased self esteem. When an individual with AN decides not to eat or to eat very little this provides positive reinforcement in the form of self esteem (a behaviourist mechanism).

2) The physiological and psychological changes they receive, such as starvation, are perceived as being the result of failure of self control, which in turn leads to a greater reliance on food restriction.

3) Due to their focus on weight, the individual engages in increased self monitoring of their weight, where any weight gain or loss perceived as being too slow leading to an increased effort to restrict intake.

Part 10 — Key Studies:

N.B. For information about the classic study of Rosenham (1973) On Being Sane in Insane Places, please see the factsheets.

Carlsson et al (1999) investigated that idea of network interactions in schizophrenia, and the therapeutic implications that such research has. Using PET scans (as detailed in Topic 3 - Biological Psychology), the researchers found that the dopamine hypothesis was supported (i.e.

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[19] Hewitt, P. L., Flett, G. L., Sherry, S. B., Habke, M., Parkin, M., Lam, R. W., . . . Stein, M. B. (2003). The interpersonal expression of perfection: Perfectionistic self-presentation and psychological distress. *Journal of Personality and Social Psychology, 84*(6), 1303-1325.

hyperdopaminergia in the subcortex and hypodopaminergia in the cortex), that dopamine is not the only neurotransmitter involved in the development of SZ (e.g. an increased focus on the role of glutamate by Moghaddam and Javitt, and that glutamate controls the release of dopamine and interaction at the post-synaptic membrane), the idea of a thalamic filter (regulates the action of neurotransmitters within the cerebral cortex), as well as the use and role of atypical and typical antipsychotics in the treatment of SZ was reconsidered.

+ There is a real-life application associated with an increased understanding of the dopamine hypothesis, as demonstrated by Carlsson et al, particularly because the use of antipsychotics depends on this understanding. This means that more effective antipsychotics can be developed, and well as pedaling further research into the role of candidate genes and target genes.

+ The brain scanning techniques used by Carlsson et al, such as PET scans, allows for reliable data to be gathered and then interpreted objectively. This means that the conclusions drawn on the basis of these findings have greater validity!

— Since the review was carried out in 2000, his findings can be said to have low temporal validity. Particularly with current technological advances, the modern understanding of schizophrenia is significantly different to that two decades ago, and so his findings may only be generalised to contribute to the understanding of the field at that specific point in time.

Scott-Van Zeeland et al reviewed the evidence for the role of EPHX2 gene variants in anorexia nervosa, using 261 individuals with early-onset and AN and then comparing these findings with 73 controls. The researchers concluded that [20]"EPHX2 is known to influence cholesterol metabolism… and found evidence for a modifying effect of a subset of variants on the relationship between cholesterol and BMI. These findings suggest a novel association of gene variants within EPHX2 to susceptibility to AN and provide a foundation for future study of this important yet poorly understood condition".

+ The use of a control group increases the validity and reliability of the findings because this group acts as a baseline measurement, through which comparisons can be made. Statistical comparisons between the control and experimental groups also dictate the confidence placed in the conclusions.

+ The large sample size of over 800 participants in total, increases the reliability of the conclusions drawn because it is less likely that the observed findings are due to chance. This means that the findings are more likely to be generalised to the general population and represent their experiences.

— However, it should be noted that the EPHX2 is only one specific candidate gene, hundreds of which contribute to the development of Anorexia Nervosa, alongside environmental influences such as enmeshment and the psychosomatic family. Therefore, it is important not to take the view of biological reductionism and explain AN purely at the lower, biological level, as this is unlikely to form a complete explanation.

[20] Scott-Van Zeeland AA, Bloss CS, Tewhey R, et al. Evidence for the role of *EPHX2* gene variants in anorexia nervosa. *Molecular Psychiatry*. 2014;19(6):724-732. doi:10.1038/mp.2013.91.