WJEC Psychology A-level

Addictive Behaviours

Notes

Addiction

Part 1 — Describing Addiction:

- A definition of addiction must include the ideas of withdrawal syndromes, tolerance and dependance (both physical and psychological).
- Withdrawal syndromes occur when the patient stops taking the addictive substance, or consumes a smaller dose, and experiences symptoms opposite to those induced by the drug. These are often very unpleasant, such as severe insomnia and nausea. Therefore, addicts are motivated to continue carrying our addictive behaviours in order to avoid withdrawal symptoms and elongate the positive effects of the addiction.
- Tolerance occurs when an increasingly high dosage of a drug does not produce the desired results or effects for the patient. This results in a spiral or cascade of usage, where increasingly larger doses are required. Such tolerance is particularly problematic in surgery, where alcohol or drug addicts need higher doses of anesthetic, due to a tolerance to sleep-inducing drugs.
- Physical dependence is characterised by withdrawal symptoms subsiding when the drug has been administered to the patient, and shows that their body cannot function normally without the drug, thus causing physiological changes. This is different to psychological dependence, which occurs when patients experience compulsions to acquire the drugs in order to experience the pleasurable effects once again, leading to the development of a habit due to this system of 'usage and reward'.

Part 2 — Genetic Risk Factors in the Development of Addiction:

- From a biological perspective, individuals may inherit candidate genes which provide them with a heredity predisposition towards developing an addiction, as opposed to a strongly deterministic standpoint.
- One of these biological susceptibility mechanisms could be abnormally low levels of D2 receptors in the brain, meaning that fewer dopamine molecules can bind to these receptors and initiate action potentials in the postsynaptic membrane.
- The second mechanism would be, as suggested by Pianezza et al (1998), abnormal gene expression of the gene coding for the CYP2A6 enzyme. Lower levels of this enzyme leads to slower rates of nicotine breakdown, reducing the concentration of nicotine metabolites within the brain's synapses and so decreasing the likelihood of developing a nicotine addiction.

+ There is evidence supporting the biological basis for a predisposition towards developing addictive behaviours. For example, Kendler et al (2012) found that adoptees with at least one biological parent with an addiction, were at a 4.4% greater risk of developing an addiction, compared to adoptees with no biological predisposition. Therefore, genetics play a crucial role in heritable predispositions.

—Purely genetic factors, such as candidate genes, may not be the only genetic factors influencing the development of addictions. For example, a genetic pre-disposition towards a lack of self-control or a tendency for impulsive behaviour may increase the rate of progression of an addiction. A child who behaves impulsively, particularly at school, is likely to be punished and so will develop a negative outlook on education and rejection from their family, leading to fewer future opportunities, and so potentially the child will look towards drugs in adulthood.

Part 3 — Psychosocial Risk Factors in the Development of Addiction:

- The 4 main psychosocial risk factors for addiction development are personality, family influences, peer influences and stress.
- Although most dispositional traits do not have a genetic basis, certain personality features, such as impulsivity and antisocial personality disorder (as suggested by Ivanov et al, 2008) which increase the risk of developing an addiction. Impulsivity in particular is associated with irrational risk-taking behaviours and little reflection, and due to its high concordance rates with addiction, suggests that there may be common neurological bases for both.
- Family influences include the extent to which the child's behaviour is being monitored (increased monitoring reduces the likelihood of addiction formation), the child's perception of their parent's attitudes towards addictive substances (e.g. regularly drinking at home during school increases the risk of excessive alcohol consumption during university, as demonstrated by Livingston et al, 2010) and a child's degree of exposure to such substances.
- Peers exert a greater influence upon the development of addictions compared to family influences, when considering adolescents. Peers provide opportunities for regular access to addictive substances, and their use informs observers of 'norms' of consumption, which they

