THE RELATIONSHIP BETWEEN ALCOHOLISM AND DEPRESSION

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Declaration

I declare that this thesis for the degree of Ph.D. has been composed solely by myself.

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ABSTRACT

The aim of this study was to investigate one aspect of heterogeneity in alcoholism, namely depression. The effect of a current diagnosis of depression on drinking outcome was examined in a random sample of 82 male and female alcoholics attending an in-patient alcoholism treatment unit. Seventy-four alcoholics were followed-up over a five month period following discharge from hospital. In addition, the relationship between drinking outcome, depressive symptomatology and cognitive measures known to be associated with depression, was explored.

Diagnosis of depression was found to vary with drinking status: in the episode of drinking which led to admission, 67% of the sample met Research Diagnostic Criteria for major depression whereas only 13% met diagnostic criteria for major depression after detoxification from alcohol. Those with a diagnosis of depression after detoxification were more likely to have received treatment for depression, both during their in-patient stay and during follow-up than those with alcoholism alone. Nonetheless, those with an additional diagnosis of depression did not differ in drinking outcome from those with a diagnosis of alcoholism alone.

Alcoholics who remained depressed after detoxification reported higher levels of hopelessness and frequency of negative thinking than non-depressed alcoholics. Depressed alcoholics were not differentiated, however, from their non-depressed counterparts on cognitive measures of dysfunctional attitudes, negative cognitive style and self-control. At first follow-up, those who were drinking alcohol, regardless of diagnosis, experienced greater hopelessness

and frequency of negative thinking than those who were abstinent. By second follow-up, however, no differences were found between the groups in affective symptomatology and cognitive measures.

The findings are discussed with reference to other studies which have examined outcome for depressed and non-depressed alcoholics. One implication of the findings is that measures of affective symptomatology and cognitive dysfunction are useful in monitoring changes across time and in exploring the characteristics of depression found in alcoholics. The results imply that depressive symptoms and dysfunctional cognitions follow, rather than precede, excessive alcohol consumption.

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INTRODUCTION

In 1985, the investigator began working with individuals with alcohol problems. As a practitioner of Beck's cognitive therapy (Beck et al., 1979), she was struck by the depressive content of many of her patients' thinking. A brief perusal of the literature led her to believe that the depressive content of thought was not commonly noted among problem drinkers, although there was some attention paid to depressive disorder in the literature on alcoholism. At that time, the effect of depression on drinking outcome was largely unknown. Having made these observations, the study presented here was undertaken.

No single method of treatment has been shown to be optimal in the treatment of alcoholism. One possible explanation for this may be that alcoholics vary in the degree to which they resemble each other and that these differences have not been taken into account in the majority of treatment studies. Additional psychopathology and more specifically depression, both as a diagnostic category and at a symptomatic level, is recognised as being commonly found in samples of alcoholics (Peace and Mellsop, 1987). Depression may therefore be regarded as one dimension on which alcoholics will vary.

If depression was found to influence drinking outcome, this would have implications for the treatment of depression among alcoholics. The effect of depression on the course of alcoholism and on outcome of treatment are active areas of study. Research has tended to concentrate on the influence of lifetime, primary or secondary diagnosis of depression on the course and

outcome of alcoholism and has largely ignored the influence of a current diagnosis of depression on treatment outcome. In addition, the relationship between a diagnosis of alcoholism, in combination with depressive symptoms and dysfunctional thinking has not been explored.

Depression is known to respond to pharmacotherapy and to psychotherapeutic treatments such as Beck's cognitive therapy (Beck et al,. 1979; Blackburn and Davidson, 1990). A diagnosis of depression is known to be associated with dysfunctional thinking and dysphoric mood. Resolution of depression brings about changes in mood and also changes in dysfunctional cognitions (Simons et al., 1984). Depression in alcoholism has not been subject to an investigation of the psychological characteristics of depression which arise from Beck's cognitive model (Beck, 1987). It is possible that the negative cognitive style which is found to be characteristic of primary unipolar depressives may also be characteristic of those alcoholics who have a co-existing diagnosis of depression. An investigation of the psychological characteristics of depression in alcoholism would therefore seem justified on the grounds that the findings may prove useful in matching specific characteristics of those dependent on alcohol with treatment, such as cognitive therapy, which aims to change depressive symptoms and negative thinking.

The present study therefore aims to explore the relationship between a diagnosis of depression, depressive symptomatology, and outcome of treatment in alcoholism and to investigate the cognitive characteristics of depression in alcoholics.

Incorporated into the design of the study are specific measures of cognitive dysfunction which have arisen from Beck's theory of depression and have been thought to be indicative of depressive thinking (Blackburn, 1988). The study of these measures in relation to a diagnosis of depression in alcoholics and in relation to drinking outcome will add to our understanding of the nature of depression as a coexisting diagnosis in alcoholism.

Plan of the thesis

The literature review covers those areas of research which are particularly relevant to the present thesis. The aims of the first chapter are several: to indicate the extent to which alcoholism is a problem, the efficacy of treatment for alcoholism, to give an account of those studies which have examined the prevalence of depression among samples of alcoholics and those which have examined the influence of depression on drinking outcome. Lastly, some potential aetiological theories which link alcoholism and depression are reviewed. Chapter two contains a review of Beck's cognitive theory of depression. Attention is drawn to the literature on the content of depressive thinking.

Chapter three outlines the rationale for the study, the main hypothesis and the design of the study. The measures used in the study are delineated in this chapter. The results arising from the study are tabulated and summarised in Chapter four.

The final chapter provides a discussion of the findings, drawing on relevant literature from cognitive theory and the field of alcohol studies.

Note: The diagnostic label alcoholism is used in this thesis as this is the term used in Research Diagnostic Criteria (Spitzer et al,. 1975).

CHAPTER ONE

LITERATURE REVIEW

1.1 THE DEFINITION OF ALCOHOLISM

In most societies, drinking alcohol is essentially a social act. As such, for the majority of people, the consumption of alcohol is non-problematic. For some, however, the consumption of alcohol does give rise to problems both for the individual concerned and for others. There is obviously a variety of ways in which alcoholism has been described and defined throughout this century and the recent past has not been devoid of changing definitions.

The modern conception of addiction was initially advocated by Dr Benjamin Rush (Levine, 1978), a physician regarded as being the founder of the Temperance Movement in America. Rush perceived an addiction to spirits as a "disease of the will". Once an appetite or "craving" for spirits had developed, the drinker was regarded as powerless to resist the impulse to drink. The drinker was then regarded as having lost control over his drinking. The cure was seen as abstinence from spirits. In Britain, around the same time as Rush, the Edinburgh physician, Thomas Trotter, published an influential essay on drunkenness (Heather and Robertson, 1989). Trotter considered habitual drunkenness as a disease of the mind which disordered the body. He, like Rush, recommended total abstinence from all alcohol as treatment for the disease.

The main characteristic of the modern view of alcoholism, loss of control over drinking, was evident in

both Rush's and Trotter's concept of addiction to alcohol.

Levine (1978) argues that the aim of temperance, to reduce habitual drunkenness, and the view of physicians that habitual excessive drinking was an addiction, were essentially complementary to one another. However, the Temperance Movement in America and also to an extent in Britain, changed from being reformatory and educational, to became increasingly coercive in the late nineteenth century. The prohibition of alcohol which resulted from the Temperance Movement in America was maintained until 1932.

From the end of Prohibition in the United States until the mid-seventies, the dominant conception of alcoholism was that described by Jellinek (Jellinek, 1952).

1.1.1 Jellinek's concept of alcohol addiction

On the basis of a questionnaire study, Jellinek (Jellinek, 1952) described a disease concept of alcohol addiction. He differentiated between "alcohol addicts" and "habitual symptomatic excessive drinkers". Only the former group were considered to be suffering from a disease which was characterised by "loss of control" over the intake of alcohol.

Jellinek outlined phases of alcohol addiction whereby an individual would initially use alcohol for social purposes but then found that alcohol relieved tensions and would increasingly seek out drinking situations. After some time, an increase in tolerance to alcohol would develop and the drinker would require more alcohol than he did previously to achieve the same level of "sedation". At this point, Jellinek believed that a

drinker entered another phase of drinking, the prodromal phase. This phase was marked by alcohol induced "blackouts" and the drinker's behaviour changed to ensure that a supply of alcohol was available. Surreptitious drinking, a preoccupation with drinking, gulping the first few drinks and a subjective awareness that his drinking may be abnormal were the behaviours characteristic of this phase. This acute phase bore the hallmark of the disease process: loss of control over the intake of alcohol which was felt by the drinker as a "physical demand" for alcohol once any alcohol had been taken. At this stage, the drinker was described as feeling remorseful and experiencing a loss of self-esteem and the drinker's behaviour was described as deteriorating in that his ability to function socially and occupationally became markedly impaired. Outside interests became less important and the drinker's entire behaviour became centred on drinking. The description of this phase resembles that of a depressive state in which an individual is characterised by low self-esteem, has feelings of guilt and is impaired in social and occupational functioning.

Abstinence and changes in drinking patterns, including morning drinking were regarded as being commonplace in this phase which then developed into a chronic phase marked by prolonged periods of intoxication, impairment in thinking, alcoholic psychosis and sometimes a loss of tolerance to alcohol.

Jellinek emphasised that only the prealcoholic phase was the same for non-addicted alcoholics and addicts. Those who were not alcohol addicts did not experience loss of control over their intake of alcohol. He

suggested that those who become alcohol addicts may have a predisposition to becoming addicted - that they may have a factor "X" which was not present in other alcoholics. However, he was cautious about this as an explanation and pointed out that even those who are not addicted can develop serious physical and social problems as a result of their drinking.

In 1960, Jellinek published his second major contribution, "The Disease Concept of Alcoholism". This publication reflected his experience acquired internationally and with the World Health Organization. In it, he proposed a broad definition of alcoholism as "any use of alcoholic beverages that causes any damage to the individual or society or both". He made a distinction between alcoholism and alcoholics and identified five distinct "species" of alcoholism which he referred to by letters of the Greek alphabet. Alcoholics were not the same as those who suffered from alcoholism in the broad sense which he had described, but were confined to those who fitted the description of the species of alcoholism which he called diseases.

Alpha alcoholism was represented by a psychological dependence on the effects of alcohol to "relieve" bodily or emotional pain. This kind of alcoholism was the cause of disturbed interpersonal relationships. Jellinek did not regard this species of alcoholism as a disease but rather as a symptom of some underlying psychological, but unspecified, disturbance.

Beta alcoholism was associated with physical damage such as polyneuropathy, gastritis and cirrhosis of the liver, resulting from poor nutritional habits and drinking alcohol. As withdrawal symptoms were not

considered to be present in this species of alcoholism, it was not considered a disease.

Gamma and delta alcoholism, on the other hand, were regarded as being diseases. Gamma alcoholics were considered to be physically dependent on alcohol. In his earlier work, he had described gamma alcoholism as alcohol addiction. The characteristics of physical dependency were an acquired increase tissue tolerance to alcohol, adaptive cell metabolism, withdrawal symptoms and "craving" for alcohol. In these alcoholics, there was a loss of control over the intake of alcohol.

Jellinek considered this kind of alcoholism to be the predominant species in North America and in other Anglo-Saxon countries. Delta alcoholism was similar to gamma alcoholism in that it shared the first three features of gamma alcoholism but instead of loss of control, there was an inability to abstain from alcohol for any length of time without the occurrence of withdrawal symptoms. This kind of alcoholism was seen as being predominant in wine-growing countries such as France.

The last species which Jellinek identified was that of epsilon alcoholism. This was periodic alcoholism, known as "dipsomania" in Europe and Latin America but there was little known about this kind of alcoholism.

Jellinek only regarded gamma and delta alcoholism as diseases as they alone were said to involve the "physiopathological changes" comparable to those found in drug addiction. These changes were regarded as being responsible for craving and loss of control.

Since Jellinek, there have been several other definitions of alcoholism. For example, some have argued

that alcoholism is a single disease entity that has an identifiable history, symptoms and signs which form a recognisable pattern (Madsen, 1974; Johnson, 1973). Others have proposed that alcoholism cannot be defined as a single entity but only as a collection of various symptoms and behaviours that together comprise different types of syndromes (Pattison et al, 1977). These authors have emphasised the diversity of factors which may lead to problem drinking: socio-cultural influences and intra-psychic factors which lead an individual to use alcohol inappropriately. They also point to the difficulty of differentiating between alcoholics and non-alcoholics and suggest that such a distinction is unwarranted.

1.1.2 The Alcohol Dependence Syndrome

The Committee on Alcohol-Related Disabilities of the World Health Organisation endorsed the "alcohol dependence syndrome" to clarify their position that alcohol addiction was one of a family of dependence disorders (Mandell, 1983). Based on the work of Edwards and Gross (1976), the essential elements of the syndrome were seen as being a narrowing of the drinking repertoire, salience of drink seeking behaviour, increased tolerance to alcohol, repeated withdrawal symptoms, repeated relief or avoidance of withdrawal symptoms by further drinking, subjective awareness of a compulsion to drink and reinstatement of the syndrome after abstinence.

Edwards and Gross did not specify which of these elements should be present in order to meet criteria for alcohol dependence but emphasised that they would tend to cluster together and that any element, if present, could vary in its intensity. They thought that reinstatement

of tolerance after abstinence and withdrawal symptoms were indicative of a biologically determined change in response to alcohol but also that learning factors would play a significant role in the development of the dependence. Dependence on alcohol was seen as a continuum rather than Jellinek's all or none concept of alcoholism.

The alcohol dependence syndrome has stimulated much interest. A debate surrounding both the nature and definition of this phenomenon has been one focus of attention in the British literature on alcohol problems (see Heather et al. 1985, Chick, 1980(a)).

There is currently no widespread agreement in the definition of alcoholism. Currently, the two most influential diagnostic systems, DSM-III (American Psychiatric Association, 1980) and ICD 9 (World Health Organization, 1980) do not share exactly the same definition of alcohol dependence (Caetano, 1987). DSM-III incorporated the recommendation of the WHO committees that the term addiction should be replaced by alcohol dependence (Klerman, 1990). Alcohol dependence was differentiated from alcohol abuse in DSM-III, with alcohol dependence being characterised by excessive use of alcohol associated with impairment in social, psychological functioning as well as in physical health.

In ICD 9, the term alcoholism was removed and the concept of alcohol dependence syndrome (ADS) was introduced instead. A distinction is made in ICD 9 between alcohol dependence and alcohol-related disabilities. Alcohol-related disabilities were physical, mental and social dysfunctions in which the use of alcohol was implicated.

DSM-III by contrast, contained a combination of Feighner's diagnostic criteria for use in psychiatric research (Feighner et al., 1972) which were then published in a slightly modified form as the Research Diagnostic Criteria (RDC) (Spitzer et al., 1975). The criteria for alcoholism had originally been formulated and modified by Guze (Guze et al., 1969). Guze proposed that a diagnosis of alcoholism be made on the existence of problems in at least three of five symptom groups. The groups were physical consequences, pathological drinking behaviour, frequent or heavy drinking on a daily basis, impairment in social or occupational functioning and a subjective evaluation either by the patient or another that the patient was alcoholic. All of these, except the measure of daily consumption, were incorporated into Feighner's criteria and then with minor alterations incorporated into RDC.

The main differences between ICD 9 and DSM-III are twofold: in ICD 9, the alcohol dependence syndrome was differentiated from alcohol-related disabilities whereas in DSM-III, alcohol-related disabilities could form part of the definition of dependence. Secondly, in DSM-III, alcohol dependence and abuse were both regarded as discrete categorical diagnosis whereas ICD 9, alcohol dependence is regarded as a dimensional construct.

More recently, there has been a convergence of views in that both DSM-III-R (American Psychiatric Association, 1987) and the planned ICD 10 define alcohol dependence based on the elements of the alcohol dependence syndrome proposed by Edwards and Gross (1976). Both diagnostic classifications use multiple criteria to define disorders of alcohol use. It also appears that the proposed

definitions of alcohol dependence in ICD 10 and DSM-IV may be more closely related to each other than in the previous versions of these diagnostic criteria (U.S. Department of Health and Human Services, 1990) although appeals are still been made for greater uniformity in classification (Caetano, 1987).

Attempts are being made to bring a greater degree of consensus in the definition of alcohol use disorders and it seems that the alcohol dependence syndrome is gaining wider and more uniform acceptance than other definitions. The literature however will reflect the variety of definitions and diagnostic practices that have prevailed and may therefore lead to differences in studies which use different criteria for describing alcoholism.

1.2 THE PREVALENCE OF ALCOHOLISM

Although the present study is based on a clinical sample of alcoholics, it is appropriate to describe briefly the extent to which the consumption of alcohol and alcohol related problems exist in the community.

Drinking habits and attitudes towards drinking have been found to be culture bound (Heath, 1987). As a result, there will be differences in the levels of drinking and in the association between drinking and alcohol related problems, including the prevalence of alcoholism in any particular society. Several factors are known to modify the consumption of alcohol. These are the availability and the price of alcohol, the current social pressures and the existence of educational and moral campaigns. Amongst these, economic factors are recognised as being of particular importance as consumption of alcohol has been shown to be closely related to the rise and fall in the general trade of a country. In the United Kingdom, for example, consumption of alcohol fell during economic depressions in the nineteenth century but increased at times of economic growth (Spring and Buss, 1977). Although alcoholic beverages were consumed in greater quantities in the eighteenth and nineteenth than they have been in the twentieth century, there have been fluctuations in the overall estimates of consumption of alcohol in this century. For example, per capita consumption of alcohol (in litres of pure alcohol) in the United Kingdom in 1970 was 7.02 and rose to 9.11 by 1985 (N.T.C Publications, 1990. p17). Similar changes were seen in other countries such as the United States and Italy (Horgan et al, 1986).

For the purposes of this study it is appropriate to outline those studies which specifically attempt to estimate the levels of drinking and alcoholism in Scotland and in Great Britain. This will give an indication of the extent of problematic drinking and alcoholism.

1.2.1 Surveys of the General Population

The results of a number of surveys on the prevalence of drinking in the United Kingdom are summarised below. One survey, commissioned by the Scottish Home and Health Department, of the drinking habits of the Scottish population took place in 1972 (Dight, 1976) took a representative sample of the Scottish population. Additional surveys of the prevalence of drinking in Scotland are provided by Plant and Pirie (1979), who surveyed the drinking habits of a sample of the population of four Scottish towns and Ritson (1985) who surveyed drinking patterns in Lothian as part of a World Health Organisation collaborative study on community responses to alcohol related problems. Wilson (1980) and Dunbar and Morgan (1987) provide results about drinking patterns in England and Wales. Crawford et al. (1985) examined self-reported alcohol consumption and alcohol related problems in the general population in three areas of Britain: the Highland and Tayside Regions in Scotland and East Kent in the South of England.

Overall consumption of alcohol in the general population was found to differ for men and women and varied from 15.3 units of alcohol to 21.6 units per week for men and between five and seven units for women (Dunbar and Morgan, 1987; Ritson, 1985).

Regular drinkers were found to be predominantly male (Dight, 1976; Plant and Pirie, 1979; Ritson, 1985), young (Dight, 1976; Ritson, 1985), single and more likely to have friends who also drank regularly (Dight, 1976). Social class was not found to be a discriminating variable for men who were regular drinkers but for women, there was a higher prevalence of regular drinking in social class 1 than in other social classes (Dight, 1976).

According to Dight (1976), six percent, all male, of the total population were heavy drinkers. Wilson (1980) found that six percent of males and one percent of women were heavy drinkers. Single men were most likely to be heavy drinkers (Wilson, 1980). Heavier drinking was associated with young males (Plant and Pirie, 1979; Wilson, 1980), specifically those between 17 and 30 (Dight, 1976).

Occupation was also associated with heavy drinking. Men, in manual occupations were more likely to be heavy drinkers (Plant and Pirie, 1979), but were found to drink less often but more heavily than non-manual workers (Ritson, 1985). More specifically, unemployed men and single men in the construction and drinks industry were likely to be heavy drinkers (Wilson, 1980). However, socially advantaged women (Ritson, 1985) and employed females with no children (Wilson, 1980) were found to drink heavily.

Generally agreed guidelines for "low risk" drinking have been less than 21 units of alcohol for men and less than 14 units for women, where one unit of alcohol is approximately half a pint of ordinary strength beer or a glass of wine (The Faculty of Public Health Medicine,

1991; Royal College of Psychiatrists, 1986). Drinking above these levels is associated with increasing risk of harm. Wilson (1980) identified problem drinkers as those drinkers who experienced two or more physical dependence or psychological drinking problems in the three months prior to the interview. Approximately five percent of men and two per cent of women were identified as being problem drinkers. However, when people who had reported problems with drinking regardless of their weekly consumption were included along with those who were drinking more than the recommended safe limits, 14% of men and three per cent of women were regarded as being heavy drinkers.

Ritson (1985) also enquired about personal and social problems associated with drinking during the year prior to the interview. Men had experienced more problems of both types than had women and as levels of alcohol consumption rose, more problems were encountered and more frequently.

The above studies used self-reported alcohol consumption to assess the prevalence of drinking. Crawford et al (1985) compared self-reported alcohol consumption to official rates of hospital admissions for alcohol dependence. Hospital admissions were different in the three areas and did not relate to self-reported drinking rates in the separate populations studied. For example, the Highland region had the highest official rate of alcohol related problems but it was found that Highlanders were more likely to be abstainers, consumed the lowest amount of alcohol per drinker in the previous week and were more likely to be within the lowest alcohol consumption group compared to respondents from other

areas. However, the male Highlanders experienced the highest number of adverse consequences of drinking over the previous two years compared with those in Tayside and Kent.

The authors considered several explanations for the lack of concordance between the official statistics and self-reported drinking. Amongst these explanations were a relatively poor response rate which may have led to biased samples, the under-reporting of consumption and the possibility that a binge pattern of drinking might lead to more adverse consequences of alcohol rather than total alcohol consumption per se. The most likely explanation however, came from a related study. Latcham et al (1984) found that the pattern of services for alcohol related disorders vary across regions. Disparity in official statistics such as psychiatric in-patient admissions for alcohol dependence, alcohol psychosis and alcohol abuse were due to admission policies which in turn reflected the available services in a region.

Whereas the above provides a cross sectional picture of drinking patterns in the general population, Dunbar and Morgan (1987) provided a longitudinal view by comparing their survey of the adult population of England and Wales with that of Wilson (1980). No significant differences in non-drinkers or in consumption were found between 1978 and 1985, the year of the survey. However, the proportion of women who were non-drinkers was significantly greater in 1985, indicating that of women who drank alcohol, there was an increase in consumption. Also, for men aged between 18-24 there was a decrease in the level of consumption from 1978 to 1985, with a shift from heavy to moderate and light drinking. Media

campaigns aimed at moderate drinking and the rise in unemployment were proposed as having influenced this decrease in drinking in young men though this explanation does not account for an increase in women's consumption.

American investigators have followed-up individuals at more than one point in time to investigate changes in drinking patterns within individuals rather than in a population. Cahalan and Room (1974) in two waves of a national survey of the United States in 1967 and 1969 interviewed 1561 male respondents between the ages of 21 and 59 on two occasions. The information from these surveys was supplemented by a more qualitative survey of men aged 21 to 59 living in San Fransisco during 1967/68. The overall findings were that all drinking problems show their highest prevalence to varying extents in young men aged 21 to 24. Men of other ages may have drinking problems but in general the proportion experiencing no problems or minimal problems remains fairly constant for all other ages. Being unmarried, regardless of age, belonging to a disadvantaged ethnic group and having low socio-economic status was associated with excessive drinking and higher rates of drinking problems. Drinking problems were also found to increase or decrease in relation to environmental changes such as marital satisfaction, marital status and the death of a parent or child as well as the frequency with which alcohol was available when in the company of friends (Cahalan, 1970).

In 1972, 615 of the respondents from the San Fransisco survey were recontacted (Clark and Cahalan, 1976). Of the men who had drinking problems at the time of the first interview, very few had the same problem or problems four years later. It was found that those who

had a problem with alcohol at the time of the first interview were likely to have continued having a problem but the continuity of specific problems was unlikely, indicating that drinking problems were neither a static or an inevitably worsening phenomena which progressed from less to more severe problems over time.

Another follow-up of the same population, surveyed after a 12 and 15 year interval, found that the incidence of heavy drinking and alcohol problems decreased with age (Fillmore, 1987(a)). There was evidence however that the chronicity of drinking problems was highest in the middle years. A cohort analysis indicated that except for variation in per capita consumption of alcohol, membership of a particular cohort did not effect these age specific findings for men nor could the findings be attributed to unique historical events or being a certain age at particular points in time.

It is evident from the surveys carried out both in Great Britain and in the United States that drinking problems are more prevalent in those who are young, male, unmarried, and of lower socio-economic status compared to those who do not have these specific demographic characteristics. Women, in general population surveys, are found to drink less than men, abstain from alcohol more than men, and the highest prevalence of frequent heavy drinking occurs in the middle years of life (Fillmore, 1987(b)) whereas for men this occurs in the early adult years. Once drinking problems have occurred, however, they tend to become more chronic in the middle years of life.

1.2.2 Clinical Populations

The problems associated with alcohol dependence and the number of individuals who suffer from a dependence on alcohol represent only a proportion of the total population whose lives are in some way limited or harmed by their use of alcohol. The use and misuse of alcohol is acknowledged to be associated with a wide array of social, psychological and physical problems (Royal College of Psychiatrists, 1986, Institute of Medicine, 1989). The adverse effects of alcohol in England and Wales have been estimated as costing in the region of £1500 million annually, based on 1983 prices (McDonnell and Maynard, 1985). The information on which this costing was based came from estimates of the cost to industry from loss of production through alcohol, the direct and indirect cost to the National Health Service, the use of services for problem drinkers, the cost of alcohol associated road traffic accidents and that of alcohol related criminal activity. Any such costing will be heavily dependent on the reliability of prevalence estimates and as such it is likely that the figure arrived at represents an under-estimate of the total cost of alcohol use and misuse.

The present study concentrates on those who are dependent on alcohol and who are receiving in-patient treatment in a psychiatric hospital. Rates for first time admissions to psychiatric hospitals for alcoholism and alcoholic psychosis vary greatly for different Health Boards and Districts throughout Britain. In 1976, the rate of admission for Scotland as a whole was 36.1 per 100,000, a figure four times higher than the equivalent rate for England and Wales (Kilich and Plant, 1981).

This rate of first time admissions to psychiatric hospitals is however known to fluctuate and during the seven year period from 1970, for example, was shown to rise in Scotland by 67% (Davies, 1982). An increase in the proportion of women admitted to psychiatric hospitals for alcohol related disorders was particularly noted during this time by Davies who points to an increase from 19% to 27% for the years 1970 to 1979. More recent Scottish figures indicate that for the years 1984 to 1986, the number of first admissions to psychiatric hospitals for alcohol dependence and alcoholic psychosis have in general decreased from 1970's levels but female admissions had risen to approximately one third of first admissions by 1986 (Scottish Health Statistics, 1988).

It is possible that individuals who are treated for alcohol dependence or alcoholic psychosis as in-patients in a psychiatric hospital constitute a separate group of those dependent on alcohol. They are recognised as being dependent on alcohol by their general practitioners and have either themselves sought and been offered treatment or have done so through their general practitioners. It is likely that there are others in the community who are alcohol dependent and either do not recognise that they have an alcohol problem or do not seek treatment or seek counselling through other agencies such as Alcoholics Anonymous or Councils on Alcohol. It is also thought that general practitioners identify only a proportion of their patients who have alcohol problems (Shaw et al, 1978) and thus these individuals will not be referred to hospital based treatment services and will not appear in official statistics.

In the clinic on which the present study is based, patients with alcohol problems are more likely to be treated as out-patients than as in-patient though some 30% will receive in-patient treatment at some time over the course of their contact with the treatment service (Ritson, 1990). Services, such as the one on which this study is based, therefore provide treatment for alcohol dependent patients on an outpatient as well as in-patient basis. The criteria applied to patients who are selected for in-patient treatment as opposed to outpatient treatment is likely to vary from service to service. Although these criteria are difficult to define and may be as motivated by "clinical hunches", they are likely to include severe withdrawal symptoms, underlying personality or neurotic difficulties which require further investigation, a lack of confidence and coping skills and family conflict which cannot be easily resolved or assessed as an out-patient (Ritson, 1990).

1.3 OUTCOME OF TREATMENT: GENERAL FINDINGS

The literature on research into the predictors of response to treatment is extensive and it is not possible here to review in detail all aspects of outcome research. Instead some general findings will be discussed.

Studies of the outcome of treatment have suffered from a diversity of diagnostic practices, difficulties in establishing suitable outcome measures, and from lack of homogeneity in the populations studied. Comparisons between studies are difficult to make as studies have also been hampered by the inadequacy of controlled studies of the efficacy of treatment and by the lack of knowledge of the natural course of alcohol dependence where as many as one fifth of alcohol dependent individuals may become abstinent as a relatively stable outcome without formal treatment (Schuckit, 1984). Also, as Kendell and Staton (1966) pointed out, individuals may seek other or multiple treatments during a follow-up period. It is therefore difficult to make a reliable estimate of the effect of treatment on outcome, particularly after a substantial period of time, as the treatment intervention may only be one of several factors which have an effect on an individual's life and their drinking in particular.

One major American study which examined outcome in 922 male patients who had attended any one of eight alcohol treatment centres in 1973, found that outcome at four years was dependent on the severity of alcohol dependence at entry into treatment (Polich et al, 1980). At four years follow-up, 54% of the sample followed-up were drinking with problems, described as either exhibiting symptoms of dependency on alcohol or

experiencing severe adverse consequences of drinking. Of the 46% in remission, 28% had abstained throughout the six months prior to the follow-up interview and 18% were drinking without problems. Those male alcoholics who were over the age of 40 and who were severely dependent on alcohol at admission into treatment were found to have a generally poor prognosis if they had not remained abstinent compared to those who were younger and less severely dependent on alcohol. The level of consumption of alcohol during follow-up did not in general affect prognosis if an individual was not dependent on alcohol. Those in whom abstinence was unstable and short term were more likely to experience serious episodes of drinking compared to those who abstained long term and who had the lowest rates of alcohol problems at follow-up.

On entry into treatment, this sample was severely impaired compared to the general population on indices of social and economic adjustment. Over one third were either divorced or separated and almost two thirds were unemployed. In spite of 48% of the sample being in remission at follow-up, levels of social and economic adjustment had remained poor over this period.

Psychological and psychiatric dysfunction were also found to be higher than that expected in the general population. Although long term abstainers were found to have the best psychological functioning and mental health, the rates of symptoms of depression, anxiety and general dissatisfaction with life were higher in all groups than those found in the general population. Eighty five per cent of the original cohort were followed-up. A substantial number of the original cohort had died (14.5%) in the intervening four years:

approximately two and a half times the expected rate for the general population, adjusting for age and race. Approximately half of these deaths were attributable to alcohol and included amongst the reasons for death were suicide, death due to liver disease, accidents and chronic alcoholism. Interestingly, the initial findings of the Rand Report were that approximately two thirds of this group of patients were improved at 18 months follow-up (Armor et al., 1978) indicating that the length of follow-up and the method of ascertaining improvement influences the results of outcome studies.

Gibbs and Flanagan (1977) reviewed 45 published studies on the outcome of treatment. They found that there was considerable difficulty in reaching conclusions from the sample of the literature selected as differences in operational criteria for both predictor variables and the measurement of outcome had been used by investigators. They concluded that in the studies analysed, ranging in their follow-up periods from one month to ten years, general and stable predictors of outcome were elusive.

One long term follow-up study of married male alcoholics who had received either treatment or "advice" 10 to 12 years previously (Duckitt et al., 1985) emphasised that outcome cannot be measured simply in terms of abstinence or drinking but should instead reflect the "process of outcome" as accurately as possible, whilst also doing justice to analysis and summary. Follow-up revealed that considerable variation in drinking behaviour had taken place and that no one-to-one relationship exists between drinking behaviour per se and social adjustment and mental health. Of the 68 men

interviewed from the original sample of 99, approximately one-third of ex-patients had experienced episodes of depression, suicide attempts and/or drug abuse over the course of follow-up.

One general and reasonably consistent finding is that outcome is poorest for male alcoholics, who are over the age of 40, lacking in social supports and severely dependent on alcohol.

1.3.1 Specific treatments for alcoholism

The literature on the efficacy of treatment for alcoholism is extensive. It encompasses studies which have investigated the efficacy of specific treatments, the search for predictors of good and poor outcome, short and long-term follow-up of patients treated for alcoholism. A comprehensive review of all the literature will not be attempted here. Rather, the efficacy of common psychological and pharmacotherapeutic treatments for alcoholism will be presented.

1.3.1 a *Pharmacotherapeutic Interventions*

Three main types of drugs are used in the treatment of alcoholism: alcohol-sensitising drugs, drugs which are intended to suppress alcohol consumption and psychotropics. Drugs are also used in the management of acute withdrawal states but these will not be mentioned here. Drugs such as fluoxetine and fluvoxamine are thought to reduce the reinforcing properties of alcohol (Institute of Medicine, 1989). Research into the efficacy of these serotonin uptake inhibitors in alcoholism is in its infancy and will not be reviewed here.

Alcohol-sensitising drugs

In Britain and in the United States, the most commonly used alcohol-sensitising drug is disulfiram (Antabuse). Disulfiram, when taken in regular doses, produces an adverse reaction on consumption of alcohol. It can produce a reaction of varying degrees of severity when alcohol is consumed and when used correctly can have relatively minor side effects (Schuckit, 1985).

Disulfiram is used to provide help to individuals who wish to maintain abstinence and acts as a deterrent for individuals who may be tempted to drink on the spur of the moment. Many patients will refuse disulfiram or fail to comply with the treatment regimen (Peachey, 1981). There is evidence that those who agree to take it and who comply with medication believe that disulfiram will be an effective means of achieving abstinence and minimise the possible problems associated with its use compared to rejectors of disulfiram (Brubaker et al., 1987).

Controlled studies of the oral administration of disulfiram to alcoholic patients have shown that compliance with medication has been associated with abstinence (Fuller and Williford, 1980) but that there were no significant differences on measures of total abstinence and other measures between those who had received disulfiram (therapeutic or inert dose) and those receiving either placebo or no disulfiram during a twelve month follow-up (Fuller et al., 1986; Powell et al., 1985; Schuckit 1985).

Recent studies have suggested that disulfiram may be a useful situational therapeutic tool in treatment and

when used in conjunction with self-control strategies (Duckert and Johnsen, 1987).

Psychotropic Medication

In addition to the serotonin uptake inhibitors mentioned earlier, lithium has been suggested as a drug which may reduce the desire for alcohol and consequently reduce consumption. Although the use of lithium has been associated with fewer days drinking at outcome of treatment, its use has also been associated with reduced scores on depression rating scales, reduced violent behaviour and fewer re-admissions to hospital (Jaffe, 1984).

More recent studies have cast doubt on the efficacy of lithium in the treatment of alcoholism, whether depression is present or not (Fawcett et al., 1987; Powell et al., 1987).

The coexistence of alcoholism and other psychopathology, particularly depression is common and will be reviewed in a later section. There is some evidence that in general, untreated concomitant psychopathology is a prognostic indicator of poor outcome (Rounsaville et al., 1987). Although it is found that concomitant psychopathology frequently remits in abstinent alcoholics, some disorders will persist and require separate and additional treatment. In general, the treatment outcome for alcoholics with "dual diagnoses" has been disappointing, even when pharmacotherapy has been prescribed (O'Sullivan et al., 1988; Penick et al.,1984).

In summary, pharmacotherapy is used as an adjunct to treatment and no single medication has been shown to be a "cure" in the overall treatment of alcoholism, with or

without concomitant psychopathology. Continued attention to subgroups in alcoholism, specifically those patients who have "dual" diagnoses, may be useful in delineating which pharmacotherapeutic approaches might be useful in improving outcome.

1.3.1.b *Behaviour Therapy*

Aversion Therapy

Aversion therapy is possibly one of the earliest behaviourally oriented treatments to be applied to the treatment of alcoholism. The theoretical basis for this treatment comes from the principles of classical conditioning and counterconditioning. The basic premise of treatment is that positive associations with alcohol are replaced by an adverse reaction to alcohol. Alcohol is then associated with either an unpleasant experience or image and this should consequently decrease the desire and increase avoidance of alcohol.

There are three main types of aversion therapies which have been used in the treatment of alcoholism. These are chemical, electrical and covert sensitization. Electrical aversion conditioning was initially more widely used than chemically induced aversion conditioning due to the precision offered by electric shock as the unconditioned stimulus. The results of uncontrolled studies (Blake, 1965; Blake, 1967; Miller, 1976) of electrical aversion are generally difficult to interpret but one controlled study (Vogler et al., 1970), carried out over a median follow-up period of eight months, demonstrated that patients receiving aversion conditioning did not differ in terms of proportion of relapse to those in the other treatment conditions

although they were found to have taken longer to relapse than patients in the other groups.

In chemical aversion therapy, alcohol is paired with nausea or vomiting induced by emetic drugs. Uncontrolled studies (Neuberger et al., 1982; Lemere and Voetglin, 1950; Weins et al., 1976) have been inconclusive as to the efficacy of chemical aversion therapy. Controlled studies have been rare and have shown it to be of no advantage when compared to electrical aversion (Jackson and Smith, 1978; Cannon et al., 1981) and of no advantage over a control group receiving standard hospital treatment (Cannon et al., 1981). Chemical and electrical aversion therapies therefore appear to add little, if any, advantage over other treatments.

Covert sensitization, involving the learning of a conditioned response between alcohol and unpleasant imagery, has advantages over both electrical and chemical aversion therapies in that it is less intrusive, requires no shock or drugs and can be administered more easily to patients on an out-patient basis. It has been shown to produce conditioned aversion to alcohol and like chemical aversion, the strength of conditioning is predictive of treatment outcome (Elkins, 1980; Cannon et al., 1981: Cannon et al., 1988).

Although behaviour therapy, including covert sensitisation, may suppress drinking in alcoholics more than either milieu therapy or transactional analysis alone (Olson et al., 1981), it may not produce results different from that of either electrical aversive conditioning or aversive imagery (Wilson and Tracy, 1976), or problem solving and group discussion (Sanchez-Craig and Walker, 1982). As the latter authors pointed

out, it is unlikely that any single treatment programme could best meet the needs of a group of chronic alcoholics that is unlikely to be homogeneous.

There is little evidence from the above studies regarding which types of alcoholic patients are likely to respond to aversion conditioning treatments although it may be suggested that those who did respond were highly motivated to overcome their alcohol problem given the nature of these treatments. It is also unclear if covert sensitization would lead to beneficial effects in the long-term outcome of alcoholism due to the difficulty of recreating and maintaining both aversive imagery and emotional response over time (Litman and Topham, 1983).

