Describe and evaluate the disease model of alcohol dependence

Addictions are complex behaviours. Most researchers believe that it is not only the action of the drug which plays a signification role in the addiction, but it is the result of the interaction of social, personal, family and lifestyle factors. However, as yet researchers still don't fully understand how these factors interact to create complex addictive behaviour. Hammersley (1999) suggests theories are largely concerned with the influence that biological factors have on dependence, and therefore see dependence on substances such as alcohol as a disease.

The 1800s first saw alcoholism conceptualised as a 'disease', with alcoholics being the victims as they passively succumbed to its addictive influence. This initial model of alcohol dependence as a disease regarded the substance as the problem and called for the treatment of excessive drinkers.

During the twentieth century, attitudes towards alcoholism changed, and the disease model was reviewed. The revised model no longer saw the substance as the problem but pointed the finger at those individuals who became addicted. Within this perspective, the small minority who consumed alcohol to excess were seen as having a problem, but for the rest of society alcohol consumption once more became an acceptable social habit. This perspective legitimised the sale of alcohol, recognising the resulting benefits to governments (increased revenue) and emphasised the treatment of addicted individuals. Alcoholism was regarded as an illness developed by certain individuals who therefore needed support and treatment. Thus 1935 saw the formation of Alcoholics Anonymous (AA).

AA sees alcoholism as an allergy, with people being physiologically susceptible to its effects. Based on his questionnaire of AA members, Jellinek (1946, 1952) proposed that alcoholism was a biological illness with a highly characteristic and predictable course comprising four major stages.

During the first stage, the 'pre-alcoholic' phase, alcohol provides the drinker with a means of reducing tension and increasing their self-confidence. However, during the second stage (the 'prodromal' phase), the alcoholic begins to drink heavily and in secret, and also experiences blackouts. The 'crucial' phase sees the alcoholic beginning to lose control and engage in benders, they also experience severe withdrawal symptoms. The final 'chronic' phase sees the alcoholic drink almost constantly and neglect almost all social and occupational responsibilities.

Jellinek (1960) also went on to identify five 'species' of alcoholics: the 'alpha' alcoholic who drinks to minimise tension; the 'beta' alcoholic who experiences physical damage, such as cirrhosis of the liver, from their drinking, but is not alcohol dependent; 'delta' alcoholics are unable to abstain from drinking; 'epsilon' alcoholics lose control of their drinking behaviour and go on periodic benders; finally, the 'gamma' alcoholic, who not only loses control of their drinking, but also experiences withdrawal symptoms – they are physically dependent.

This disease model of alcohol dependence was the single most influential theory for much of the twentieth century and is still the dominant view underlying many treatment programmes. However, Lilienfeld (1995) argues that alcoholism appears to be far more variable than this, and many drinkers don't comfortably fit in with Jellinek's theories. Peele (1989) highlighted six major assumptions made in this disease model of alcohol dependence. These include the

fact that alcoholics drink too much, not because they intend to, but because they can't control their drinking. Also, that alcoholics inherit their alcoholism and so are born as alcoholics. This assumption, according to Lilienfeld (1995), therefore implies that all individuals drink heavily for the same or similar reasons. Furthermore it was suggested that alcoholism is a disease that can strike any individual, from any socio-cultural background, and that alcoholism always gets worse without treatment; those alcoholics can therefore never cut back or quit on their own. Therefore treatment based on the AA principles can consequently be the only effective treatment. Finally, those who reject the AA principles or observers who reject any of the above are in denial.

However, Cloninger (1987) proposed that group 1 alcoholics are at risk from type 1 alcoholism; they drink primarily to reduce tension, are predominantly female, are prone to anxiety and depression, and tend to have relatively late onset of problem drinking. By contrast, group 2 alcoholics are at risk of type 2 alcoholism; they drink primarily to relieve boredom, to give free rein to their tendency towards risk-taking and sensation-seeking, are predominantly male, prone to antisocial and criminal behaviour, and tend to have relatively early onset of drinking behaviour.

Although the evidence for Cloninger's model is tentative and indirect, it challenges the disease model in a quite fundamental way. If he's correct, alcoholism may represent the culmination of two very different pathways (Lilienfeld, 1995).

Alcohol dependence syndrome (ADS: Edwards, 1986) is a later version of the disease model. It grew out of dissatisfaction with 'alcoholism' and with the traditional conception of alcoholism as a disease. 'Syndrome' adds flexibility, and suggests a group of concurrent behaviours that accompany alcohol dependence. They needn't always be observed in an individual, nor observed to the same degree in everyone. For example, instead of loss of control or inability to abstain, ADS describes 'impaired control'. This implies that people drink heavily because, at certain times and for a variety of psychological and physiological reasons, they choose not to exercise control (Lowe, 1995). Lowe maintains that:

> 'Simple disease models have now been largely replaced by a more complex set of working hypotheses based, not on irreversible physiological processes, but on learning and conditioning, motivation and self-regulation, expectations and attributions.'

Although many researchers still emphasise a disease model of addiction, there are several criticisms of this perspective. For example, the disease model encourages treatment of alcoholism through life-long abstinence, yet life-long abstinence is very rare and may be difficult to achieve. The model therefore fails to take relapse into consideration, and may inadvertently promote relapse by encouraging individuals to set unreasonable targets for abstinence. However, the description of controlled drinking (Davies, 1962; Sobel & Sobel, 1976, 1978), that suggests that alcoholics can return to normal drinking patterns, challenging the central ideas of the disease model. This model suggests that perhaps an addiction is not irreversible and that abstinence may not be the only treatment goal.