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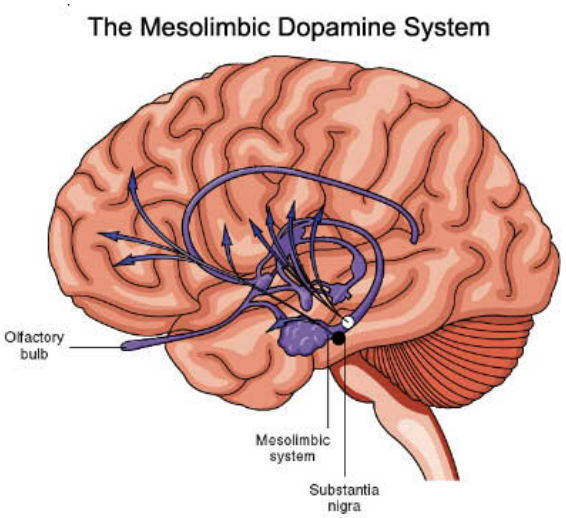

The mesolimbic dopamine system may be the "reward center" of the brain where pleasure and displeasure arising from many sources (including psychoactive drugs) is registered.

often aim to exceed due to their incorrect perceptions.

•When considering the influence of stress, it may be useful to take on an interactionist approach, as suggested by Andersen and Teicher (2008). Chronic stress does not always lead to addiction, despite a strong correlation between the two. Instead, chronic stress acts as a predisposition towards stress (a diathesis) which then must be paired with an environmental stressor (e.g. childhood rape, in the case of Epstein et al's 1998 study) to result in addiction.

+There is evidence supporting the utility of adopting an interactionist approach towards understanding addiction, as suggested by Mayes and Suchman (2006). No one factor has been found to be more important than another in determining the likelihood of developing an addiction, but rather the combinations of different factors help to inform us of the severity and type of addiction which may be caused. Therefore, we are incorrect in saying that one factor will indefinitely lead to addiction, as some variations of factors may actually reduce this risk, such as increased parental monitoring and changing one's peers.

— The main issue with risk factors of addiction is that only correlations exist. This means that there is only a link between one risk factor, such as family influence, and the increased likelihood of developing an addiction. Correlational studies suffer from the 'third factor' problem, which suggests that there may be a third, unstudied factor which has influenced both outcomes. The file drawer problem means that correlational studies may be biased in that they only include studies which have found significant results. Overall, correlational studied can never establish a 'cause and effect' relationship between two factors, thus limiting the utility of such explanations.

+ Hawkins et al (1992) have suggested that there is a real-life application in an improved understanding of the various risk factors and the extent to which they interact. For example, this can be useful for social services for identifying families at risk of their children developing addictions, as well as creating interventions for adolescents, to discourage them from smoking and teach them social skills in order to politely but firmly refuse cigarettes without ridicule or embarrassment, as suggested by Tobler et al (2000).

Part 4 — Explanations for Nicotine Addiction: Brain Neurochemistry:

- Dani and Heinemann's 1996 desensitisation hypothesis!
- The researchers suggest that there is an antagonistic effect between the levels of acetylcholine and dopamine, particularly in the areas of the brain which control feelings of pleasure and pain, such as the ventral striatum and the nucleus accumbens.
- Nicotinic receptors (on the postsynaptic membranes of synapses in the nucleus accumbens) allow both acetylocholine (a neurotransmitter) and nicotine to bind. This results in the generation of an action potential in the postsynaptic membrane and the consequent breakdown of acetylocholine. However, the presence of nicotine means that the receptors undergo a process of deregulation/desensitisation, whereby they become less sensitive, reducing neurotransmission within these areas, and leading to feelings of heightened pleasure.
- The process above also means that the neurotransmitter dopamine is released into the mesolimbic system, which interacts with the nicotinic receptors to produce these feelings of euphoria and heightened pleasure.
- Withdrawal symptoms occur when there is upregulation (the opposite of deregulation) - this occurs when decreased nicotine levels causes the nicotinic receptors to become sensitive again to acetylcholine, leading once again to an increased rate of action potential generation within the nucleus accumbens. This results in feelings of anxiety and potentially nausea. The only way

to relieve these feelings is to increase nicotine consumptions once again to provoke deregulation.
- Over time, the concentration of nicotine required to provoke deregulation increases periodically, and so the individual is said to have developed a tolerance towards nicotine.

+ There is evidence supporting the role of dopamine in the deregulation of nicotinic receptors, as suggested by McEvoy et al (1995). Patients taking the dopamine antagonist Haloperidol, which binds to and blocks dopamine receptors, smoked significantly more than those who were not taking the drug. This suggests a higher consumption of nicotine was needed to maintain the deregulation of nicotinic receptors, due the blocking of dopamine action. This in turn suggests that an increased level of nicotine is needed to compensate for decreased dopamine levels, thus supporting the role of dopamine in the maintenance of nicotine addictions.