Scant attention is paid to coexisting psychopathology in alcoholics in these studies. One report on psychological test data on alcoholics receiving pharmacological aversion (Zielinski, 1979) found that 42% of patients scored at or above levels indicating clinical depression on the Beck Depression Inventory (Beck et al., 1961), the Zung Self-Rating Scale (Zung, 1965) and on the Minnesota Multiphasic Personality Inventory (Hathaway and McKinley, 1951). Unfortunately, the report is unclear about the timing of assessment and whether or not patients had completed detoxification and no attempt was made to relate the findings to response to treatment.

Contingency Management

The application of instrumental learning principles to the treatment of chronic alcoholism has been successfully demonstrated by "community reinforcement programmes" (Hunt and Azrin, 1973; Azrin, 1976; Azrin et al., 1982). In all of these studies, alcoholics have been assigned to either an experimental condition, the

community reinforcement programme or to a control group, standard in-patient or out-patient treatment. The community reinforcement approach involves several treatment components such as behavioural training in job finding skills, marital therapy, restructuring daily activities, reinforcing leisure activities and the use of disulfiram to reduce impulsive drinking. Although the components of the community reinforcement programme have varied from the original package, the principle of systematically and consistently reinforcing abstinence has been applied in an attempt to make an abstinent life-style more rewarding than drinking. This is in contrast to the "natural world" in which drinkers are often given more attention from helping agencies and family and friends when experiencing a "crisis" or when drinking.

The results of these studies have shown that contingency management procedures are highly effective in achieving improvement in several areas of functioning including more time abstinent and in employment at six months follow-up compared to those in the control groups who had mostly relapsed and spent more time unemployed. The effectiveness of contingency management may lie not in the strength of the reinforcers used but in the individual's perception of the relative importance of the rewards and punishments that these programmes utilised (Litman and Topham, 1983).

In the "natural world", a drinker is likely to find that the consequences of his drinking behaviour and the benefits of not drinking are rarely articulated and neither predictably or systematically responded to. As a result the perceived benefits of changing drinking behaviour are likely to be less powerful in changing

behaviour. The goals and behaviours sought in community reinforcement programmes are clearly expressed and agreed by therapist and subject, are consistently reinforced and a great deal of time and effort are involved in attaining these.

Behavioural Self-Control Training

Behavioural self-control training (BSCT) has received increasing attention in the 1980's as social learning theories have gained ascendancy (eg. Bandura, 1977). Unlike classical and operant conditioning theories which are based on the premise that behaviour is determined and shaped by the external environment, the basic premise of self-control theories is that behaviour can be shaped by an individual's environment which is under the control of that individual. An individual can organise his environment so as to increase the likelihood of occurrence of certain behaviours or to avoid other behaviours.

BSCT involves a set of self-management procedures designed to decrease or stop the consumption of alcohol. Included in these are such procedures as functional analysis of the antecedents of drinking behaviour, self monitoring, coping strategies and goal setting.

Miller et al (1981) examined the effectiveness of BSCT administered either with minimal therapist contact or therapist directed. Self referred problem drinkers were randomly assigned to the two treatment conditions and did not differ on outcome measures. The improvement rate of 81% for the minimal treatment group is greater than that of 41.9% quoted by Emrick (1975) for untreated or minimally treated cases indicating that BSCT

interventions are more effective than no treatment, at least in the short term.

In clinical populations, the results are more varied. In one study (Foy et al., 1984), in-patient alcoholics were randomly assigned to two treatments: BSCT, aimed at moderating drinking behaviour, in addition to an abstinence based treatment programme, and an abstinence programme alone. Those who had received the additional training did worse during the first six months after treatment ended, although there was no difference in treatments at long term follow-up of five years (Rychtarik et al., 1987). As there is no control over what happened in the intervening five years from treatment, the evidence suggests that BSCT aimed at controlled drinking for more severely dependent alcoholics has a detrimental effect in the short term compared to abstinence treatment.

Cue exposure treatment

Cue exposure and response prevention are recognised as being effective methods of treatment in obsessive compulsive disorder (Rachman and Hodgson, 1980). Operant and classical conditioning theories have been used to explain the mechanism by which cue exposure might decrease the desire to drink in alcoholics, thereby avoiding relapse. Conditioned responses to alcohol cues develop after repeated administrations and it is possible to extinguish these responses during unreinforced cue exposure.

Early reports of studies with small numbers of patients and single case studies suggested that cue exposure treatment might be promising in achieving abstinence and in reducing the desire to drink (Pickens

et al., 1973; Hodgson and Rankin, 1976; Blakey and Baker, 1980). Controlled studies involving in-patient subjects given a priming dose of alcohol then being asked to refrain from drinking alcohol (in vivo cue exposure) in the experimental group, and control groups given imaginal cue exposure have found that a priming dose of alcohol decreases the desire for alcohol, decreases the difficulty in resisting drinking alcohol, and increases the time required to consume a dose of alcohol in a subsequent behaviour test (Rankin et al., 1983; Laberg and Ellertsen, 1987).

These studies can be criticised as they employed small numbers of dependent alcoholics and it is unclear if cue exposure would generalise to a natural setting or produce long-term gains. These studies also do not address the question of the efficacy of cue exposure in comparison with other treatments and no conclusion can be made regarding the advantage of this treatment over others. Recent theoretical criticisms of cue exposure have suggested that treatment may only increase the latency to relapse and not the extent of relapse and that the link between conditioned responses to alcohol related cues and relapse has not been proven (Drummond et al., 1990).

1.3.1.c *Psychotherapeutic Approaches*

Of the controlled treatment studies published before 1980, Miller and Hester (1980) found no persuasive evidence for the effectiveness of psychodynamic psychotherapy in the treatment of alcoholism.

In a large scale study (Brandsma et al., 1980) alcoholic out-patients were randomly assigned to one of four treatments: a rational behaviour therapy group, an

insight oriented therapy group, an Alcoholics Anonymous group and a no-treatment control group. Only 116 of the original 260 patients completed treatment. At 12 month follow-up, those patients who had been in any of the active treatment groups did significantly better overall in terms of reduction in drinking and social and legal problems than those in the control group. However, those in the control group were also found to improve over time although not to the extent of those in treatment.

One of the problems in determining the efficacy of psychotherapy in the treatment of alcoholism is the lack of an agreed definition of what constitutes psychotherapy or counselling and in the integrity of the interventions and their delivery to patients (Institute of Medicine, 1989).

1.3.1.d *Cognitively Oriented Treatments*

A social-learning approach to problem drinking emphasises the multiple determinants of drinking behaviour and recognises that drinking will be influenced by social, cultural and individual factors in addition to the physiological effects of alcohol. Treatment strategies which provide the drinker with skills to identify risk situations, elucidate the consequences of drinking, and increase the likelihood of generating alternative behaviours to drinking have been encompassed in skills training approaches. Broad spectrum approaches to the treatment of alcoholism have also included skills training. These treatments aim to tackle problems that may be functionally related to drinking behaviour as well as drinking behaviour itself.

Social Skills Training

Social skills training can be considered as a cognitively oriented behavioural treatment. Ferrell and Galassi (1981) found that in-patient alcoholics, identified as having poor social skills and given assertiveness training in addition to milieu therapy, did significantly better than those attending human relations training on a measure of self expression but not of anxiety at six week follow-up. At two year follow-up, those in the assertiveness group had remained abstinent for more months and had significantly lower self ratings on an anxiety measure and higher ratings on a self expression scale than those in the human relations group.

A Norwegian study (Eriksen et al., 1986) randomly assigned in-patient alcoholics to group social skills training and to a control discussion group. Social skills training was aimed at increasing clients' social skills and assertiveness behaviour. In addition, clients in both groups participated in the traditional abstinence oriented treatment programme. Throughout the 12 month follow-up, lottery tickets were used successfully as an incentive for clients to return self-report questionnaires detailing drinking, work behaviour, nights slept at home and use of disulfiram. Clients in the social skills training group did significantly better on drinking measures, employment, admission rates to institutions and nights spent sleeping at home during follow-up than those who had taken part in the control discussion group.

Chick et al (1988) compared two "advise" groups to extended treatment which included social skills training in addition to milieu and group therapy based treatment.

Two years following treatment, those who had received extended treatment were found to have less continuing problems than those in the "advise" groups.

These studies suggest that social skills and assertiveness training appears to be an effective adjunct to traditional or milieu treatment for alcoholics, whether or not they are selected for deficiencies in social skills.

One early study investigated the effectiveness of problem solving skills training to reduce drinking behaviour and relapse rates in in-patient alcoholics (Chaney et al., 1978). Subjects were randomly assigned to one of three treatment groups: a problem solving skills training group, a discussion group and a no-additional treatment control group. The skills group received modelling, role-playing, and coaching to generate alternatives to drinking and to learn problem solving strategies based on the work of D'Zurilla and Goldfreid (1971). Those who had received training in problem solving skills had drunk significantly less and on fewer days as well as having significantly shorter duration of relapse than those in the other groups at one year follow-up. Analysis of relapse situations revealed that the largest number of relapses were associated with negative emotional states. The study indicated that a problem solving approach significantly enhanced the effect of "standard" treatment in reducing drinking and duration of relapse up to one year after treatment. Although negative emotional states were found to be the most commonly endorsed reason for relapse, no attempt was made to specifically modify emotional states *per se* in treatment. It remains a possibility that treatment aimed

at modifying emotional states using a problem solving approach would be effective in the treatment of alcoholics who experienced these difficulties.

Oei and Jackson (1982) evaluated the long term efficacy of cognitive-behavioural skills training methods in patients admitted to a treatment center for alcohol problems. On the basis of difficulties in assertion, patients were assigned to one of four treatment groups: social skills training, cognitive restructuring, a combination of social skills training and cognitive restructuring and a control group consisting of supportive therapy. Cognitive restructuring entailed "rational persuasion" to modify any irrational beliefs and Meichenbaum's "self talk" procedures. Patients were followed-up at three, six, and 12 months after treatment. Those patients who had received cognitive restructuring and a combination of social skills and cognitive restructuring showed significantly greater rate of improvement on all follow-up measures, including behavioural and drinking measures, compared to controls and to those receiving social skills alone. Those who had received cognitive restructuring, either alone or in combination, had significantly lower alcohol consumption over the year's follow-up compared with those in the social skills group, who in turn consumed less alcohol than the control group. Although those receiving social skills training improved more quickly than other groups on self ratings of fear of negative evaluation, this lead was not maintained in the follow up period where those who had received cognitive restructuring, either alone or in combination, continued to improve compared to the social skills group. Reinforcement of positive self

statements, relevant self-disclosure by therapists and modelling and rehearsal of coping behaviour seemed to be important components of the cognitive behavioural treatment (Oei and Jackson, 1984). This study suggests that cognitive restructuring resulted in cognitive changes manifested by continuing improvement in overt behaviour, even in the cognitive restructuring alone group whose behaviour was not directly modified.

As cognitive therapy (Blackburn et al., 1981; Blackburn and Davidson, 1990; Teasdale et al., 1984), interpersonal psychotherapy and cognitive therapy (Elkin et al., 1989) have been rigorously evaluated and shown to be effective in the treatment of out-patients with major depression, alcoholics with coexisting depression may benefit particularly from these treatments, not only in terms of depression but also in reducing alcohol consumption or maintaining abstinence.

One study reports four case studies of alcoholics (McCourt and Glatz, 1980) treated with Beck's cognitive therapy (Beck, 1976). The authors suggested that these four patients responded positively to the treatment, which took place in groups, and continued to report improvement in drinking behaviour at one year follow-up. However, the study does not report the overall findings of group cognitive therapy and it is unclear if depressed alcoholic patients, or whether any alcoholic, would specifically benefit from this treatment. To date, no properly controlled studies have evaluated the effectiveness of interpersonal or cognitive therapy in depressed alcoholic patients (Institute of Medicine, 1989).

Relapse Management

One of the critical problems in the treatment of alcoholism, and addictions in general, is the maintainance and generalisation of behavioural change over time. Although many studies report improvement in drinking behaviour after treatment, abstinence is rarely a stable outcome (Armor et al., 1978). Roughly two-thirds of persons addicted to alcohol, heroin and cigarettes have been found to have similar temporal patterns of relapse within the first three months following treatment (Hunt et al., 1971).

The work of Marlatt (1985) has suggested that there may be common behavioural and cognitive factors associated with relapse, regardless of the addictive substance. Others have found that abstinent alcoholics rated unpleasant mood states, social anxiety, external situations and decreased cognitive vigilance as circumstances which were potentially the most dangerous in terms of precipitating relapse (Litman et al., 1979).

Annis and Davis (1988) designed a relapse prevention programme based on self-efficacy theory (Bandura, 1977) The focus of treatment was on drinking situations recognised as being of risk to heavy drinkers. The treatment was in two phases: the first involving the identification of drinking risk situations and homework assignments graded in difficulty according to ratings of self-efficacy. The second phase involved strategies to help maintenance of behavioural changes and to improve perceived self-efficacy in coping with previous challenging drinking situations by gradually withdrawing therapist support and encouraging clients to develop self-monitoring abilities.

At three months, 47% of the 38 clients followed-up reported total abstinence and at six months follow-up, 29% had continued to be abstinent. For those who continued to drink, there were marked decreases in the frequency of drinking and quantity drunk on each occasion over the follow-up.

One of the predictions from the study was that changes in drinking behaviour would be associated with changes in self-efficacy. This was confirmed. Although substantial decreases were noted in the adverse consequences of drinking and social and personal functioning improved, more than one-quarter of clients continued to report interpersonal, vocational and affective problems. Given that all clients were employed and over one-third had been pressurised into seeking treatment by employers and families, some continuing difficulties in interpersonal and vocational areas might be expected to arise from the reactions of others as most individuals continued to drink, albeit at lower levels than before treatment. The fact that affective problems continued to be reported is of particular concern as negative emotional states were the most common antecedent of heavy drinking in this group and therefore clients reporting affective problems might be regarded as being most vulnerable to relapse (Chaney et al., 1978).

The lack of a control group in Annis's study does not allow comparisons with no treatment or another treatment. The study can only be regarded as a pilot study which nonetheless has added to our understanding of the process of relapse by operationalising a theoretical model. Information on the client's psychiatric status would have been helpful in clarifying the nature of

negative emotional states as it is possible that clients were suffering from a depressive disorder in addition to their alcohol problem.

1.3.1.e *Other treatment approaches*

The studies mentioned above constitute research into some of the main treatments of interest in recent years. This review is not designed to cover all treatments. Some have been omitted such as research into the efficacy of Alcoholics Anonymous (Emrick, 1987) and marital therapy (O'Farrell et al., 1985).

1.3.1.f *Summary*

There appears to be no one optimal treatment for alcoholism. Rather there are a range of pharmacological, psychosocial, cognitive and behavioural interventions, some of which have been shown to be efficacious in the treatment of alcoholism and problem drinking. Although there is increasing emphasis on the heterogeneity of alcoholic populations (Meyer and Kranzler, 1990), little attention has been paid to this in the treatment literature to date (Miller and Hester, 1986). Client characteristics, including co-existing psychiatric disorder, could be considered as likely to have an influence on treatment outcome.

Depression has consistently been associated with alcoholism (Schuckit, 1983; Hasin et al., 1988; Halikas et al., 1981). Over the past twenty-five years, pharmacotherapy and cognitive therapy in the treatment of unipolar depression have been extensively evaluated and have been shown to be efficacious in the treatment of this disorder (Murphy et al., 1984; Beck et al., 1985(a); Blackburn et al., 1981). Prophylactic antidepressant medication is known to be helpful although relapse rates

are nonetheless high in unipolar depression: 45% of patients relapse within one year and 70% by three years (Glen et al., 1984). Cognitive therapy may play a role in the prevention of relapse in depression (Blackburn et al., 1986(a); Kovacs et al., 1981; Beck et al., 1985(a)). This knowledge has not been systematically applied to the treatment of alcoholics with co-existing affective disorder. The extent to which depression or depressive symptomatology contributes to drinking outcome in alcoholism has however not been fully studied.

1.4 COMORBIDITY: DIAGNOSTIC ISSUES

Feinstein (1970) introduced the term "comorbidity", defined as "any distinct additional clinical entity that has existed or that may occur during the clinical course of a patient who has the index disease under study" (pp.456-457). The term comorbid is restricted to diseases or disorders and, strictly speaking, does not apply to symptoms.

There are several uses of the term comorbidity in psychiatric research and practice (Maser and Cloninger, 1990). Clinical studies use the concept of comorbidity to describe the fact that more than one disorder can be diagnosed in the same individual, whereas in psychiatric epidemiological studies, the term is used to indicate the relative risk of disorders, other than the index disorder, being present within an individual patient.

In the diagnosis of mental disorders, no one sign or symptom is sufficient to define a disease. Diagnostic criteria have been arrived at on the basis of the type, number, sequence of onset and duration of multiple nonspecific signs and symptoms and on the natural history of a disorder. Symptoms differ in their relative importance for a particular diagnosis. For example, the diagnosis of generalised anxiety disorder in DSM-III-R requires evidence of apprehensive expectation (criterion A), but any 6 of 18 other anxiety symptoms (criterion D) are also necessary. As a result of this partial lack of symptom specificity, patients with the same diagnosis may be heterogeneous in their symptom profile and are likely to vary a great deal in the extent to which they have common prominent features.

Klerman (1990(a)) in a review of comorbidity, hypothesised that psychiatry's recent and growing interest in comorbidity arose out of a paradigm shift in psychopathology which began post World War II. He argued that a "neo-Kraepelinian" paradigm has become dominant in research centres concerned with psychopathology, diagnosis and nosology. This new paradigm which began in the United Kingdom and in North America was a "re-affirmation" and modification of the 19th century continental European approach to psychiatry which was dominated by the "medical model". The "medical model" paradigm proposed multiple disorders of which the cure would be found in biological causes. The concept of multiple, discrete disorders again gained scientific respectability after much criticism, particularly during the 60's from outwith and within psychiatry (Szasz 1962; Menninger 1963; Illich 1976). From within psychiatry came criticisms of the unreliability of diagnosis, the lack of universality of diagnosis, the culture bound nature of diagnostic systems, the problems of working with a system that described the phenomena of mental illness in a categorical manner when, it was argued, a dimensional model might be better. From outwith psychiatry, the anti-psychiatrists and labelling theorists made themselves heard with their descriptions of the dehumanising effect of the process of diagnosis and labelling as psychiatry's way of serving society's need to control "deviant" behaviour.

During the 70's, there was a general move towards the adoption of many of the ideas and methods of the neo-Kraepelinian paradigm. Structured interviews and operationalised criteria for the assessment of

psychopathology were used along with agreed diagnostic criteria and statistical methods to assess the reliability and validity of diagnoses. Klerman (1990(a)) states that the paradigm shift culminated with the publication in 1980 of the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III).

1.4.1 Approaches to comorbidity

There are several approaches to the study of comorbidity. These will be described in the following sub-sections.

1.4.1.a *Multiaxial diagnostic systems*

Multiaxial diagnostic systems, such as DSM-III and DSM-III-R, explicitly encourage multiple diagnosis to be made. The relationship between diagnoses made on different axis is deliberately left unspecified in DSM-III and DSM-III-R. These diagnostic systems expose the problems of comorbidity. Questions of aetiology, causation of one diagnosis by another, the relative importance of each diagnosis in determining prognosis, treatment and outcome are left unanswered.

1.4.1.b *Primary-Secondary Distinction*

There are several uses of this distinction (Klerman, 1990(b)). The approach to comorbidity proposed by the Washington University group for the purpose of research, restricts the terms of primary and secondary to refer to the chronology of disorders. The goal here was to reduce the heterogeneity of samples in research so that there would be less variability in subject selection. Here, the disorder which is regarded as primary is that which occurs first chronologically. Again, there is no

implication as to causation or to which disorder is to be regarded as the more clinically important.

The foundations of the concept of secondary depression were laid in the 1950's by the members of the Department of Psychiatry at Washington University. The basic assumption is that depression which occurs alone will differ in aetiology, family history, course and response to treatment from depression which follows another psychiatric illness.

A second use of the primary-secondary distinction comes from general medicine. Classification systems within this field of medicine using this distinction imply a causal connection between disorders. For example, tuberculosis secondary to silicosis. In this form of classification, there is a clear indication as to which disorder is of the greater clinical importance and which should receive precedence in treatment. DSM-III-R incorporates this use of the primary-secondary distinction for a number of conditions such as organic anxiety disorder and organic personality disorder where the causal implication is explicit. The principle of a hierarchical classification is also evident in that organic conditions are assumed to be causal or aetiological in the disorder.

The final use of this distinction is by clinicians and refers to which disorder is to be regarded as being of major importance by the clinician treating a patient. There are no studies of the reliability of this judgment in either clinical practice or in research (Klerman 1990(b)).

1.4.1.c *Lifetime disorders*

The advent of structured psychiatric interviews and diagnostic criteria has allowed researchers to investigate the extent to which psychiatric disorders co-exist within individuals across their lifetimes. This approach to comorbidity is neither hierarchical nor does it allow for causal hypothesis. It has been useful in providing estimates of the prevalence of multiple psychiatric disorders in community and clinical samples. Confusion can however arise if current diagnosis is included in prevalence rates of lifetime diagnosis, particularly if this information is then used to predict outcome of illness. Structured interviews such as the SADS-L (Endicott and Spitzer, 1978) are specifically designed to give information leading to diagnosis of lifetime psychopathology but its use is cautioned where there is likely to be a current disorder. This practice has sometimes been ignored and lifetime and current diagnosis have not been clearly distinguished.

1.4.1.d *Spectrum Disorders*

The concept of spectrum disorders comes mainly from research on the hereditability of disorders. The basic concept involves the idea that certain disorders which are clinically related may share a common "underlying" genetic link. This concept has been used in adoption, cross-rearing and family aggregation studies in psychiatry and has more recently been integrated into DSM-III for disorders such as cyclothymic disorders.

1.4.1.e *The Radical Empirical Approach*

Some researchers argue that diagnostic systems which make use of hierarchical principles obscure valuable information about the occurrence and nature of disorders

and that causal hypotheses cannot be properly examined as these are excluded by the operation of hierarchical principles. Although DSM-III-R has suspended some of the older principles of hierarchical diagnostic systems, these researchers would argue it has not gone far enough. In DSM-III-R, for example, the hierarchy for major depression precludes an independent diagnosis of generalised anxiety disorder or of panic disorder if these syndromes occur concurrently with a depressive condition.

There are however other approaches to the concept of comorbidity which do not fit with the neo-Kreapelinian model with its reliance on observable symptoms and behaviour. These approaches take the existence of high levels of comorbidity as given and seek to investigate underlying biological and psychopathogenic processes using genetic, physiological and psychological studies.

In general, as diagnostic systems become more inclusive of diseases and as the rules of exclusion of specific diagnosis are relaxed and hierarchical classification systems become less rigid, the more likely it is that morbidity and comorbidity will be increasingly detected. The degree to which comorbidity represents a "true" underlying relationship or, at the other extreme, is an artifact of more inclusive diagnostic systems needs to be explored carefully if we are to increase our understanding of the pathogenesis of mental illness.

1.4.2 Primary and secondary depression

In an overview of ten studies examining the significance of secondary depression, defined as depression in an individual who has one or more pre-existing non-affective disorders, Clayton and Lewis

(1981) found that secondary depression was more frequently diagnosed in out-patient and in-patient settings than in community surveys. Primary and secondary depression are virtually indistinguishable in terms of symptom pattern except for psychomotor retardation which is more common in primary depression. Patients with secondary depression are likely to have a younger age of onset of depression and a more chronic course of illness compared to patients with primary depression. Primary depressives were likely to be women rather than men, more middle class, of a higher educational level and less likely to be divorced or separated than secondary depressives. Many of these differences were accounted for by the samples of patients from which those with secondary depressives derived; the most common primary diagnoses being alcoholism, schizophrenia, opiate addiction, sociopathy and hysteria. Secondary depressives were more likely to have a family history of alcoholism whereas bipolar illness and suicide were found to be more common in primary depressives. However, a family history of depression was common to both primary and secondary depressives.

There is some evidence that primary and secondary depressives can be distinguished by neurophysiological variables such as EEG sleep measurements (Coble et al., 1976; Kupfer et al., 1978). Although these findings indicate that primary and secondary depressions may not be biologically homogeneous, there is reason to be cautious about these findings. Kupfer and his colleagues (1978) found significant differences between patients with secondary depression with and without concurrent medical disease. They did not compare these groups with

primary depressives separately and it is possible that the differences found between primary and secondary depressives could be associated with the primary illness of one of the sub-groups of secondary depressives. For example, some of those with secondary depression had a primary diagnosis of alcoholism and there is increasing evidence to suggest there may be central nervous system changes subsequent to prolonged alcohol abuse that could persist beyond the two weeks abstinence period Kupfer required before sleep recordings were taken (Begleiter and Porjesz, 1984).

1.4.3. Alcoholism and Comorbidity

Meyer and Kranzler (1990) in a recent review of alcohol abuse, alcohol dependence and comorbid depression, emphasised that it was no longer tenable to regard alcoholics as constituting a homogeneous group or to make the assumption that all psychiatric symptomatology in alcoholics was aetiologically related to the development of the disorder. They concluded that the concept of comorbidity was a useful one in exploring the complexity of mood disorders and alcoholism.

Studies that have investigated the prevalence of psychiatric disorder and alcoholism have used both community and clinical samples. Different diagnostic instruments have been employed and the sample may have been gathered for purposes other than purely the investigation of the co-occurrence of psychiatric disorder and alcoholism. The studies were carried out at different points in time and across different locations.

In an attempt to obtain estimates of the prevalence of comorbidity in alcoholism and the relationship between alcoholism and other psychiatric disorders, some studies

have been carried out on patients with affective syndromes although the majority have used alcoholic samples.

1.4.4 The Prevalence of Psychiatric Disorder in Alcoholism

Investigators have predominantly examined the co-occurrence of psychiatric disorders in alcoholism over the course of an individual's lifetime. Some have specified whether the disorder was primary (occurred first) or secondary (occurred after the onset of the primary disorder).

A number of studies have shown that between 54% and 75% of alcoholics, either in the community (Weissman and Myers, 1980) or in clinical populations (Powell et al., 1982; Hesselbrock et al., 1985; Herz et al., 1990) suffer from another psychiatric disorder. The study with the lowest prevalence of comorbidity (54%)(Herz et al., 1990) may have underestimated the extent of comorbidity because of the small sample size (n=74). If the latter study is not considered, the prevalence of comorbidity ranges from 63% to 75%. When primary alcoholism is differentiated from secondary alcoholism, the prevalence of primary alcoholism with secondary psychiatric disorder in male alcoholics was found to be 43% (Schuckit, 1983).

Male and female alcoholics have been shown to differ in the type of disorder from which they have suffered. Community studies have shown that female alcoholics were more likely than male alcoholics to have an additional diagnosis. They were more likely to have had any diagnosis, except for personality disorder which was more common amongst males (Helzer and Pryzbeck, 1988). This finding was reflected in clinical samples, in which depression and phobias were more likely to be found

amongst women whereas antisocial personality disorder and substance abuse was more common amongst men (Hesselbrock et al., 1985).

1.4.5 The Prevalence of Affective Disorder in Alcoholism

The prevalence of affective disorder in alcoholism has been examined in both community and clinical samples. It has been claimed that male alcoholics in community studies have a similar rate of depression as in males in the general population (Helzer and Pryzbeck, 1988). For female alcoholics however, depression has been found to occur twice as often as that found in females in the general population (Helzer and Pryzbeck, 1988). This study, using DSM-III criteria, and drawing on data from the five sites in the NIMH Epidemiological Catchment Area (ECA) study, had a sample of 20,000. In a smaller community sample of 510, Weissman and Myers (1980) found that, of the 34 subjects who had ever been alcoholic, 44% had a diagnosis of major depression and 18% of minor depression, using Research Diagnostic Criteria. There was no difference between male and female alcoholics in the lifetime diagnosis of depression (Weissman and Myers, 1980).

The prevalence of depression in clinical samples has been assessed using two main diagnostic systems: RDC and DSM-III criteria. Studies using DSM-III criteria have reported a wide range of prevalence of depression (16% to 42%). Two of these studies by Herz et al (1990) and Halikas et al (1981) were carried out on small samples of patients presenting for treatment (n=74 and 71 respectively). Consequently, the prevalence reported in these studies (16 to 24%) may be low. Two studies using larger sample sizes reported a prevalence of depression

ranging from 38% to 42% (Hesselbrock et al., 1985; Powell et al., 1982). Using RDC, rather than DSM-III criteria to diagnose depression, Hasin et al., (1988) found a higher prevalence of depression (68%) amongst alcoholic in-patients. It is likely that the difference in prevalence found is due to the structured interviews used to assess diagnosis: the Diagnostic Interview Schedule (DIS)(Robins et al., 1981), used to reach diagnosis on DSM-III, and SADS-L used in conjunction with RDC, are known to show poor agreement in the assessment of affective disorder in patients with substance abuse. The SADS-L produces a considerably higher number of cases of major depression then the DIS (Hasin and Grant, 1987).

When only lifetime diagnosis of primary alcoholism is considered, the prevalence of secondary depression in males has been reported to be approximately 25% (O'Sullivan et al., 1983; Schuckit, 1983). Secondary depression has been found to develop later in life in male alcoholics than in female alcoholics (Cadoret and Winokur, 1974).

The simultaneous co-occurrence of alcoholism and depression in community samples has been found to be low. Weissman and Myers (1980) have reported that 2.9% of their community sample had at one point in their lives received both a diagnosis of alcoholism and depression. However, at the time of the survey, only 0.4% of the sample met diagnostic criteria for both alcoholism and either major or minor depression (Weissman and Myers, 1980). When depressive symptomatology, rather than diagnostic criteria are considered, it has been found that 4% of both males and females showed both alcoholism and depressive symptoms at any one time (Midanik, 1983).

Helzer and Pryzbeck (1988) postulated that these results do not necessarily contradict findings of a stronger association between alcoholism and depression in clinical samples as the co-occurrence of alcoholism and depression is likely to lead individuals into treatment. This would therefore increase the prevalence of depression found in clinical alcoholic samples. This phenomenon is known as Berkson's bias: the increased tendency for persons having two or more diagnoses to seek and receive treatment and thus fall into study populations drawn from treatment services.

In hospitalised alcoholics, the prevalence of depression in the index episode of alcoholism has been examined using the change version of the SADS and found to be 30.2% when assessed not later than five days after admission (Dackis et al., 1986). However, of the 49 patients initially depressed, only 10 (20.4%) continued to fulfil diagnostic criteria for major depression after 10 to 14 days of abstinence. Patients who continued to meet criteria for depression were significantly more likely than those who recovered to have a positive dexamethasone suppression test and to have a positive family history of affective disorder. The authors suggested that those initially diagnosed as depressed may have had alcohol-induced, organic affective syndromes and not major depression which would not have been expected to remit spontaneously in such a short space of time. Another possibility is that those patients whose depression did not remit were simply recovering more slowly from an alcohol-induced depression.

Bernadt and Murray (1986) in a study of in-patients, explored the links between psychiatric disorder and

drinking. This study also attempted to answer Morrison's (1974) criticism that it was not established that depression occurs more frequently in alcoholics than it does in other psychiatric disorders. Every male and every second female admitted to hospital included in the study were interviewed on the Present State Examination (PSE) (Wing et al. 1984).

Thirty seven (10%) of patients admitted received an RDC diagnosis of alcoholism. A total of 73 patients were found to be drinking heavily, at least 8.1 drinks per day in the year before admission. Only the alcoholics as a group were drinking above the mean reported, although those with personality disorders were also drinking heavily on a daily basis. Heavy drinking was not common amongst patients with major depression, anxiety and phobic disorders, obsessive-compulsive disorders and schizophrenia. Only the alcoholic group had increased their consumption of alcohol in the month prior to admission. The authors found that alcoholics and non-alcoholic groups were similar in terms of prevalence of secondary disorders. Affective disorder was the most frequently given secondary diagnosis. In this sample, 15.8% of alcoholics received a PSE second sub-class allocation of depression as did 15.3% of other psychiatric patients.

There is evidence that a diagnosis of depression in the current episode of alcoholism may change with abstinence (Dackis et al., 1986; Nakamura et al., 1983). As depression does not remit rapidly, depression in the current episode may be due to the chronic effect of alcohol intoxication. This may account for the relatively high prevalence of both current and perhaps,

lifetime diagnosis of depression in alcoholics. However, depression as a co-existing diagnosis in an index episode, may be as common in non-alcoholic samples as it is in alcoholic samples (Bernadt and Murray, 1986).

1.4.5.a *Alcohol Abuse in Patients with Affective Syndromes*

There are relatively few studies specifically looking at alcohol abuse in samples of patients with affective disorder. Hasin et al (1985), using out-patients and in-patients from five treatment centres across the U.S.A. who participated in the NIMH Clinical Research Branch Collaborative Program on the Psychobiology of Depression, looked at alcohol and drug abuse in patients with current major depression or mania. All 835 patients were assessed by the SADS and met RDC criteria for a definite major depressive or manic syndrome on admission to the study. The sample contained more women than men (57.6% versus 42.4%). Approximately 24% of the sample scored three or more on the SADS alcohol scale indicating at least "minor interference" in functioning due to alcohol and 9% scored at this level on the drug scale. Men were found to be more likely than women to abuse alcohol and drugs. Men in the lowest socio-economic group were found to be most likely to report alcohol problems and younger rather than older patients, regardless of sex were more likely to be abusers of both substances.

As such, these patients resemble those found in samples from alcohol treatment units.

1.4.5.b *Comparison of Alcoholics with and without Depression*.

Cadoret and Winokur (1974), using Feighner's criteria (Feighner et al., 1972) found that male

alcoholics with secondary depression had significantly more "alcoholic paranoia" and a more frequent binge pattern of drinking than men without secondary depression. Female alcoholics with both primary and secondary depression showed significantly more frequent and longer periods of abstinence than female alcoholics without depression. Women with primary depression were significantly older when they developed alcoholism compared to women who had developed alcoholism with and without secondary depression. When both male and female alcoholics with depression, either primary or secondary, were compared with those with alcoholism alone, a highly significant difference emerged in the frequency of past multiple suicide attempts: 27% of the depressives in comparison to only 5% of the non-depressed alcoholics.

In a study of 285 male alcoholics, Schuckit (1983) compared the clinical course and family history of alcoholics with and without secondary affective disturbance. Primary and secondary labels were applied on the basis of the chronology of development of symptoms. Seventy of the alcoholics (30%) were found to have secondary depression and 163 were primary alcoholics with no secondary disturbance (70%), using DSM-III criteria. The two remaining groups were very similar in terms of their drinking although the affective group tended to have more alcohol related problems. There were no significant differences in demographic characteristics between the two groups. Alcoholics with affective disorder were more likely to have had past psychiatric admissions and out-patient treatment than those without affective disturbance. In terms of family psychiatric history, which was checked with a relative, no overall

differences emerged between the groups for either affective disorder or alcoholism.

Schuckit et al (1969) in an earlier study of female alcoholics found that female primary depressives had a significantly higher incidence of affective disorder in first degree relatives than primary alcoholics. In the Cadoret and Winokur (1974) study, there was no difference in the incidence of affective disorder in parents and siblings for secondary depressives, indicating that a familial factor may not be involved in the aetiology of secondary depression in contrast to primary depression in alcoholics. However, when the sample was enlarged to 259 hospitalised alcoholics, Winokur et al. (1971) analysed the morbid risk of alcoholism and depression in relatives of probands with primary alcoholism, primary affective disorder (depression-alcoholics) and sociopathy. The authors found that male relatives in all three groups were more likely to show an increase of alcoholism than females, and female relatives likely to show an increased risk of depression than males. Winokur proposed a spectrum hypothesis to explain this finding, suggesting that alcoholism manifests itself in a spectrum of conditions, one of which is alcoholism, another depression. In Winokur's description of depressive spectrum disease (Winokur, 1979), women tend to have depression and male family members, alcoholism and antisocial personality disorder.

Depression is commonly recognised as being twice as common in women as it is in men (Weissman and Klerman, 1977). Recent evidence suggests that there is an increase in the reported rates for depression, particularly for young men, in Western countries and that

there is a trend for the sex ratios to equalise (Paykel, 1991). If Winokur's depression spectrum hypothesis is correct, it follows that there would be a concomitant decrease in the prevalence of alcoholism as more men become depressed.

O'Sullivan et al (1983), using Feighner's diagnostic criteria to detect primary and secondary depression, found that those with alcoholism alone reported more traffic violations and more alcoholic blackouts than those with either primary or secondary affective disorder. There were no differences in the history of drinking between the three groups, nor in terms of items that were related to severity of dependence on alcohol, although no separate measure of severity of dependence was taken. No significant differences were evident in length of abstinence after in-patient treatments in the past between the groups, though the two groups with co-existing affective disorder had received more in-patient treatment for alcoholism in the past. In general, those with co-existing affective disorder had received more hospital treatment in the past. For the index episode, those with primary affective disorder with secondary alcoholism had the shortest duration of relapse before admission, perhaps indicating that these patients are more energetically treated by their psychiatrists or that they seek treatment at an earlier point due to their mood disturbance.

1.4.5.c *The effects of additional psychiatric disorder on social, occupational functioning and alcohol related variables*.

Depression in alcoholics does not appear to be associated with social and occupational impairment (O'Sullivan et al., 1979; Hasin et al., 1988). However,

phobias and depression have been associated with a higher number of psychological and physical complaints (Hesselbrock et al., 1985). Anti-social symptoms, on the other hand, have been associated with social and occupational impairment (Hasin et al., 1988).

Additional psychiatric disorder also appears to hasten the development of alcoholism. Such affected individuals were younger when they received a diagnosis of alcoholism (Herz et al., 1990) and in addition have been found to have more admissions for alcoholism in the past (O'Sullivan et al., 1979). The severity of dependence on alcohol has been associated with two additional psychiatric disorders, substance abuse and major depression (Hasin et al., 1988).

1.4.6 Summary

Alcoholism is not a unitary or homogeneous disorder. There is substantial evidence to suggest that in both community and particularly in clinical samples, additional psychopathology is common. Although estimates of additional psychopathology differ according to the samples studied and the instruments used to classify disorders, up to two-thirds of clinical samples of alcoholics are likely to have a lifetime diagnosis of another psychiatric disorder. Affective disorder and antisocial personality disorder appear to be the most commonly and consistently reported additional disorders. Alcoholism with secondary affective disorder would appear to be more common in male alcoholics than is primary affective disorder, secondary alcoholism. Women more than men appear to suffer from additional psychopathology, including secondary and primary

depression although this evidence is weakened by there being fewer studies carried out on mixed sex populations.

Antisocial personality disorder appears to be more common in male alcoholics and depression more common in female alcoholics.

Having an additional psychiatric diagnosis appears to alter the course of alcoholism: it may hasten the development of alcoholism and may bring individuals to the attention of treatment agencies more quickly.

More recent studies have examined the relationship between alcoholism and affective disorder in the current episode. There is consistent evidence to suggest that a diagnosis of depression in the current episode may change to one of alcoholism alone, once detoxifiction or abstinence has been achieved. The prognosis of those who continue to be depressed remains unclear.