— However, Berrendero et al (2010) have suggested that there has been too much emphasis on the role of dopamine in the maintenance of nicotine addictions, and that there is not one isolated system or neurotransmitter which is solely responsible. For example, modern research has shifted their focus onto GABA and serotonin, as well as the role of endorphins which may produce the 'euphoric' and 'painless' feelings associated with a hit of nicotine. Therefore, a broader scope of research is required.

+ An interactionist approach may be a better explanation for nicotine addiction, whilst acknowledging the role of biological factors. The key problem with a reliance on dopaminergic explanations, as suggested by Choi et al (2003) is the idea of biological reductionism, where nicotine addiction is reduced to the activity of neurotransmitters. Despite such neurotransmitter activity being a biological predisposition or 'diathesis', psychosocial risk factors for the development of addictions may be more important, such as peer and family influences. This may therefore explain the statistic that only 50% of regular smokers develop a nicotine addiction, as they may be surrounded by 'protective' psychosocial factors e.g. a family with strong -anti-smoking' views.

Part 5 — Explanations for Nicotine Addiction: Learning Theory:

- Psychologists have suggested that nicotine addictions can be explained using social learning theory: it is a learned behaviour, and can be acquired through both classical and operant conditioning. A key feature of operant conditioning would be reinforcement, both negative and positive. This increases the likelihood of a certain behaviour being repeated.
- Positive reinforcement suggests that we carry out certain behaviours because we are motivated by the consequent rewards. Smoking can be positively reinforced by the reward of the euphoric feelings of nicotine stimulating the dopaminergic mesolimbic system, increasing the likelihood that we will smoke again to achieve the same reward.
- Negative reinforcement suggests that we carry out certain behaviours because we want to avoid unpleasant consequences. Smoking can be negatively reinforced by avoiding unpleasant withdrawal symptoms, through progressively increasing nicotine intake and therefore prolonging the period of desensitisation or deregulation of nicotinic receptors in the nucleus accumbens.
- Cue reactivity suggests that there are certain environmental cues, such as attending parties or social gatherings, which increase the likelihood of engaging in addictive behaviours, such as smoking. These environments contain both primary reinforcers (the pleasurable effects of smoking) and secondary reinforcers which coincide with the benefits of smoking (such as the smell of cigars, the cardboard feel of the packet and the neat arrangement of cigarettes). Primary and secondary reinforcers work together, through the process of cue reactivity (made up of behavioural responses, physiological responses and our own personal attitudes towards smoking) to reinforce and maintain excessive smoking habits.

+ Learning theory is able to explain gender differences in smoking, as suggested by Carpenter et al (2014). Women are less likely to give up smoking and more likely to relapse, which may be due to their poor self-efficacy i.e. not believing that they are able to give up smoking. This may also be due to women potentially being more social than men, being engaged in more social events and caring about interpersonal relationships, all of which could increase their sensitivity to cues which trigger smoking, and thus more easily lead to an addiction through the mechanism of cue reactivity.

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+ There is also evidence from animal studies that increased self-administration of nicotine may be the result of the pleasurable effects associated with increased dopaminergic stimulation of the mesolimbic system and the deregulation of nicotinic receptors. This was suggested by Levin et al (2010) who found that rats rapidly increased their intravenous intake of nicotine periodically, resulting in up to 100 licks/ attempts per infusion. Therefore, this gives strong support for the role of cue reactivity and positive reinforcement in the development of nicotine addictions.

+ The use of learning theory as an explanation for nicotine addiction has particularly useful real-life applications because aversion therapy and covert sensitisation is based upon these same principles. For example, Smith (1988) found that aversion therapy using electric shocks produced nicotine addiction recovery rates that were at least 32% higher compared to people who had simply decided to stop smoking. Therefore, an improved understanding of learning theory as an explanation for nicotine addiction may serve as as economical implication of psychological research, where improved NHS and public health service guidelines for the treatment of such a common addiction could be beneficial for sufferers.

Part 6 —Explanations for Gambling Addiction: Learning Theory:

- Gambling addictions can also be explained using social learning theory, with specific emphasis being placed on cue reactivity and five types of reinforcement (positive, negative, variable, partial and vicarious).
- All types of reinforcement increase the likelihood that the specific behaviour will be displayed again.

•Cue reactivity serves as the main explanation for the particularly high relapse rates associated with gambling addictions. There are many 'low-level' cues which continuously provoke sufferers to further engage with gambling activity, such as the presence of gambling shops on many high street, glamorous TV reports of the latest lottery winners and discounted entry for various gambling websites. These act as secondary reinforcers because they are associated with the pleasurable effects of gambling.

•Partial reinforcement is based on the idea that, as demonstrated by Skinner, behaviours most resistant to extinction are not produced through the consistent rewarding of desirable behaviours. Instead, the occasional winning bet experienced by gamblers is almost as rewarding compared to winning each time, due to the anticipation. The idea that the gambler may win on occasion, and so is partially reinforced/rewarded for their efforts, motivates them to continue trying to win.

- Variable reinforcement is based on the concept that a gambler has a certain statistical chance of winning e.g. an average of 20 throws of the dice. However, this does not necessarily mean that every 20th throw will result in winning a reward, and so they are motivated by this continuous temptation and 'statistical justification' to continue betting. Thus, persistency is key!
- Positive reinforcement maintains gambling behaviours by rewarding gamblers for their successful bets through the thrill of winning, the admiration and praise from others, the associated glitz and glamour of horse racing and the numerous success stories of lottery winners. These last few examples in particular are examples of vicarious reinforcement, where gamblers see others being rewarded for displaying addictive behaviours, and so they are motivated themselves to display these same behaviours in an effort to achieve the same rewards.
- Negative reinforcement maintain gambling behaviours through providing the gambler with an escape from their day to day struggles, due to the shift of focus.

— Learning theory cannot explain all stages of the acquisition and development of gambling behaviours, as suggested by Brown (1987). For example, positive reinforcement and variable reinforcement appear to be particularly important as part of the initial development of gambling,

which may be due to the immediate effects of pleasure which arise from successful bets, and the respite this provides from life crises. However, cognitive explanations may be better suited to explaining the long-term maintenance of addictive behaviours, with a particular focus on the cognitive biases and developing lack of educational insight into the ideas of chance/probability, being key factors.

— Similarly, learning theory suggests that, through the idea of cue reactivity, that all cues which have triggered gambling behaviours will continue to do so in all individuals. However, this is not always the case, as pointed out by Griffiths and Delfabbro (2001). Therefore, it may be more useful to adopt an interactionist approach as a means of explaining gambling, where reactive cues may act as the diathesis (vulnerability) but the surrounding psychosocial risk factors may act as stressors e.g. peer and family influences. An individual is unlikely, for example, to develop a gambling addiction if their life-long partner refuses to talk about or enter gambling shops.

+ However, there is evidence supporting the role of positive and vicarious reinforcement in the development of serious gambling addictions, as suggested by Dickerson (1979). In his field study of two Birmingham betting shops, the high-frequency betters were found to often place their bets in the final 2 minutes before the start of the race. This is also associated with the heightened frenzy of the brokers, increasingly tense commentary and the overall 'buzz' from the betting shop. Therefore, such betting behaviour may be positively reinforced by providing the better with an outlet to engage in the same buzz.

Part 7 — Explanations for Gambling Addiction: Cognitive Theory:

- The cognitive theory of gambling addiction is based on the two premises of expectancy theory and cognitive biases.
- Gamblers have an irrational idea of expectancy - they believe that the benefits of gambling must exceed the associated costs. This irrationality is further emphasised through a gambler's selective attention towards focusing on the positives and rewards of their addiction, as opposed to the very real losses.
- Cognitive biases play an important role in their distorted views of expectancy, as suggested by Rickwood (2010). There are 4 types of cognitive biases which include faulty perceptions (focusing on "gambler's fallacy", which suggests that losses cannot continue forever and must inevitably be broken by a win), selective recall (only remembering the positives and rewards of their addiction), skills and judgements (particularly focusing on their irrational illusion of control) and their personal traits (believing in luck or upholding superstitious beliefs about specific betting machines or games).
- To illustrate the effects of cognitive biases, Griffiths (1994) found that regular gamblers demonstrated 11.5% more instances of 'irrational verbalisations', compared to occasional gamblers, despite no differences between the two groups in terms of winning more money. Examples of such irrational verbalisations would be "I have a knack for this machine" and "Everyone knows I can choose winning numbers!". Therefore, this shows that irrationality and selective attention are key to upholding cognitive biases/distortions and a deluded idea of expectancy.