1.5 OUTCOME OF COMORBID ALCOHOLISM AND DEPRESSION

Several studies have examined the effect of the presence of comorbid diagnoses on outcome. Penick et al (1984) provided data on 117 male and female alcoholics at outcome of one year. Two groups were followed-up, one with no additional diagnoses and one with additional diagnoses of antisocial personality disorder, depression and mania diagnosed by the Psychiatric Diagnostic Interview (PDI)(Othmer et al., 1981). This study appears to have had a surprisingly high follow-up rate (100%).

Other studies have specifically compared drinking outcome for alcoholics with and without depression. Rounsaville et al (1987) reported a follow-up rate of 84.3% of surviving male and female alcoholics at one year. The effect of lifetime diagnosis and, more specifically, primary diagnosis (DSM-III), in addition to severity of psychopathology at entry into treatment on drinking outcome was examined. O'Sullivan et al (1988) investigated the effect of lifetime diagnosis of affective disorder in male alcoholics on drinking outcome. Eighty-five per cent of men attended three follow-up occasions over a two year period.

The effect of current affective state on drinking outcome in alcoholics was examined in two studies. Hatsukami and Pickens (1982) carried out a cross-sectional study whereby 44% of 711 alcoholics, who had received treatment for alcoholism, responded to mailed questionnaires. The relationship between the severity of depressive symptomatology, as opposed to diagnostic category, and excessive drinking was investigated. Hasin et al (1989) used a different approach by selecting depressed male and female patients, who had an RDC

current diagnosis of alcoholism. The patients were followed up at six monthly intervals over a two year period to investigate the likelihood of remission from alcoholism. These patients had been recruited into the NIMH Collaborative Study on the Psychobiology of Depression and only 72% of the 127 alcoholics had received treatment for alcoholism over the time of follow-up.

Penick et al (1984) found that alcoholics with and those without additional lifetime diagnoses had both made "substantial" and significant improvement from baseline measures of drinking and social problems at one year follow-up. Nonetheless, those with additional lifetime diagnoses continued to show more impairment due to drinking in comparison to the alcoholic only group. This group however, also displayed greater impairment than the alcoholic only group at the beginning of the study.

O'Sullivan et al (1988) found that patients with lifetime diagnoses of unipolar affective disorder had received significantly more treatment for drinking bouts by the second and third follow-up than those patients with alcoholism alone and those with alcoholism and bipolar affective disorder. There was however no significant differences in the rate of abstinence, or in the number of days drinking between the groups on any of the follow-ups. Those with alcoholism alone were significantly poorer attenders at appointments with the hospital psychiatrist treating them compared to the other affectively disordered groups.

Predictably, the affectively disordered groups reported more affective disturbance at follow-up and received more treatment for this than those with

alcoholism alone. Depression was given as a reason for relapse in only a minority of cases. Patients reported that drinking during follow-up had been a response to an opportunity to drink (eg. a social situation) rather than in response to a stressful situation.

This study suggests that those with a lifetime diagnosis of affective disorder in addition to alcoholism had a poorer outcome than those with alcoholism alone. They receive more treatment both for their drinking and for affective disorder over the two years following the index admission. However, there was no significant difference between the groups in terms of rate of abstinence, despite exposure to extra treatment by the affectively disordered group. The sample of alcoholics in this study were demographically relatively homogeneous and was described by the authors in an earlier paper (O'Sullivan et al., 1979) as being "stable" and "middle class". As a result, the similarities in outcome may reflect the homogeneity of the sample and it is these factors which may be of more importance in determining outcome rather than the effect of an affective disorder on alcoholism.

Rounsaville et al (1987) provided a detailed follow-up study of 321 alcoholics, previously described by Hesselbrock et al (1985). Eight subjects had died and 47 could not be located or refused to take part in follow-up. Outcome measures relating to medical status, psychosocial functioning, drinking behaviour and its consequences and treatment for alcoholism were examined. The prognostic significance of the most prevalent additional lifetime psychiatric diagnoses (which included the current episode) in this sample of alcoholics was

investigated; those with major depression, antisocial personality disorder, and drug abuse. These three disorders were each contrasted with two other groups: those with no lifetime DSM-III diagnosis other than alcoholism and those who had a lifetime history of any other DSM-III disorder in addition to alcoholism.

There were significant differences in outcome for men and women, depending on their lifetime, including current, diagnosis. Men with major depression, drug users and those with "other diagnosis" had a worse prognosis in terms of receiving more treatment for alcohol problems, drinking more frequently and more heavily and scored more highly on the Minnesota Multiphasic Personality Inventory (MMPI)(Hathaway and McKinley, 1951) at follow-up than the group with no additional disorders. However, in contrast to men, drinking related outcome for women was worse for those who had no additional lifetime disorder and was roughly the same for those with "other diagnosis" and with major depression, indicating better outcome for women with a lifetime, including current diagnosis, of major depression.

Men with antisocial personality disorder and with "other disorders" had poorer outcomes than those with no additional lifetime diagnosis. In addition, the severity of alcohol dependence predicted poorer outcome in men. Women with antisocial personality disorder, drug abuse and with no additional diagnosis had similarly poor outcome. Looking at a dimensional approach to outcome involved correlating outcome measures with MMPI scores and a global severity rating taken at admission. The degree of dependence on alcohol at intake was

significantly correlated with poorer outcome for men. Higher average MMPI scores were significantly correlated with poorer outcome on several measures such as a pathological pattern of drinking and physical condition due to drinking.

The authors examined the prognostic significance of primary and secondary psychiatric diagnosis, on the basis of which disorder occurred first in an individual's lifetime. This reduced the size of the groups with major depression as 82% of depressed men and 53% of depressed women had a first depressive episode after the onset of alcoholism, drug abuse or antisocial personality disorder. When treatment outcome was compared in patients with primary diagnosis of alcoholism, antisocial personality disorder, depression and alcoholism, very few significant differences were noted. Those with a primary diagnosis of major depression had poorer ratings on social adjustment and higher ratings on the MMPI at follow-up compared to the other groups. In general, a lifetime diagnosis of an additional psychiatric disorder was shown to have greater prognostic power than a primary diagnosis, partly because categorization into primary diagnostic groups yielded heterogeneous groups.

Hatsukami and Pickens (1982) investigated the severity of depressive symptomatology and excessive alcohol or drug abuse in a postal cross-sectional follow-up in a sample of subjects who had been treated for alcohol or drug abuse. Roughly one third of the group received a postal questionnaire and the Zung Self Rating Depression Scale one month after discharge, one third received the same package after six months discharge, and the remaining one third, 12 months after discharge.

Instead of establishing depression by diagnostic criteria, depressive symptoms were said to exist if an individual scored at or above 50 on the Zung scale. Consequently, unlike the studies reviewed above, this study specifically examined the association between current symptomatology, rather than lifetime diagnosis, on drinking outcome. The authors defined relapse as any use of drugs or alcohol on more than one occasion during follow-up. Non-responders to the mailing, interviewed by telephone, were found to have relapsed significantly more than responders, but had a significantly lower rate of depressive symptoms than the responders.

Of those responding to the mailing, relapse rate and depressive symptoms were found to increase over time, although the severity of depressive symptoms did not increase significantly with time. Those subjects who had relapsed had significantly higher rates of depressive symptoms (ie. scoring 50 or more on the Zung) than those who had abstained both at one month after treatment and at one year. Mean Zung scale scores were similarly significantly higher for those who had relapsed compared to those who had abstained both at six months and at one year. Subjects who had not relapsed had scores on the Zung scale within the normal range.

No significant differences were found in relapse rates between men and women. Women reported significantly higher rates of depressive symptoms in the one year group and higher mean Zung scale scores on all occasions than did men.

From this study it appears that there is a positive association between relapse and increases in depressive symptomatology. It is possible that the occurrence of

high rates of depressive symptoms at follow-up was due to the effect of drinking per se, as many patients may return to drinking and indeed, may drink at levels approaching pre-treatment levels. Laboratory studies have shown that alcohol increases depression in alcoholic subjects (Warren and Raynes, 1972; Mendelson et al., 1964; Goodman and Gilman, 1975).

Hasin et al (1989) reported on a two year follow-up of affectively disordered patients with current alcoholism, previously described in Hasin et al (1985). Apart from the study by Hatsukami and Pickens (1982), which examined the effects of current depressive symptomatology on drinking outcome, the study by Hasin et al (1989) is the only study to have examined the effect of current additional psychiatric diagnosis on drinking outcome. Using a structured interview, the Longitudinal Interval Follow-up Evaluation, 84.3% of the original patients were interviewed every six months over a period of two years. Remission from alcoholism was defined as "26 weeks or more with no evidence of any RDC alcohol symptoms". Relapse was defined as "any occurrence of RDC alcohol symptoms following 26 weeks of remission".

The authors calculated the cumulative probability of remission from RDC alcoholism to be 0.67 at two years. In general, patients continued to remit throughout the two year period. Of the 48 patients who had not remitted during the two years, eight were dead at the end of follow-up: four had committed suicide, two had died whilst heavily intoxicated, one died of cancer and one had been shot.

Outcome was quite variable for these depressed alcoholic patients. Roughly two thirds of these patients

remitted after treatment and continued to do well, whereas those who did not remit tended to have a poor prognosis with several committing suicide or dying whilst intoxicated. Unfortunately, no information was given on alcohol consumption or depression during outcome and this in combination with the rather categorical definition of remission and relapse, limits the analysis of change in drinking and in mood or depression over time. Higher alcohol dependency and having a diagnosis of schizo-affective disorder were associated with poor outcome. Antisocial personality disorder was not associated with poor outcome in this study. It may be that depressed alcoholics with antisocial personality disorder are in some way different from those without depression and that this combination of diagnoses may lead to a better prognosis. This result is in contrast to Rounsaville et al's (1987) finding that alcoholics with antisocial personality disorder had a poor prognosis. The latter authors did not differentiate between depressed and non-depressed alcoholics with antisocial personality disorder which may account for the difference in results.

1.5.1 Summary

Only one of these studies examined the effect on outcome of a current diagnosis of depression, as opposed to a lifetime diagnosis, on outcome in alcoholism (Hasin et al., 1989). Unfortunately, this study only examined remission from alcoholism and as such, drinking outcome is overly rigidly defined. It also considered the effect of depression as a diagnostic category, rather than depression as a condition with varying degrees of severity. In addition, this study did not compare

depressed alcoholics with alcoholics alone, so the effect of depression on outcome cannot be properly evaluated.

However, one study did examine current depressive symptomatology and found that depressive symptoms increase as relapse rates increase during follow-up. Those alcoholics who remain abstinent score within the normal range on a self-rated depression scale, but those who had relapsed scored significantly higher on the scale.

The outcome for patients with alcoholism and any additional disorder, whether current or lifetime, appears to be poorer than for those with alcoholism alone. Having an additional diagnosis of affective disorder appears to worsen prognosis. There is some evidence that more treatment for both drinking and depression is given at follow-up for those with co-existing affective disorder, although on measures of drinking outcome, no differences were observed.

One study (Rounsaville et al., 1987) found that men with lifetime diagnosis of alcoholism and affective disorder have a poorer prognosis than men with alcoholism alone. This pattern was reversed for women: those with an affective disorder had a better prognosis than those with alcoholism alone.

The evidence suggests that severity of dependence on alcohol and additional psychopathology, such as an affective disorder and antisocial personality disorder, have prognostic value in groups of alcoholic patients. The prognostic significance of a current diagnosis of depression in alcoholic patients is unknown.

1.6 THE TRANSMISSION OF ALCOHOLISM AND DEPRESSION

In order to explore further the relationship between alcoholism and depression, evidence for the familial transmission of the co-occurrence of alcoholism and depression will be reviewed. The relative contributions of genes, environment and their interaction, family studies, studies of twins and families with adopted children have been used to gain information on the relationship between alcoholism and other psychiatric disorders.

Genetic factors and sharing the same environment can endow members of a family with a predisposition to the same disease. The prevalence of mental illness among the relatives of alcoholics has been used to elucidate the relationship between depression and alcoholism.
Although single genes have been found to be responsible for some disorders, the blended effect of several genes (polygenic traits) often accounts for some disorders (Williams, 1988). Diseases are however more likely to be the product of a combination of environmental, single genes and polygenic traits.

1.6.1 Familial Incidence

Cotton (1979), in a widely cited review of 39 studies found that the incidence of alcoholism was substantially higher in the relatives of alcoholics than in the relatives of non-alcoholics. However, depression and psychopathic features, variously described, were also found in families of alcoholics. Cotton briefly reviewed the studies of Winokur and his colleagues on the relationship between the familial incidence of alcoholism and depression, concluding that a "strong relationship

was found between alcoholism and affective disorder in the relatives of patients having affective disorder".

For example, Pitts and Winokur (1966) in a family history study of both male and female probands, selected affectively disordered and alcoholic patients from admissions to a psychiatric hospital. They selected 62 index alcoholics, 25 of whom were women, and matched these patients with 62 medically ill controls. Alcoholism was significantly more frequent in the fathers but not the mothers of the alcoholic probands compared to the matched control group. Affective disorder and alcoholism were reported significantly more frequently in the siblings of alcoholics than in the siblings of the controls.

This study also reported on the incidence of alcoholism and affective disorder in 366 affectively disordered probands, matched with 180 controls. There was a significant excess of affective disorder in both parents of the depressed probands compared to the control group. The depressed probands had a significant excess of alcoholism in their fathers compared to the matched controls. There was also a significant excess of affective disorder and alcoholism in the siblings of the depressed probands compared to the siblings of the control group.

The overall findings were therefore an excess of alcoholism in the fathers and an excess of both affective disorder and alcoholism in the siblings of the alcoholic probands. In the depressed probands there was an excess of affective disorder in mothers, fathers and siblings. In addition, alcoholism was more common in the fathers

and siblings of the affectively disordered probands compared to their matched controls.

In a study of 259 hospitalised alcoholics, including 103 females, personal interviews were carried out on 507 first degree relatives (Winokur et al., 1970) to investigate the possibility that there was a sex difference in the prevalence of alcoholism and affective disorder in the relatives of male and female probands. Alcoholism was more prevalent in male as opposed to female relatives of the alcoholic probands, whereas affective disorder was more frequently seen in the female relatives of the alcoholic probands. Sociopathy was significantly more prevalent in the male rather than female relatives of alcoholic probands.

In a later study of 100 unipolar depressive probands (Winokur, Cadoret et al. 1971, 1975), family members were personally interviewed in order to assess the prevalence of psychiatric disorder in first degree relatives. The authors found that there was a significantly greater risk for depression in the females compared to male relatives of depressed probands. Male relatives were more likely than females to have a diagnosis of alcoholism or sociopathy. The probands were divided into early and late onset depression (ie. before and after 40 years of age). Taking into account that the risk of depression will vary according to age and sex, familial affective disorder was significantly greater in the female rather than the male relatives of the early onset probands. Familial alcoholism was significantly greater in the early onset depressed probands compared with the late onset probands. This was in contrast to depressives who had a later onset of illness where no significant

difference was noted in the prevalence of affective disorder in the male and female relatives of late onset depressive probands. Winokur and his colleagues suggested that the early onset group be considered as representing a type of depressive illness which he called "depressive spectrum disease" and the second group, "pure depressive disease". In depressive spectrum disease, the illness would "appear to be not limited only to depression but also associated with alcoholism and sociopathy". Essentially, depressive spectrum disease would manifest itself usually in early onset women and there would be an excess of depression in female relatives and an excess of alcoholism and sociopathy in male relatives. In pure depressive disease, typically represented as males with late onset depression, there would be no excess of antisocial disorder or alcoholism in relatives and there would be equal rates of depression in male and female relatives.

The data appeared to support a familial association between alcoholism and depression. Winokur's hypothesis of a depressive spectrum disease suggested that the two disorders may be aetiologically related to one another.

Cloninger (1979) examined Winokur's data further, comparing it to other studies, including his own. He found that the primary diagnosis of the proband was important in determining the diagnosis of first degree relatives. If an alcoholic proband had a primary diagnosis other than alcoholism, for example antisocial personality disorder, Cloninger found that the relatives would be significantly more likely to have the same diagnosis as the proband rather than a diagnosis of depression or alcoholism. The diagnosis of relatives was

therefore more likely to reflect the primary diagnosis of the proband, whether this was alcoholism or not. It was argued that where there were sex differences in the prevalence of alcoholism, regardless of the primary diagnosis, this difference reflected the expected sex distribution of alcoholism in the population.

Cloninger's re-examination of Winokur's data demonstrated that primary depressives with alcoholism had fewer alcoholic relatives than primary alcoholics. Consequently, it is unlikely that early onset primary depression and primary alcoholism were alternative expressions of the same underlying familial disposition. According to the depressive spectrum hypothesis, any differences between alcoholism and depressive disorders are due to non-familial factors, and the two disorders are indistinguishable in terms of their family histories of psychiatric disorder. Cloninger's re-examination of Winokur's data suggests that the co-occurrence of depression and alcoholism is not fully accounted for by the depressive spectrum hypothesis and that non-familial factors, such as sex, may affect the prevalence of these disorders.

In a later study examining the nature of the association between alcoholism and depression, Merikangas et al (1985) found that depressives with no history of alcoholism did not transmit alcoholism. However, probands with alcoholism and depression tended to transmit both depression and alcoholism. Depressed probands were classified according to the presence or absence of alcoholism. Of the 215 probands, 19 of the depressed group were alcoholic. The onset of depression in these 19 probands preceded the onset of alcoholism as

determined by a modified version of the SADS-L. Direct interviews of first degree relatives were carried out in 30% of cases, otherwise information was obtained from medical records and family history information was taken from multiple informants. The depressed probands were divided into two groups: those with alcoholism, and those without alcoholism. These groups were then compared with a control group who had no evidence of psychiatric disorder.

Rates for all psychiatric disorders were highest in probands with depression and alcoholism compared to probands with depression alone. The relatives of probands with depression and without alcoholism had significantly higher rates of depression, anxiety disorders, and antisocial personality disorder than did normal controls. However, there was no difference in the prevalence of alcoholism in the relatives of depressed probands without alcoholism and normal controls. The increased rate of alcoholism in the relatives of depressed probands was therefore accounted for by the presence of alcoholism in the proband.

If depression and alcoholism were alternate manifestations of the same underlying disorder, as suggested in the depressive spectrum hypothesis, then subjects with depression only would be equally likely to transmit alcoholism and depression as subjects with depression and alcoholism. Merikangas' findings suggest that this is not the case: depressives who then develop alcoholism transmit both alcoholism and depression but those with depression alone do not transmit alcoholism. This study's findings agree with those of Cloninger et al (1979) who argued that alcoholism and depression were

transmitted independently in a sample of probands who were alcoholic rather than depressed.

Penick et al (1987) interviewed 568 male alcoholics using the Psychiatric Diagnostic Interview to determine the diagnosis of the probands and with a structured interview, determined the prevalence of psychiatric diagnosis in their first degree relatives. The probands were then divided into two groups according to whether they had a positive history of alcoholism in their relatives or a negative family history of alcoholism. Most of the probands (65%) had a positive family history of alcoholism in their relatives, where at least one first degree relative had experienced problems with drinking at some time in their lives. These probands and their relatives, were more likely to have had psychopathology, other than alcoholism. They were more likely than those without a family history of alcohol abuse to have had an earlier onset of problem drinking and also to have experienced greater impairment in social and occupational functioning. They were also more likely to meet diagnostic criteria for other psychiatric disorders such as depression, drug abuse, antisocial personality disorder, panic attacks and obsessive compulsive disorder.

The relatives of this group, male or female, were more likely to have had a diagnosis of depression, mania, hypochondriasis, panic attacks, schizophrenia and suicide attempts than the relatives of alcoholics without a positive family history of alcoholism. Problem drinking and antisocial personality disorder were significantly associated with the male relatives, not the female relatives, of the probands with a family history of

alcoholism. This study therefore underlined the psychiatric heterogeneity of alcoholics with a family history of alcoholism and underlined that this heterogeneity is mirrored in their first degree relatives. In this study, there was therefore a co-occurrence of alcoholism and a range of psychiatric disorders in a sub-group of alcoholics and their families.

These studies, spanning several decades, differ in methodology, probands studied and in diagnostic criteria used to determine the prevalence of psychopathology. Some studies collected information about relatives using the family history method, whilst others interviewed relatives directly. Later studies have benefited from using more widely accepted diagnostic criteria and structured interviews (Penick et al., 1987; Merikangas et al., 1985).

In summary, it would appear that later studies, using more reliable psychiatric diagnostic criteria, have found that the primary diagnosis of the proband is likely to be reflected in the psychiatric history of first degree relatives. Alcoholics who have a family history of alcoholism are likely to suffer from a variety of psychiatric disorders. Their relatives, of both sexes, are also likely to have other psychiatric disorders. Depressed probands, on the other hand, are no more likely to have a positive family history of alcoholism than normal controls. It seems unlikely that depression and alcoholism are alternative manifestations of the same disorder as they are not transmitted equally in the relatives of depressed or alcoholic probands. The mechanism by which these disorders are transmitted is

unclear, although given the heterogeneity of psychiatric disorders associated with alcoholism, it would seem that environmental influences (family life), in addition to genetic factors, are likely to account for the association between alcoholism and depression.

1.6.2 Adoption Studies

The most powerful evidence of a genetic contribution of alcoholism is likely to come from studies of children who were adopted from their biological parents at an early age. Where children of alcoholic parents have been reared apart from their parents from an early age, the effects of environmental influences arising from living in a home with an alcoholic parent are minimised.

Goodwin et al (1973), out of a large pool of Danish adoptees, who had been separated from their biological parents before six weeks of life, selected 55 adoptees who had a biological parent hospitalised for alcoholism. The group of adoptees with an alcoholic biological parent were compared to two control group of adoptees which were combined for the purposes of analysis. The control groups were one group whose biological parents had no history of psychiatric hospitalisations and a second group who had a biological parent who had been hospitalised, but with a diagnosis other than alcoholism. They found that the adopted sons of alcoholics were nearly four times more likely to be alcoholic than the adopted sons of non-alcoholics.

There were no significant differences between the control group and the adopted sons of alcoholics in the prevalence of psychiatric disorder, other than alcoholism. The finding raised the possibility that

there may be a genetic component in the transmission of alcoholism.

Goodwin et al (1974) then compared the adopted-away sons of alcoholics with their own brothers who had been raised by the biological parent. A total of 85 subjects were interviewed: 50 non-adopted controls subjects selected from census records and 35 siblings of adopted sons of alcoholics. Alcoholism rates and problems associated with alcohol were found to be similar in the two groups. However, although the length of exposure to the alcoholic parent was not associated with the development of alcoholism, increasing severity of alcoholism in the biological parent was related to an increased tendency towards developing alcoholism in the sons. The adopted sons, in this study, had significantly more overall psychopathology (excluding alcoholism) and significantly more personality disturbance than the non-adopted siblings. This suggested that the presence of other psychopathology may be related to environmental factors in this group.

Goodwin and his colleagues argued that their results indicated that environmental factors did not significantly contribute to the development of alcoholism in the sons of severe alcoholics.

However, two weaknesses of this work have been noted (Murray et al., 1983). There was a poor follow-up rate which may have biased the results in favour of those who were not problem drinkers. Secondly, the definition of alcoholism which, if widened to include problem drinking, was found to negate the evidence for a genetic predisposition for alcoholism. In Murray at al's (1983) reconstitution of the data, the control group adoptees

were more frequently categorised as "heavy" or "problem drinkers" than the index adoptees. This result would then contradict Goodwin's finding and would suggest that alcoholism, and less severe alcohol abuse are not genetically determined.

Cadoret and Gath (1978) in an adoptee study designed to examine the relationship between childhood behaviours and a diagnosis of alcoholism in later life, also examined the relationship of the biological parent's diagnosis to childhood and adult behaviours in the adopted-away sons. There was a significant relationship between primary alcoholism in the adult adoptee and having a biological parent who was alcoholic. However, there was no significant relationship between a secondary diagnosis of alcoholism and having a biological parent who was alcoholic. There was also no relationship between the presence of psychiatric disorder in the biological family and alcoholism in the adoptee.

Although there is some evidence that genetic factors predispose individuals to developing alcoholism in adulthood, some of these adoption studies do not clearly substantiate the genetic transmission hypothesis. There is evidence that parental psychiatric history of disorders, other than alcoholism, does not increase the risk of alcoholism in adult adoptees beyond that of normal controls.

1.6.3 Twin studies

Methodologically twin studies are useful in answering questions of inheritance of traits as the method relies on the fact that monozygotic (MZ) twins will share exactly the same set of genes, whereas nonidentical, dizygotic (DZ) twins are no more alike than

siblings. It is reasoned that if MZ twins are found to be more similar than DZ twins for a given characteristic, then the excess concordance is assumed to be due to their greater genetic similarity.

Twin studies have found evidence supporting the view that genetic factors play a role in the development of adult drinking patterns (Kaprio et al., 1987; Heath and Martin, 1988). Genetic factors were more influential than shared environment in accounting for weekly alcohol consumption in twin teenagers once alcohol consumption had begun (Heath and Martin, 1988). Kaprio et al (1987) used hierarchical linear regression to demonstrate that, among co-twins in the community, genetic factors still contributed to shared alcohol drinking patterns, even after the effects of age and frequency of social contact were removed.

In the United Kingdom, the Maudsley Hospital Twin Register has been used to investigate the inheritance of various psychiatric disorders, including alcoholism. This register consists of twins who have attended the hospital since 1948. Gurling et al (1984), using the SADS-L to yield RDC diagnoses of alcoholism selected 28 MZ and 28 DZ twins. The concordance rates for alcohol dependence in MZ twins was 21% and for DZ twins 30%, indicating no evidence of a genetic effect in the development of alcoholism. Seventeen of the co-twins were also alcoholic. Of these 17, eleven (65%) were depressed. Of the remaining 39 (non-alcoholic) co-twins, 20 (51%) were also depressed. These results show that depression can occur, at a high rate, independently of alcoholism in co-twins of alcoholic probands. The high prevalence of depression in the non-alcoholic co-twins

demonstrates that the occurrence of depression in twins, concordant for alcoholism, is not strictly due to genetic factors but also due to common environmental factors.

In general, concordance for all diagnoses other than alcoholism was greater in MZ twins (48%) than in DZ twins (21%). The authors concluded that there was a considerable amount of depression in the alcoholic probands and in their co-twins, whether alcoholic or not. Unlike the first published twin study (Kaij, 1960), they found no evidence that a genetic effect exists for the transmission of alcoholism. Gurling et al's (1984) sample differs in several ways from Kaij's (1960) sample. It contained both men and women, was based on a psychiatric hospital population and different diagnostic criteria was used. Kaij's sample was based on male twins in Sweden where one or both twins had been reported to the Temperance Board. Alcoholism was medically rather than socially defined as the presence of at least two symptoms such as blackouts and physical dependence. It is possible that Kaij's sample was biased towards alcoholism associated with antisocial behaviour as individuals were often registered with the Temperence Boards by authorities such as the police.

Mullan et al (1986) investigated the relationship between alcoholism and neurosis by selecting MZ and DZ twins who had an ICD 8 (International Classification of Diseases) (W.H.O., 1965) diagnosis of episodic drinking, habitual excessive drinking, alcohol addiction or any form of alcoholic psychosis. Approximately one third of the alcoholic probands had a lifetime diagnosis of a neurotic illness, including neurotic depression, panic disorder and obsessive compulsive disorder. For their

co-twins, neurotic disorder was twice as common in those who also had a diagnosis of alcoholism compared to non-alcoholic co-twins of alcoholic probands. This pattern was irrespective of zygosity of the co-twin and alcoholism was found to be independent of concordance for neurosis. The excess of drinking in alcoholic compared to non-alcoholic co-twins appeared to have resulted from alcoholism rather than a genetic predisposition to neurosis having "caused" alcoholism. In each pair of twins, Eysenck's neuroticism score was higher in the twin who was more dependent on alcohol, indicating that alcoholism may result in higher neuroticism scores.

From these twin studies, there is some evidence that the pattern of drinking, rather than alcoholism per se, may be genetically determined (Kaprio et al., 1987; Heath and Martin, 1988). Alcoholism and psychopathology were investigated in two studies (Gurling et al. 1984; Mullan et al., 1986). Depression and neurosis appear to be prevalent in the co-twins of alcoholic probands. The evidence suggests that the association is more likely to be due to environmental rather than genetic factors as neurotic disorder was found in the co-twins of alcoholic probands, whether or not they themselves were alcoholic and regardless of zygosity.

1.6.4 Summary

Taking the evidence from family, adoption and twin studies together, alcoholism and depression do occur together within the same families. Alcoholics are likely to suffer from a wide variety of psychiatric disorders, as are their first degree relatives. Alcoholics with a family history of alcoholism, and their relatives, are more likely to show other psychopathology than are

alcoholics without a family history of alcoholism. There is no strong evidence that alcoholism per se is a disorder which is transmitted genetically. Nonetheless, there is some evidence from adoption and twin studies that patterns of drinking may be, in some part, genetically determined. There is no convincing evidence that the co-occurrence of alcoholism and neurotic disorder, including affective disorders, is genetically determined. Rather, where a relationship is found, the association appears to be due to other factors which are environmental and perhaps familial in nature.

1.7 ALCOHOL AND MOOD

Two popularly held beliefs are first, that people drink alcohol because they are low in mood and that alcohol will enable them to "drown their sorrows" and second, that people drink to reduce anxiety. If this were found to be true, then alcoholism and depression might be regarded as being causally related. However, it has not yet been clearly established that people actually do drink alcohol for its mood altering properties.

Of the studies which have investigated the relationship between alcohol and mood, the majority have concentrated on the relationship between alcohol consumption and the behavioural and psychological response to stressful stimuli. Consequently, there has been a greater emphasis on the effect of alcohol on anxious rather than depressive affect.

To study the effect of alcohol on mood, a similar procedure has been used across studies. Subjects have been administered alcohol and, at various points in the procedure, standardised self-rating scales of mood and in some studies, measures of physiological arousal have been taken.

Experimental studies have shown that there are a wide range of conditions which influence affective changes induced by alcohol. For example, in common with other studies of emotion, there are inconsistent findings between measures of physiological arousal, subjective reports of emotions and behaviour (Wilson et al., 1980). Physiological state may be only one determinant of reported emotion and changes in self-reported affect may not correlate with other physiological changes.

Determinants of self-reported mood changes in response to alcohol include the dose of alcohol (Williams, 1966; Warren and Raynes, 1972), the phase of the subjects' blood alcohol concentration (McCollam et al., 1980), prior mood state (Russell and Mehrabian, 1975), subjects beliefs about the effect of alcohol (Rohsenow, 1983), the context within which drinking takes place (Pliner and Cappell, 1974) and the instructions given to subjects (Marlatt et al., 1973).

More recent research has employed a "balanced placebo" design to enable the instructional and pharmacological effects of alcohol to be distinguished from each other. Two studies using a balanced placebo design have shown that despite similar beliefs about the effect of alcohol on anxiety, males and females respond differently in interactions involving social evaluation (Abrams and Wilson, 1979; Wilson and Abrams, 1977). These two studies illustrate that sex may differentiate the effects of alcohol on anxiety or arousal, although the authors were cautious in interpreting the results as indicating a sex difference *per se*. It is possible that the difference found was due to cognitive factors: women perceiving the social consequences of intoxication as negative and exercising increased self restraint in order to produce a more effective coping response.

Despite this wide range of conditions that can influence both the nature and extent of mood changes induced by alcohol, the results of these studies have been broadly in agreement: at low doses of alcohol, mood is enhanced, with subjects reporting happiness, relaxation and even euphoria whereas at higher doses, subjects report more dysphoric mood states such as

anxiety and depression (Freed, 1978; Russell and Mehrabian, 1975; Tucker et al., 1982). Given that these findings indicate that drinking at high doses may be aversive, they do not explain why individuals are motivated to drink heavily. One possibility is that dependent drinkers are insensitive to these effects of alcohol on mood. Another possibility is that they experience some relief from the aversive consequences of drinking, including affective disturbance at higher doses of alcohol.

1.7.1 Alcohol and Stress

Multiple factors such as social, cultural and physiological may underly the consumption of alcohol. In recent years, there has been a growing body of literature on psychological theories which have been helpful in exploring the relationship between stress and drinking. Some of these theories, such as the tension reduction theory (Conger, 1956) and self-awareness theory (Hull, 1981), may increase our understanding of the relationship between alcohol and mood.

The original tension reduction theory (Conger, 1956) originated from drive reduction theory and proposed that increased tension is a heightened drive state. Alcohol is consumed for its tension reduction properties and has the property of lowering drive by reducing tension. Tension reduction then acts as a reinforcer of drinking behaviour.

The tension reduction model proposes that alcohol alters behaviour by its direct pharmacological effects on affective and motivational states. The self-awareness model (Hull, 1981), in contrast, assumes that some of the causes and effects of alcohol cannot be explained by the

pharmacological effects of alcohol alone and this model proposes that alcohol affects behaviour indirectly by reducing self-awareness. Alcohol is assumed to decrease negative self-evaluation following failure. This is assumed to be a sufficient condition to induce and sustain alcohol consumption.

1.7.1.a *The Tension Reduction Hypothesis*

In an early review, Cappell and Herman (1972) concluded that there was little support for the proposition that alcohol reduces tension. A more recent review by Cappell and Greeley (1987) agreed with Hodgson et al (1979) that there is some evidence that alcohol reduces tension but the findings are often contradictory with respect to the tension reduction hypothesis. The current priority is to establish under what conditions alcohol reduces tension.

Studies which have examined whether alcohol reduces tension in human subjects have used two main categories of stressors, noxious stimuli (pain) or social stressors (social interaction). The following studies have investigated the tension reducing properties of alcohol, whilst assessing the contribution of drinking history and personality factors to tension reduction in individuals.

Noxious stimuli have been used to examine the difference between heavy and moderate drinking on the effect of alcohol and tension reduction. For example, problem drinkers were reported to experience more pain reduction at high doses of alcohol than at lower doses whereas moderate drinkers experience pain reduction at lower doses of alcohol and were more susceptible to pain at higher doses (Brown and Cutter, 1977). The connection between the tension reduction hypothesis and pain

attenuation is based on the assumption that pain relief by an analgesic drug involves a reduction in the emotional reaction component. The greater analgesic effect of alcohol experienced by problem drinkers compared with moderate drinkers may be related to their specific capacity to gain relief from aversive stimulation at high doses. Why this mechanism for reducing the emotional reaction component should be different in the two groups remains obscure.

Prior drinking history has been found to be unrelated to the effects of dose of alcohol in an anxiety arousing social interaction (Wilson et al., 1980) but the effect of alcohol on anxiety was found to be dependent on tolerance to alcohol (Lipscomb et al., 1980). Compared to subjects low in alcohol tolerance, subjects with high tolerance had a much lowered heart rate at high doses of alcohol. Other measures of anxiety failed to demonstrate this effect. It is possible that the stress manipulation was ineffective in subjects low in tolerance.

Another study investigated the relationship between risk for alcoholism and the effect of alcohol on response to a speech stressor (Sher and Levenson, 1982). Based on scores on personality inventories which have been shown to detect those at risk for alcoholism, subjects who were out-going, aggressive, impulsive and antisocial had a more pronounced reduction in tension after alcohol consumption that was not found in low risk subjects. Sher and Levenson suggested that this response to alcohol might provide a unique opportunity for tension reduction as a mechanism for reinforcement in high risk subjects. These results along with those of Brown and Cutter (1977) and Lipscomb et al (1980) suggest that some individuals

are more likely than others to be sensitive to the tension reducing properties of alcohol and thereby more vulnerable to developing alcoholism. There is a suggestion that there may be a biological or genetic basis for these differences. However, there is some evidence that the association between tension reduction and pre-alcoholic personality characteristics may not be a robust finding (Schuckit et al., 1981; Sher, 1987).

The evidence for the tension reduction hypothesis appears to be mixed. It appears that alcohol does reduce tension more effectively in individuals who are at risk for alcoholism than in low risk individuals. In addition, there is some evidence that high doses of alcohol may have tension reducing properties in heavy drinkers and in individuals with high tolerance to alcohol.

1.7.2 Alcohol and Depression

The literature on the prevalence of depression in alcoholism reveals a strong association between affective disorder and drinking. The extent to which negative affect or mood disorder is a motivating factor in drinking is relatively unexplored and unclear.

There appears to be a dose-dependent biphasic effect of alcohol on emotions: at moderate doses, alcohol would increase positive emotions whereas at large doses, alcohol would be likely to be associated with dysphoric mood states (Russell and Mehrabian, 1975; Freed, 1978; Tucker et al., 1982). Russell and Mehrabian (1975) hypothesised that a depressed individual should experience relief from depression at moderate blood alcohol concentrations but should experience an increase in dysphoria at higher levels of blood alcohol. This

implies that depressed drinkers would only find relief from dysphoria at low levels of drinking. Although this hypothesis does not explain why some alcoholics persist in drinking when depressed, it may partially account for the initiation of drinking.

Two studies have specifically examined the effect of experimentally manipulated depression on alcohol consumption. In one study, subjects mood was manipulated by being told that they had either done poorly or well on an intelligence test (Pihl and Yankofsky, 1979). The mood manipulation was successful as self-ratings of anxiety and depression were higher in those subjects who had been told they had done poorly. The results were opposite to what would be predicted from the tension reduction hypothesis: those subjects who experienced negative affect drank less in a subsequent taste test procedure than control subjects whose affect was unchanged or slightly elevated. The result may thus provide some evidence for Russell and Mehrabian's hypothesis (1975).

Noel and Lisman (1980) conducted a series of experiments to investigate the relationship between alcohol consumption, depressed mood and learned helplessness. In the first of the series, male and female students were classified as light, moderate or heavy drinkers. Female subjects who scored at levels indicating at least mild depression on a depression inventory were found to be significantly more likely to be heavy drinkers than light drinkers. There was no comparable relationship between depression scores and drinking category in men suggesting a sex difference and as a result, the remainder of Noel and Lisman's studies

involved only women. Two subsequent experimental studies involved giving subjects unsolvable problems in a learned helplessness paradigm. Although the success of the learned helplessness manipulation was questionable, it did appear to induce increases in depression and hostility scores. In both studies, those subjects given unsolvable problems drank more beer in a taste rating procedure than did control subjects, although the actual amounts of beer consumed by both groups of subjects were small. The discrepancy between the results of this series of studies and those of Pihl and Yankofsky (1979), suggest that negative affect might best be considered as a limited source of motivation for drinking. There may however, be conditions or situations in which the consumption of alcohol may be affected by negative mood.

1.7.2.a *Self-Awareness Theory*

Hull (1981) proposed that alcohol reduces self-awareness by interfering with the encoding of information relevant to a state of self-awareness. This is then assumed to decrease an individual's sensitivity to self-relevant cues in the environment regarding appropriate behaviour and the ability to evaluate past behaviour through feedback. As alcohol inhibits information that would act as a source of self-criticism and negative affect, it is regarded as providing psychological relief. This process is then assumed to both induce and sustain alcohol consumption.