— There are individual differences in terms of each gambler's cognitive distortions and particularly, as suggested by Burger and Norris (1985), the extent to which they believe they can control the events in their lives - this is a similar concept to locus of control. For example, those who believe they have a high degree of control may displace those feelings onto truly random events, from a statistical point of view, such as the lottery. Therefore, in an attempt to prove that they can still maintain this degree of control, they continue to partake in such gambling behaviours, which eventually leads to an addiction.

+ There is evidence to support the irrationality and impulsive nature of gamblers, as suggested by Michalczuk et al (2011). A group of 30 patients with gambling addictions were compared to a neurotypical control group of 30 others. The researchers found that the gamblers had an increasingly impulsive nature, often demanding instant gratification as opposed to waiting for smaller, but more likely rewards. These two traits suggests that, alongside the cognitive distortion of a deluded illusion of control, there is a cognitive element involved in gambling addictions and that it is emphasised through certain dispositional traits, such as impulsivity.

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+ The cognitive theory of gambling addictions has practical applications in terms of sparking new research into the biological/neurochemical basis of gambling (as suggested by Clark, 2010) and an improved system of CBT as a potential treatment for such addictions. For example, an improved understanding of the cognitive biases of gambling addictions, particularly the illusion of control and gambler's fallacy, would therefore be more easily identifiable by therapists during CBT, and such distorted beliefs could then be challenged as irrational. Therefore, cognitive theories have both practical and theoretical values.

Part 8 — Reducing Addiction: Behavioural Interventions:

- The two types of behavioural interventions are aversion therapy (a historical approach which is less regularly used today) and covert sensitisation (more commonly used today).
- Aversion theory is based upon classical conditioning and aims to counter-condition the individual, through repeated exposures between the addictive stimulus (e.g. alcohol) and unpleasant consequences (e.g. vomiting), so that the patient associates the two together (in line with the law of contiguity) and the link between the addictive substance and pleasure is broken.
- Aversion therapy is used for alcohol addictions. The patient would either be given an emetic after consuming alcohol, or a drug such as disulfiram. The emetic causes vomiting, whilst the disulfiram creates the immediate feeling of a hangover. Either way, the patient associates alcohol with these unpleasant consequences, and so their addiction undergoes extinction (in classical conditioning terms). The alcohol is the unconditioned stimulus, whilst the drug is the neutral stimulus (look back at Learning Theory!).
- Aversion therapy is also used for gambling addictions, where the patient would receive a two-second electric shock when reading out words associated with their addiction. The mechanism of this is similar as above.
- However, some patients may have pre-existing medical conditions which makes such therapy impossible, as well as raising ethical issues. For example, if a patient vomits at work, they may be given disciplinary action and must explain that they are undergoing treatment for an alcohol addiction, which may be considered embarrassing or even dehumanising.
- Therefore, covert sensitisation is considered a suitable substitute. This uses the same mechanisms as described above, but the patient simply imagines these negative consequences, as opposed to actually experiencing them. The descriptions of provoking situations must be especially graphic to induce feelings of disgust, such as involving the patient's pre-existing phobias, as suggested by McMurran (1994).

+ There is evidence to suggest that the modern methods of covert sensitisation may be a more effective treatment of gambling addictions, compared to the historical use of aversion therapy. For example, McConaghy et al (1983) found that covert sensitisation produced a 60% greater recovery rate compared to the use of traditional electrical aversion therapy, strongly suggesting that this should be the pioneering method in addiction treatments, especially considering the practical and ethical issues associated with aversion therapies.

— There are significant ethical issues associated with aversion therapies, and consequences for recovery rates. Essentially self-inflicting pain and nausea may be considered as breaching the BPS ethical guidelines of a participant's right to be protected both psychologically and physically. Such extremely unpleasant consequences may demotivate others from engaging or starting these therapies, thus doing little for their motivation in tackling their addiction.
— A lack of a double-blind procedure when evaluating studies of the effectiveness of aversion therapies allows for researcher bias when coming to conclusions, as has been argued by Hajek and Stead (2001) in their review of 25 aversion therapy studies for nicotine addictions. Due to the researchers knowing which groups were experimental or placebo, their preconceived ideas about the effectiveness of these therapies may have affected the validity and reliability of the conclusions they had drawn.

Part 9 — Reducing Addiction: Cognitive Behaviour Therapy (CBT):

- CBT assumes that faulty cognitive processing and cognitive biases are the key to a patient's addiction, and so must be challenged. This is possible through functional analysis (where the key risk factors for triggering addictive behaviours are identified) and skills acquisition (which acts as an alternative coping mechanism for the patients, apart from the addictive source).
- Functional analysis aims to identify the situations and stimuli which trigger the start of carrying out addictive behaviours, as well as considering the emotions felt before, during and after such

behaviours. For example, an alcoholic may admit that social situations in which there are many strangers, triggers their social anxiety and so leads them to drinking excessively in order to 'loosen up'. The key to this interaction is the quality of the patient-therapist relationship, which must be warm enough to build rapport whilst still maintaining a psychological distance so that the therapist can be firm in their own beliefs. The therapist will identify and challenge irrational beliefs or cognitive biases.

- Functional analysis is an ongoing process and is not restricted to the beginning of therapy, but is often revisited in the case of relapse.
- Skills training is made up of cognitive restructuring, specific skills and social skills.

1. The key to cognitive restructuring is the therapist explaining the irrationality of the patient's cognitive biases and offering other rational/logical alternatives. This may also be educational - in the example of a recovering alcoholic who drinks excessively to deal with their stress, the therapist may point out that alcohol only aggravates the body, weakening the immune system and making the patient more vulnerable to illness, which would only increase their stress further.
2. Specific skills involves the therapist and the patient making links between the patient's life problems and their current disorder, thus giving CBT the description of a 'broad-spectrum' treatment for this awareness. Anger management training, such as identifying the emotions which act as a precursor for aggression (Novaco et al), as well as assertiveness training (increasing social communication skills and realising that violence is not the single best way to resolve a conflict) are both examples.
3. Social skill development involves the therapist teaching the patient methods in which to ignore or reduce the impact of triggering stimuli. For example, in the case of a drug addict, the therapist may suggest that the patient avoids situations in which drugs are frequently used (e.g. parties) and come up with 'excuses' that could be used when being offered drugs (e.g. a lack of funds). The therapist will model such methods which the patient will copy, through role play and a 'directive' therapy.

+ There is research supporting the importance and effectiveness of a high-quality therapist-patient relationship. For example, Petry et al (2006) found that the experimental group of chronic gamblers (who attended Gambler's Anonymous meetings and had 8 CBT sessions) experienced a significant reduction in their gambling behaviours 1 year afterwards, compared to the control group (without CBT). However, critical to these improvements was the use of a therapist, as opposed to a book, in the CBT treatment. This not only supports CBT but also the use of a 'broad-spectrum' treatment of addictions, where problems in other areas of the patient's life appears to be a major contribution to the development of addiction.

+ CBT also takes a positive outlook on relapse, viewing it as inevitable in some patients and acknowledging it as an opportunity for further treatment e.g. a reconsideration of the original functional analysis and new cognitive restructuring. This may be considered as motivational for some patients, who would no longer see themselves as failures for relapsing, but positive in their efforts to change and opening an avenue for even more effective treatment methods.

— CBT is a cognitively demanding treatment, requiring addicts to face up to their addictions and combat them 'head-on', as opposed to other indirect therapies such as token economy systems. Therefore, attrition (drop-out) rates are 5 times higher in CBT compared to other therapies, as suggested by Cuijpers et al (2008) and are often triggered by crises in the lives of the patient subsiding, which may have been their original reason for undertaking therapy. Therefore, CBT may be most effective as an initial treatment, to be replaced at later stages of recovery by an alternative treatment.