Research in this area has suggested that alcohol impairs the acquisition of new information and that individuals who are high in self-consciousness are more likely to recall self-relevant material than subjects low in self-consciousness under placebo conditions but when

alcohol has been consumed no differences are noted between high and low self-conscious subjects (Hull, 1987). One investigation arising from self-awareness theory (Hull and Young, 1983) found that the consumption of alcohol of subjects high in self-consciousness varied as a function of whether they had been given positive or negative feedback of results of an intelligence type task. Consumption of alcohol of subjects low in self-consciousness did not vary as a function of success or failure. These results suggested that alcohol is consumed as a function of the quality of past performance and of the subjects degree of self-consciousness. After completing this part of the experiment, subjects took part in another ostensibly unrelated experiment in which they rated their mood and took part in a wine tasting procedure. Those individuals who were high in self-consciousness and who had received negative feedback in the previous experiment reported significantly more negative mood and drank more wine than those high in self-consciousness who had received positive feedback. Mood scores and amount of alcohol consumed by those low in self-consciousness were found to fall in between the extremes and not to vary according to feedback given.

Although research on the self-awareness model has been largely limited to males and to experimental studies under laboratory conditions, the model has also been used to predict patterns of alcoholic relapse (Hull et al., 1986). Alcoholics high in self-consciousness who experience life events indicative of personal failure were predicted to resort to alcohol use more than both high self-conscious individuals who experience positive self-relevant life events and individuals low in self-

consciousness. Thirty-five males who had received in-patient detoxification were followed-up at three and six months.

Although the predictive ability of the variables was less at six months follow-up, at three months those high in self-consciousness who had experienced self-relevant negative life events were more likely to be drinking at levels similar to pre-treatment compared with the other groups. Whilst these results are consistent with the self-awareness theory, they do not demonstrate that the stress reducing effects of alcohol are mediated through self-awareness.

Self-relevant information is known to receive a greater amount of automatic processing than neutral information (Geller and Shaver, 1976). The interaction of active attentional and automatic processing of self-relevant information was studied by Bargh in a dichotic listening task (Bargh, 1982). It was found that there was an automatic attention response to self-relevant information presented outside of the subjects awareness indicating that self-relevant information was processed automatically. These findings suggest that information that is self-relevant will be processed automatically. This casts some doubt on Hull's theory that individuals will vary in the degree to which they respond to self-relevant information. Hull (1987) proposes that alcohol can affect behaviour indirectly through cognitive processes. More specifically, the theory proposes that alcohol reduces self-awareness by inhibiting the use of information-processing strategies essential to the self-aware state (Hull, 1987, p272). If information that is self-relevant requires little attentional effort and is

processed automatically as Bargh (1982) has shown, then Hull's theory may need revision. The encoding processes that are supposed to be disrupted by alcohol are the sensitivity to and selection of self-relevant information. These processes may be disrupted but further analysis of how these processes affect behaviour with alcohol consumption needs to be detailed (Wilson, 1983).

In addition, the results of two experiments by Keane and Lisman (1980) on the effect of alcohol and a placebo beverage on socially anxious males in a social situation in which they were instructed to make a favourable impression on a female confederate do not fit with the model proposed by Hull. In the first experiment, they found that alcohol significantly increased self-evaluative worries in subjects when compared to those who had the placebo beverage. Subjects who had received alcohol also rated the quality of their performance and seemed to be unaware of the deterioration in their performance which would confirm Hull's notion of alcohol reducing negative evaluation following failure. However, in a second experiment, alcohol subjects rated their performance in the social interaction as being inferior compared to controls, indicating a degree of self-awareness. This finding that alcohol did not blunt self-awareness does not fit with Hull's self-awareness model.

Despite these criticisms, both the tension-reduction model and the self-awareness model have in common the assumption that alcohol is consumed to avoid or escape negative affective states. As such, there are some similarities between these theories. However, in the self-awareness model, alcohol is assumed to produce a

change in affective state indirectly through cognitive processes, whereas in the tension reduction model, alcohol is assumed to have a direct physiological effect on arousal. The mechanism by which alcohol reduces arousal is still unknown. Nonetheless, the factors which influence the effect of alcohol on stress are beginning to be delineated.

One hypothesis related to the findings, arising from the self-awareness and tension reduction models, is that alcoholics who are also depressed will be more likely than those who are not depressed to be motivated to consume alcohol in order to reduce negative affect and the negative bias in cognitive processes.

CHAPTER TWO

LITERATURE REVIEW

2.0 COGNITIVE THEORY OF DEPRESSION

Cognitive theories of psychopathology have become increasingly abundant over the past two decades. The major postulate of cognitive theories is that experience is translated into meaningful internal representations through cognitive processes (Gilbert, 1984). Put simply, we cannot know things in themselves but can only know our interpretations of events. In depression, this internal representation of reality, which is the product of cognitive processing, is regarded as being potentially pathogenic and the cognitive processing of information can be regarded as the pathogenic agent. One of the most influential of the cognitive theories of emotion has been Beck's cognitive theory (Beck, 1967).

2.1 BECK'S COGNITIVE MODEL

According to Beck's cognitive model (Beck, 1967; Beck et al., 1979; Beck, 1987) depressed individuals, and those who are at risk of becoming depressed as adults, have acquired dysfunctional cognitive schemata as a result of certain types of negative experiences in childhood. In adulthood, these cognitive schemata become activated when the individual is exposed to negative events which in some way echo the experiences on which the early schemata were based. The content of depressogenic schemata are related to loss and once activated are assumed to affect the encoding, storage and retrieval of information. They determine the biases or distortion in information processing which shape the interpretation of experience. The biases in information

processing are implied from the negative content of thinking. Depressed individuals are seen as having negative perceptions of themselves, the world and the future (negative cognitive triad) and make systematic logical errors when processing information. These systematic processing errors include arbitrary inference, selective abstraction, overgeneralisation, minimisation and maximisation and personalisation. Kovacs and Beck (1978) have described these aspects of dysfunctional thinking in depression as disorders of the content of thought, the process of thought and the structure of thought.

This model of depression has led to specific predictions about the cognitive processing of information in depressed individuals.

2.1.2 Information Processing

Beck's theory predicts that depressed individuals will encode, store and retrieve information in a more negatively biased way than non-depressed individuals (Beck et al., 1979; Beck, 1967). Hollon and Kriss (1984) have proposed that depressed patients will have a negative expectation set due to their pre-existing negative cognitive schemata which affects the way in which stimuli are attended to, perceived, encoded and retrieved. Several studies have been reported which are experimental in design and a review of these shall be mentioned briefly here as they do not rely on self-report methods where there are greater difficulties controlling for external factors. Although these studies are largely inferential in nature in that the results are based on observations of verbal and behavioural reactions to stimuli, they may help in elucidating some of the

cognitive processes which Beck views as central to depression. Attention bias and retrieval from memory are two aspects of information processing in depression which are reviewed below.

With regard to attention bias in depression, it has been proposed that depressed individuals show an over-sensitivity to negative information. Gotlib and McCann's (1984) study found that mildly depressed students were significantly slower at naming the colours of negative words than manic (positive) or neutral content words in a modified Stroop word colour naming task. Non-depressed subjects, in contrast, showed no differential response latencies to the three word types. The differential response latency is considered to be the result of cognitive interference. The findings were consistent with the hypothesis that increased accessibility for negative constructs produced interference for the competing task of naming the colours of depressed content words in depressed individuals. These findings were replicated in depressed psychiatric patients (Gotlib and Cane, 1987). A more recent study (Gotlib et al., 1988), attempted to disentangle whether depressives selectively attend to the depressive content of stimuli or whether depressive content words are more elaborately processed as either of these processes could have accounted for the results of the above studies (Gotlib and McCann, 1984; Gotlib and Cane, 1987). Selective attention to one member of a pair of words was assessed in depressed and non-depressed subjects using tachistoscopic presentation of three types of pairs of words (manic-neutral, depressed-neutral and manic-depressed). The results were contrary to the authors predictions. Mildly depressed

subjects did not show an attentional bias to any of the three groups of words suggesting that depressed individuals do not selectively attend to negative stimuli and experiences. In contrast, the non-depressed subjects were found to attend to manic-content words more frequently than to neutral or depressed-content words. These results were interpreted as indicating that there was not a negative attentional bias in the depressed group, at least for early stage processing. Another interpretation of these results is that depressives do attend to negative stimuli but not in favour of other stimuli, whereas normal controls show a bias in attention to positive stimuli. Ingram et al. (1983) have suggested that one of the characteristics of depression is a deficit in the ability to process positive information rather than an oversensitivity to negative information.

There may be difficulties in generalising from findings from sub-clinical to clinical levels of psychopathology (Depue and Monroe, 1978). A study utilising P300 responses, assumed to be an electrophysiological measure of central processing, supported Beck's model of depression (Blackburn et al., 1990). The occurrence of the P300 wave is usually associated with an unexpected stimulus or with the absence of an expected stimulus. The size of the P300 wave is assumed to vary with the degree to which a stimulus is expected. Depressed patients were shown to have a smaller amplitude of P300 in response to negative words compared to positive words whereas normal controls showed the reverse response. Depressed patients, presumably due to their underlying negative schemata, were therefore oriented to receive and process negative

stimuli whereas normals expected to process more positive stimuli.

Models of depressive information processing suggest that depressives should have greater accessibility at a self-referent level to negative information and should therefore process negative information more efficiently than non-depressives (Ingram and Reed, 1986). Several studies have confirmed this hypothesis. Depressed patients recalled more self-referent negative adjectives than positive adjectives compared to non-depressed psychiatric and normal controls (Derry and Kuiper, 1981). Depressed in-patients have also been shown to be superior to controls in correctly identifying unpleasant words presented through a tachistoscope (Powell and Hemsley, 1984). However, mildly depressed students showed enhanced recall of both negative and positive self-referent adjectives (Kuiper and Derry, 1982) indicating that severity of depression may enhance the encoding of negative information although this should not be taken to imply that depressives are unable to process positively self-referent information.

In relation to autobiographical memory recall, there is evidence that increases in the severity of depression leads to increased likelihood of retrieving negative memories (Fogarty and Hemsley, 1983; Lloyd and Lishman, 1975) but these findings are difficult to interpret as they do not take into account objective differences in the base rate of negative events in the lives of the depressed and non-depressed control groups studied (Blaney, 1986). Clark and Teasdale (1982) overcame this problem by using within subject comparisons. They tested patients at two times of day where depth of depression

varied due to diurnal mood variation and found that whilst more depressed, patients recalled more unhappy and fewer happy memories compared to when less depressed.

The evidence described above suggests that these dysfunctional cognitive processes are stable characteristics of a categorical diagnosis of depression. However, there is some limited evidence that dysfunction in some cognitive processes, such as retrieval of memories (Lloyd and Lishman, 1975; Teasdale and Fogerty, 1979) and self-referent encoding (Dobson and Shaw, 1987) may be more state-dependent, that is, they may vary with the degree of depressive mood, regardless of diagnosis.

In a review of information processing, Ingram and Reed (1986) provided a useful summary of findings which suggested the following trends. Compared to non-depressed controls, depressives recall more encoded negative semantic information than positive information. In the processing of information relevant to self-knowledge and behaviour, there appears to be a bias leading to an under-estimation in the encoding of positive information rather than an over-estimation of negative information. These findings fit with those of Gotlib et al (1988) and are consistent with the results of some other studies which have investigated different aspects of depressed subjects functioning, such as self perception of social competency (Lewinsohn et al., 1980) and expectancy of success in a dice-rolling task (Golin et al., 1979) which indicated a positive or self-serving bias in non-depressed subjects and "even-handedness" in depressed subjects.

It is also important to note that altered information processing has been shown to occur in other

emotional disorders such as anxiety disorder, although the effect of anxiety in the processing of information may be different from that found in depression (Williams et al., 1988).

2.1.3 The Content of Thought in Depression

In Beck's model of depression, there is a negative bias in the content of thought which is manifested by a negative view of self, the world and the future. This has been labelled the negative cognitive triad. Depressive thinking is also thought to be characterised by the operation of depressogenic schemata which influence how an individual interprets environmental events (Beck, 1976). These depressogenic schemata of self-deprecation and self-blame lead to errors in thinking and the manifestation of cognitive and behavioural symptoms of depression. Various measures have been derived from the theory which have attempted to measure these descriptive aspects of cognitive dysfunction.

2.1.3.a *Frequency of Negative cognitions*

A measure of the frequency of negative cognitions, the Automatic Thoughts Questionnaire (ATQ)(Hollon and Kendall, 1980) has been shown to discriminate between depressed and non-depressed subjects (Blackburn et al., 1986(b)) and between depressed and remitted patients (Eaves and Rush, 1984). However in the latter study, the ATQ did not discriminate between endogenous and non-endogenous depressives, nor did it discriminate between depressed patients with unipolar and bipolar depression, and patients with depression secondary to substance abuse, in a study testing the specificity of depressed cognitions in clinical depression (Hollon et al., 1986).

The frequency of negative cognition, as measured by the ATQ, has also been shown to be state dependent rather than trait dependent (Simons et al., 1984; Blackburn et al., 1986(b)). These findings support Beck's model of depression and indicate that the high frequency of negative self-statements is a general characteristic of depressive thinking. This characteristic, however, does not feature in the thinking of non-depressed and recovered depressed subjects and as such the presence of these cognitions should be considered as a symptom, and not a predictor, of depression.

2.1.3.b *Negative Cognitive Style*

The Cognitive Style Test (CST)(Wilkinson and Blackburn, 1981) was designed to measure the three aspects of the negative cognitive triad, a negative view of self, world and future. Using a revised version of this scale, Blackburn et al (1986(b)) confirmed that depressed patients have a negative view of the world and of the future compared to non-depressed controls and recovered depressed patients. The self sub-scale of the CST did not however differentiate between recovered and depressed patients indicating that a negative view of self may be a relatively enduring aspect of depressive thinking. Compared to non-depressed controls, depressed patients and recovered depressives have been shown to describe themselves more negatively (Bradley and Mathews, 1988) although depressed patients have a more negative view of themselves when depressed than when in remission (Myers et al., 1989).

2.1.3.c *Hopelessness*

Another measure of depressive content of thinking is the Hopelessness Scale (HS)(Beck et al., 1974) which was

designed to measure one aspect of Beck's cognitive triad, namely a negative view of the future. Having a psychiatric disorder, particularly a diagnosis of depression or alcoholism is associated with a higher risk of suicide (Hawton, 1987). Hopelessness has been suggested to play a critical role in suicide (Beck, 1967) and the seriousness of suicidal intent has been found to be more highly correlated with a measure of hopelessness than with depression. In an analysis of consecutive suicide attempters, hopelessness was a more powerful indicator of suicidal intent than depression at the time of the index admission and the statistical association between intent and depression was due to their joint relationship with hopelessness (Minkoff et al., 1973). The measure of hopelessness used in this study was later to become the Hopelessness Scale. Other studies have also confirmed that hopelessness is an important predictor of eventual suicide in depressed patients with suicidal ideation (Wetzel, 1976) and in parasuicides (Dyer and Kreitman, 1984).

Hopelessness has been shown to be a better predictor of patients' wishes to die than depression in a group of depressed and schizophrenic patients (Kovacs et al., 1975). In a ten year prospective follow-up study of patients hospitalized for suicidal ideation (Beck et al., 1985(b)), those patients who committed suicide were compared with those who survived during the five to ten year follow-up. Although the suicides did not differ from those who survived on their initial scores on depression and suicidal ideation, those who eventually committed suicide were more pessimistic about the future than those who survived. Beck postulated that in

depression, hopelessness about the future may arise out of activation of underlying cognitive patterns and that even if individuals suffered from similar degrees of depression, there will be some who will develop more pessimistic attitudes towards the future than others. If hopelessness can be considered as an underlying schema activated in depressed mood, then those who are particularly high in hopelessness in one episode may be at increased risk of suicide not just for that episode, but also for episodes of depression in the future.

On the basis of five general population studies, Roy and Linnoila (1986) estimated the prevalence of suicide in alcoholics to be as high as 21.2%. Although chronic intoxication may in itself be a sufficient condition for suicide or a suicide attempt, there is evidence that alcoholics who attempt or complete suicide may also suffer from additional psychopathology, particularly depression (Murphy et al., 1979; Berglund, 1984; Hesselbrock et al., 1988).

Although it is likely that hopelessness may be an important factor in suicide in alcoholics, Beck and Steer (1989) in a prospective follow-up study examining clinical predictors of eventual suicide found that hopelessness, measured by the HS, did not predict eventual suicide amongst a group of patients hospitalized for suicide attempts. However, a diagnosis of alcoholism at the time of the index admission increased the risk of eventual suicide by a factor of five compared with the risk for non-alcoholics. Hopelessness, in this study was assessed after the index attempt and not retrospectively as has been done in some other studies (Dyer and Kreitman, 1984). It is therefore a possibility that

having survived a suicide attempt, subjects were less pessimistic about the future than they had been before making the attempt. Another difference is that the populations differed in both studies: one study investigated individuals who had attempted suicide (Beck and Steer, 1989), the other, patients with suicidal ideation (Beck et al., 1985(b)). It is possible that those patients with suicidal ideation differ from those who attempt suicide in that a previous attempt is considered to be the best indicator of increased risk of eventual suicide (Sainsbury, 1978).

Given that patients with a diagnosis of alcoholism are at risk of suicide and that moderate levels of hopelessness have been found in in-patient alcoholics (McGovern, 1986), the relationship between hopelessness, depression and suicide in alcoholics needs further exploration. Although Beck's model of depression would not predict that a negative view of the future in one depressive episode would necessarily recur in a future episode or have a bearing on the prediction of suicide, it is possible that hopelessness might remain a stable construct within individuals and across episodes. Those alcoholics who are also depressed may be particularly vulnerable to experiencing a sense of hopelessness which may place them at greater risk of suicide than those who have a diagnosis of alcoholism alone.

2.1.3.d *Depressive Schemata*

Beck's theory would predict that those at risk for depression will have acquired dysfunctional schemata through their negative experiences in childhood. A depressogenic schema is assumed to be an enduring or trait dimension in an individual's cognitive

organisation. A depressogenic schema is hypothesised to remain prepotent unless, as an adult, the schema is reactivated by an event similar in negative experience to that experienced in childhood. As a result of being reactivated, attention will be directed to those features which are consistent with the schema. Perception, thought and memory will tend to be dominated by the negative themes consistent with the schema.

According to Beck's theory, in depression the maladaptive schemata and negative cognitive patterns account for the affective, motivational, behavioural, vegetative as well as cognitive symptoms of depression. The concept of schemata is therefore fundamental to the cognitive theory of depression and depressogenic schemata are regarded as rendering an individual vulnerable to depression (Beck, 1987).

Utilising measures which are assumed to represent these underlying cognitive structures such as the Dysfunctional Attitude Scale (DAS)(Weissman and Beck, 1978) and the Cognitive Style Test (CST)(Wilkinson and Blackburn, 1981), several researchers have examined whether dysfunctional schemata act as vulnerability factors for depression. Beck's theory would predict that the schemata of individuals who have recovered from depression would remain more dysfunctional than the schemata of a control group.

Various researchers have emphasised the enduring aspects of depressogenic schemata and have investigated whether it is possible to identify a depressogenic cognitive style in individuals who are not currently depressed which would increase vulnerability to depression over time. Studies using the DAS (Simons et

al., 1985; Hamilton and Abramson, 1983) and the CST (Wilkinson and Blackburn, 1981) have found that the scores of recovered depressed patients do not differ significantly from those of control groups and where elevated scores are found in recovered depressed patients, doubt has been expressed as to the extent of recovery from depression (Teasdale, 1988). These findings do not fit readily with Beck's predictions and suggest that schemata may be characterised more as state dependent structures rather than as more persistent trait structures.

Other researchers have emphasised a different aspect of Beck's schema theory which has resulted in two other hypotheses concerning cognitive vulnerability to depression. One of these concerns both the latent and stimulus specific aspects of the schema and can be thought of as a schema-event vulnerability hypothesis (Stiles and Gotestam, 1988). The conjecture here is that depression develops when a depressogenic schema is activated by an event which is congruent with the schema. This has been called the principle of specificity (Zuroff and Mongrain, 1987). In addition, it is assumed that the schema can only be measured when it is activated.

In a longitudinal prospective study investigating the relationship between depression and life events, Hammen et al (1985) classified college students into schema types according to those who were predominantly dependent (dependent on others for gratification) and those who were self-critical (dependent on achievement for gratification) on the basis of a measure assessing the content of recalled stable self-schemata. These schemata types relate to Beck's personality structures of

autonomy and sociotropy (Beck et al., 1983). Beck's theory would predict that subjects whose self-schemata are predominantly dependent will be more likely to experience negative interpersonal events as more depressing than negative achievement events. Those subjects whose self-schemata are predominantly self-critical will show the opposite pattern. In the prospective follow-up, interview and self-report measures of both depression and life events confirmed the hypothesis. Subjects who were characterised by dependent self-schemata were found to be significantly more vulnerable to depression on experiencing negative interpersonal life events compared with negative achievement life events. Results for the self-critical subjects however were less convincing, suggesting that the schema-event specificity principle did not apply. The authors suggested that since achievement events were more salient in both groups of these college subjects, these events were more normal and therefore featured prominently in both groups, thus lending less possibility of discrimination between the groups.

Two other studies have investigated the schema-event specificity hypothesis (Zuroff and Mongrain, 1987; Olinger et al., 1987). The first of these studies (Zuroff and Mongrain, 1987) was experimental in design and results were similar to those found in Hammen at al's (1985) naturalistic study: dependent and self-critical subjects showed increased vulnerability to depression following exposure to audiotapes of interpersonal rejection and achievement failure. Those subjects who predominantly relied on achievement for gratification experienced feelings of worthlessness and inferiority

(introjective depression) in response to both achievement and interpersonal rejection audiotaped themes whereas those subjects who predominantly relied on others for gratification experienced feelings of helplessness and weakness (anaclitic depression) in response to interpersonal rejection. Zurroff and Mongrain, on the basis of their findings, have proposed that the level of activation of a schema is a continuous function of the congruity between a given situation and the content of the schema. As such, schemata might be better understood as being activated to varying degrees rather than as being activated or not activated. The results of the study by Olinger et al (1987) is also supportive of the schema event specificity hypothesis in that depression was evident in cognitively vulnerable individuals only when the life events were related to the individuals dysfunctional attitudes. These studies offer at least some support for the schema event specificity hypothesis.

Lastly, Teasdale and Dent (1987) have proposed a related hypothesis. In this version, a depressogenic schema is activated by depressed mood and the schema is not accessible unless there is sufficiently adequate activation. However this hypothesis, the differential activation hypothesis, assumes that the type of events, or change in mood, that would lead to severe and persistent depression in a minority of people would produce at least mild and short-lasting depression in most people. It is also proposed that the negative cognitions that are activated by depressed mood do not necessarily relate to the event that initially produced the depression.

Miranda and Persons (1988) tested the hypothesis that dysfunctional attitudes are vulnerability factors to depression but are unlikely to be reported unless the mood of an individual is depressed at the time of assessment. They found that those women who had previous episodes of depression reported more dysfunctional attitudes than did women without a positive history of depression but the effect was a function of current mood state. Those who had no history of depression had low scores on dysfunctional attitudes regardless of current mood whereas, for women with a history of depression, dysfunctional attitudes were positively related to negative mood state. This is consistent with the differential activation hypothesis as vulnerability to clinically significant or persistent depression "is related to the type of cognitive constructs and process that become active and accessible in depressed mood, but these dysfunctional constructs and process may not be demonstrable in the absence of depressed mood" (Teasdale, 1988, p260).

Teasdale and Dent (1987) examined whether schemata could be differentially activated. Recovered depressives were compared with never depressed controls on a self-schema task after a mood induction procedure. The self-schema task involved reading positive and negative adjectives, some of which included global negative traits such as "worthless", "failure" and "pathetic". They found that recovered depressed subjects recalled more negative adjectives which had been previously been rated as self-descriptive. The results supported the differential activation hypothesis in that individuals with a history of depression differed from those who had

never been depressed in the type of cognitions activated by mildly depressed mood. As recovered depressives scored more highly on neuroticism compared to the control group, both when tested following the experiment and three years previously, the personality dimension of neuroticism was found to act as a vulnerability factor to depression. This would suggest that depression prone individuals differ from non-depressed controls in that they may have persistent biases in the way that information is processed. However, this finding differs from the schema specificity hypothesis in that it does not require negative life events to be congruent with dysfunctional schemata in order to render the individual vulnerable to depression.

The process and conditions by which depressogenic schemata become activated continues to be elaborated and the hypothesis that deprossogenic schemata render individuals vulnerable to clinical depression requires further longitudinal studies. There is however evidence that access to depressogenic schemata is dependent on mood state (Simons et al., 1985; Hamilton and Abramson, 1983; Miranda and Persons, 1988).

These studies illustrate some of the various hypotheses which have followed from Beck's theory relating to cognitive vulnerability to depression. This theory has been both influential and useful in the study of depressed mood in depression. The theory and associated cognitive measures may also be useful in studying the characteristics and effects of depressed mood in alcoholism.

2.2 SELF-CONTROL THEORY

Rehm's (1977) cognitive behavioural model of depression is based on Kanfer's (1970) self-regulation model which proposes that the self-monitoring of behaviour is essential for self-regulation and self-control. Rehm's (1977) model of depression involves dysfunction in a set of three interrelated processes: self-monitoring, self-evaluation and self-reinforcement. It is proposed that depressives will attend selectively to immediate rather than to delayed outcomes of their behaviour. They will fail to make accurate internal attributions about causality and set overly strict performance criteria for self-evaluation. Lastly, depressives are expected to show relatively low rates of self-reward and high rates of self-punishment. In this model, cognitive symptoms of depression are regarded as reflecting self-control deficits.

Several studies have investigated these hypotheses and results have confirmed some aspects of Rehm's theory. With regard to self-monitoring, depressives have been shown to monitor more negative and fewer positive behaviours compared to non-depressives (Roth and Rehm, 1980). Depressed patients were found to be less likely than non-depressed patients to reward themselves on a word recognition task despite no differences between the two groups in actual performance (Rozensky et al., 1977). Depressives have also been shown to evaluate their performance as poorer than non-depressives (Lewinsohn et al., 1980). Rokke and Kozak (1989) also failed to find that depressives evaluations of their performance was unrealistic and that they were less self-rewarding compared to non-depressed controls. When the variability

in self-evaluation was controlled for in this study and in another (Lobitz and Post, 1979), differences in self-reinforcement were no longer evident, indicating that depressed subjects may reward themselves according to their performance. Deficits in self-reinforcement therefore seem to be dependent on self-evaluation, indicating that subjects' perception of their abilities may be the predominant dysfunction in depression. These studies have used behavioural, rather than self-report cognitive measures, as dependent variables as Rehm's theory has not led to the development of any such cognitive measures.

Rosenbaum (1980) has developed a measure of self-control, the Self-Control Schedule (SCS), which is based on a cognitive-behavioural conception of self-control. Although it does not measure depressive cognitions, it does contain items concerned with perceived self-efficacy, the application of problem solving strategies, the ability to delay immediate gratification and the use of "self-statements" to control unpleasant psychological experiences such as anxiety and depression. Simons et al. (1985) in a trial comparing cognitive therapy and antidepressant medication, found that SCS scores were the best single predictor of success in cognitive therapy. Depressed patients, with high self-control scores at the beginning of treatment responded more favourably to cognitive therapy than did patients who were low in self-control. The opposite pattern was found for responders to medication. The authors concluded that "learned resourcefulness" in depressed patients appears to be an important predictor of response to cognitive therapy.

Although the SCS has not been used in research in alcoholism, the face validity of the measure suggests that it may be useful in that excessive and problematic drinking can be considered as a failure of self-control. It is likely that alcoholics, particularly those who are not severely dependent on alcohol (Pendery et al., 1982), and who show high levels of learned resourcefulness will show a greater ability to abstain from drinking.

CHAPTER THREE

THE PRESENT STUDY

3.1 RATIONALE OF THE PRESENT STUDY

There is increasing awareness of heterogeneity amongst alcoholics. One aspect of this heterogeneity is the extent to which alcoholics suffer from a wide range of psychiatric disorders. One of the most commonly reported of these is depression. Although additional psychopathology is known to affect on the history of alcoholism, its effect on outcome of alcoholism is less clear. Recent studies (Rounsaville et al., 1987, O'Sullivan et al., 1988) have suggested that a lifetime diagnosis of depression is associated with poor prognosis in male alcoholics: those with a lifetime diagnosis of depression receive more treatment at follow-up compared to those with alcoholism alone. However, from the available evidence it is not clear if a lifetime diagnosis of depression is associated with a poorer drinking outcome.

Aside from these few studies which have investigated the effect of lifetime depression, there are no studies which have specifically examined the association between drinking outcome and a current diagnosis of depression in male and female alcoholics. It is possible that drinking outcome for those with a current diagnosis of depression will differ from those without such a diagnosis, as depression existing in the current episode, as opposed to lifetime depression, is more likely to influence immediate behaviour.

A negative emotional state, such as that experienced in depression, is thought to be one of many factors which

influences the consumption of alcohol. One prediction arising from self-awareness and tension reduction models of alcohol consumption is that depressed individuals will be more likely than those who are not depressed to consume alcohol in order to reduce negative affect and reduce the negative bias in thought.

Two aspects of depressive symptomatology are negative mood and thinking. These are thought to be associated with a diagnosis of depression and are also known to be state dependent and therefore may not be specific to a diagnosis of depression. Negative emotional states in alcoholics have been regarded as one cause of relapse. However, there is also evidence that depressed mood decreases with abstinence from alcohol. The relationship between measures of depressive symptomatology and thinking in alcoholism has not been investigated.

3.2 AIMS OF THE STUDY

The main aim of this study was to investigate the heterogeneity of alcoholism and specifically the relationship between depression, depressive symptomatology and drinking outcome in a clinical sample of alcoholics.

There are few studies which have examined the relationship between depression and drinking outcome. Most of these studies have only examined the relationship between a diagnosis of depression, either as a lifetime or primary diagnosis, and drinking outcome. No one has yet investigated the effects of either a current diagnosis of depression on outcome or the depression-specific cognitive characteristics of depression on drinking outcome in alcoholics.

3.2.1. The main hypothesis

The main hypothesis is that patients with a diagnosis of alcoholism who have an additional current diagnosis of depression will differ from those with alcoholism alone as follows:

1. in a random sample of alcoholics attending an in-patient treatment unit, a minority will present with alcoholism alone, the majority will have an additional diagnosis of depressive disorder;

1.a a much smaller proportion of subjects will be depressed following detoxification from alcohol;

2. those with alcoholism and depression in the index episode will not differ from those with alcoholism alone in demographic variables or family psychiatric

history, but will differ in terms of drinking history and past psychiatric history;

3. those with alcoholism and a diagnosis of depression in the episode leading to admission and those who are depressed after detoxification from alcohol will differ in the treatment they receive both as in-patients and at follow-up. They will also differ in drinking outcome from those with alcoholism alone;

4. those with a diagnosis of depression will differ from those without depression on measures of depressive symptomatology and cognitive style. In addition, depressive symptomatology and cognitive style will predict and be associated with drinking behaviour at outcome.

3.3 DESIGN OF THE STUDY

The study was carried out whilst the investigator was employed as a senior Clinical Psychologist in the Department of Clinical Psychology of the Royal Edinburgh Hospital. As part of her work, she provided a clinical service to the Alcohol Problems Clinic.

3.3.1 The Alcohol Problems Clinic

The Alcohol Problems Clinic has recently been described by Ritson (1990). Originally established in 1963, the unit provides a clinical service to problem drinkers on both an outpatient and in-patient basis. The 14 bed in-patient unit, is situated within the Royal Edinburgh Hospital, a psychiatric hospital which serves the population of Edinburgh and its surrounding area.

The initial assessment of patients takes place in the outpatient clinic. Approximately 425 new outpatients are referred per year and about one third of all patients referred will be admitted to the in-patient treatment programme.

In-patient treatment takes the form of detoxification, if necessary, followed by a three week basic treatment programme which is eclectic in nature. Not all patients admitted for detoxification enter the programme. The in-patient programme consists of group sessions concerned with alcohol education, problem-solving skills, social skills training, stress management and relaxation training. Some experience of group psychotherapy is also given which is aimed at increasing self-awareness. In addition, each patient is assigned a key worker who sees the patient regularly to assess their individual needs and who may, if possible, also involve the patient's family or friends in treatment.

The treatment programme is ideally considered as a start to treatment and the patient then attends individual sessions with their key worker to continue the task of changing their drinking pattern to a style consistent with a more adaptive life style. Many patients are encouraged to seek additional support from Alcoholics Anonymous and from the local Council on Alcohol after leaving the in-patient programme.

Ritson (1990) has suggested the following factors as influencing the decision to admit: patients who are likely to experience severe withdrawal symptoms; those who need to gain confidence and skills in coping with environmental and intrapsychic pressures; those who have personality difficulties requiring investigation; those who require a break from family pressures in order to explore family and interpersonal difficulties; those patients who would benefit from meeting others with similar problems. These factors are best thought of as influencing the decision to admit a patient rather than being hard and fast criteria applied in a consistent and objective manner.

The study began in October 1986 and patients were admitted into the study over a three year period. During the time of the study, the structure of the three week in-patient programme changed. Initially, patients were admitted on a weekly basis to a three week rolling programme. This practice changed to one in which a single cohort of patients were admitted for three weeks.

3.3.2 Selection of Subjects

Subjects were randomly selected from amongst those patients who had been admitted to the Alcohol Problems

Clinic of the Royal Edinburgh Hospital for the three week in-patient programme.

On average, five patients were admitted weekly to the programme. Initially, the investigator estimated that one patient could be admitted into the study each week and every third patient on the admission list was approached. This list of patients names was written by different members of the nursing staff as the patient's name was given to them by medical or nursing staff following a decision to admit the patient. As such, the order of names appearing on the list could be considered as having been placed on the list at random. The list was kept in the nursing duty room.

When the programme was changed to admit a single cohort of patients once every three weeks, attempts were made to select and interview two randomly selected patients from the list of patients admitted. However, due to the investigator's clinical commitments to other areas, it was often only possible to select one patient from the list. Again the patient whose name was third on the admission list was approached. If two subjects were to be approached, then the third and the sixth named patients were approached.

Grounds for exclusion from the study were as follows:

1. Patients over the age of 65 years and less than 18 years.
2. Patients whose residence was outwith reasonable travelling distance from Edinburgh.
3. Patients who had no fixed abode.
4. Patients who had severe medical complications.
5. Patients who had gross brain damage.

If an individual refused to take part in the study, the patient whose name was next on the list was approached. This only occurred on one occasion where a patient expressed a desire to leave the unit and thought that he should not take part in the study.

The majority of patients who were to attend the in-patient programme were first admitted for detoxification in the week preceding the start of the programme. It was rare for a patient to attend the programme without having first been admitted for detoxification. This allowed the investigator to approach the selected subject in the week before the programme began.

Although the investigator was a member of staff of the unit and ran some of the groups which patients would attend in the programme, she was introduced to the patient by another member of staff, usually the nurse on duty at the time who had already talked to the patient. It was felt that this procedure would allow the patient to give consent to taking part in the study more freely as it disentangled the joint role of the investigator as both clinician and researcher.

After being introduced to the patient in the clinic, the investigator gave an outline of the study to the patient and answered any questions. If the patient showed an interest in taking part, they were then given a written outline of the study (Appendix I). A written consent form was also given out for completion should the patient agree to take part. The patient was then thanked for their time and a further appointment was made for the following week, the start of the in-patient programme. The investigator then returned to see the patient and if they then agreed to take part in the study, written

consent was sought. An interview was arranged for the following day at a time which would not coincide with the patient attending part of the programme.

3.3.3 Procedure

3.3.3.a *The Initial Interview*

The patient was interviewed in the investigator's office, situated in the Psychology Department within the hospital. This environment was quiet and its normal use was as a patient consulting room. One and a half hours were set aside for this first interview. Figure 1 gives a summary of the tests and information sought during admission.

Figure 1 Table of testing during admission

ADMISSION TO UNIT	ATTENDANCE IN-PATIENT PROGRAMME		
Detoxification	WEEK 1	WEEK 2	WEEK 3
	SADS		
	Family History		
	MMS		
	SADQ		
	Drinking History		
	Drinking Diary		
	MADRS	MADRS	MADRS
	BDI	BDI	BDI
	STAI	STAIS	STAIS
	ATQ		
	HS		
	CST		
	DAS		
	SCS		

SADS : Schedule for Affective Disorders and Schizophrenia
MMS : Mini Mental State
SADQ : Severity of Alcohol Dependence Questionnaire
MADRS: Montgomery-Asberg Depression Rating Scale
BDI : Beck Depression Inventory
STAI : State-Trait Anxiety Inventory
STAIS: State Anxiety Inventory
ATQ : Automatic Thought Questionnaire
HS : Hopelessness Scale
CST : Cognitive Style Test
DAS : Dysfunctional Attitude Scale
SCS : Self-Control Schedule

The investigator began the interview by asking readily answerable questions such as the patient's address, occupation and some other personal details. Once the patient seemed at ease with the investigator, the Mini-Mental State (MMS)(Folstein et al., 1975), a test of cognitive function was administered. This was given to exclude any patient who may have been suffering from severe cognitive impairment from taking part in the study. None of the patients selected were excluded on this basis.

The Schedule for Affective Disorders and Schizophrenia (SADS)(Endicott and Spitzer, 1978) was administered on the first occasion the patient was seen. The aim, during the time allocated, was to cover the history and symptomatology of the patient's current episode. Information was sought on the episode which led to admission and about the nature of symptoms since detoxification. The pace of the interview was, within limits, dictated by the patient. No attempt was made to resolve problems patients mentioned but they were listened to with an attentive and empathic manner. No attempt was made to reach a diagnosis at this time or at any time throughout the time a patient was seen, including follow-up. The information pertaining to diagnosis was analysed after the patient had been discharged from the study.

At the end of this interview, the investigator completed the Montgomery-Asberg Depression Rating Scale (MADRS)(Montgomery and Asberg, 1979). The patient was then accompanied back to the Alcohol Problems Clinic and a further appointment was made for later that week.

Second session

The second session also took place over a period of an hour and a half. The SADS, including past psychiatric history, was completed if this had not been achieved in the first session. The aims in this session were firstly, to obtain a family psychiatric history from the patient and secondly, to obtain a detailed drinking history, including the amount of alcohol and pattern of drinking in the recent past. The Severity of Alcohol Dependence Questionnaire (SADQ)(Stockwell et al., 1979) was completed. The patient was also requested to complete measures of affective symptomatology and cognitive questionnaires. At the end of this interview, the patient was again thanked for their help and accompanied back to the Alcohol Problems Clinic. If the patient had not completed these questionnaires in the allotted time, they were asked to complete them within the following twenty-four hours, at a time when they would be alone and undisturbed. If this occurred, the investigator collected the questionnaires from the patient the following day.