Part 10 — Applying the Theory of Planned Behaviour to Addictive Behaviour:

- Ajzen (1985) suggested that addictive behaviours arise from having intentions to do so, in line with the Theory of Planned Behaviour (TPB). Our intention to do so depends upon our personal attitudes towards addiction, our perceived attitudes of others (favourable or unfavourable) and our preconceived ideas about our ability to tackle the addiction (e.g. based upon the resources we have available).
- Therefore, it should be possible to predict behaviour on the basis of known intentions, and this applies equally to addiction.
- Most addicts will have both favourable and unfavourable personal attitudes about their addictions, but balance these two sides to produce a general or overwhelmingly positive/

negative view. For example, an alcoholic will insist that alcohol is the only escape from their stressful life, but also acknowledge that the increasingly financial strain associated with supporting this addiction may add to their stress even further.

- A key element of an individual having the intention to embark or tackle an addiction would be the perceived social norms and favourable/unfavourable attitudes of significant peers in their life. For example, if a woman's partner disapproved of her drinking excessively on occasion and refused to help her financially to do so, the individual will experience a smaller intention or 'drive' to be entangled in an alcohol addiction. It is important to note, however, that our perceptions of the attitudes of our peers may not reflect their actual attitudes.
- Perceived behavioural control is based upon self-efficacy, which describes our own personal beliefs about how likely we are to tackle our addiction. This in turn depends upon the resources we have available (e.g. an Alcoholics Anonymous hotline, willpower) and can lead to indirect behavioural change (e.g. simply making preparations to change an addiction or evaluating the benefits and costs) or direct behavioural change (e.g. actively seeking out help).
- Therefore, according to TPB, the key factors associated with an individual's intentions (and hence the likelihood that they will experience addictions) depends on their own attitudes, the perceived attitudes of others and the self-efficacy or possibility of tackling the addiction. The emphasis here is on perceptions of others.

— The subjective nature of the TPB means that it is difficult to objectively assess and measure, as seen through its reliance on self-report measures. Despite perceived norms and attitudes being subjective concepts, as argued by Ogden (2003), there is the key problem of many addicts suffering from denial and demotivation, meaning that they are unlikely to be able to give accurate accounts of their own views and that of others. This is especially the case when considering that addiction is generally treated as a taboo subject and an undesirable characteristic, meaning that the results will be further skewed by social desirability bias (a type of demand characteristic). Finally, as argued by Sniehotta et al (2014), there is only correlational evidence of a link between intention and actual behaviour, meaning that the 'third factor problem' has not been considered and a 'cause and effect' relationship cannot be established.

+ There is evidence supporting the utility of TPB as an explanation for some types of addiction, and particularly alcohol-related disorders, as suggested by Hagger et al (2011). These researchers found that intended behaviours influenced the number of alcohol units individuals consumed, whilst perceived behavioural control accurately predicted actual alcohol consumption. This supports TPB's principles of personal attitudes, subjective norms and perceived behavioural control. However, Hagger also pointed out that TPB cannot explain all types of addiction or even all types of alcohol-related addictions, such as binge-drinking disorder. Therefore, TPB is best considered an effective explanation for some types of addictions, thus reducing its universality.

— TPB incorrectly makes the assumption that the formation of an intention to start an addiction is rational, and heavily involved with logical decision-making processes. This is not always the case, particularly with drug and alcohol abuse cases where there are other irrational contributory factors. These include past emotions, childhood abuse, associations between addictive stimuli and traumatic events and preconceived ideals about addiction. This reduces the validity of TPB as an explanation for addiction, due to not acknowledging the fact that addiction, by definition, is an irrational way of dealing with external pressures, even if not perceived in this way by the patient.

Part 11 — Applying Prochaska's Model of Behaviour Change to Addictive Behaviour:

- Prochaska and DiClemente (1983) proposed a cyclical model of behavioural change, where the duration and number of stages differs between person to person. This depends upon how ready the person is for change, and also determines the extent to which behavioural intervention treatments will be effective. The 6 stages are: pre-contemplation, contemplation, preparation, action, maintenance and termination.
- Stage 1 = Pre-contemplation = This occurs when the patient is thinking about change, but is not ready for it and so does not implement it. This may be due to denial (refusing that they have a problem) or demotivation (e.g. arising from previous failed attempts at stopping their addiction).
- Stage 2 = Contemplation = This occurs when the patient begins to evaluate the costs and benefits of tackling their addiction. Many people remain within the contemplation phase for

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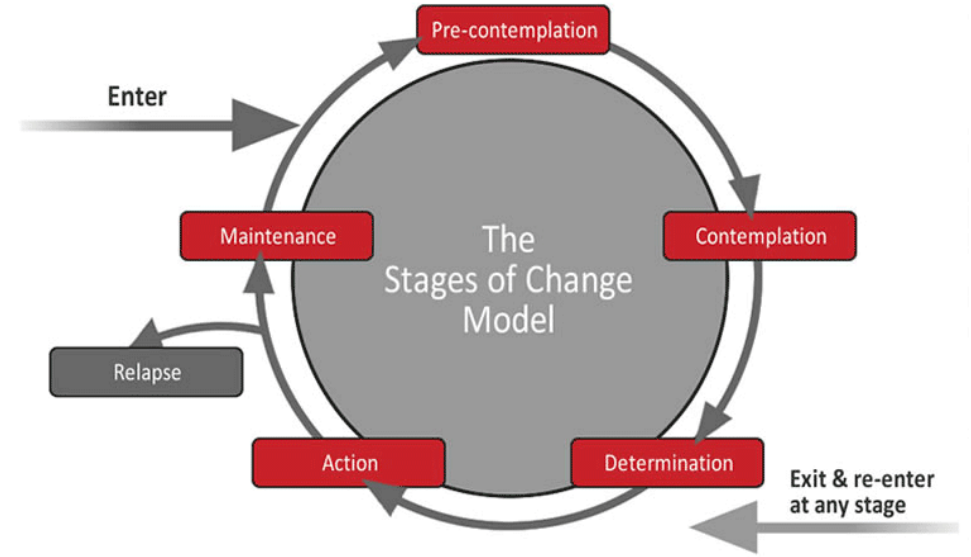

extended periods of time, and so behavioural intervention treatments applied here are not particularly effective.
•Stage 3 = Preparation = The patient has decided that they are ready for change because they believe that the benefits of tackling their addiction outweighs the cost of withdrawal symptoms, changing lifestyles etc. The patient may make a GP appointment or call a hotline e.g. Alcoholics Anonymous.

- Stage 4 = Action = The patient makes decisive actions about tackling their addiction, such as throwing away drugs or pouring away alcohol. These actions must reduce the risk posed by the original addiction e.g. pouring all the alcohol down the sink, as opposed to switching to alcohol-free wines or beers. Therefore, behavioural interventions are particularly effective when applied here.
- Stage 5 = Maintenance = The focus here is on preventing relapse, through providing the patients with coping mechanisms for loneliness, stress and withdrawal symptoms, other than the original addiction.
- Stage 6 = Termination = This occurs when the patient experiences no further reliance on their addictive substance or behavioural patterns. Since some patients may not be able to reach this stage, such as in the case of chronic drug abuse, the emphasis would be shifted upon reducing the likelihood of relapse and giving the patient skills to progress through the initial 4 stages more quickly.

+ Prochaska's six-stage model of addictive behaviour, as suggested by DiClemente et al (2004), takes a positive outlook on relapse. Whilst some other models, such as the theory of planned behaviour may view relapse as a failure, the six-stage model views relapse as a necessity, which reflects the dynamic and 'messy' nature of recovery from addiction. This, despite potentially underestimating the severity of relapse and the consequences in terms of the time needed to reach the maintenance or termination stages again, may support those entering recovery in reassuring them that this is a natural response to breaking addictions, rather than branding them as failures.

— However, there is evidence refuting the effectiveness or applications of the six-stage model. For example, Taylor et al (2006) and Cahill et al (2010) found that alternatives to tackling nicotine addictions were equally as effective as the stage approach to treating addictions, suggesting that Prochaska's model may have no real practical value. This was based upon reviews of the relevant studies carried out by NICE (the National Institute for Health and Care Excellence). Therefore, this suggests that the six-stage model has greater theoretical value, and so is limited in this sense.

+ As opposed to other models which explain addictions as being part of a static process with a set sequence of stages through which all patients progress (i.e. the theory of planned behaviour), the six-stage model offers a refreshing alternative. Prochaska's model acknowledges that addiction is a dynamic process, where stages can be 'recycled' or even omitted, depending upon the individual. This emphasis on time and the occasional inevitability of relapse means that such a model can 'tailor' its explanations for each individual addiction case!