3.3.3.b *Change in protocol during study*

Initially, only those patients who scored at or above certain levels on the MADRS and State Anxiety Inventory (STAIS) which forms part of the State-Trait Anxiety Inventory (STAI)(Speilberger et al., 1970) were re-interviewed in the two remaining weeks in the programme. The rationale for this procedure was that the study was concerned with changes in depressive symptomatology over time and only those scoring at least at a mildly depressed or anxious level would be re-interviewed as they would be more likely to show changes in mood over that time period. However, after

approximately twenty patients had been interviewed, the investigator decided to change the original protocol and ask all patients to complete measures of depressive symptomatology and anxiety, including MADRS, throughout the three weeks in hospital. The reason for this change was that the investigator had originally been informed that patients remained on detoxification medication for five days. It became evident from scrutiny of the drug Kardex that some patients remained on detoxification medication, usually chlordiazepoxide, longer than an average of five days. As this may have affected their initial ratings of affective symptoms, it was decided to monitor affective symptoms of all patients once per week for the three weeks of the programme.

The investigator arranged to interview each patient at an appointed time each week. On these occasions the patient completed the Beck Depression Inventory (BDI)(Beck et al., 1961) and STAIS, self-rating questionnaires. The patient was then asked questions relevant to the MADRS which was completed by the investigator. If the patient had other concerns at these times, the investigator listened sympathetically to these and suggested that these be raised with the patient's key worker. The patient was then thanked for their cooperation.

3.3.3.c *Follow-up of Patients*

All patients were informed at the outset of the study that they would be requested to attend two follow-up interviews. The first follow-up session took place one month after leaving the in-patient programme and the second follow-up was scheduled for three months following the first follow-up. The first follow-up appointment was

arranged with the patient before they left the unit and to avoid inconvenience, the time was arranged to coincide with the day the patient had an appointment with their key worker. A letter reminding the patient of the appointment with the investigator was sent in the week before the follow-up. If a patient did not attend for follow-up, a second letter was sent, reminding them of the study and asking them to attend for follow-up.

As with the initial interviews, all follow-up interviews took place in the investigator's office. Patients were asked to attend for follow-up at the Outpatient Department of the hospital which is situated within the hospital. This was the usual practice for patients attending the Department of Clinical Psychology. The key worker was informed that their patient had attended to take part in the study and if there was any reason for concern about the patient, this was expressed to the key worker with the patient's consent. If a patient did not attend for follow-up, their case notes were examined to find out if they were attending the unit for regular follow-up and information was sought from their key worker.

If a patient did not attend for follow-up after two written requests, a letter was sent to the patient to ask if a home visit would be convenient. If the investigator did not receive an affirmative answer, the patient was considered lost to follow-up.

On a few occasions, the patient was followed-up at home by the investigator and another researcher, conducting a related study and who was interested in contacting the patient for a follow-up appointment. This arrangement was only possible in a few cases and the

patient's agreement was sought for both researchers to visit them.

On rare occasions, patients who did not respond to the first two written requests to attend follow-up were not sent a letter asking permission to visit them at home. This was due to the investigator feeling unsure of the safety to herself in carrying out such a visit.

3.3.3.d *Format of Follow-up Interviews*

Figure 2 gives an outline of the tests used and information sought at follow-up.

Figure 2 Table of testing during follow-up

FIRST FOLLOW-UP 1 MONTH	SECOND FOLLOW-UP 4 MONTHS
Drinking Diary	Drinking Diary
Medication	Medication
Re-admission	Re-admission
MADRS	MADRS
BDI	BDI
STAI	STAI
ATQ	ATQ
HS	HS
CST	CST
DAS	DAS
SCS	SCS

MADRS: Montgomery-Asberg Depression Rating Scale
BDI : Beck Depression Inventory
STAI : State-Trait Anxiety Inventory
ATQ : Automatic Thought Questionnaire
HS : Hopelessness Scale
CST : Cognitive Style Test
DAS : Dysfunctional Attitude Scale
SCS : Self-Control Schedule

After receiving the patient in the her office, the investigator made a general enquiry as to the well-being of the patient. The patient was then asked about their drinking over the period since they were last seen by the investigator. A calendar was provided to facilitate the patient in remembering dates and events on which they could base their recall of drinking. The investigator

completed the retrospective assessment of the patient's drinking and then showed the assessment to the patient in order to check the information. It was then noted whether or not their drinking had resulted in any problems for them in the intervening period.

After the investigator had completed the retrospective diary of drinking, the patient was asked to complete the self-rating scales of affective symptoms and cognition. The patient was then asked more specific questions regarding their mood and any depressive symptoms, following which the investigator completed the Montgomery-Asberg Depression Rating Scale.

All patients were asked on each follow-up occasion if they had been prescribed medication, either by a doctor in the Alcohol Problems Clinic or their General Practitioner, in the period since they last saw the investigator. They were also asked if they had been taking the medication prescribed. Their case notes and drug record were also read to check this information.

At the end of the first follow-up interview, the patient was thanked for their time and cooperation in taking part in the study and a second follow-up appointment was arranged.

The conduct and content of the second follow-up interview was identical to that of the first.

3.4 MEASURES

3.4.1 Diagnostic Instrument

The Schedule for Affective Disorders and Schizophrenia.

The Schedule for Affective Disorders and Schizophrenia (SADS)(Endicott and Spitzer, 1978) was developed in conjunction with Research Diagnostic Criteria (RDC)(Spitzer et al., 1975) as a means of improving the reliability and validity of psychiatric diagnostic practice. The SADS specifically aims to reduce the amount of variance in information a clinician receives from a patient by providing the clinician with a structured interview with which to assess a patient's psychopathology and level of functioning.

There are three versions of the SADS: the regular version (SADS), the lifetime version (SADS-L) and a version for measuring change (SADS-C). This study used the regular version of the SADS (SADS) and only this version will be described here.

The SADS is similar in design to a clinical interview which focuses on differential diagnosis. Part 1 of the SADS is concerned with the patient's current episode of illness. The interview focuses on the time during the current episode of illness when symptoms were at their worst and the extent to which these symptoms have changed in severity, if at all, in the week prior to the interview. A progression of questions, items and criteria are provided by the schedule to aid in ruling in or out specific RDC diagnosis. Although summary scales can be obtained using the SADS, this was not carried out in this study as this would have resulted in the investigator coming to a judgement on diagnosis. Although not always possible, it was felt that the

investigator should remain "relatively blind" to the diagnosis of patients so as not to influence their treatment beyond that which normal clinical practice would dictate.

Part 2 of the SADS is similar to SADS-L in that this part of the interview concentrates on past psychiatric disturbance and illness. The criteria for a given diagnosis are specified and the interviewer can use as many sources of information as are available to make a judgement on whether the disorder has been present. In this study, hospital case notes were used as the main source of information in addition to the patients' own accounts of any psychiatric disturbance they may have experienced.

The current section of the SADS has been shown to be a reliable method of rating psychopathology (Endicott and Spitzer, 1978). High levels of agreement were found for both test-retest evaluations (r greater than 0.6 in 82% of cases) and intraclass evaluations (r greater than 0.6 in 90% of cases).

From the information obtained from the SADS interview, the patient's social class was derived from the Office of Population, Census and Surveys (1980) Classification of Occupations. A housewife was classified according to her husband's occupation. If working, women were classified by their own occupation or last occupation. Likewise, men were classified according to their own occupation or last occupation.

3.4.2 Research Diagnostic Criteria

The main aim of RDC is to increase the reliability of psychiatric diagnostic practice by making explicit the inclusion and exclusion criteria for psychiatric

diagnoses (Spitzer et al., 1978(a)). The specific diagnostic criteria refer to the presence or absence of symptoms, signs, the duration or course of an illness, and also level of impairment. Operationalised definitions are given of diagnostic criteria.

The RDC were designed to be used in conjunction with the SADS and therefore the clinician is expected to use all available sources of information in reaching a diagnosis. Some of the diagnoses, such as antisocial personality disorder, are based on the long-term functioning of an individual, including early adolescence. For the main part, however, diagnoses are based on the present episode of illness.

The reliability of RDC categories was investigated in three studies and was found to be highly reliable, even under test-retest conditions (Spitzer et al., 1978(b)). Where joint interviews were conducted with the same patient, there was high agreement on the diagnosis of patients. For example, the Kappa coefficient of reliability was equal to 0.90 for depression and equal to 1.00 for alcoholism. In addition, it was possible for an interviewer, using the lifetime version of the SADS (SADS-L), and who was "blind" to the patient's original diagnosis, to diagnose past episodes of illness which agreed well with raters who reviewed the patient at the time of the illness and who had available several sources of information (for example, Kappa coefficient of reliability for major depressive disorder equal to 0.76).

The term major depressive disorder is used in RDC as a broad category of depression which encompasses many different sub-divisions of depression such as neurotic

and psychotic depression. Although based on the original Feighner criteria for depressive illness (Feighner et al., 1972), RDC require a shorter duration, two weeks, of illness for a definite diagnosis of major depression and only one week for a probable diagnosis of depression. An investigator can however use more stringent criteria, if required. In this study, RDC were used and the duration of illness was noted separately. RDC for primary major affective disorder have been found to be over-inclusive: 72% of patients with reactive depression, which responded to changes in psychosocial environment, were found to meet such criteria (Nelson et al., 1978). Another study examined the usefulness of RDC sub-types of depression in predicting response to treatment (Prusoff et al., 1980). Although both situational and endogenous depression were found to respond to a combination of short-term interpersonal psychotherapy (IPT) and amitriptyline, those patients with endogenous depression did not respond to IPT alone and those with a situational depression responded to either treatment alone. This finding indicates that the distinctions between these sub-types of depression are valid in that they predicted a differential response to treatment.

RDC also provide a relatively broad and inclusive definition of alcoholism and many patients with minor drinking problems are likely to meet RDC. However, when comparisons are made with other diagnostic systems, such as Feighner criteria (Feighner et al., 1972) and the Diagnostic and Statistical Manual (DSM) III criteria (American Psychiatric Association, 1980), there is 86% agreement between these criteria on the diagnosis of alcoholism in a sample of factory workers (Leonard et

al., 1984). When clinical samples have been investigated for concordance of criteria for alcoholism using the same diagnostic criteria, even greater agreement (92%) was reached (Singerman et al., 1981). The sample in this study was considered by the staff of the clinic as warranting in-patient rather than outpatient treatment for alcoholism, and had been deemed by referring agents, usually general practitioners, to have problems warranting referral to a specialised treatment unit for alcohol problems.

Feighner's primary-secondary distinction is used in RDC. Primary major depression is defined as "a period of major depressive disorder that has not been preceded by any of the specific list of nonaffective disorders" (Spitzer et al., 1978(b) p777). Where a patient had a diagnosis of a disorder, other than alcoholism in the past, this disorder was considered primary and lifetime diagnoses were considered separately from current episode diagnosis.

For the main part, however, this study concentrated on the present illness. A patient can be given more than one diagnosis according to RDC. On the basis of information from the SADS, RDC were applied to reach a diagnosis for the present episode as well as past episodes of illness for each patient. For each patient a diagnosis was obtained for both the current episode at its worst and for the time interval since stopping drinking and the SADS interview. All patients had been abstinent from alcohol for at least seven days at the time of the SADS interview. RDC for definite major depressive disorder require that dysphoric features be present for more than two weeks and, for a diagnosis of

probable major depressive disorder, the symptoms should be present for between one and two weeks. If a patient had met criteria for definite major depressive disorder for the time in the current episode when at its worst, and continued to meet criteria for definite major depressive disorder up to and including the time period of the week previous to the interview, then they were defined as meeting criteria for definite major depressive disorder. On the other hand, if a patient had not met criteria for definite major depressive disorder during the current episode but had symptoms of depression arising after having stopped drinking, the depressive disorder could not be classified as being definite as the symptoms would not have been present for more than two weeks. Only probable major or minor depressive disorder could be diagnosed.

For the purpose of analysing the data, definite and probable diagnosis of major depression were combined.

The RDC convention of counting only those manifestations of antisocial personality which cannot be clearly attributed to alcohol or alcoholism was used for the diagnosis of antisocial personality disorder.

3.4.3 Family History

The family history method was used to collect data about the first degree relatives of patients. This method was chosen in preference to the family study method as the latter method would have been more costly in time and because of the practical difficulties in collecting information by directly interviewing all first degree relatives. Therefore the family history method was used and the patient was the source of information on psychiatric illness in their family. Information was

systematically collected on all first degree relatives, including parents, siblings and offspring, regardless of whether they were alive or dead. Information was also collected about the psychiatric history of the patient's spouse, where appropriate. If hospital records were available for a family member, these were also searched to verify the diagnosis.

Diagnosis of family members was made according to the Family History RDC (FH-RDC) (Andreasen et al., 1977). When the family history method has been compared with the family study method, the family history method was found to have greater sensitivity than some other family history methods although it gave an underestimate of familial rates of affective disorder (by approximately 50%) compared to the family study method. However, in some cases, a more valid estimate of the rate of familial alcoholism, substance abuse and antisocial personality disorder was obtained using the family history method (Andreasen et al,. 1986). In general, the specificity of the family history method is high with very few unaffected individuals wrongly classified as having a particular disorder when compared with information received directly from the individual concerned (Thompson et al., 1982). In addition, sensitivity was highest for the diagnostic categories of depression and alcoholism compared with other diagnostic categories. Thus, although the family history method may have some limitations, it has reasonable sensitivity for the major psychiatric diagnosis of particular interest in this study, namely alcoholism and depression, and has the obvious advantages in practical and cost terms over the family study method.

3.4.4 Drinking Information

3.4.4.a *The Retrospective Diary Method*

Information about the quantity of alcohol consumed by a patient was obtained using the retrospective diary method described by Latcham (Latcham, 1984). This information was sought on three occasions: during admission and on the first and second follow-up occasions.

During admission, patients were asked to recall the amount they had drunk on the seven days before admission. As this method was retrospective, they were asked to recall what they had drunk on the day before admission, the day previously and so on until the seventh day before admission was reached. The type of alcohol consumed and quantity was specified to estimate the number of units of alcohol consumed (a unit of alcohol is 8 grammes). They were also asked to recall the circumstances surrounding each drinking occasion. The date and day of the week, the time of day, the duration of the drinking session, where drinking had taken place and who had been present on each occasion. This information was recorded and was sought primarily as an aide memoir.

They were then asked if this pattern of drinking was typical of their "usual drinking pattern" in the previous three months before admission. If atypical, a typical week's drinking was recorded. It was found that many patients had altered their drinking pattern before admission to hospital.

At follow-up, this method of recalling alcohol consumption was also used and a calendar was provided to help the patient recall the circumstances of their drinking in the intervening period. Case notes were also

searched for information about alcohol consumption as a means of checking self-report of alcohol consumption. Unfortunately, there was seldom specific details of alcohol consumption recorded. Sometimes it was noted if the patient had been drinking or abstinent but more often there was no record of drinking.

There are a large number of self-report measures of alcohol consumption but the two most common methods are summary measures of drinking and recall of recent drinking episodes. The most commonly used summary method involves a quantity-frequency measure (QF) where respondents are asked how much alcohol they consume on a "usual" drinking occasion. This method has a particular disadvantage in that it produces estimates of the "usual" amount of alcohol consumed and this could be interpreted as the "mean" level of consumption. Both the quantity and frequency of drinking occasions are found to distributed asymetrically with a positive skew and thus it is likely that there will be a bias towards the under-reporting of unusually light and heavy alcohol consumption (Webb et al., 1990).

The advantage of the retrospective diary method over a QF method is that it provides a continuous measure of consumption and does not rely on subjects having to decide on an estimate of their "usual" drinking behaviour. It is commonly thought that alcoholics may be unreliable self-reporters of alcohol consumption, often under-reporting their consumption or "denying" drinking and associated problems. Clinical studies which have used collateral's reports to validate self-reports have indicated that there is a high degree of agreement between the reports of patients and collaterals,

particularly when the collateral is a spouse (Midanik, 1982). In addition, where discrepancies arose between reports of collaterals and patient, the bias was not in any one direction. This would indicate that self-reports may be at least as reliable as collateral reports of alcohol consumption in clinical populations.

One major disadvantage in using the retrospective diary method is difficulty in remembering details of drinking episodes as the length of time increases at follow-up. However, the QF method of measuring consumption would also be liable to this source of error and, as this requires the subject to give estimates of typical drinking, variation in drinking may not be so readily described.

Each subject was also asked about the number of episodes of alcoholism he or she had experienced. An episode was defined according to SADS and RDC. An episode was considered to have ended if diagnostic criteria were no longer fulfilled or the subject described an absence of alcohol related problems. If an episode had resulted in hospital treatment, then case notes were checked before making a decision about when an episode ended.

3.4.4.b *Alcohol Related Problems*

Details of the type and number of alcohol related problems reported by patients were noted. The categories of alcohol related problems were those specified in the SADS. At follow-up, the same method was applied to obtain information about alcohol related problems although the time interval was specified as being the interval since the patient was last interviewed by the investigator.

Sobell and Sobell (1978) checked the veridicality of self-reported alcohol related behaviour with official records in three groups of alcoholics: voluntary out-patients, court referred outpatients and voluntary in-patients. In general, the in-patient group gave the lowest percentage of valid answers (68%) to questions where a discrepancy could be determined compared to the outpatient groups. In this group, the amount of over-reporting (56%) did not differ significantly from under-reporting (44%). The outpatient groups, in contrast, significantly over-reported answers ($p<.05$). As some of the information requested by the Sobells referred to events in the past, and memory dysfunction was not taken into account, details such as the number of times arrested may have been forgotten. It would seem prudent to regard information about events in the distant past with caution.

In this study, other than when diagnosing alcoholism, information on alcohol related problems concerned the current episode and the intervals between follow-up interviews.

3.4.4.c *Severity of Alcohol Dependence Questionnaire*

The Severity of Alcohol Dependence Questionnaire (SADQ)(Stockwell et al., 1979) is the earliest scale for measuring severity of dependency on alcohol. It was designed to cover those features of the alcohol dependence syndrome most amenable to change. The SADQ is a self-completion questionnaire consisting of 20 items. The focus of time is on "a recent month of heavy drinking". The questionnaire is divided into five sections: physical symptoms of withdrawal, affective symptoms of withdrawal, craving and withdrawal-relief

drinking, typical daily consumption and the rapidity of reinstatement of symptoms after a period of abstinence. Items concerned with withdrawal symptoms refer to how the respondent felt upon waking up during a heavy drinking spell as this is the most common time for such symptoms to arise. Each item on the questionnaire is scored from zero to three, depending on the frequency of occurrence of each item. The maximum total possible score is 60. Mean scores of 33.6 (s.d. 16.1) have been reported for 103 patients attending an alcoholism treatment unit and 33.8 (s.d. 13.7) for 59 patients at a detoxification unit. These have been given as aids to interpretation (Stockwell et al., 1983).

The SADQ was completed by the original sample of 104 individuals reporting to an Alcoholism outpatient clinic (Stockwell et al., 1979). The individual sections of the SADQ were found to correlate significantly with each other ($r = .51$ to $r = .80$) and correlated (between 0.69 and 0.80) with total SADQ score which was consistent with the view that these symptoms and behaviours develop in a parallel and related fashion with each other as severity of dependence increases. After dropping three items from the original 23, factor analysis of pooled items resulted in a first factor accounting for 53% of the variance. A score of 36 and above was found to correlate with clinician ratings of severe, rather than moderate, degree of dependence in 82% of cases, suggesting that the instrument was a reasonably valid measure of dependency on alcohol.

Later investigation of the SADQ (Stockwell et al., 1983) showed that the questionnaire had high test-retest reliability ($r = .85$, $p < .001$) when given to in-patients

tested on two occasions two weeks apart. The authors also tested, in some part, the construct validity of the SADQ. The SADQ does not contain items referring directly to the narrowing of the drinking repertoire which is postulated as a core element of the alcohol dependence syndrome (Edwards and Gross, 1976). The diversity of an individual's drinking repertoire was examined by Stockwell and his colleagues to investigate the hypothesis that the more stereotyped this repertoire, both within and between drinking days, the greater the degree of alcohol dependence. Alcohol clinic attenders were interviewed about their drinking behaviour. A multiple analysis of variance, with SADQ as the dependent variable, showed that stereotyped drinking behaviour, (ie. limited variability both within and between heavy drinking days) contributed significantly ($p < .001$) to the variance, independently of the effects of other drinking parameters, such as duration of heavy drinking, included in the analysis. Further analysis revealed that "mainly continuous" and "mainly binge drinkers" tended to have higher SADQ scores than "occasional drinkers". In addition, significant, albeit small correlations were found between a physician's ratings of withdrawal severity, between subject's ratings of craving for alcohol and amount of medication administered throughout the withdrawal period and total SADQ scores.

Self-completion questionnaires have been criticized on the grounds that they do not precisely define the complexity of experience relating to alcohol dependence (Chick, 1980), and may not measure more subtle aspects of the proposed alcohol dependence syndrome (Orford, 1987). However, the SADQ appears to provide a short, reliable

and valid means of measuring severity of dependence on alcohol.

3.4.5 Mini-Mental State

One of the exclusion criteria of this study was that any patient who exhibited gross cognitive deficits was to be excluded from the study. The British adaptation (Dick et al., 1984) of the "Mini-Mental State" (MMS)(Folstein et al., 1975) was used to assess cognitive performance. This is a clinician administered, eleven item questionnaire and represents a scored form of the mental status examination employed by clinicians but without questions concerning mood, abnormal mental experiences and the form of thinking.

Scores on the MMS have been found to agree with clinical opinion of cognitive deficits in three diagnostic groups in an elderly population (Folstein et al., 1975). The mean score on MMS for a group of normal elderly controls was 27.6 and this was found to be significantly different ($p < .001$) from patients with dementia (mean 9.7), patients with affective disorder with cognitive impairment (mean 19.0) and affective disorder without notable cognitive deficits (mean 25.1). In addition, when patients were tested before and after treatment, those patients with dementia showed no significant change in scores on MMS whereas those groups expected to change, showed significant increases in scores ($p < .025$) indicating that MMS is a valid test of cognitive function in patients with dementia and depression as well as a useful means of quantifying the severity of cognitive impairment. Inter-rater reliability was found to be high ($r = 0.83$, $p < .0001$) and 28 day test-retest reliability was found to be similarly

high (r= 0.99, p< .0001). The concurrent validity of MMS was determined by correlating MMS scores with the Weschler Adult Intelligence Scale (WAIS)(Weschler, 1955). Scores on MMS correlated highly with the Verbal and Performance scales of the WAIS; r= 0.78 (p< .0001) for Verbal Intelligence Quotient and r= 0.66 (p< .001) for Performance Intelligence Quotient.

Dick et al (1984) reported similar results to Folstein et al in a population of younger neurologically impaired adults. They examined the sensitivity and specificity of the MMS to detect cognitive impairment and found that a score of 23 or less gave 4.3% false positive results in the cognitively normal group and detected 76% of the cognitively impaired group of neurological patients. This study used Dick et al's cut off score of 23. The MMS is therefore satisfactory as a reliable indicator of cognitive function although it may be relatively insensitive to right hemisphere damage (Dick et al., 1984). It provided a brief and quantifiable measure of cognitive function in the sample studied.

3.4.6 Measures of Affective Symptomatology

3.4.6.a *Montgomery-Asberg Depression Rating Scale*

The Montgomery-Asberg Depression Rating Scale (MADRS)(Montgomery and Asberg, 1979) was designed to be a clinician rating scale, sensitive to changes in depression. The final version of the scale consisted of the ten items, out of a possibility of seventeen, which were most sensitive to changes in depression during treatment, and which represented most of the core symptoms of depression. Responses to broadly phrased questions about symptoms and more detailed questions allow the clinician to rate the severity of each symptom

present. Each item is scored on a seven point scale: a score of zero signifies the absence of a symptom and a score of six, the most extreme rating of the symptom. Scores on MADRS range from zero to sixty indicating both the intensity and frequency of occurrence of symptoms. The time frame of the scale can be determined by the investigator and in the case of this study, was taken to be the seven days prior to interview.

The scale has been shown to be a reliable and valid measure of depression. Scores on MADRS correlated significantly with scores on the Hamilton Rating Scale for Depression ($p<0.001$)(HRSD)(Hamilton, 1967) indicating that MADRS is a valid measure of severity of illness. The scale has good inter-rater reliability ($r= 0.89$). Preliminary validation of the MADRS scale compared the performance of MADRS and HRS scores with an experienced clinician's global judgement of response to treatment. MADRS (17 and 10 item versions) and the HRS capacity to identify responders and non-responders to treatment revealed that the 10 item version was found to discriminate best between responders and non-responders to treatment. The point biserial correlation between response category and change scores was $r= 0.70$ for MADRS 10 item version versus $r= 0.67$ for MADRS 17 item version and $r= 0.59$ for HRS (Montgomery and Asberg, 1979).

Using the Hospital Anxiety and Depression scale (HAD)(Zigmond and Snaith, 1983), Snaith and Taylor (1985) reported that MADRS had high concurrent validity with HAD Depression ($r=0.81$) but lower correlations with HAD Anxiety ($r=0.37$) which lends support to the view that MADRS is a valid measure of depression capable of differentiating depression from anxiety.

One disadvantage of MADRS is that there are no means of classifying patients according to a grade of severity. However, Snaith and his colleagues (Snaith et al., 1986) have suggested that scores between 20 and 34 indicate moderate depression and scores between 7 and 19, mild depression on MADRS.

3.4.6.b *Beck Depression Inventory*

The Beck Depression Inventory (BDI) is a widely used self-rating scale for the measurement of severity of depression. The original BDI (Beck et al., 1961) has 21 items, each consisting of a graded series of statements whose scores range from zero to three depending on severity. Items refer to the vegetative, cognitive and mood symptoms of depression which Beck regarded as characteristic of depression (Beck, 1967). The patient selects the statement in each set which best represents his or her condition. The range of scores is from zero to 63, reflecting both the number of symptoms endorsed and the severity of each.

The BDI has been found to have high levels of internal consistency with a mean coefficient of 0.86 reported for psychiatric populations (Beck et al., 1988). The split-half reliability has also been found to be satisfactory (r= 0.86) (Beck and Beamesderfer, 1974). The BDI compares favourably with measures of global clinical assessment of depression (correlations of between 0.62 and 0.77)(Hamilton, 1982) indicating good concurrent validity.

Beck has recommended a cut-off score of thirteen as a screening device for the detection of depression and for research purposes a score of twenty-one has been recommended as identifying a relatively pure group of

depressed patients (Beck and Beamesderfer, 1974). The Center for Cognitive Therapy has more recently advocated the use of the following cut-off scores in depressed patients: scores of between 10 and 18 indicate mild to moderate depression, between 19 and 29 moderate to severe depression and 30 and above severe depression (Beck et al., 1988).

The validity of change in scores on the BDI was investigated by Beck et al (1961). They found that 85% of change in BDI score was reflected in changes in clinician global ratings. Bech et al. (1975) found a correlation of 0.82 between change in BDI score and change in global rating and 0.56 between change in BDI and change in HRS score.

Beck et al (1988) have reviewed studies which have a bearing on the construct validity of the BDI. Biological correlates of depression, suicidal and drinking behaviours, indicators of psychosocial adjustment and stress related symptoms have been associated with BDI scores.

3.4.6.c *Speilberger State-Trait Inventory*

The State-Trait Anxiety Inventory (STAI)(Speilberger et al., 1970) is a widely used self-report measure of subjective anxiety. It comprises of two 20 item scales, one requiring subjects to report feelings of anxiety at the time when they are completing the questionnaire (State) and the second scale requiring subjects to report how they generally feel (Trait). About half of each scale is made up of items where anxiety is rated as "present" and half "absent". Respondents rate each item on a four point scale.

The construct validity of the state anxiety scale was assessed by asking students to complete the scale under several stressful conditions and a relaxation condition. The mean scores were lowest under the relaxation condition and highest for watching a stressful film indicating that the scale has adequate construct validity (Speilberger et al., 1970).

Thompson (1989) has suggested that although the concurrent validity of the trait scale against other personality scales of anxiety and neuroticism has been found to vary from 0.53 to 0.85, trait anxiety may not measure an unvarying trait. There appears to be a consensus that trait and state anxiety will both show similar directional changes. Under conditions of low stress, the correlation of state and trait scales is approximately 0.65. Test-retest reliability of the state scale varies from 0.16 to 0.62 whilst the trait scale reliability is higher at 0.65 to 0.82. This indicates that scores on the trait scale will vary less than those on the state scale but under conditions of low stress, as expected, the two scales will correlate highly.

3.4.7 Cognitive Measures

3.4.7.a *Automatic Thought Questionnaire*.

The Automatic Thought Questionnaire (ATQ) was developed as a measure of the negative content of thinking (Hollon and Kendall, 1980). The ATQ is a 30 item self-rating questionnaire, each item being rated on a five point scale which indicates the frequency of occurrence of thoughts in the past week. The range of scores on the ATQ is 30 to 150. The original validation study of the ATQ was carried out in a sample of undergraduate students who were divided into depressed

and non-depressed groups on the basis of their scores on two measures of depression, above 11 on the BDI and above 26 on the Minnesota Multiphasic Personality Inventory, Depression Scale (MMPI)(Hathaway and McKinley, 1951). The ATQ was found it to be a valid measure of the negative content of thought in that it discriminated significantly ($p < 0.01$) between depressed and non-depressed students. The ATQ was also found to correlate highly with the two depression scales used to differentiate depressed from non-depressed students and with Speilberger's Trait Anxiety Inventory indicating that negative thinking may not be specific to depression.

Dobson and Breiter (1983) investigated the internal reliability, concurrent validity and the correlation of ATQ with other cognitive measures using a large sample of 456 undergraduate students. They found that the ATQ had high internal reliability (Cronbach's coefficient alpha =.96 for men and .95 for women). Concurrent validity was more modest but still significant at $p < .001$ ($r = .62$ with the BDI and $r = .36$ with the Dysfunctional Attitude Scale), indicating that the ATQ may be a sensitive indicator of level of depression.

The original study has been criticized for using undergraduates diagnosed on the basis of self-rating scales but more satisfactory studies, including some using clinical populations, have attested to the reliability and validity of the ATQ (Blackburn, 1988).

3.4.7.b *Cognitive Style Test*.

The Cognitive Style Test (CST)(Wilkinson and Blackburn, 1981; Blackburn et al., 1986(b)) is a measure of the three elements of Beck's cognitive triad. It was designed to assess the degree of negative interpretations

in both pleasant and unpleasant situations. The test consists of 30 statements, 10 relating to situations concerning the self, 10 concerning the world and 10 the future. Half of each of these elements concern pleasant and half unpleasant events. Four possible responses are available for each situation and the respondent is requested to choose one which most closely resembles how he or she would respond. An extremely negative response obtains a score of four and an extremely positive response, a score of one, with scores of two and three representing relatively positive or negative reactions. From the test, a total score can be obtained as well as scores on the different components of the triad and between interpretations of pleasant and unpleasant events.

Blackburn et al (1986(b)) revised the CST to increase its face validity for depressed patients and to increase the sensitivity of the test. They compared four groups on their scores on CST with patients meeting RDC criteria for major or minor depressive disorder; anxious patients, normal controls, recovered depressed patients, and recovered anxious patients. Analysis of covariance, with age as covariate, showed that the depressed group scored significantly higher on all six sub-scales of the CST. One scale, CST-S (CST, self sub-scale), did not differentiate depressed and recovered depressed patients. This suggests that negative thinking relating to the self may be a relatively enduring component of depressed thinking regardless of age. The CST (total score) was found to correlate with the BDI ($r = .49$, $p < .01$) but not with the HRSD or with the STAI. The absence of a significant correlation with the HRSD is not surprising

given that discrepancies have been noted between observer and self-reported depression which are attributed to raters and ratees response sets (Paykel et al., 1973). The CST therefore appears to be adequate conceptually. The CST was found to be internally consistent in that the correlations between sub-scales were highly significant ($p< .001$) within the depressed sample which would suggest that negative thinking tends to generalise across all elements of the cognitive triad.

Concurrent validity of the CST was shown by significant correlations ($p< .01$) with other measures of depressive thinking: the Hopelessness Scale ($r= 0.50$), Automatic Thought Questionnaire ($r= 0.57$) and the Dysfunctional Attitude Scale ($r= 0.44$).

The CST has therefore been shown to be internally consistent and to have reasonable construct and concurrent validity.

3.4.7.c *Dysfunctional Attitude Scale*.

The Dysfunctional Attitude Scale (DAS)(Weissman and Beck, 1978) is a self-report inventory of basic attitudes or beliefs, derived from Beck's (1967) cognitive theory of depression, which are assumed to underlie depressive thinking. The scale has three forms, two short forms and an original 100 item version. They contain items which relate to idiosyncratic beliefs which were collected from patients undergoing treatment; themes of achievement, love, approval, perfectionism and autonomy.

Each item is scored on a seven-point Likert scale which measures degree of agreement with the statement of belief. The total score on the two short forms of DAS is obtained from adding the score on each item and ranges from 40 to 280. Two parallel forms of the scale are

available (A and B), each scale having 40 items, from an original pool of 100 items, chosen after a factor analysis of the data obtained from 275 undergraduate students. The two forms correlate highly (r= .79) and are regarded as being equivalent to each other. They showed high internal consistency (p< .001) and to be reliable at re-test (p< .001).

The psychometric properties of the DAS were investigated by Oliver and Baumgart (1985) using an unselected adult population, mainly hospital staff and their spouses. The 275 subjects completed the BDI and DAS. Comparisons of the two parallel forms (A and B) revealed that they were less similar than had been previously thought: a principal components analysis revealed three factors on DAS A, characterised as need for approval, perfectionism and avoidance of risks with a fourth unnamed factor. Factor analysis of DAS B, on the other hand, produced four other factors: need for approval, need to impress others, need for success and need to control feelings. This finding, plus modest item-total correlations (50% of the correlations between .30 and .50 and 25% between .20 and .30) indicated that the two short forms may be different both in content and structure from each other. The authors suggested that DAS is a valid measure of depressogenic attitudes but would be better used in its original form rather than in the two shortened forms.

This study however, like many others, used the DAS A version. Cane et al (1986) in a principal-factors factor analysis with Varimax rotation of DAS A, found a similar factor structure to that found by Oliver and Baumgart (1985). The former authors found two factors which they

labelled performance evaluation and approval by others. These factors accounted for 47% and 14% of the variance respectively.

Several studies in clinical populations have noted that DAS scores decrease upon recovery from depression with the implication that dysfunctional beliefs are not a trait-like phenomena (Hamilton and Abramson, 1983; Simons et al., 1984; Blackburn et al., 1986(b)). Hollon et al (1986) examined the specificity of depressogenic cognitions in bipolar and unipolar depression and, included patients diagnosed as depressed secondary to substance abuse disorder, amongst the seven control groups. The DAS A was found to be state-dependent in that scores were within normal values for the recovered depressed groups. Those with depression secondary to substance abuse did not differ significantly from those with unipolar, bipolar depression and general psychiatric disorder which included schizophrenic patients in terms of their scores on DAS A. This suggests that the DAS is susceptible to non-specificity in relation to depression when used with non-depressed schizophrenic patients. This study did not test the specificity of the DAS with respect to disorders where high levels of anxiety might be found.

3.4.7.d *Hopelessness Scale*.

The Hopelessness Scale was designed to measure the future element of Beck's cognitive triad (HS)(Beck et al., 1974). The HS consists of 20 items which the respondent rates as either true or false, giving a total maximum score of 20. The internal reliability coefficient was found to be .93 in the initial study of 294 patients hospitalised after making recent suicide

attempts. Item-total correlation coefficients were all statistically significant and ranged from .39 to .76 suggesting that the total score is more reliable than any single item (Beck et al., 1974).

Concurrent validity was assessed by a comparison of scores on the HS with clinician ratings of hopelessness, total BDI scores and with scores on the BDI item of pessimism. All of these were significantly ($p < .001$), although moderately, correlated.

The construct validity of the HS has been established by testing hypotheses relevant to the construct of hopelessness. Dyer and Kreitman (1984) summarised six studies in which the relationship between hopelessness, depression and suicidal intent was investigated in suicide attempters and ideators. They found that there was considerable support for the view that suicidal intent is related to hopelessness rather than to level of depression: when level of depression was controlled, hopelessness was significantly correlated with suicidal intent in all but two studies.

Beck et al (1985(b)) followed-up 207 depressed patients who had been hospitalised because of suicidal ideation for a five to ten years period. The HS, assessed at time of admission, and the pessimism item of the BDI were the only measures found to predict the eventual suicide of 14 patients. However, in a prospective study of suicide attempters, Beck and Steer (1989) found that neither HS nor BDI scores at time of admission were found to be associated with increased risk of suicide in a cohort of patients with a variety of diagnosis. Of the measures used in the study, only the precautions sub-scale of the Suicidal Intent Scale

(SIS)(Beck et al., 1974) was significantly associated with increased risk of suicide.

Greene (1981) has reported normative data on the HS from a randomly selected sample of the general population. A mean score of 4.45 (s.d 3.09) was reported, lower than the reported means for clinical groups (Nekanda Trepka et al., 1983) where a mean of 13.05 (s.d 5.17) was reported for 86 depressed out-patients, and lower than a mean of 5.3 (s.d 4.2) found in a sample of 50 alcoholics at the beginning of treatment (McGovern, 1986).

3.4.7.e *Self-Control Schedule*.

The Self-control Schedule (SCS)(Rosenbaum, 1980) is a measure of self-control which, unlike the above cognitive measures, was not developed to measure cognition but to assess various aspects of self-control behaviours: combating unpleasant emotional and physical states, solving everyday problems, delaying gratification, and evaluating one's situation and oneself. The schedule consists of 36 items, each scored on a six point Likert scale. The total score is the sum of all the responses.

Rosenbaum (1980) found that the schedule had high test-retest reliability (r=.86) and internal consistency (alpha coefficient range .78 to .88) when assessed in four student samples, and one of Israeli men. The concurrent validity of the SCS was assessed by two measures which were conceptually related to the SCS; modest correlations were found between SCS and externality on Rotter's I-E scale (Rotter, 1966) ($r=-.40$, $p< .01$) and with total score on the Irrational Beliefs Test (IBT)(Jones, 1968)($r=-.48$, $p< .001$). Rude (1983)

failed to find an association between the SCS and depressive symptoms (r=-.08 between BDI and SCS and r=-.28 between SCS and 30 item MMPI Depression scale). Otherwise, there is little information on the validity of the SCS.

Simons et al.(1985) reported that depressed patients scoring high in SCS at the beginning of treatment did better in treatment with cognitive therapy than those who scored low on SCS. Patient who did better with pharmacotherapy showed the opposite pattern. They concluded that "learned resourcefulness" was a good predictor of response to cognitive therapy. This finding needs replication and in addition suggests that SCS may be worthy of further exploration in clinical populations.

3.5 ANALYSIS OF DATA

Data collated during the present study were entered on the University of Edinburgh main-frame computer network and SPSS-X (release 3.0) was used to analyse data.

Categorical data were analysed using chi-square and the Fisher's exact test. On the whole, other analyses of data were carried out using parametric univariate statistics: Pearson correlation, analysis of variance, independent-measures t statistic. The distribution of all affective symptomatology and cognitive variables were distributed approximately normally on the first occasion of testing. At first and second follow-up, this distribution continued to approximate a normal distribution and was not significantly improved by performing a logarithmic transformation.

Multivariate statistics were used in the repeated analyses of variance. Univariate repeated measures ANOVA with more than 1 df for the repeated measure requires homogeneity of covariance (Tabachnick and Fidell, 1989). As variables measured closer in time tend to be more highly correlated than variables measured further apart in time, the assumption of homogeneity of covariance is likely to be violated, leading to increased Type 1 error. Several strategies are available to analyse data in the event of violation of this assumption. A multivariate approach to repeated measures, is a statistically acceptable alternative to repeated measures ANOVA because multiple dependent variables replace the within-subjects independent variables and the assumption of homogeneity of covariance is no longer required. Although other assumptions such as homogeneity of variance-covariance

matrices and absence of multicollinearity and singularity must be met, they are less likely to be violated.

One preferred solution to univariate repeated measures ANOVA is a trend analysis (Tabachnick & Fidell, 1989). As statistical tests of trends and other contrasts use single degrees of freedom of the within-subjects independent variables, there is no possibility of violation of homogeneity of covariance.

The main aim of the repeated measures analysis was to investigate change in scores of affective symptomatology and cognitive measures over time. As the interval between each occasion of testing was not equal, polynomial contrasts were applied to the variables (Hand and Taylor, 1987). The results could then be used to describe the overall change across time and the rate at which the scores changed with time.

Principal components analysis was used to describe the relationship between affective symptoms and cognitive scores on separate occasions of testing. The main aim was to produce an empirical summary of scores of affective symptomatology and cognitive variables, not to test a specific hypothesis relating to these variables. Principal components analysis was therefore used in preference to factor analysis. Varimax orthogonal rotation was performed to simplify factors and maximise the loadings within factors. Factor scores were used to provide estimates of the scores subjects would have received on each of the components had they been measured directly.

CHAPTER FOUR

RESULTS

4.1 DESCRIPTION OF THE SAMPLE

Table 4.1.1 contains a description of the general characteristics of the sample. One third of the sample was female and two-thirds were male. The average age of the sample was over forty. Roughly two-thirds were in social classes III, IV and V with the remainder in social classes I and II. Only one quarter of the sample was employed, the majority having been unemployed between one month and five years. Approximately two-thirds of the sample had minimal educational qualifications. Approximately two-thirds were owner-occupiers or lived in rented accommodation, and one quarter lived either in their parental home or in their partner's home.

Table 4.1.2 provides a description of the sample on both follow-up occasions. Eight patients were lost to the study in that they did not attend any follow-up appointments. Seventy eight percent of the patients taking part in the study attended the first follow-up and 83 percent attended the second follow-up.

The average number of days from discharge to the first follow-up appointment was 44 days or just over six weeks. There was an average of 112 days or 16 weeks from the first follow-up appointment until the second follow-up. Added together, the average total length of follow-up was 22 weeks, roughly five and a half months from discharge.

At the first follow-up, 45 percent were abstinent from alcohol. Fifty five percent had been drinking during the first follow-up period. Among those who had been drinking, alcohol was consumed on an average number of six days during that time. In the period since

discharge, the mean number of units of alcohol consumed was 70, although the range varied from zero to 779 units. The average number of units of alcohol consumed on the heaviest drinking day was 14. The average number of units of alcohol consumed on the lightest drinking day was four but again there was a considerable range in the number of units consumed.

Fourteen patients did not attend the second follow-up appointment. During the time period to the second follow-up, only nineteen percent of patients had remained abstinent, eighty one percent had been drinking during this time. An average of approximately one third of the days during this time had been drinking days, although some patients had been drinking on most days.

The total number of units of alcohol consumed over this period was higher than in the first follow-up period. For example, the average amount consumed on the heaviest day's drinking had increased by one third from the first follow-up period to 21 units.

Table 4.1.1 Description of Sample (n=82)

	n	%
Sex		
male	55	67
female	27	33
Social Class		
I and II	26	32
III	31	38
IV and V	25	30
Marital Status		
single	19	23
married/cohabiting	27	33
separated/divorced	31	38
widowed	5	6
Education		
degree/diploma	16	20
SED Highers	15	18
"O" level or 10 years at school	51	62
Work Status		
full employment	20	24
unemployed:		
1-6 months	25	30
6-12 months	10	12
1-3 years	16	20
3-5 years	5	6
never worked	3	4
retired (ill-health)	3	4
Housing		
owner-occupier	29	35
rented	25	30
tied housing	4	5
lodging house	3	4
parental/partner	21	26
Age (mean sd,range)	41.13	(10.43, 19-60)

Table 4.1.2 Description of Outcome for Those Attending Both Follow-up Occasions

	First follow-up	Second follow-up
Number attending follow-up n(%)	64 (78)	68 (83)
Number not attending follow-up n(%)	18 (22)	14 (17)
Number of days to follow-up	44(20)(20-112)	112(55)(25-284)
Abstinent: n (%)	29(45)	13(19)
Drinking: n (%)	35(55)	55(81)
Number of days drinking	6(15)(0-112)	42(46)(0-200)
Total number of units consumed	70(129)(0-779)	623(879)(0-5281)
Units of alcohol consumed per day	2(3) (0-24)	6(7) (0-40)
Number of units drunk on heaviest drinking day	14(17)(0-58)	21(18)(0-70)
Number of units drunk on lightest drinking day	4(6)(0-30)	8(10)(0-61)!!

!! 2 cases missing
Unless indicated otherwise, all values in the table are mean (sd)(range).

4.2 DEPRESSIVE DISORDER IN ALCOHOLISM

4.2.1 Prevalence of Depression in Alcoholism

For the episode of drinking that led to admission, the majority of patients were diagnosed as having major depressive disorder in addition to alcoholism, according to RDC. However, after detoxification from alcohol, this situation was reversed; the minority were diagnosed as having major depressive disorder, with 52 having no depression (Table 4.2.1).

Table 4.2.1 Diagnosis of Depression in Alcoholics: Pre-Admission and Post Detoxification

	Pre-Admission	Post Detoxification
	n=82	n=82
No depression	12	52
Minor depression	15	19
Major depression	55	11

Of the 55 patients diagnosed as having had major depressive disorder in the pre-admission episode, 31 had no depression after detoxification, 14 had probable minor depressive disorder and 10 had a diagnosis of definite major depressive disorder. Twelve of the 15 patients diagnosed as having minor depressive disorder had no depression after detoxification, two continued to have minor depressive disorder and one got worse and was diagnosed as having probable major depressive disorder. On the whole those with no depression in the pre-admission episode continued to experience no depression, although three patients experienced symptoms of depression which met criteria for a diagnosis of probable minor depression.

Of the 11 patients with a post detoxification diagnosis of major depression , 10 had major depressive disorder during the pre-admission episode and one patient had probable minor depressive disorder. Therefore only one patient had a diagnosis of probable major depressive disorder post-detoxification, the remaining ten continued to meet diagnostic criteria for definite major depressive disorder.

Figure 3

Diagnosis of Depression: Changes following Detoxification

Pre-admission			**Post-detoxification**	
Diagnosis	n		n	Diagnosis
		->->	10	Major
Major	55	->->	14	Minor
		->->	31	No Depression
		->->	1	Major
Minor	15	->->	2	Minor
		->->	12	No Depression
		->->	0	Major
No	12	->->	3	Minor
Depression		->->	9	No Depression

Summary Most alcoholics who presented for in-patient treatment had an additional diagnosis of depression. However, following detoxification from alcohol, only a minority remained depressed. These results provide evidence in support of hypotheses 1 and 1a.

4.2.2 General description of the sample in terms of pre-admission and post-detoxification diagnosis

Table 4.2.2 contains demographic data for patients who were diagnosed as having no depression, minor or major depression in addition to alcoholism during the pre-admission episode. One way analysis of variance revealed no difference in age between the groups ($F=1.80$, df 2,79 ns).

In order to compare categorical variables, the diagnostic groups were collapsed: no depression and minor depression were combined and compared to those with major depression. These variables were then analysed using the chi-square test.

For the pre-admission diagnosis, a chi-square analysis showed no difference between the groups on sex distribution (chi-square=0.00, df 1, ns). There was a significant difference in social class between the diagnostic groups (I and II compared with III and with IV and V) (chi-square=9.12, df 2, $p<0.05$). Patients in social class I and II were less likely to have major depression than those in other social classes. In order to analyse marital status, married and cohabiting patients were compared to the other categories combined together. Chi-square analysis revealed no difference in marital status between the groups (chi-square=0.09, df 1, ns). Educational level was analysed by combining those with degrees, diplomas and Scottish Highers or equivalents and this combination was compared with those who had achieved "O" levels or had completed at least 10 years of education: no difference was found between the groups in level of education achieved (chi-square=0.02, df 1, ns). In terms of work status, three groups were compared: those in full employment, those who had been

unemployed between one and six months and those who had been unemployed for longer than six months (including those who had either never worked or who had been retired on grounds of ill-health). No significant differences were noted between the three groups in terms of work status (chi-square=1.19, df 2, ns). Owner-occupiers were compared with those in rented accommodation and with the other categories combined. There was no difference in category of housing between the groups (chi-square=1.45, df 2, ns).

Table 4.2.3 contains a summary of demographic variables for patients who were diagnosed as having no depression, minor or major depression, once detoxification had been completed. The patients described here are the same patients who are described in table 4.2.2 relating to pre-admission diagnosis. The same variables were analysed to explore whether the characteristics of the population change after detoxification from alcohol in terms of demographic and alcohol related variables.

One way analysis of variance revealed no significant difference between the three groups in age (F=0.11, df 2,79, ns).

For the analysis of categorical variables, the diagnostic groups were again collapsed. On this occasion, minor and major depression were combined into a single depressed group and this group was compared to those patients with no depression post-detoxification. There were no significant differences in sex distribution between the groups (chi-square=0.31, df 1, ns). There was no significant difference in social class between the groups (social classes I and II compared to III and to IV

and V) (chi-square=0.57, df 2, ns). No significant difference was noted between the groups in marital status when those who were married or cohabiting were compared to the other categories combined (chi-square=0.09, df 1, ns). Approximately 37% of those who were depressed and 31% of those who were not depressed were either married or cohabiting. There was no difference between the groups in terms of education (chi-square=0.76, df 1, ns). Those who had obtained at least Scottish "Highers" or above were compared with those who had obtained "O" levels or less.

A significant difference was noted between the groups in work status (chi-square=6.82, df 2, $p<.05$). Further analysis revealed that those who had been unemployed for between one and six months were less likely to be depressed than those in full employment and long term unemployment combined.

In terms of housing, there were no significant differences between the groups (chi-square=0.99, df 2, ns). Owner occupiers were compared to those in rented accommodation and with those who lived in either lodgings, tied housing and the homes of either parent or partner combined.

Table 4.2.2 Description of Sample (Pre-Admission)

	No Depression n=12 n	MinorDepression n=15 n	Major Depression n=55 n	p
Sex;				
Male/Female	9/3	9/6	37/18	ns
Social Class;				(.05
I and II	7	7	12	
III	2	3	26	
IV and V	3	5	17	
Marital Status;				ns
single	3	3	13	
married/cohabiting	5	5	17	
widowed	0	3	2	
separated/divorced	4	4	23	
Education;				ns
degree/diploma	4	2	10	
SED highers	2	3	10	
"O" levels, 10 years school	6	10	35	
Work Status;				ns
full employment	4	3	13	
unemployed:				
1-6months	3	7	15	
6-12months	2	0	8	
1-3years	2	4	10	
3-5years	1	0	4	
never worked	0	0	3	
retired (ill-health)	0	1	2	
Housing;				ns
owner-occupier	6	6	17	
rented	1	6	18	
tied house	0	0	4	
lodging house	1	0	2	
parental/partner	4	3	14	
Age				
mean(sd)	43.3(9.1)	44.9(11.4)	39.7(10.3)	ns

Table 4.2.3 Description of Sample (Post-Detoxification)

	No Depression n=52 n	Minor Depression n=19 n	Major Depression n=11 n	p
Sex;				
male/female	36/16	12/7	7/4	ns
Social class;				ns
I and II	18	7	1	
III	19	8	4	
IV and V	15	4	6	
Marital Status;				ns
single	13	4	2	
married/cohabiting	16	6	5	
widowed	3	1	1	
separated/divorced	20	8	3	
Education;				ns
degree/diploma	11	4	1	
SED highers	11	1	3	
"O" level, 10 years school	30	14	7	
Work Status;				<.05
full employment	10	7	3	
unemployed				
1-6 months	21	3	1	
6-12 months	3	4	3	
1-3 years	10	3	3	
3-5 years	3	1	1	
never worked	2	1	0	
retired(ill-health)	3	0	0	
Housing;				ns
owner-occupier	20	6	3	
rented	14	8	3	
tied	3	0	1	
lodging house	1	1	1	
parental/partners	14	4	3	
Age;				
mean(sd)	41.5(10.8)	40.8(10.3)	39.9(9.8)	ns

Table 4.2.4 describes the relationship between pre-admission diagnosis and variables related to alcohol problems and consumption. Analysis of all variables in the table was carried out with one way analysis of variance. The groups did not differ significantly on their scores on the SADQ (F=1.77, df 2,79, ns) or on the MMS (F=0.00, df 2,79, ns). There were no significant differences between the groups in the age at which problems with drinking began (F=0.60, df 2,79, ns) or in the number of years of problem drinking (F=1.65, df 2,79, ns). There a significant difference between the groups in the number of past episodes of problem drinking (F=1.34, df 2,78, ns) or in the number of alcohol related problems reported by the groups on admission (F=0.53, df 2,79, ns). There were no significant differences between the groups in the total number of units of alcohol consumed in the week prior to admission (F=0.83, df 2,79, ns), nor in the number of days on which alcohol was consumed in the week prior to admission (F=1.56, df 2,79, ns).

In general, in the week prior to admission, all groups had decreased their intake of alcohol from the level consumed in a typical week in the three months before admission. There were, however, no significant differences between the groups on the number of units of alcohol consumed in a typical week in the three months prior to admission (F=1.28, df2,79, ns).

Table 4.2.4 Description of alcohol related variables (Pre-Admission)

	No Depression mean(sd)	Minor Depression mean(sd)	Major Depression mean(sd)	p
	n=12	n=15	n=55	
SADQ	25.0(13.6)	23.5(12.4)	29.5(11.8)	ns
MMS	28.2(1.9)	28.2(1.9)	28.2(1.5)	ns
age problem drinking began	25.9(8.4)	28.9(9.7)	26.3(8.4)	ns
years problem drinking	15.0(10.7)	11.7(5.1)	10.7(7.3)	ns
episodes of problem drinking	1.8(0.9)	3.2(4.9)!	2.2(1.5)	ns
number of alcohol related problems	11.1(2.1)	11.0(3.8)	10.4(2.4)	ns
units alcohol in pre-admission week	87.7(80.4)	117.2(139.9)	128.5(90.7)	ns
days drinking in pre-admission week	4.5(2.8)	4.1(3.2)	5.3(2.3)	ns
units alcohol in typical week before admission	117.3(85.5)	172.9(123.1)	156.5(84.1)	ns

! 1 case missing
SADQ: Severity of Alcohol Dependence Questionnaire
MMS : Mini Mental State

Table 4.2.5 describes the three diagnostic groups after detoxification in relation to variables measuring alcohol problems and consumption. One way analysis of variance was used to analyse all variables.

No significant differences between the three groups were noted in scores on the SADQ (F=3.02, df 2,79, ns) or on the MMS (F=1.98, df 2,79, ns). The average age reported for the onset of problem drinking was 26 years for all groups and no significant difference was found between the groups (F=0.01, df 2,79, ns). On average, 11 years problem drinking was reported. In terms of the number of episodes of drinking throughout lifetime, the groups were not significantly differentiated (F=1.44, df 2,78, ns).

The number of alcohol related problems experienced in the episode of drinking which led to admission was not significantly different between the groups (F=0.42, df 2,79, ns). There was no significant difference between groups in alcohol consumption in the week prior to admission (F=2.58, df 2,79, ns). Nor was there a significant difference between the groups in the number of days in which alcohol was consumed in the week preceding admission (F=0.79, df 2,79, ns). The number of units of alcohol consumed in a typical week in the three month period leading to admission was also not significantly different between the groups (F=0.02, df 2,79, ns).

Table 4.2.5 Description of alcohol related variables (Post-Detoxification)

	No Depression n=52 mean(sd)	Minor Depression n=19 mean(sd)	Major Depression n=11 mean(sd)	p
SADQ	27.0(12.2)	25.1(9.6)	35.7(14.6)	ns
MMS	28.5(1.5)	27.8(1.9)	27.6(1.5)	ns
age problem drinking began	26.8(8.3)	26.8(9.1)	26.5(9.7)	ns
years problem drinking	11.6(7.4)	11.3(8.2)	11.4(8.2)	ns
episodes of problem drinking	2.1(1.4)	3.1(4.3)	1.9(0.7)!	ns
number of alcohol related problems	10.5(2.7)	10.4(2.8)	11.3(2.0)	ns
units alcohol in pre-admission week	119.1(89.5)	92.1(71.2)	176.0(160.3)	ns
days drinking in pre-admission week	5.2(2.6)	4.4(2.5)	4.9(2.7)	ns
units alcohol in typical week before admission	153.5(95.3)	156.4(86.4)	150.3(99.3)	ns

! 1 case missing
SADQ: Severity of Alcohol Dependence Questionnaire
MMS : Mini Mental State

4.2.3 Past Psychiatric History

Table 4.2.6 Number (%) of past psychiatric diagnoses for patients with Major Depression and with either Minor or no Depression in the pre-admission episode

Pre-admission Diagnosis	ALC	DEP	ANX	DRUG	EAT	ASP	SUIC
Depressed N=55	38(69)	11(20)	11(20)	2(4)	2(4)	6(11)	7(13)
Minor or No Depression N=27	19(70)	5(19)	3(11)	2(7)	0(0)	7(26)	17(63)
Total Population	57(70)	16(20)	14(17)	4(5)	2(2)	13(16)	24(29)

NB A patient can have more than one diagnosis

ALC :Alcoholism
DEP :Depression (unipolar and bipolar)
ANX :Anxiety, Obsessive Compulsive Disorder
DRUG:Drug Dependence
EAT :Anorexia Nervosa, Bulimia Nervosa
ASP :Antisocial Personality Disorder
SUIC:Suicidal behaviour

For the pre-admission episode, those patients with minor and no depression were collapsed into one group and were compared with those patients with major depression.

No significant difference were noted in the prevalence of the following past psychiatric disorders: alcoholism (chi square=0.00, df 1, ns); depression (chi square=0.00, df 1, ns.); anxiety and obsessive compulsive disorder (chi square=0.48, df 1, ns); drug abuse (Fisher's exact probability=0.40, ns); eating disorder (Fisher's exact probability=0.45); antisocial personality disorder (chi square=1.94, df 1, ns); past suicidal behaviour (chi square=0.04, df 1, ns).

Table 4.2.7 Number (%) of Past Psychiatric Diagnosis for Depressed and Non-Depressed Patients at Post-Detoxification

Post Detoxification Diagnosis	ALC	DEP	ANX*	DRUG	EAT	ASP	SUIC
Depressed N=30	19(63)	9(30)	9(30)	2(7)	2(7)	8(27)	12(40)
Non-depressed N=52	38(73)	7(13)	5(10)	2(4)	0(0)	5(10)	12(23)
Total population	57(70)	16(20)	14(17)	4(5)	2(2)	13(16)	24(29)

NB A patient can have more than one diagnosis

ALC :Alcoholism
DEP :Depression (unipolar and bipolar)
ANX :Anxiety, Obsessive Compulsive Disorder
DRUG:Drug Dependence
EAT :Anorexia Nervosa, Bulimia Nervosa
ASP :Antisocial Personality Disorder
SUIC:Suicidal behaviour

* $p < 0.05$

The past psychiatric history of the depressed (minor and major) and non depressed groups post-detoxification are described in table 4.2.7. The depressed group had a significantly higher prevalence rate of anxiety disorders (including obsessive compulsive disorder) compared to the non-depressed group (chi square=4.24, df 1, $p < 0.05$).

However no significant differences were noted in the prevalence of the following psychiatric disorders or in suicidal behaviour between the two post detoxification diagnostic groups: alcoholism (chi square=0.45, df 1, ns); depression (chi square=2.34, df 1, ns); drug abuse (Fisher's exact probabilty=0.47); eating disorders (Fisher's exact probability=0.13); antisocial personality disorder (chi square=0.00, df 1, ns); past suicidal behaviour (chi square=1.88, df 1, ns).

4.2.4 Family History Of Depression.

The relationship between family history of depression and the diagnosis of depression in patients during the episode of drinking which led to admission and after detoxification was investigated. If any first degree relative with a history of depression met RDC criteria for depressive disorder, then this was considered to be a positive family history of depression.

Table 4.2.8 and table 4.2.9 show that there was no significant association between a positive family history of depression and pre-admission diagnosis of depression (chi-square=0.00, df 1, ns) or post-detoxification diagnosis (chi-square=0.05, df 1, ns).

Table 4.2.8 Relationship between Positive Family History of Depression and Pre-admission Diagnosis

	Minor and No Depression	Major Depression
Positive family history depression	5	9
No family history of depression	22	46

Table 4.2.9 Relationship between Positive Family History of Depression and Post-detoxification Diagnosis

	No Depression	Major and Minor Depression
	n	n
Positive family history depression	8	6
No family history of depression	44	24

4.2.4.a *Relationship of primary depression to family history and diagnosis in the pre-admission and post-detoxification episode.*

Table 4.2.10 shows that there was no relationship between having a primary diagnosis of depression and family history of depression (Chi-square=0.01, df 1, ns).

The relationship between a primary diagnosis of depression and a diagnosis during the current episode was also explored. Table 4.2.11 and 4.2.12 show that there was no relationship between a primary diagnosis of depression and diagnosis in the pre-admission episode (Chi-square=1.17, df 1, ns) or post-detoxification (Chi-square=2.57, df 1, ns)

Table 4.2.10 Relationship between primary diagnosis of depression and family history of depression

	primary depression n	other disorders primary n
Positive family history depression	2	12
No family history of depression	6	62

Table 4.2.11 Relationship between primary diagnosis of depression and other psychiatric disorder and a diagnosis of depression in the pre-admission episode.

	Minor and No Depression n	Major Depression n
primary depression	4	4
other disorders primary	23	51

Table 4.2.12 Relationship between primary diagnosis of depression and other psychiatric disorder and diagnosis of depression after detoxification.

	No Depression n	Minor and Major Depression n
primary depression	3	5
other disorders primary	49	25

Summary of findings relating to the second hypothesis.

On the whole, few differences were found between alcoholics with and without depression on demographic variables. There were two exceptions: those in social classes I and II in comparison with other social classes were less likely to have a diagnosis of major depression in the episode leading to admission and secondly, those unemployed for between one and six months were less likely to have a diagnosis of depression post-detoxification than those in other categories of employment.

No relationship was evident between family history of depression and diagnosis of depression in the index episode. Nor was there a relationship between having a primary (first ever) diagnosis of depression and having a diagnosis of depression in the index episode.

There were no differences between those with and without depression in drinking history or in alcohol consumption before admission. Depressed alcoholics had not therefore been drinking more alcohol than those without depression before admission.

In terms of past psychiatric history, compared to alcoholics without depression, those alcoholics with a diagnosis of depression after detoxification had a significantly higher prevalence of past psychiatric diagnoses of anxiety disorders.

4.2.5 Treatment during inpatient stay

Patients with a post-detoxification diagnosis of depression were more likely to receive antidepressant medication than were other patients (chi-square=5.53, df 1, $p<0.05$) (table 4.2.13). The category "depression" includes both minor and major depression. However, table 4.2.14 shows that there was no significant relationship between receiving antidepressant medication and pre-admission diagnosis of depression (chi-square=1.21, df 1, ns).

Table 4.2.13 Post-detoxification diagnosis and treatment for depression during inpatient stay

	No depression n	Depression n
received antidepressant medication	2	7
no antidepressant medication	50	23

Table 4.2.14 Pre-admission diagnosis and treatment for depression during inpatient stay

	Minor and No Depression n	Major Depression n
received antidepressant medication	1	8
no antidepressant medication	26	47

4.2.6 Treatment for depression at outcome

Table 4.2.15 shows that fourteen patients received either ECT or antidepressant medication during the follow-up period.

Table 4.2.15 Number(%) of patients receiving medication and other treatment at follow-up

Treatment	n (%)
ECT	1 (1)
antidepressant medication	13 (16)
anxiolytic (withdrawal) medication	21 (26)
other medication	7 (9)
antabuse (1st follow-up)	33 (40)
antabuse (2nd follow-up)	21 (26)

Table 4.2.16 demonstrates that there was no significant difference in treatment for depression at follow-up between those patients with a pre-admission diagnosis of major depression and those with a pre-admission diagnosis of minor or no depression (chi-square=3.77, df 1, ns). However, table 4.2.17 shows that those patients with a post-detoxification diagnosis of minor or major depression were more likely to have received treatment for depression during follow-up than those with no depression (chi-square=7.12, df 1, $p<0.01$).

Table 4.2.16 Pre-admission diagnosis and treatment for depression at outcome

	Minor and No Depression n	Major Depression n
received treatment for depression	1	13
no treatment for depression	26	42

Table 4.2.17 Post detoxification diagnosis and treatment for depression at outcome

	No depression n	Depression n
received treatment for depression	4	10
No treatment for depression	48	20

4.2.7 Treatment for withdrawal states at outcome

Table 4.2.19 shows that there was no significant relationship between post-detoxification diagnosis of depression and receiving treatment for withdrawal states (chi-square=0.00, df 1, ns), nor was there a significant difference between pre-admission diagnosis of depression and receiving medication (chi-square=0.73, df 1, ns) (table 4.2.18).

Table 4.2.18 Pre-admission diagnosis of depression and treatment for withdrawal states at outcome

	Minor and No Depression n	Major Depression n
received treatment for withdrawal states	9	12
no treatment for withdrawal states	18	43

Table 4.2.19 Post-detoxification diagnosis and treatment of withdrawal states at outcome

	No depression n	Depression n
received treatment for withdrawal states	13	8
no treatment for withdrawal states	39	22

4.2.8 Relationship between diagnosis of depression and re-admission to hospital

Table 4.2.20 shows that there was no significant relationship between re-admission to hospital during

follow-up and pre-admission diagnosis of depression (chi-square=0.00, df 1, ns). Table 4.2.21 demonstrates that a post-detoxification diagnosis of depression also did not have a significant relationship with the number of re-admissions to hospital at follow-up (chi-square=1.9, df 1, ns).

Table 4.2.20 Re-admission to hospital during follow-up: Pre-admission diagnosis

	Minor and No depression	Major Depression
Re-admission	6	13
No re-admission	21	42

Table 4.2.21 Re-admission to hospital during follow-up: Post-detoxification diagnosis

	No depression	Depression
Re-admissions	9	10
No re-admissions	43	20

Summary of treatment relating to hypothesis 3.

Compared to alcoholics with and without depression in the pre-admission episode and to alcoholics without depression after detoxification, those alcoholics who remained depressed after detoxification were more likely to have received treatment for depression during admission and at follow-up. Those who had either a pre-admission or a post-detoxification diagnosis of depression were not more likely than those who were not depressed to have had treatment for withdrawal states or to be re-admitted to hospital at follow-up.

4.2.9 Diagnostic Groups at Follow-up: relationship between diagnosis and drinking outcome.

Table 4.2.22 demonstrates that there was no significant relationship between pre-admission diagnosis and abstinence at first follow-up (chi-square=0.06, df 1, ns) or at second follow-up (chi-square=0.21, df 1, ns).

Table 4.2.23 similarly shows that no significant relationship existed between post-detoxification diagnosis and abstinence at first follow-up (chi-square=0.00, df 1, ns) or at second follow-up (chi-square=0.00, df 1, ns).

Table 4.2.22 Relationship between abstinence from alcohol at follow-up and pre-admission diagnosis

	Minor & no depression		Major depression	
	1st f-up	2nd f-up	1st f-up	2nd f-up
Abstinent	10	6	19	7
Drinking	10	19	25	36

Table 4.2.23 Relationship between abstinence from alcohol at follow-up and post-detoxification diagnosis

	No depression		Major & Minor depression	
	1st f-up	2nd f-up	1st f-up	2nd f-up
Abstinent	18	8	11	5
Drinking	22	36	13	19

4.2.9.a *Relationship between pre-admission diagnostic groups and drinking variables at outcome (Drinkers only)*

One way analysis of variance was used to analyse all variables in table 4.2.24. There was no significant difference between the three pre-admission diagnostic groups (including those abstinent) in length of time to first follow-up (F=0.73, df 2,61, ns). There was no significant difference between the groups in the number of days to the first drink (F=0.88, df 2,32, ns). The number of days drinking in the first follow-up period was not significantly different between the groups (F=0.44, df 2,32, ns). There was no significant difference between the groups in the total quantity of units of alcohol consumed during the first follow-up (F=1.54, df 2,32, ns), nor in the number of units of alcohol consumed per day of follow-up (F=1.14, df 2,32, ns). There were no significant differences between the groups in the number of units of alcohol consumed on the heaviest drinking day (F=0.91, df 2,32, ns) or on the lightest drinking day (F=0.03, df 2,32, ns).

Table 4.2.25 shows that there were no differences between the pre-admission diagnostic groups on any of the drinking variables at second follow-up. All variables in Table 4.2.25 were analysed with a one way analysis of variance. No significant difference was evident between the groups in the number of days from the first follow-up to the second follow-up (including those abstinent) (F=0.86, df 2,65, ns). For the 55 patients known to have relapsed, there was no significant difference in the number of days to the first relapse in this follow-up period (F=0.05, df 2,48, ns). Nor were there any significant differences between the groups in the number of days in the second follow-up that were spent drinking

(F=1.34, df 2,52, ns), or in the total number of units of alcohol consumed in that period (F=0.22, df 2,52, ns) or in the number of units consumed per day of follow-up (F=0.29, df 2,52, ns) or in the number of units of alcohol consumed on the heaviest drinking days (F=0.01, df 2,52, ns) or on the lightest drinking days (F=1.26, df 2,51, ns).

Table 4.2.24 Drinking Outcome at First Follow-up for Pre-admission Diagnostic Groups (Drinkers only)

	No Dep N=6 mean(sd)	Minor Dep N=4 mean(sd)	Major Dep N=25 mean(sd)	p
Number of days from discharge to 1st f-up: n=9/11/44!	51.8(23.3)	42.9(21.3)	43.1(19.1)	ns
Number of days from discharge to 1st drink:	20.8(34.0)	5.8(5.7)	17.8(14.7)	ns
Number of days drinking:	7.0(8.3)	6.0(4.1)	13.4(22.0)	ns
Total number of units alcohol drunk:	64.3(47.9)	50.0(31.5)	156.1(170.5)	ns
Units alcohol per day follow-up	1.3(1.3)	1.5(0.8)	3.7(4.7)	ns
Number of units drunk on heaviest day:	22.0(15.2)	16.5(9.1)	26.7(15.5)	ns
Number of units drunk on lightest day:	6.5(7.6)	6.8(7.8)	7.2(7.3)	ns

No dep : No Depression, Alcoholism only.
Minor Dep: Minor Depression and Alcoholism
Major Dep: Major Depression and Alcoholism
f-up : follow-up
! : total number attending

Table 4.2.25 Drinking Outcome at Second Follow-up for Pre-admission Diagnostic Groups (Drinkers only)

	No Dep N=10 mean(sd)	Minor Dep N=9 mean(sd)	Major Dep N=36 mean(sd)	p
Number of days from 1st f-up to 2nd f-up n=12/13/43!	108.9(59.2)	129.8(69.3)	107.3(49.0)	ns
Number of days from 1st f-up to 1st drink: n=10/8/33!!	22.0(25.6)	23.5(34.9)	25.7(39.2)	ns
Number of days drinking	41.1(40.8)	73.6(57.3)	50.2(43.0)	ns
Total number of units alcohol drunk	925.8(971.9)	821.3(637.1)	713.8(977.3)	ns
Units alcohol per day follow-up	8.5(8.8)	5.9(3.3)	6.8(8.2)	ns
Number of units drunk on heaviest day: mean (sd)	26.5(13.6)	25.7(13.6)	26.6(17.6)	ns
Number of units drunk on lightest day: n=10/9/35!!	12.7(14.5)	4.9(3.4)	9.3(10.7)	ns

No dep : No Depression, Alcoholism only.
Minor Dep: Minor Depression and Alcoholism
Major Dep: Major Depression and Alcoholism
f-up : follow-up
! : total number attending
!! : number in group

Table 4.2.26 shows the relationship between alcohol related problems and pre-admission diagnostic groups. One way analysis of variance found no significant differences between the pre-admission diagnostic groups in the total number of alcohol related problems (F=0.19, df 2,71, ns), nor in the number of alcohol related social problems at outcome (F=0.36, df 2,71, ns), nor in the number of physical dependency problems at outcome (F=0.03, df 2,71, ns).

Table 4.2.26 Alcohol Related Problems across the Follow-up Period for Pre-admission Diagnostic Groups

	No Dep n=11 mean(sd)	Minor Dep n=14 mean(sd)	Major Dep n=49 mean(sd)	p
Total number of problems	5.3(3.8)	4.8(4.4)	5.5(3.9)	ns
Number of social problems	2.1(1.5)	1.9(1.7)	2.3(1.6)	ns
Number of physical dependency problems	3.4(2.5)	3.1(2.7)	3.3(2.7)	ns

No dep : No Depression, Alcoholism only.
Minor Dep: Minor Depression and Alcoholism
Major Dep: Major Depression and Alcoholism

4.2.9.b *Relationship between post-detoxification diagnostic groups and drinking variables at outcome (drinkers only)*

One way analysis of variance was used to analyse all variables in table 4.2.27. There were no significant differences between the post detoxification diagnostic groups (including those abstinent) in length of time to first follow-up (F=1.26, df 2,61, ns). The average time period to the first follow-up was 44 days or approximately 6 weeks. No significant differences were found in the number of days from discharge to the first drink taken by patients in the groups who relapsed (F=1.36, df 2,32, ns). The average number of days of drinking in the first follow-up period was not significantly different for the three groups (F=2.22, df 2,32, ns). No significant differences were noted between the groups in the total amount of units of alcohol consumed in the period up to the first follow-up (F=0.88, df 2,32, ns) or in the number of units of alcohol consumed per day of follow-up (F=0.44, df 2,32, ns). There was no significant difference in the number of units of alcohol consumed on the heaviest drinking day (F=0.73, df 2,32, ns), nor was there a significant difference in the amount of alcohol consumed on the day of lightest drinking (F=0.93, df 2,32, ns).

As in table 4.2.27, drinking variables in table 4.2.28 were analysed using one way analysis of variance. There was no significant difference in the length of follow-up between the three groups (including those abstinent), from the first follow-up to the second (F=0.50, df 2,65, ns). The average length of the second follow-up for the total sample was 112 days or approximately 16 weeks after the first follow-up. There was no significant difference between the groups in the

number of days from the first follow-up until relapse (F=0.48, df 2,48, ns). The number of drinking days during this follow-up period was not significantly different between the three groups (F=0.12, df 2,52, ns). In relation to the quantity of alcohol consumed during this follow-up period, no significant differences were noted between the groups on any of the variables measuring quantity (total units of alcohol, F=0.94, df 2,52, ns; number of units of alcohol consumed per day of follow-up, F=0.62, df 2,52, ns); number of units consumed on the heaviest drinking day, F=0.41, df 2,52, ns; number of units consumed on the lightest drinking day, F=0.02, df 2,51, ns).

Table 4.2.29 shows the number of alcohol related problems for the three post-detoxification diagnostic groups at outcome. The groups were not significantly differentiated from each other in the number of alcohol related problems at outcome using one way analysis of variance (F=0.11, df 2,71 ns). Analysis of variance, similarly, did not significantly differentiate the groups at outcome on the number of physical dependency problems (F=0.03, df 2,71, ns), nor in the number of alcohol related social problems (F=1.08, df 2,71, ns).

Summary of findings relating to hypothesis 3 on drinking at follow-up

Analysis of variance demonstrated no significant relationship between either pre-admission or post-detoxification diagnosis and consumption of alcohol at follow-up or to the number or type of alcohol related problems. Independent t-tests, comparing minor and no depression with major depression (pre-admission diagnosis) and minor and major depression with no

depression (post detoxification) corroborated these results.

Table 4.2.27 Drinking Outcome at First Follow-up for Post-detoxification Diagnostic Groups

	No Dep N=22 mean(sd)	Minor Dep N=8 mean(sd)	Major Dep N=5 mean(sd)	p
Number of days from discharge to 1st f-up: n=40/14/10!	47.2(19.2)	37.9(17.1)	41.5(25.9)	ns
Number of days from discharge to 1st drink:	23.1(29.0)	34.1(54.0)	18.4(37.5)	ns
Number of days drinking:	9.4(8.6)	7.3(6.8)	27.4(47.3)	ns
Total number of units alcohol drunk	143.5(179.1)	66.4(42.1)	160.2(115.9)	ns
Units alcohol per day follow-up	3.3(5.0)	1.8(1.3)	3.7(2.5)	ns
Number of units drunk on heaviest day	27.0(14.9)	20.1(17.4)	21.8(11.2)	ns
Number of units drunk on lightest day	8.0(8.3)	4.0(3.4)	8.0(6.2)	ns

No dep : No Depression, Alcoholism only.
Minor Dep: Minor Depression and Alcoholism
Major Dep: Major Depression and Alcoholism
f-up : follow-up
! : total number attending

Table 4.2.28 Drinking Outcome at Second Follow-up for Post-detoxification Diagnostic Groups

	No Dep N=36 mean(sd)	Minor Dep N=11 mean(sd)	Major Dep N=8 mean(sd)	p
Number of days from 1st f-up to 2nd f-up: n=44/14/10!	110.6(58.6)	123.4(57.6)	101.2(30.2)	ns
Number of days from 1st f-up to 1st drink: n=33/10/8!!	23.1(29.0)	34.1(54.0)	18.4(37.5)	ns
Number of days drinking:	51.0(48.1)	58.5(41.3)	50.0(43.0)	ns
Total number of units alcohol drank:	668.4(687.6)	1101.8(1527.2)	770.4(763.8)	ns
Units of alcohol per day follow-up	6.2(5.8)	9.1(11.8)	7.8(8.6)	ns
Number of units drank on heaviest day:	25.1(14.8)	28.2(17.3)	30.3(20.9)	ns
Number of units drank on lightest day: N=35/11/8!!	9.1(12.5)	8.9(4.7)	9.9(9.5)	ns

No dep : No Depression, Alcoholism only.
Minor Dep: Minor Depression and Alcoholism
Major Dep: Major Depression and Alcoholism
f-up : follow-up
! : total number attending
!! : number in group

Table 4.2.29 Alcohol Related Problems across Follow-up for Post-detoxification diagnostic Groups

	No Dep n=49 mean(sd)	Minor Dep n=14 mean(sd)	Major Dep n=11 mean(sd)	p
Total number of problems:	5.2(4.0)	5.8(3.9)	5.4(3.9)	ns
Number of social problems:	2.1(1.5)	2.7(1.8)	1.9(1.6)	ns
Number of physical dependency problems:	3.3(2.8)	3.2(2.3)	3.5(2.5)	ns

4.3 AFFECTIVE SYMPTOMATOLOGY AND COGNITIVE MEASURES: DIAGNOSIS

Table 4.3.1 shows the relationship between measures of affective symptomatology and cognitive measures at baseline (after detoxification). Pearson's correlations (two-tailed) test showed that measures of anxiety and depression all correlated significantly with each other and with cognitive measures. The association between self-control and dysfunctional attitudes, clinician rated depression and state anxiety gave lower correlations than did other measures, as did the relationship between clinician ratings of depression and self-ratings of dysfunctional attitudes.

Table 4.3.1 Correlation of affective symptomatology and cognitive measures at baseline

	STAIT	STAIS	BDI	MADRS	ATQ	DAS	CST	HS
STAIS	.71***							
BDI	.72***	.75***						
MADRS	.63***	.63***	.61***					
ATQ	.72***	.72***	.80***	.50***				
DAS	.53***	.45***	.48***	.27*	.53***			
CST	.61***	.45***	.53***	.37***	.57***	.54***		
HS	.65***	.66***	.72***	.63***	.69***	.47***	.60***	
SCS	-.52***	-.31**	-.42***	-.26*	-.47***	-.29*	-.66***	-.41***

STAIT: Spielberger Trait Anxiety Inventory
STAIS: Spielberger State Anxiety Inventory
BDI : Beck Depression Inventory
MADRS: Montgomery-Asberg Depression Rating Scale
ATQ : Automatic Thought Questionnaire
DAS : Dysfunctional Attitude Scale
CST : Cognitive Style Test
HS : Hopelessness Scale
SCS : Self-Control Schedule

*** p<0.001
** p<0.01
* p<0.05

4.3.1 Differences between the diagnostic groups on affective symptomatology and cognitive measures on each occasion across the study.

One way analysis of variance was carried out on each measure and on each occasion to determine whether the diagnostic groups were differentiated from each other on any occasion. Table 4.3.2 shows scores on MADRS for the

three groups and the results of one way analysis of variance. There were significant differences between the diagnostic groups on the first to the fourth occasion of testing. There were no differences between groups on their scores on MADRS at the final follow-up occasion. A Scheffe post-hoc test showed a significant difference between the non-depressed group and the major and minor depressed groups and between the minor and major depressed groups in the first week after detoxification. In the following two weeks there was a significant difference between major and non-depressed and between major and minor depressed groups. At first follow-up, there was a significant difference on MADRS between the group with major depression the group with no depression.

Table 4.3.3 shows the analysis of variance for the diagnostic groups which again revealed significant differences between the diagnostic groups from the first week after detoxification until the first follow-up on the BDI. There was no difference between the diagnostic groups at second follow-up. A Scheffe post-hoc test showed that the significant difference in scores was between the depressed (major and minor) groups and the non-depressed group at baseline, and between the major depressed group and both the minor depressed and non-depressed groups in the second week after detoxification. At the third week and at first follow-up, the difference was between the major and the non-depressed group.

Table 4.3.4 shows that there was a significant difference between the diagnostic groups on their scores on STAIS on the first three occasions, the three weeks following detoxification. At baseline, a Scheffe post-hoc test revealed that the major and minor depressed

groups were different from the non-depressed group. The non-depressed and the major depressed groups differed at weeks two and three, and the major depressed differed from the minor depressed group at week two.

Table 4.3.5 shows the results of one way analysis of variance on STAIT scores for the groups on each occasion. There was a significant differences between the groups on their scores on STAIT at baseline and at first follow-up. A Scheffe post hoc-test demonstrated that there was a difference between both the major and minor depressed groups' scores and those of the non-depressed group at week one. At first follow-up, the major depressed group was significantly different from the non-depressed group.

Legend: tables 4.3.2 to 4.3.10

Occ: Occasion
1 baseline
2 week 2
3 week 3
4 first follow-up
5 second follow-up

Gp : Diagnosis
1 No Depression
2 Minor Depression
3 Major Depression

Table 4.3.2 MADRS Scores on Each Occasion

Occ	Gp	n	Mean	(s.d)	F	p	Scheffe
1	1	52	6.33	(4.60)			
	2	19	14.89	(8.38)			
	3	11	23.27	(7.48)	41.69	<.001	3,2v1;3v2
2	1	37	7.32	(4.85)			
	2	16	10.25	(6.48)			
	3	11	23.73	(13.39)	21.36	<.001	3v1;3v2
3	1	36	5.42	(4.23)			
	2	16	10.06	(6.96)			
	3	10	19.20	(12.87)	15.76	<.001	3v1;3v2
4	1	39	6.36	(6.72)			
	2	14	10.00	(7.99)			
	3	9	15.22	(11.80)	4.97	=.01	3v1
5	1	43	8.47	(9.95)			
	2	15	12.60	(11.99)			
	3	10	14.00	(12.35)	1.55	n.s.	

Table 4.3.3 BDI Scores on Each Occasion.

Occ	Gp	n	Mean	(s.d)	F	p	Scheffe
1	1	52	16.12	(10.00)			
	2	18	25.22	(8.03)			
	3	11	31.27	(12.71)	13.33	<.001	3,2v1
2	1	37	12.62	(8.29)			
	2	15	18.00	(10.16)			
	3	11	28.73	(12.67)	12.12	<.001	3v1;3v2
3	1	35	9.49	(8.30)			
	2	16	15.06	(12.26)			
	3	10	24.30	(15.76)	7.51	<.01	3v1
4	1	39	8.67	(9.81)			
	2	14	16.36	(12.18)			
	3	9	20.56	(15.14)	5.39	<.01	3v1
5	1	44	10.36	(10.18)			
	2	15	16.33	(15.61)			
	3	8	16.25	(15.42)	1.79	n.s.	

Table 4.3.4 STAIS Scores on Each Occasion

Occ	Gp	n	Mean	(s.d)	F	p	Scheffe
1	1	52	45.04	(12.81)			
	2	18	61.67	(9.70)			
	3	11	65.18	(8.96)	22.05	<.001	3,2v1
2	1	37	45.49	(12.00)			
	2	16	51.56	(12.13)			
	3	10	65.10	(13.77)	10.13	<.001	3v1;3v2
3	1	36	41.53	(13.12)			
	2	16	49.50	(15.36)			
	3	10	59.10	(16.89)	6.38	<.01	3v1
4	1	39	39.49	(17.68)			
	2	14	49.21	(17.57)			
	3	9	47.11	(17.32)	1.89	n.s.	
5	1	44	42.48	(14.62)			
	2	15	48.27	(17.25)			
	3	8	46.13	(21.40)	0.79	n.s.	

Table 4.3.5 STAIT Scores on Each Occasion

Occ	Gp	n	Mean	(s.d)	F	p	Scheffe
1	1	51	46.00	(12.00)			
	2	18	55.39	(9.54)			
	3	11	61.82	(9.08)	11.55	<.001	3,2v1
4	1	37	41.68	(12.32)			
	2	14	49.21	(12.84)			
	3	9	54.89	(13.53)	4.80	<.05	3v1
5	1	44	42.20	(12.65)			
	2	14	47.29	(14.28)			
	3	8	48.88	(15.43)	1.36	n.s.	

Tables 4.3.6 to 4.3.10 show the scores for the diagnostic groups on cognitive measures across occasions. One way analysis of variance was used to test for differences on cognitive measures between the groups.

Table 4.3.6 shows that analysis of variance indicated significant differences between the diagnostic groups at baseline and at first follow-up on the ATQ. However, the differences between the groups was not sufficient to satisfy the Scheffe test criteria at first follow-up. There were no differences between the groups at second follow-up. A Scheffe post-hoc test showed a significant difference between the scores of the non-depressed group and those with major depression. Table 4.3.7 shows that there was a significant difference between the diagnostic groups on the DAS at first follow-up. A Scheffe post-hoc test shows that the difference in scores on the DAS was between the major depressed group and the non-depressed group.

Tables 4.3.8 and 4.3.9 show that there were no differences between the scores of the groups on the CST or SCS on any occasion. Table 4.3.10 shows that there were significant differences between diagnostic groups on their scores on HS at baseline and at first follow-up. There was no difference between the groups at second follow-up. A Scheffe post-hoc test shows that the difference was between the non-depressed group and those of the depressed (major and minor) groups at baseline and

between the major and non-depressed groups at first follow-up.

Table 4.3.6 ATQ Scores on Each Occasion.

Occ	Gp	n	Mean	(s.d)	F	p	Scheffe
1	1	51	73.33	(25.96)			
	2	18	89.28	(29.83)			
	3	11	115.18	(29.87)	11.24	<.001	3v1
4	1	38	57.84	(27.42)			
	2	14	69.00	(29.00)			
	3	9	85.22	(38.67)	3.34	<.05	-
5	1	43	61.33	(32.00)			
	2	15	67.47	(39.26)			
	3	7	73.71	(48.60)	.45	n.s.	

Table 4.3.7 DAS Scores on Each Occasion.

Occ	Gp	n	Mean	(s.d)	F	p	Scheffe
1	1	51	132.76	(33.56)			
	2	18	142.28	(37.93)			
	3	11	159.55	(38.31)	2.75	n.s.	
4	1	39	120.13	(35.18)			
	2	13	130.62	(51.28)			
	3	9	158.56	(27.97)	3.73	<.05	3v1
5	1	43	126.47	(42.93)			
	2	15	125.20	(54.08)			
	3	6	131.67	(23.10)	.05	n.s.	

Table 4.3.8 CST Scores on Each Occasion.

Occ	Gp	n	Mean	(s.d)	F	p	Scheffe
1	1	51	62.02	(11.76)			
	2	18	66.44	(12.71)			
	3	11	70.82	(12.89)	2.77	n.s.	
4	1	39	60.13	(14.15)			
	2	14	69.14	(15.91)			
	3	9	69.67	(12.64)	2.98	n.s.	
5	1	43	58.93	(13.07)			
	2	15	64.60	(16.07)			
	3	6	58.00	(7.13)	1.07	n.s.	

Table 4.3.9 SCS Scores on Each Occasion.

Occ	Gp	n	Mean	(s.d)	F	p	Scheffe
1	1	51	10.75	(30.52)			
	2	17	8.94	(30.09)			
	3	11	-13.18	(23.13)	2.92	n.s.	
4	1	39	19.49	(34.75)			
	2	14	9.29	(25.74)			
	3	9	-2.22	(34.44)	1.72	n.s.	
5	1	43	16.74	(33.33)			
	2	14	18.64	(37.82)			
	3	7	20.71	(30.84)	.05	n.s.	

Table 4.3.10 HS Scores on Each Occasion.

Occ	Gp	n	Mean	(s.d)	F	p	Scheffe
1	1	51	5.63	(5.16)			
	2	18	9.78	(4.72)			
	3	11	12.36	(4.84)	10.54	<.001	3,2v1
4	1	39	5.03	(4.93)			
	2	14	8.21	(6.41)			
	3	9	11.33	(7.16)	5.28	<.01	3v1
5	1	43	5.42	(5.59)			
	2	14	7.00	(6.97)			
	3	8	7.88	(8.84)	.70	n.s.	

4.3.2 Comparison of scores on cognitive measures between non-depressed alcoholics and normal controls

Table 4.3.11 shows scores on cognitive measures for normal control groups and those alcoholics who were not depressed after detoxification. Independent t-tests demonstrated that there were significant differences between normal control groups and those diagnosed as non-depressed alcoholics after detoxification. (ATQ: $t=5.82$, df 81, $p< 0.01$; DAS: $t=3.74$, df 81, $p< 0.01$; HS: $t=2.36$, df 445, $p< 0.05$; SCS: $t=4.67$, df 154, $p< 0.01$; CST: $t=1.59$, df 80, ns).

Table 4.3.12 shows scores on cognitive measures for normal control groups and those alcoholics who were not depressed in the pre-admission episode. Independent t-tests showed no significant differences between those alcoholics who were not depressed before admission and normal control groups on baseline measures of HS, CST and SCS (HS: $t=0.06$, df 406, ns; SCS: $t=0.77$, df 115, ns; CST: $t=0.40$, df 41, ns). However, significant differences were found between those alcoholics who were not depressed before admission and normal control groups on measures of ATQ and DAS (ATQ: $t=2.97$, df 42, $p< 0.01$; DAS: $t=3.46$, df 42, $p< 0.01$). Those alcoholics who were not depressed before admission have higher scores on the DAS and ATQ than normals.

Normal Control Groups in tables 4.3.11 and 4.3.12 from:
ATQ: Hollon et al., 1986
DAS: Hollon et al., 1986
HS : Greene, 1981
SCS: Rosenbaum, 1980
CST: Blackburn et al., 1986

Table 4.3.11 Comparison of Non-depressed patients* and Normal controls on Baseline Cognitive Measures

	Non-Depressed Alcoholics (*Post-Detoxification)			Normal Controls			
	n	mean	s.d	n	mean	s.d	p
ATQ	51	73.33	25.96	32	45.12	11.02	<.01
DAS	51	132.76	33.56	32	108.25	19.68	<.01
HS	51	5.63	5.16	396	4.45	3.09	<.05
CST	51	62.02	11.76	31	58.10	9.0	ns
SCS	51	10.75	30.52	105	31.3	23.2	<.01

Table 4.3.12 Comparison of Non-depressed patients* and Normal controls on Baseline Cognitive Measures

	Non-Depressed Alcoholics (*Pre-Admission)			Normal Controls			
	n	mean	s.d	n	mean	s.d	p
ATQ	12	58.33	17.79	32	45.12	11.02	<.01
DAS	12	133.5	26.02	32	108.25	19.68	<.01
HS	12	4.50	3.90	396	4.45	3.09	ns
CST	12	59.25	7.23	31	58.10	9.0	ns
SCS	12	25.83	23.10	105	31.3	23.2	ns

4.3.3 Comparison of depressed alcoholics and depressed groups from other studies on cognitive measures

Table 4.3.13 shows scores on cognitive measures for depressed groups and those alcoholics who were depressed after post-detoxification. Independent t-tests showed no significant differences between depressed alcoholics and depressed patients on baseline measures of DAS, HS and CST (DAS: t=1.58, df 25, ns; HS: t=0.4, df 97, ns; CST: t=0.02, df 29, ns). A significant difference was found between unipolar depressed patients and those alcoholics diagnosed as depressed after detoxification on the ATQ

(ATQ: t=2.93, df 25, p< 0.01). Those alcoholics who were depressed after detoxification have higher scores on the ATQ than unipolar depressed patients.

Table 4.3.13 Comparison of Depressed Alcoholics* and Depressed Groups on Baseline Cognitive Measures

	Depressed			Depressed Alcoholics (*Post-detoxification)			
	n	mean	s.d	n	mean	s.d	p
ATQ	16	85.00	23.62	11	115.18	29.97	<.01
DAS	16	140.88	23.08	11	159.55	38.31	ns
HS	88	13.01	5.17	11	12.36	4.84	ns
CST	20	70.90	10.80	11	70.82	12.89	ns

Depressed Groups from:
ATQ: Hollon et al., 1986
DAS: Hollon et al., 1986
HS : Nekanda-Trepka et al., 1983
CST: Blackburn et al., 1986.

Summary of findings presented above relating to hypothesis 4

Measures of affective symptomatology and cognitive meaures at baseline were significantly correlated with each other. There were significant differences on measures of affective symptomatology between the depressed and non-depressed alcoholics at baseline, and when measures had been taken throughout admission. The depressed and non-depressed groups continued to be differentiated on their scores of clinician and self-rated depression and on trait anxiety at first follow-up but not at second follow-up.

Depressed and non-depressed alcoholics were significantly differentiated on baseline measures of the frequency of negative thoughts and hopelessness but were not differentiated by their baseline scores of negative

cognitive style, dysfunctional attitudes or self-control. At first follow-up, those alcoholics who had been depressed following detoxification scored differently from those who were not depressed on measures of hopelessness, the frequency of negative thoughts and dysfunctional attitudes.

By comparing those alcoholics who were not depressed after detoxification with normal controls from other studies, it was found that non-depressed alcoholics scored significantly higher than normals on all cognitive measures, except the CST. Scores on cognitive measures for those alcoholics who had not met criteria for a depressive disorder, either pre-admission or after detoxification, were then compared with normal controls. It was found that alcoholics (without depression) scored significantly higher on measures of the frequency of negative thinking and dysfunctional attitudes than normals.

In order to explore the nature of depression in alcoholics further, scores on cognitive measures for those alcoholics who were depressed after detoxification were compared with depressed groups from other studies. It was found, on the whole, that scores of depressed alcoholics on cognitive measures were not different from those of other depressed groups. However, depressed alcoholics were found to have significantly higher scores on the ATQ, a measure of the frequency of negative thoughts.

4.3.4 Principal Components Analysis of baseline affective symptoms and cognitive measures

The purpose of carrying out a principal components analysis was to produce an empirical summary of scores on affective symptoms and cognitive variables. Factor scores were then used to provide estimates of the scores subjects would have received on each of the factors had they been measured directly. Table 4.3.14 shows the result of principal components analysis with Varimax rotation. The measures of affective symptoms and cognition at baseline produced two factors. Those measures loading higher than 0.50 are displayed in bold type face. Only one measure, STAIT, loaded highly on both factors. The two factors accounted for 72% of the variance of mood and cognition scores.

Table 4.3.14 Principal components analysis of baseline affective and cognitive measures (with Varimax rotation).

	Factor 1	Factor 2
BDI	**.82**	.32
MADRS	**.81**	.09
STAIT	**.70**	**.51**
STAIS	**.87**	.18
ATQ	**.73**	.45
HS	**.74**	.37
DAS	.42	**.57**
CST	.27	**.87**
SCS	-.13	**-.85**

Factor 1 eigenvalue 5.39, variance 59.9%
Factor 2 eigenvalue 1.09, variance 12.1%

Table 4.3.15 shows scores for the pre-admission diagnostic groups at baseline on the two factors. One way analysis of variance demonstrated that there was a significant difference between the groups on scores in the first factor: those with major depression scored higher on factor 1 than the non-depressed group. There

was no significant difference between the groups on factor 2.

Table 4.3.16 shows scores for the post-detoxification diagnostic groups at baseline on the two factors. One way analysis of variance demonstrated that there was a significant difference between the groups on scores in the first factor: those with major and minor depression scored higher on factor 1 than the non-depressed group. There was no significant difference between the groups on factor 2.

Table 4.3.15 Mean scores for factored aaffective symptoms and cognitive measures at baseline for each pre-admission diagnostic category

Groups:	Factor 1	Factor 2
No depression	-0.72 (0.68)	-0.28 (0.70)
Minor depression	-0.36 (1.00)	-0.22 (0.68)
Major depression	0.25 (0.97)	0.12 (1.11)
F, df	6.30, 2,75	1.15, 2,75
p	< 0.01	ns
Scheffe	3v1	-

Table 4.3.16 Mean scores for factored affective symptoms and cognitive measures at baseline for each post-detoxification diagnostic category

	Factor 1	Factor 2
No depression	-0.47 (0.74)	-0.03 (0.97)
Minor depression	0.58 (0.76)	-0.15 (1.13)
Major depression	1.26 (0.82)	0.36 (0.93)
F, df	30.35, 2,75	0.90, 2,75
p	< 0.001	ns
Scheffe	3,2v1	-

4.3.5 Relationship between affective symptomatology and cognitive measures at baseline and alcohol related variables

Table 4.3.17 shows Pearson's correlation (two-tailed) of affective measures at baseline with the pre-admission alcohol related variables. There was a significant relationship between the SADQ and measures of anxiety and depression. No association was found between affective measures at baseline and the number of years problem drinking, or in the number of units of alcohol consumed in the week before admission or in a typical week.

Table 4.3.18 shows Pearson's correlation (two-tailed test) of cognitive measures at baseline and alcohol related variables. A significant relationship was found between the SADQ, the ATQ and HS at baseline. The ATQ at baseline was also significantly associated with the number of units of alcohol consumed in the week preceding admission. No other significant associations were found between cognitive measures at baseline and alcohol related variables.

Table 4.3.17 Relationship between measures of affective symptomatology at baseline and drinking related variables before admission.

	SADQ	years problem drinking	units in week before admission	units in typical week
STAIT	.24*	-.11	-.00	-.08
STAIS	.24*	-.14	.11	-.03
BDI	.32**	-.04	.14	.01
MADRS	.23*	-.01	.09	-.01

SADQ:Severity of Alcohol Dependence Questionnaire
** p<0.01
* p<0.05

Table 4.3.18 Relationship between cognitive measures at baseline and drinking related variables before admission

	SADQ	years problem drinking	units in week before admission	units in typical week
ATQ	.32**	.00	.27*	.01
DAS	.14	.14	-.00	-.11
CST	.07	-.04	.01	-.07
HS	.25*	.08	.08	.06
SCS	-.16	.11	-.16	-.02

SADQ:Severity of Alcohol Dependence Questionnaire
** p<0.01
* p<0.05

Partial correlations demonstrated that the scores derived from BDI, ATQ and HS were equivalent in their association with SADQ. For example, when controlling for BDI, the correlations between SADQ, ATQ and HS were not significant (r=0.12 and r=0.04 respectively). When controlling for ATQ or HS, the correlation between SADQ and BDI was also not significant (r=0.10 and r=0.19 respectively).

4.3.6 Hopelessness and Suicidal Behaviour

Scores on the Hopelessness Scale at baseline were divided into high and low hopelessness, taking a cut off point of 13, the mean plus one standard deviation for this sample, and the average score for depressed patients (Nekanda-Trepka et al., 1983). Table 4.3.19 shows the relationship between hopelessness and past suicidal behaviour. A significant relationship was found between high hopelessness and past suicidal behaviour (chi-square=6.16, df 1, p=0.01).

Table 4.3.19 Hopelessness and history of suicidal behaviour

	No history n	Positive history n
low hopelessness	50	14
high hopelessness	8	10

Summary of findings presented above relating to hypothesis 4

Principal components analysis of baseline mood and cognitive measures produced two factors. Measures of affective symptomatology, ATQ and HS loaded highly on the first factor. DAS, CST, SCS and state anxiety loaded highly on the second factor. Significant differences were found between depressed and non-depressed alcoholics on the first factor, but not on the second factor.

Scores on the SADQ correlated significantly with measures of affective symptomatology and the ATQ and HS. The BDI, a measure of affective symptomatology, the ATQ and HS were equivalent in their association with SADQ. The ATQ also correlated significantly with the number of units of alcohol consumed in the week before admission. A significant relationship was found between high scores on the Hopelessness Scale and past suicidal behaviour.

4.4 MEASURES OF AFFECTIVE SYMPTOMATOLOGY AND COGNITIVE MEASURES: PREDICTION OF DRINKING AT FOLLOW-UP

This section concerns the relationship between affective symptoms and cognitive variables measured at baseline and drinking at follow-up. The relationship between drinking and abstinence and mean scores on the factors at baseline are shown in table 4.4.1. Independent t-tests showed that there was no significant relationship between scores on the two factors at baseline and drinking status. (Factor 1: t=0.89, df 59, ns; two-tailed test; Factor 2: t=0.61, df 59, ns, two-tailed test).

Table 4.4.2 shows the relationship between abstinence and drinking at second follow-up and mean scores on the factors at baseline. Independent t-tests demonstrated that there was no significant relationship between these variables at second follow-up. (Factor 1: t=0.28, df 63, ns, two-tailed test; Factor 2: t=0.48, df 63, ns, two-tailed test).

Table 4.4.1 Scores on factored affective measures and cognitive measures at baseline: relationship with drinking and abstinence at first follow-up

	Abstinent n=28 mean (sd)	Drinking n=33 mean (sd)
factors		
1	-0.15 (1.05)	0.08 (0.97)
2	0.13 (0.82)	-0.02 (1.05)

Table 4.4.2 Scores on factored measures of affect and cognition at baseline: relationship with drinking and abstinence at second follow-up.

	Abstinent n=13 mean (sd)	Drinking n=52 mean (sd)
factors		
1	-0.05 (1.19)	0.04 (1.03)
2	0.19 (0.73)	0.04 (1.02)

Table 4.4.3 shows the association between the number of units of alcohol per day of follow-up and scores on the factors at baseline. There was no significant correlation between these measures at either first or second follow-up (Pearson's correlation: 2-tailed test).

Table 4.4.3 Scores on factored measures of affect and cognition at baseline: Pearson correlation with amount of alcohol consumed (drinkers only) at outcome.

Units alcohol per day of follow-up	Factor 1	Factor 2
Follow-up 1	0.24	0.13
Follow-up 2	0.07	0.23

Summary of findings presented above relating to hypothesis 4

Scores on baseline measures of affective symptomatology and cognitive measures did not predict amount of alcohol consumed at follow-up, and were not related to either drinking or abstinence.

4.4.1 Self-control, antabuse and drinking at outcome

Scores on the SCS at baseline were divided at the median to give an estimate of high and low self-control. No significant difference was noted between the baseline measure of high and low self-control and abstinence from alcohol at first follow-up (chi-square=0.08, df 1, ns), or at second follow-up (chi-square=0.00 df 1, ns)(Table 4.4.4).

Table 4.4.5 shows the relationship between high and low scores on SCS and the prescription of antabuse at follow-up. No significant difference was found between baseline measures of low and high self-control and receiving antabuse at the first follow-up (chi-

square=0.00, df 1, ns), or at the second follow-up (chi-square=0.78, df 1, ns).

Table 4.4.4 Self-control schedule scores at baseline and abstinence at follow-up

	Abstinent n		Drinking n	
Follow-up	1st	2nd	1st	2nd
High Self-control	16	6	17	28
Low Self-control	13	7	18	27

Table 4.4.5 Self-control Schedule scores at baseline and prescription of antabuse at follow-up

	Prescription of Antabuse			
	1st follow-up n		2nd follow-up! n	
	yes	no	yes	no
High Self-control	16	24	8	31
Low Self-control	17	25	13	28

! unable to establish prescription in two cases

4.5 AFFECTIVE SYMPTOMATOLOGY AND COGNITIVE MEASURES: ASSOCIATION WITH DRINKING AT OUTCOME

Table 4.5.1 shows the association between measures of affective symptomatology and cognitive variables at first follow-up. Pearson's correlation (2-tailed test) demonstrated that all measures of affective symptoms and cognition were highly correlated at first follow-up.

Table 4.5.1 Correlation of affective symptomatology and cognitive measures with each other at first follow-up

	STAIS	STAIT	BDI	MADRS	ATQ	DAS	CST	HS
STAIT	.78***							
BDI	.82***	.86***						
MADRS	.69**	.72***	.78***					
ATQ	.80***	.85***	.89***	.79***				
DAS	.48***	.58***	.59***	.46***	.54***			
CST	.45***	.62***	.58***	.36**	.55***	.68***		
HS	.70***	.77***	.80***	.65***	.79***	.75***	.70***	
SCS	-.48***	-.56***	-.56***	-.35**	-.51***	-.55***	-.56***	-.65***

** p< 0.01 *** p< 0.001

Table 4.5.2 shows the association between affective symptomatology and cognitive measures at second follow-up. Pearson's correlation (2-tailed test) demonstrated that all measures of affective symptomatology and cognitive measures correlated highly and significantly at second follow-up.

Table 4.5.2 Correlation of affective symptomatology and cognitive measures with each other at second follow-up

	STAIS	STAIT	BDI	MADRS	ATQ	DAS	CST	HS
STAIT	.83***							
BDI	.80***	.84***						
MADRS	.72***	.65***	.71***					
ATQ	.84***	.86***	.92***	.75***				
DAS	.58***	.64***	.61***	.54***	.65***			
CST	.64***	.67***	.63***	.47***	.68***	.66***		
HS	.69***	.72***	.74***	.54***	.77***	.68***	.60***	
SCS	-.56***	-.69***	-.55***	-.52***	-.62***	-.64***	-.59***	-.57***

*** p<.001

Table 4.5.3 shows scores on affective symptomatology measures and cognitive measures for those who were abstinent and drinking at first follow-up. Independent t-tests were used to analyse all symptomatology and cognitive measures. At first follow-up, those who were abstinent scored significantly lower on all affective symptomatology measures when compared to those who were drinking, regardless of amount of alcohol consumed during that period of time. (BDI: t=2.31, df 60, p< 0.05; MADRS: t=2.23, df 60, p< 0.05; STAIS: t=2.67, df 60, p=0.01; STAIT: t=2.45, df 58, p< 0.05).

On cognitive measures, those who remained abstinent scored significantly lower than those who were drinking, on the ATQ (t=2.85, df 59, p< 0.01) and on the HS (t=2.43, df 60, p< 0.05). No significant differences were found between those who were drinking and those who were abstinent on the CST (t=1.14, df 60, ns), DAS (t=1.1,7 df 59, ns), and on SCS (t=1.83, df 60, ns).

Table 4.5.4 shows scores on measures of affective symptomatology and cognitive measures at second follow-up

for those who were abstinent and drinking. Independent t-tests found no significant differences between those who were abstinent and those who were drinking on any of the measures of affective symptomatology and cognition at second follow-up. (BDI: t=1.18, df 63, ns; MADRS: t=1.90, df 64, ns; STAIS: t=1.13, df 63, ns; STAIT: 0.54, df 62, ns; ATQ: t=1.75, df 61, ns; DAS: t=1.70, df 60, ns; CST: t=0.78, df 60, ns; HS: t=1.20, df 61, ns; SCS: t=1.05, df 60, ns).

Table 4.5.3 Measures of affective symptomatology and cognitive dysfunction at first follow-up and abstinence

	Abstinent N=27~ mean (sd)	Drinking n=35~ mean (sd)	p
BDI	8.3(12.0)	15.1(11.3)	<.05
MADRS	5.9(6.6)	10.5(9.1)	<.05
STAIS	36.2(15.2)	47.9(18.3)	=.01
STAIT N=26/34	40.8(13.8)	49.0(12.1)	<.05
ATQ N=27/34	52.6(29.4)	73.9(28.7)	<.01
DAS N=27/34	121.3(35.5)	133.4(42.9)	ns
CST	61.1(15.7)	65.4(14.0)	ns
HS	4.6(5.7)	8.2(5.9)	<.05
SCS	22.7(23.8)	7.4(38.1)	ns

~ for two cases, no measures of cognitive style or affective symptomatology were available

Table 4.5.4 Measures of affective symptomatology and cognitive dysfunction at second follow-up and abstinence

	Abstinent N=12 ~ mean (sd)	Drinking n=53 ~ mean (sd)	p
BDI	8.9(8.8)	13.6(13.0)	ns
MADRS N=12/54	5.2(4.7)	11.7(11.5)	ns
STAIS	40.3(13.0)	45.9(16.3)	ns
STAIT N=12/52	42.7(9.7)	45.0(14.0)	ns
ATQ N=12/51	49.3(24.8)	68.8(36.7)	ns
DAS N=11/51	107.8(44.1)	132.3(43.1)	ns
CST N=11/51	57.5(11.2)	61.0(14.1)	ns
HS N=12/51	4.3(6.8)	6.7(6.2)	ns
SCS N=12/50	24.3(14.6)	13.5(34.9)	ns

~ for three cases, no measures of cognitive style or affective symptomatology were available

Principal Components Analysis of affective symptomatology and cognitive measures at follow-up.

As with baseline data, principal components analysis were carried out on measures of affective symptomatology and cognitive dysfunction for each occasion of follow-up. Table 4.5.5 shows the result of a principal components analysis with Varimax rotation. The measures of symptomatology and cognition at first follow-up produced two factors. Those measures loading higher than 0.50 are displayed in bold type face. HS loaded highly on both factors. The two factors accounted for 81.3% of the variance of affective symptomatology and cognition scores.

Table 4.5.6 shows the result of principal components analysis of measures of symptomatology and cognition at

second follow-up. Only one factor was produced, accounting for 72.2% of the variance of measures.

Table 4.5.5 Principal components analysis (with Varimax rotation) of affective symptomatology and cognitive measures at first follow-up.

	Factor 1	Factor 2
BDI	.84	.44
MADRS	.88	.17
STAIT	.79	.47
STAIS	.85	.29
ATQ	.87	.37
HS	.63	.68
DAS	.30	.82
CST	.25	.85
SCS	-.27	-.76

Factor 1 eigenvalue 6.27, variance 69.7%
Factor 2 eigenvalue 1.04, variance 11.6%

Table 4.5.6 Principal components analysis of affective symptomatology and cognitive measures at second follow-up.

	Factor 1
BDI	.90
MADRS	.85
STAIT	.91
STAIS	.88
ATQ	.93
DAS	.79
HS	.83
CST	.81
SCS	-.73

Factor 1 eigenvalue 6.50, variance 72.2%

Table 4.5.7 shows the association between the factors on measures of affective symptomatology and cognitive dysfunction at baseline and first and second follow-up. There was a significant correlation between factor 1 at baseline and factor 1 at first follow-up. Factor 2 at baseline was significantly correlated with factor 2 at follow-up 1 and with the factor at follow-up 2. Factor 2 at follow-up 1 correlated significantly with the factor at the second follow-up.

Table 4.5.7 Relationship between factor scores at different time periods: Pearson correlation of factor scores (two-tailed test)

	Baseline		Follow-up 1	
	F1	F2	F1	F2
F1	0.60***	-0.13		
Follow-up 1				
F2	0.10	0.67***	0.00	
F1	0.22	0.40**	0.27	0.64***
Follow-up 2				

** p< 0.01
*** p< 0.001

Table 4.5.8 shows scores on factor 1 and factor 2 at first follow-up for those who were abstinent and drinking. Independent t-tests demonstrated that there was a significant difference between those who were drinking and abstinent on factor 1 (t=2.56, df 56, p< 0.02, two-tailed test). However, no differences were found between these groups on factor 2 (t=0.92, df 56, ns, two-tailed test).

Table 4.5.9 shows the association between units of alcohol consumed per day and scores on the factors at first follow-up. There was a significant correlation (Pearson's correlation, two-tailed test) between alcohol consumption and scores on factor 1.

Table 4.5.8 Scores on factored measures of affective symptomatology and cognition at first follow-up: relationship with drinking and abstinence at first follow-up

	Abstinent n=26 mean (sd)	Drinking n=32 mean (sd)	p
factors			
1	-0.36 (0.85)	0.29 (1.03)	<.02
2	-0.13 (0.75)	0.11 (1.17)	ns

Table 4.5.9 Scores on factored measures of affective symptomatology and cognitive style at first follow-up: Pearson correlation with amount of alcohol consumed (drinkers only)

Units alcohol per day	Factor 1	Factor 2
Follow-up 1	0.51**	0.10

** p<0.01

Table 4.5.10 shows mean scores on the factor of affective symptomatology and cognitive measures at second follow-up for those who were abstinent and drinking. At second follow-up, independent t-tests demonstrated that there was no significant difference between those drinking and those abstinent at this time on factor 1 (t=1.56, df 55, ns, two-tailed test). However, table 4.5.11 shows that there was a significant correlation between the number of units of alcohol consumed per day at second follow-up and scores on factor 1 at second follow-up.

Table 4.5.10 Scores on factored measures of affective symptomatology and cognitive style at second follow-up: relationship with drinking and abstinence at second follow-up

	Abstinent n=11 mean (sd)	Drinking n=46 mean (sd)	p
factor 1	-0.38 (0.56)	0.14 (1.05)	ns

Table 4.5.11 Scores on factored measures of affective symptomatology and cognition at second follow-up: correlation with amount of alcohol consumed at second follow-up (drinkers only)

	Factor score
Units alcohol per day of follow-up	0.46**

** p<0.01

Summary of findings presented above relating to hypothesis 4.

At each follow-up, affective symptomatology and cognitive measures were found to be highly and significantly correlated.

At first follow-up, those who were drinking had significantly higher scores on all measures of affective symptoms and on the cognitive measures ATQ and HS. Principal components analysis of measures of affective symptoms and cognitive measures corroborated these findings: scores on the first factor at follow-up 1 differentiated drinkers from those abstinent. However, no significant differences were found between those abstinent and drinking on the second factor on which the cognitive measures DAS, CST, HS and SCS loaded highly.

At second follow-up, no significant differences were found between those drinking and abstinent on individual symptom and cognitive measures. Principal components analysis produced only one factor and scores on this factor corroborated this finding: no significant differences were found between those drinking and abstinent at second follow-up.

For those drinking at follow-up, scores on the first factor at first follow-up and on the single factor at second follow-up were significantly associated with the amount of alcohol consumed during each follow-up.

4.5.1 Changes in affective symptomatology following cessation of drinking: Relationship to post-detoxification diagnosis

The following results are based on diagnosis after detoxification from alcohol. During the three weeks following admission, the patients were inpatients and assumed not to be drinking. Measures of affective

symptoms were taken weekly during this time. In order to explore the pattern of change on measures of depression, anxiety and cognitive measures for the diagnostic groups across time measures were analysed using analysis of variance with repeated measures. Only subjects who completed all three weekly rating scales are included in the analyses. The major question of interest is whether scores on depression and anxiety differ between the diagnostic groups across time. This can be explored by studying the pattern of scores over time by transforming the repeated measures over time into polynomial contrasts.

Table 4.5.12 shows scores on MADRS for the diagnostic groups during the three weeks inpatient stay. Multivariate tests of significance showed an interaction of diagnostic group and time for scores on the MADRS. The scores of the groups on this clinician-rating scale of depression therefore did not have the same pattern of change across time. Inspection of the means revealed that the non-depressed group changed little across the three time points, those with minor depression showed a decrease in mood between occasion 1 and occasion 2 and those with major depression remained more depressed than the other two groups throughout the three week period and only showed a decrease in depression between weeks 2 and 3.

Table 4.5.13 shows scores on the BDI across inpatient stay. There was no interaction of group and time on the BDI, indicating that the three diagnostic groups had the same pattern of change on scores of depression across the three weeks of inpatient stay.

Table 4.5.14 shows STAIS scores across the inpatient stay for the diagnostic groups. There was no interaction of group and time on the State Anxiety Inventory. The three diagnostic groups had the same pattern of change on scores of state anxiety across the three week period.

Table 4.5.12 MADRS Scores across Three Occasions

	No Dep	Minor	Major
	n=36	n=16	n=10
	mean(s.d)	mean(s.d)	mean(s.d)
Occasion 1	7.56 (4.38)	15.50 (8.97)	23.30 (7.89)
Occasion 2	7.28 (4.91)	10.25 (6.48)	25.00(13.40)
Occasion 3	5.42 (4.23)	10.06 (6.96)	19.20(12.87)

Effect: Diagnosis by Occasions. Wilk's Lambda=0.799, df 4,116, $p < 0.05$
Effect: Occasions. Wilk's Lambda=0.707, df 2,58, $p < 0.001$

Table 4.5.13 BDI Scores across Three Occasions

	No Dep	Minor	Major
	n=35	n=15	n=10
	mean(s.d)	mean(s.d)	mean(s.d)
Occasion 1	18.86 (9.54)	26.60 (8.09)	30.60 (13.20)
Occasion 2	12.14 (8.10)	18.00(10.16)	28.50 (13.33)
Occasion 3	9.49 (8.30)	15.47(12.58)	24.30 (15.76)

Effect: Diagnosis by Occasions. Wilk's Lambda=0.923, df 112,4, ns.
Effect: Occasions. Wilk's Lambda=0.590, df 2,56, $p < 0.001$

Table 4.5.14 STAIS Scores across Three Occasions

	No Dep	Minor	Major
	n=36	n=16	n=9
	mean (s.d)	mean (s.d)	mean (s.d)
Occasion 1	50.19 (11.82)	61.13(10.16)	65.33 (9.07)
Occasion 2	45.56 (12.16)	51.56(12.13)	67.11(12.96)
Occasion 3	41.53 (13.12)	49.50(15.36)	61.78(15.50)

Effect: Diagnosis by Occasions. Wilk's Lambda=0.897, df 4,114, ns.
Effect: Occasions. Wilk's Lambda=0.805, df 2,57, $p < 0.01$

4.5.2 Changes in Symptomatology and Cognition over Study

4.5.2.a *Measures of Depression and Anxiety*

Measures of depression and anxiety were taken throughout the time of the study, during the three weeks of inpatient treatment for alcohol problems, and on both follow-up occasions. Cognitive measures and a measure of trait anxiety (STAIT) were taken on three occasions, at the beginning of treatment for alcohol problems, after

detoxification had been completed, then on the two follow-up occasions. The relationship between post-detoxification diagnosis, affective symptoms and cognitve measures over time was explored using the same analysis as above, analysis of variance with repeated measures. The following tables show the results for the symptomatology measures across the time period of the study using a polynomial contrast weighted for the time interval between measures (ie. 1, 2, 3, 9, 25 weeks).

Table 4.5.15 shows scores on MADRS for the diagnostic groups across the time of the study. Multivariate tests of significance showed a significant interaction of group and time for scores on the MADRS. The groups therefore did not change in the same way across the time of the study. Those with major depression showed a steady decrease in scores across time whereas those with minor depression showed more fluctuation in scores across time. Table 4.5.16 shows scores on BDI for the diagnostic groups across time. There was no significant interaction of group and time for scores on the BDI. The groups therefore did not differ in their pattern of change over the 5 occasions. However, the scores showed a significant decrease over time. Table 4.5.17 shows scores on STAIS across time for the three groups. The scores of the groups changed in the same way across time. There was a significant main effect for time. The scores across time points fluctuated but generally decreased over time. Table 4.5.18 shows scores for the groups across time on the STAIT. Univariate ANOVA revealed a significant difference between the groups and a significant effect for time but no interaction between group and time.

Table 4.5.15 MADRS Scores across Five Occasions

	No Dep	Minor	Major
	n=24	n=11	n=8
	mean(s.d)	mean (s.d)	mean (s.d)
Occasion 1	7.83 (4.67)	15.73 (8.56)	20.50 (5.93)
Occasion 2	7.38 (5.40)	10.18 (6.40)	22.50(13.30)
Occasion 3	5.08 (4.65)	12.09 (7.27)	16.13(11.00)
Occasion 4	6.08 (7.22)	11.46 (8.37)	14.38(12.32)
Occasion 5	7.50 (8.70)	14.27(12.58)	11.38(10.58)

Effect: Diagnosis by Occasions. Wilk's Lambda=0.612, df 8,74, p< 0.05
Effect: Occasions. Wilk's Lambda=0.586, df 4,37, p< 0.001

Table 4.5.16 BDI Scores across Five Occasions

	No Dep	Minor	Major
	n=25	n=11	n=7
	mean (s.d)	mean (s.d)	mean (s.d)
Occasion 1	18.24 (9.43)	27.09 (9.22)	31.00(11.56)
Occasion 2	11.44 (8.22)	19.09(11.05)	25.57(11.90)
Occasion 3	8.12 (6.29)	17.09(13.32)	20.14(13.89)
Occasion 4	9.12(11.36)	16.36(13.40)	17.57(15.42)
Occasion 5	9.20(10.28)	15.73(14.89)	17.43(16.26)

Effect: Diagnosis by Occasions. Wilk's Lambda=0.903, df 8,74, ns.
Effect: Occasions. Wilk's Lambda=0.336, df 4,37, p< 0.001

Table 4.5.17 STAIS Scores across Five Occasions

	No Dep n=25 mean (s.d)	Minor n=11 mean (s.d)	Major n=6 mean (s.d)
Occasion 1	50.60 (10.95)	62.00(10.36)	67.67 (6.98)
Occasion 2	45.04 (11.87)	54.00(12.51)	69.50 (9.92)
Occasion 3	40.84 (10.40)	53.55(16.31)	62.00(14.56)
Occasion 4	41.28 (17.94)	45.82(17.61)	47.83(19.10)
Occasion 5	42.76 (13.45)	48.18(15.99)	48.67(24.36)

Effect: Diagnosis by Occasions. Wilk's Lambda=0.764, df 8,72, ns.
Effect: Occasions. Wilk's Lambda=0.522, df 4,36, $p < 0.001$

Table 4.5.18 STAIT Scores across Occasions 1,4 and 5

	No Dep n=33 mean (s.d)	Minor n=12 mean (s.d)	Major n=7 mean (s.d)
Occasion 1	45.76 (11.72)	57.75(10.81)	61.86 (9.39)
Occasion 4	39.67 (11.09)	50.08(13.58)	52.20(14.02)
Occasion 5	42.12 (13.23)	50.17(12.99)	49.43(16.58)

Diagnosis by Occasions: F=0.96, df 98,4, ns.
Occasions: F=10.91, df 98,2, $p < 0.001$

4.5.2.b Cognitive Measures

Table 4.5.19 shows scores on the DAS across time for the three diagnostic groups. There was no significant interaction of group and time on DAS scores, nor was there an effect for time, indicating no change in scores on the DAS.

Table 4.5.20 shows scores on the CST for the three diagnostic groups across time. The data met the assumptions of univariate analysis of variance with repeated measures. There was no interaction of group and time on the CST, nor was there an effect of time.

Table 4.5.21 shows scores across time for the diagnostic groups on HS. Multivariate tests of significance demonstrated that there was no interaction between group and time on the HS. Like the DAS and CST,

there was also no significant time effect, indicating no overall change in scores on the HS.

Table 4.5.22 shows scores on the ATQ for the groups. There was no significant interaction of group and time on scores on the ATQ. There was however a significant change in scores over time.

Table 4.5.23 shows scores on the SCS for the groups across time. The assumptions of univariate ANOVA were met and analysis revealed no significant interaction between group and occasion. There was however, a significant difference in scores across time.

Table 4.5.19 DAS Scores across Occasions 1, 4 and 5

	No Dep	Minor	Major
	n=34	n=12	n=5
	mean (s.d)	mean (s.d)	mean (s.d)
Occasion 1	128.24(30.40)	149.17(42.42)	154.60(32.42)
Occasion 4	119.32(33.20)	134.75(51.25)	141.00(19.38)
Occasion 5	122.59(39.35)	127.08(58.40)	128.80(24.60)

Effect: Diagnosis by Occasion. Wilk's Lambda=0.951, df 4,94, ns.
Effect: Occasion. Wilk's Lambda=0.899, df 2,47, ns.

Table 4.5.20 CST Scores across Occasions 1, 4 and 5

	No dep	Minor	Major
	n=34	n=13	n=5
	mean (s.d)	mean (s.d)	mean (s.d)
Occasion 1	62.09 (11.14)	68.92(11.91)	62.20 (9.42)
Occasion 4	60.50 (13.92)	69.46(16.51)	59.80 (4.82)
Occasion 5	59.06 (13.67)	66.00(16.86)	57.20 (7.66)

Diagnosis by Occasions: F=0.13, df 98,4, ns.
Occasions: F=1.46, df 2,98, ns.

Table 4.5.21 HS Scores across Occasions 1, 4 and 5

	No Dep n=34 mean (s.d)	Minor n=12 mean (s.d)	Major n=7 mean (s.d)
Occasion 1	5.27 (4.85)	10.25 (4.43)	10.57 (4.93)
Occasion 4	4.41 (4.57)	8.17 (6.70)	10.29 (7.72)
Occasion 5	5.00 (5.77)	7.83 (7.22)	8.71 (9.20)

Effect: Diagnosis by Occasion. Wilk's Lambda=0.947, df 4,98, ns.
Effect: Occasion. Wilk's Lambda=0.957, df 2,49, ns.

Table 4.5.22 ATQ Scores across Occasions 1, 4 and 5

	No Dep n=33 mean (s.d)	Minor n=13 mean (s.d)	Major n=7 mean (s.d)
Occasion 1	72.33 (26.09)	92.54 (28.92)	120.29(22.21)
Occasion 4	54.49 (25.38)	68.46 (30.11)	79.00(41.84)
Occasion 5	60.30 (31.34)	72.39 (39.98)	73.71(48.60)

Effect: Diagnosis by Occasions. Wilk's Lambda=0.914, df 4,98, ns.
Effect: Occasion. Wilk's Lambda=0.598, df 2,49, p< 0.001

Table 4.5.23 SCS Scores across Occasions 1, 4 and 5

	No Dep n=35 means (s.d)	Minor n=11 means (s.d)	Major n=6 means(s.d)
Occasion 1	13.09 (26.47)	0.91 (27.79)	-9.33(24.77)
Occasion 4	21.09 (34.72)	8.91 (27.91)	13.83(16.73)
Occasion 5	17.83 (33.96)	17.09 (30.42)	27.67(27.12)

Diagnosis by Occasions: F=2.44, df 4,98, ns
Occasions: F=9.89, df 2,98, p<.001

Summary of affective symptomatology and cognitive measures across the study.

During the time of admission, measures of self-rated depression and anxiety showed the same pattern of change: all diagnostic groups showed a decrease in scores over the three weeks. Diagnostic groups showed a different pattern of change on clinician-rated depression with the non-depressed group showing very little change and the depressed groups showing different rates of change across the three weeks.

There was a general decrease in scores on self-rated depression and self-rated anxiety across the duration of the study which was shown by all three diagnostic groups. Clinician rated depression showed a more complex pattern of change in that the diagnostic groups did not have the same pattern of change across time. Although groups showed a decrease in depression, those who had a diagnosis of major depression showed a greater decrease in depression over time than other diagnostic groups.

There was no significant change in dysfunctional attitudes over time, negative cognitive style or in hopelessness. However, there was an overall increase in measures of self-control and in the frequency of negative automatic thoughts for all groups.

4.6 RELATIONSHIP OF OTHER VARIABLES TO OUTCOME

4.6.1 Sex Differences

Sex differences in alcohol related characteristics were investigated using two-tailed independent t-tests. Several significant differences were found (table 4.6.1). Women were significantly older than men in the age at which problem drinking began ($t=3.73$, df 80, $P<.001$). Women had been problem drinking for significantly fewer years than men ($t=3.82$, df 80, $p< 0.01$). Women had consumed significantly fewer units of alcohol in the week before admission than men ($t=2.48$, df 80, $p< 0.05$), had been drinking for significantly fewer days than men in the week before admission ($t=2.33$, df 80, $p< 0.05$) and drank significantly less in a typical week before admission than did men ($t=2.63$, df 80, $p< 0.01$). There were no significant differences between the sexes on scores on the Severity of Alcohol Dependence Questionnaire ($t=1.21$, df 80, ns), on the Mini Mental State ($t=1.41$, df 80, ns), in the number of episodes of problem drinking in the past ($t=0.88$, df 80, ns), or in the number of alcohol related problems experienced during the current episode ($t=0.40$, df 80, ns).

Table 4.6.2 shows the relationship between sex and past psychiatric history. A chi-square analysis revealed no significant differences in past psychiatric disorders between the sexes. (Alcoholism: chi-square=0.02, df 1, ns; Depression: chi-square=3.67, df 1, ns; Aanxiety: chi-square=0.00, df 1, ns; Drug Disorder: Fisher's exact probability=0.40; eating Disorder: Fisher's exact probability= 0.11; Antisocial Personality Disorder: chi-square=0.00, df 1, ns; Suicidal Behaviour: chi-square=1.80, df 1, ns).

Table 4.6.1 Sex Differences in Alcohol Related Characteristics

	Male N=55 mean(sd)	Female N=27 mean (sd)	p
SADQ	28.9(12.9)	25.4(10.9)	ns
MMS	28.0(1.6)	28.6(1.6)	ns
Age problem drinking began	24.4(7.7)	31.4(8.5)	<.001
Years of problem drinking	13.3(8.2)	7.8(4.4)	<.01
Past episodes of problem drinking	2.5(2.9)	2.0(0.9)	ns
Number of alcohol problems	10.7(2.4)	10.4(3.0)	ns
Days drinking in pre-admission week	5.4(2.4)	4.07(2.6)	<.05
Units of alcohol in week before admission	139.0(106.1)	82.7(73.5)	<.05
Units of alcohol in typical week	172.0(100.5)	116.7(60.8)	.01

Table 4.6.2 The relationship between Sex and Past Psychiatric Diagnosis

	ALC	DEP	ANX	DRUG	EAT	ASP	SUIC
sex:							
male (n=55)	39	7	10	2	0	9!	13
female (n=27)	18	9	4	2	2	4	11

NB A patient can have more than one diagnosis
! 1 case missing
ALC :Alcoholism
DEP :Depression (unipolar and bipolar)
ANX :Anxiety, Obsessive Compulsive Disorder
DRUG:Drug Dependence
EAT :Anorexia Nervosa, Bulimia Nervosa
ASP :Antisocial Personality Disorder
SUIC:Suicidal behaviour

Table 4.6.3 shows the relationship between sex and primary diagnosis. Sex did not influence the presence of

a primary diagnosis of depression (chi-square=3.51, df 1, ns).

Table 4.6.3 The relationship between Sex and Primary Psychiatric Diagnosis

	primary diagnosis depression	other primary diagnosis
sex		
male (n=55)	3	52
female (n=27)	5	22

Table 4.6.4 shows the relationship between sex and drinking outcome. Two-tailed independent t-tests revealed no significant differences between the sexes in the total number of units of alcohol consumed during the first follow-up (t=1.05, df 62, ns) and during the second follow-up (t=1.34, df 66, ns). There was no difference between the sexes in the number of days drinking at first follow-up (t=0.91, df 62, ns) or at second follow-up (t=-1.12, df 66, ns). There was no significant difference between sexes in the number of units of alcohol consumed per day at first follow-up (t=0.99, df 62, ns) or second follow-up (t=1.52, df 66, ns). Nor was there any significant difference between the sexes in the number of days in the first follow-up (t=0.29, df 62, ns) or the second follow-up (t=0.53, df 66, ns). Again no significant differences were evident between men and women in the number of units consumed on the heaviest drinking day in the first follow-up (t=0.02, df 62, ns) or in the second follow-up (t=0.11, df 66, ns). Nor was there a significant difference between men and women in the number of units drank on the lightest day's drinking (t=0.43, df 62, ns) at first follow-up or at second follow-up (t=-0.14, df 64, ns).

Table 4.6.5 shows the relationship between sex and number of alcohol related problems at outcome. Women

experienced more alcohol related problems than men at outcome (t=2.25, df 72, p< 0.05).

Table 4.6.4 Relationship between drinking outcome and sex of patient

	Follow-up 1 mean (sd)		Follow-up 2 mean (sd)	
	male n=44	female n=20	male n=46	female n=22
total units of alcohol	81.5 (147.9)	45.1 (65.9)	524.8 (894.5)	827.6 (868.1)
units alcohol per day follow-up	2.0 (4.0)	1.0 (1.3)	4.7 (7.2)	7.6 (7.8)
number of days drinking	7.4 (17.8)	3.7 (5.9)	38.1 (45.4)	51.3 (46.3)
number of days to follow-up	44.8 (19.87)	43.2 (20.73)	109.4 (56.4)	117.0 (52.7)
units on heaviest drinking day	13.5 (17.5)	13.5 (14.8)	21.6 (18.6)	21.0 (16.6)
units on lightest drinking day	4.1 (7.1)	3.4 (4.6)	7.4 (11.3)!	7.8 (8.3)!

! 1 case missing

Table 4.6.5 Number of alcohol related problems at outcome

	Male N=49	Female N=25	p
number of alcohol related problems at outcome:mean(sd)	4.6(3.7)	6.8(4.2)	p<.05

Summary

Women in the sample had started problem drinking at a younger age than men, and had experienced fewer years of problem drinking. They also consumed less alcohol in the week before admission, and on fewer days, and less alcohol in a typical week in the three months prior to admission, than did men. Nonetheless, men and women did not differ in the number of past episodes of drinking or in the number of alcohol related problems experienced in

the index episode or in their scores on the Severity of Alcohol Dependence Questionnaire and on a scale of cognitive impairment. In addition, sex was not found to influence the presence of a primary diagnosis of depression in the sample.

No differences were found between the sexes in drinking outcome although women had experienced a greater number of alcohol related problems at follow-up.

4.6.2 Attenders and non-attenders at follow-up

Eight people did not attend any follow-up appointments. Table 4.6.6 shows general characteristics of attenders and non-attenders. There was no significant difference in the sex of patients who did not attend follow-up (chi-square=0.81, df 1, ns), nor was there a significant difference in age between attenders and non-attenders (t=0.60, df 80, ns). There was no significant differences between attenders and non-attenders in social class (chi-square=0.69, df 1 ns), in marital status (chi-square=0.81, df 1 ns), in education (Fishers Exact Probability=0.53, ns), in work status (Fishers Exact Probability=0.35, ns), or in housing (chi-square=0.00, df 1 ns).

Table 4.6.7 shows scores on MMS and alcohol related variables before admission for those attending and not attending follow-up appointments. Significant differences between those who attended follow-up and those who did not were evident on the MMS (t=4.15, df 80, $p< 0.001$) and in the number of past episodes of problem drinking (t=3.59, df 79, p=0.001). No significant differences were noted between those attending and not attending for follow-up on the SADQ (t=0.59, df 80, ns), in the age problem drinking began (t=0.26, df 80, ns), in

the number of years problem drinking (t=0.04, df 80, ns), in the number of alcohol related problems reported at admission (t=1.45, df 80, ns), in the number of units of alcohol consumed in the week preceding admission (t=0.14, df 80, ns), in the number of days drinking in the pre-admission week (t=0.01, df 80, ns), nor in the number of units of alcohol consumed in a typical week before admission (t=0.66, df 80, ns).

Table 4.6.6 Comparison of attenders and non-attenders at follow-up

	Attenders n=74 n	Non-attenders n=8 n	p
Sex; Male/Female	48/26	7/1	ns
Age; mean(sd)	40.9(10.3)	43.3(11.8)	ns
Social Class; I and II	25	1	
III,IV and V	49	7	ns
Marital Status; married/cohabiting	26	1	
single,widowed, separated/divorced	48	7	ns
Education; degree/diploma, SED highers	29	2	
10 years school, truant	45	6	ns
Work Status; full employment, unemployed: 1-6 months	41	4	
unemployed: 6 months to 5 years or never worked	33	4	ns
Housing; owner-occupier or rented	49	5	
tied, lodging house or with parent/partner	25	3	ns

Table 4.6.7 Comparison of attenders and non-attenders at follow-up on alcohol related variables

	Attenders N=74 mean(sd)	Non-attenders N=8 mean(sd)	p
SADQ	28.0(12.5)	25.3(10.3)	ns
MMS	28.4(1.4)	26.1(2.0)	<.001
age problem drinking began	26.8(8.9)	26.0(5.3)	ns
years problem drinking	11.5(7.7)	11.4(7.2)	ns
episodes of problem drinking N=73/8	2.0(1.1)	5.0(6.6)	=.001
number of alcohol related problems	10.5(2.6)	11.9(2.4)	ns
units alcohol in pre-admission week	119.9(101.9)	125.1(82.1)	ns
days drinking in pre-admission week	5.0(2.6)	5.0(2.6)	ns
units alcohol in typical week before admission	151.5(93.5)	174.5(88.0)	ns

Table 4.6.8 shows the relationship between attenders and non-attenders at follow-up and a diagnosis of depression both pre-admission and post-detoxification. For pre-admission diagnosis, there was no significant difference between those with minor and no depression and those with major depression in attendance at follow-up (chi-square=0.00, df 1, ns)

Likewise, for post-detoxification diagnosis, there was no significant difference at follow-up between those with no depression and those with minor and major depression combined (chi-square=0.00, df 1, ns).

Table 4.6.8 Comparison of pre-admission and post-detoxification diagnosis of attenders and non-attenders

	Attenders n=74	Non-attenders n=8	p
Pre-admission:			
minor and no depression	24	3	
major depression	50	5	ns
Post-detoxification:			
minor and major depression	27	3	
no depression	47	5	ns

Summary of Differences between attenders and non-attenders

There were two significant differences between those who attended and did not attend for follow-up: those who did not attend had lower scores on the MMS indicating greater cognitive impairment and had experienced a greater number of past episodes of problem drinking.

4.6.3 Length of follow-up

Table 4.6.9 shows the mean number of days in the follow-up periods for those drinking and abstinent. There was no significant difference in the length of follow-up between those who were abstinent and those drinking at follow-up. Table 4.6.10 describes the association between measures of alcohol consumption at follow-up and length of follow-up. There were no significant correlations between measures of alcohol consumption and length of follow-up.

Summary

Length of follow-up was not influenced by drinking status, nor was the amount consumed at follow-up associated with the length of follow-up.

Table 4.6.9 Number of days in follow-up periods for those abstinent and drinking (two-tailed t-tests)

	Abstinent mean (sd)	Drinking mean (sd)	t	p
Follow-up 1 n=29/35	40.86(17.75)	47.11(21.51)	1.25	ns
Follow-up 2 n=13/55	98.62(43.39)	115.02(57.24)	0.97	ns

Table 4.6.10 Pearson correlations between measures of alcohol consumption and the number of days in the follow-up period.

Measures of alcohol consumption	Number of days	
	Follow-up 1	Follow-up 2
Total units	0.09	0.23
Units per day	-0.18	-0.08

4.6.4 Severity of Alcohol Dependence

Table 4.6.11 shows the association between scores on the Severity of Alcohol Dependence Questionnaire and drinking at outcome. A Pearson's correlation (two-tailed test) demonstrated that there was no significant correlation between SADQ and measures of drinking at outcome. Table 4.6.12 describes the differences between those abstinent and drinking at follow-up in scores on the Severity of Alcohol Dependence Questionnaire. There was no difference in scores on the SADQ.

Table 4.6.11 Pearson correlations between severity of alcohol dependence (SADQ) and drinking at outcome

	Severity of alcohol dependence	
Measures of drinking at outcome	Follow-up 1	Follow-up 2
Total units	0.14	0.09
Units per day of follow-up	0.15	0.09

Table 4.6.12 Differences between those abstinent and drinking at follow-up on the SADQ (two-tailed t-tests)

	Abstinent mean (sd)	Drinking mean (sd)	t	p
Follow-up 1 n=29/35	27.83 (12.86)	29.60 (12.65)	0.55	ns
Follow-up 2 n=13/55	22.54 (11.51)	29.60 (12.91)	1.81	ns

Summary

Severity of dependence on alcohol, measured at admission to treatment, was not found to be related to drinking at outcome.

4.6.5 Influence of lifetime diagnosis of depression on outcome.

The influence of a lifetime diagnosis of depression on drinking outcome was considered. Independent t-tests, comparing those with a lifetime diagnosis of depression with alcoholics without such a lifetime diagnosis revealed no differences in drinking outcome at either first (table 4.6.13) or second follow-up (table 4.6.14). At first follow-up, one subject with a lifetime diagnosis of depression had not attended for first follow-up until the 112th day following discharge. As a result, the number of days on which he had been drinking far outnumbered those of the others in this category as he had more opportunity to consume alcohol. Consequently, data specifically pertaining to the number of days drinking at follow-up for this subject was removed from the analysis.

There was no significant difference in rate of abstinence at first follow-up (chi-square=0.13, df 1, ns) or at second follow-up (chi-square=1.01, df 1, ns) in those with and without a lifetime diagnosis of depression (table 4.6.15).

Table 4.6.13 Comparison of drinking outcome at first follow-up for those with and without a lifetime diagnosis of depression.

	No lifetime Depression mean (sd) n=52	Lifetime Depression mean (sd) n=12	t	p
Number of days drinking N=52/11!	4.4 (6.5)	5.3 (9.4)	0.35	ns
Total units alcohol drunk	67.2 (124.9)	82.8 (148.7)	0.37	ns
Units alcohol per day follow-up	1.7 (3.6)	1.5 (2.7)	0.21	ns
Number of units drunk on heaviest day	14.6 (17.3)	8.7 (12.6)	1.13	ns
Number of units drunk on lightest day	4.4 (6.9)	1.6 (2.6)	1.38	ns

! one subject (outlier) removed from analysis

Table 4.6.14 Comparison of drinking outcome at second follow-up for those with and without a lifetime diagnosis of depression.

	No lifetime Depression mean (sd) n=54	Lifetime Depression mean (sd) n=14	t	p
Number of days drinking	42.8 (47.4)	40.5 (40.1)	0.87	ns
Total units alcohol drunk	572.9 (695.5)	814.9(1400.1)	0.92	ns
Units alcohol per day follow-up	5.4 (6.6)	6.6 (10.4)	0.50	ns
Number of units drunk on heaviest day	22.1 (17.4)	18.8 (20.0)	0.61	ns
Number of units drunk on lightest day	8.0 (11.3)	5.6 (5.8)	0.75	ns

Table 4.6.15 Relationship between lifetime diagnosis of depression and abstinence at follow-up

	First follow-up		Second follow-up	
	Abstinent n=29	Drinking n=35	Abstinent n=13	Drinking n=55
No lifetime Depression	23	29	9	45
Lifetime Depression	6	6	4	10

Summary

There was no relationship between lifetime diagnosis of depression and drinking outcome at follow-up.

CHAPTER FIVE

DISCUSSION

5.0 INTRODUCTION

The main hypothesis of the present study is that alcoholism is not a unitary disorder. One dimension on which alcoholics vary is in co-existing psychopathology and the present study focuses specifically on depression in alcoholics.

5.1 SAMPLE CHARACTERISTICS AND DESIGN.

The characteristics of the sample in the present study are reasonably representative of heavy and problem drinkers found in the general population, in terms of sex distribution (Dight, 1976; Wilson, 1980), and in clinical samples of alcoholics (Scottish Health Statistics, 1988) where up to one-third are women. The sample has a number of characteristics common to alcoholics in general (Edwards, 1982): relatively high rates of unmarried, divorced and separated individuals, unemployment, and alcohol related problems. The average age of the sample is illustrative of clinical populations where chronic alcohol problems occur in the middle years of life.

Although the original aim was to assess patients at one and four months after discharge, the mean lengths of follow-up obtained were six weeks and 5.5 months due to patients failing to attend at the appointed original times. This length of follow-up is at variance with most other studies which have followed-up patients between one and two years after discharge. In the main, other studies have been concerned with longer-term outcome of alcoholism, such as recurrence and remission from alcoholism and alcohol related problems. The intention of the short length of follow-up in the present study is

two-fold: to increase the likelihood of closely monitoring the association between affective symptomatology, cognitive measures and drinking behaviour, and to obtain detailed reports of drinking during the follow-up period.

In the present study, those who attended follow-up had fewer past episodes of drinking and were less intellectually impaired, as measured by the Mini-Mental State, than those who did not attend for follow-up. Those eight who did not attend for either follow-up may represent a sub-group who experienced more alcohol-related impairment.

All patients in the present study were in-patients in an abstinence-oriented treatment programme. The low rate of abstinence obtained at both first and second follow-up may reflect an unstable pattern of drinking and therefore may not be indicative of longer term drinking outcome. During admission or at follow-up, individuals may change their drinking goal from one of abstaining from alcohol to moderation of alcohol consumption. The criteria used in the evaluation of drinking outcome in the present study are precise and are intended to reflect accurately the extent of drinking and not necessarily problematic drinking.

Drinking outcome at follow-up has been reported by other investigators to be predicted by the prior degree of dependency on alcohol (Hasin et al., 1988; Polich et al., 1980). The present study does not find any such relationship.

5.2 DIAGNOSIS OF DEPRESSION

The present study differs from other studies which have examined drinking outcome for depressed alcoholics in that diagnosis of depression in the current episode is the principal focus, rather than lifetime diagnosis of depression.

As predicted, a high prevalence of major (67%) and minor depression (18%) was found for the episode leading to admission. Hasin et al (1988), using the Schedule for Affective Disorders and Schizophrenia (SADS)(Lifetime version), found that 68% of male and female alcoholics met Research Diagnostic Criteria (RDC) for lifetime major depressive disorder after detoxification, a strikingly similar proportion to that found in the pre-admissison episode in the present study. Hesselbrock et al (1985), using DSM-III computer diagnosis, obtained from the Diagnostic Interview Schedule (DIS), found that 23% of male and female alcoholics had a current diagnosis of major depression. However, in the latter study, it is not clear at which stage of in-patient treatment this diagnosis was made. Differences in prevalence of depression found could partly be due to the timing of the assessment in relation to detoxification, and to the diagnostic instrument used. In the present study, depression was assessed using the SADS and RDC. Post-detoxification diagnosis was distinguished from diagnosis in the episode leading to admission. Although current episode of depression is the primary focus in this study, lifetime diagnosis can be based on current as well as past episodes which accounts for the similar rates of depression found in the present study and that of Hasin et al (1988). This lack of differentiation between

current and past episodes is confusing, especially when diagnosis is used as a prognostic indicator.

Although both the DIS and SADS-L are commonly used diagnostic procedures in psychiatric research, they are known to show poor agreement in the assessment of affective disorder in patients with substance abuse: the SADS-L produces a considerably higher number of cases of major depression then the DIS (Hasin and Grant, 1987). The principle factor accounting for the difference in rate of detection of major depression in substance abuse populations was found to be within the structure of the DIS. This interview is designed to be used by non-clinicians, and contains questions designed to uncover drug and alcohol-induced symptoms. For example, if a symptom, such as depressed mood, was regarded by a subject to be caused by drinking alcohol or through taking medication, then the symptom is not included in generating a computer diagnosis. When this discrepancy was taken into account, Hasin and Grant (1987) found that the agreement between the SADS-L and DIS substantially improved for major depression in alcohol and drug dependent patients. Past physician-assessed diagnosis of clinical depression, evinced by treatment for depression and hospitalization for depression, was significantly and more strongly associated with diagnosis obtained using the SADS-L than with the DIS, indicating that the SADS-L was a more valid diagnostic instrument for the detection of major depression than the DIS in a sample of substance abusers.

In the present study, major and minor depression in the index episode appear to be labile diagnoses. As predicted, those patients with a diagnosis of major

depression in the episode leading to admission did not have a diagnosis of major or minor depression after admission to hospital and detoxification from alcohol. Brown and Schuckit (1988), assessing depressive symptomatology on the Hamilton Rating Scale for Depression, obtained a similar result with male alcoholics with no previous lifetime psychiatric diagnosis. However, it is also possible that the process of hospitalisation itself leads to a decrease in the severity of depression as individuals are removed from the impact of the problems they may have been experiencing, into an environment which is more sheltered from the outside world, predictable, and in which there is an expectation of change for the better.

The findings of the present study and those of others (Dorus et al., 1987; Overall et al., 1985; Brown and Schuckit, 1988) indicate that alcohol induced depression is a major contributor to the prevalence of depression found in alcoholics. At least two possible factors account for depression in alcoholism: familial and alcohol related factors.

5.2.1 Factors influencing a diagnosis of depression

Further indication of the lability of depression in alcoholism comes from the scarce differences found in demographic variables, drinking history, pre-admission alcohol consumption, personal psychiatric history and family history of depression between the diagnostic groups in the present study. As predicted, those with a diagnosis of depression do not differ from those without a diagnosis of depression, in terms of their family history of depression. Also, a primary lifetime diagnosis of depression is not associated with either

current episode diagnosis of depression or with a family history of depression in the present study. These results indicate that familial factors contributed little to diagnosis of depression in the current episode in this sample. These results do not support the idea that familial factors are important (Winokur et al., 1975) in the manifestation of depression in alcoholism.

On the whole, past psychiatric history is not significantly related to diagnosis of depression, either for the current episode or after detoxification from alcohol. However, having a diagnosis of anxiety disorder is related to a diagnosis of depression post-detoxification. There is a recognised association between depression and anxiety: the course of anxiety states has been found to be complicated by secondary depression over periods of up to nine years (Clancy et al., 1978) and long term outcome for patients with anxiety disorders has been found to be poor, with at least one third of patients suffering from recurrent or chronic illness (Murphy et al., 1986). One explanation for the association between post-detoxification depression and a past diagnosis of anxiety disorder found in this study is that those with a diagnosis of an anxiety disorder in the past may be particularly likely to experience depression post-detoxification, due to having developed depression secondary to both alcoholism and anxiety disorder.

The lack of difference between the diagnostic groups on pre-admission alcohol consumption or in past drinking history suggests that a diagnosis of depression does not influence, nor is it reflected in the intensity of drinking, at least after heavy drinking has become

established. In the present study, the diagnostic groups did not differ in the severity of alcohol dependence in the index episode. Hasin et al (1988) found that a lifetime diagnosis of major depression was associated with higher scores on an alcohol dependence scale. Although, this difference in findings may be attributable to the use of current as opposed to lifetime diagnosis of depression in the present study, O'Sullivan et al (1983) using Feighner's criteria (Feighner et al., 1972) for lifetime diagnosis of depression also found no relationship between depression and indicators of severity of alcohol dependence.

5.3 OUTCOME FOR DIAGNOSTIC GROUPS

One prediction at the outset of the present study was that alcoholics with co-existing depression would differ in drinking outcome at follow-up from those alcoholics with no depression. This prediction was not confirmed. Those alcoholics with minor or major depression post-detoxification do not differ on drinking outcome measures from those with alcoholism alone. Nor does the finding change when diagnosis in the pre-admission episode is used to examine drinking outcome.

Other follow-up studies on drinking outcome of depressed and non-depressed alcoholics have reported mixed results. Rounsaville et al. (1987) found that a lifetime (including current) diagnosis of depression was related to significantly poorer drinking outcome in men, but better drinking outcome for women, when compared to alcoholics who had no additional psychopathology. In contrast, O'Sullivan et al (1988) found no significant difference in abstinence rates or in the total number of days drinking between male alcoholics with or without a lifetime diagnosis of unipolar depression or bipolar depression over a 24 month follow-up period. The unipolar depressed alcoholics had however, received significantly more treatment for drinking by one and two years follow-up compared to the other groups and in addition, reported more depression at follow-up than those with no depression. Nonetheless, the unipolar depressed group did not differ significantly in terms of drinking outcome from those alcoholics who were not depressed.

In the present study, those who were depressed after detoxification are more likely to have received treatment

for depression, during admission and during follow-up, than those without depression. However, despite this additional treatment, no differences are found in drinking outcome between the groups. In addition, the fourteen subjects in the present study with a lifetime diagnosis of depression do not differ in drinking outcome from those who have not experienced depression in their lifetimes. The present study corroborates the findings of O'Sullivan et al (1988).

The main difference in findings between Rounsaville et al's (1987) study and the present study is that the former study found drinking outcome for alcoholics with depression to vary according to sex. As the findings in the present study differ from those in Rounsaville et al's study (1987), the similarities and differences between the studies warrant further comment. The samples in both studies included randomly selected men and women, unlike other studies which have examined the effect of diagnosis of depression on outcome of alcoholism in men only (O'Sullivan et al. 1988; Penick et al., 1984). In addition, both the present study and that of Rounsaville et al (1987) used similar multiple outcome measures of drinking and alcohol related problems.

However, there are several important differences between the studies. Rounsaville et al (1987) followed-up 266 subjects from the original group of 321 alcoholic in-patients (Hesselbrock et al., 1985) whereas the present study follows-up 74 out of 82 alcoholic in-patients. Although the follow-up rates do not differ widely, the number of subjects in the present study is approximately a quarter of the size of Rounsaville et al's (1987) sample. The sample in this study was

obtained from one National Health Service treatment facility whilst Rounsaville et al (1987) obtained their sample from three separate treatment facilities, with only one state-funded, in Connecticut.

The differences in findings between the present study and that of Rounsaville et al (1987) may reflect differences in the psychiatric morbidity of their original sample (Hesselbrock et al., 1985) and the diagnostic instruments used in both studies. As described above, the criteria for depression depend on the instrument used to detect depression. Consequently, the nature of depression detected in the two samples may be considerably different.

An additional difference between the present study and Rounsaville et al (1987) is the use of lifetime diagnosis, including current, of depression in the latter. The present study indicates that there is no influence of lifetime diagnosis on drinking outcome, when lifetime diagnosis is distinguished from current depression. The effect of having an additional current diagnosis of minor or major depression is the main focus in the present study as current psychopathology is more likely to influence short-term behaviour, emotion and thinking than an episode of illness which may have arisen in the more distant past.

In addition, primary diagnosis is not considered as an independent variable in the assessment of outcome of drinking in the present study as only eight patients had experienced depression at an earlier age than alcoholism. Those with a primary diagnosis of depression are not more likely than those without a primary diagnosis of depression to reach diagnostic criteria for depression in

the episode before admission or after detoxification. Nor are those with a primary diagnosis of depression more likely to have a positive family history of depression when compared with those without depression as a primary diagnosis. Secondly, some of those individuals with a primary diagnosis of alcoholism had also experienced an episode of depression (5 patients), independently of alcoholism, since they were originally diagnosed as alcoholic. Those with alcoholism as a primary diagnosis represented a heterogeneous group, not only in terms of having later episodes of depression, but also, in reaching diagnostic criteria for other psychiatric diagnosis during their lifetimes.

From the findings of the present study, it would appear that there is strong evidence that a dual diagnosis of depression and alcoholism occurring in the index episode, or after detoxification, does not indicate worse outcome for drinking problems, when compared to alcoholics without co-existing depression. The only follow-up study (Rounsaville et al., 1987) which found that depression influenced drinking outcome found that men, with a lifetime diagnosis of depression and alcoholism, and men with other psychiatric diagnoses, had a poorer outcome compared to men with alcoholism alone. Other broadly similar studies have not confirmed this finding for male alcoholics (O'Sullivan et al., 1988; Penick et al., 1984).

There are several implications arising from the findings of the present study. It is possible that a diagnosis of depression does not have utility in predicting drinking behaviour, once dependence on alcohol is established. Other implications concern the nature of

depression in those who are dependent on alcohol. It is possible that depression, both as a diagnostic category and as a dimensional phenomenon, is transitory in alcoholics once treatment for alcoholism has begun and, by implication, short term abstinence has been achieved.

5.4 RELATIONSHIP BETWEEN AFFECTIVE SYMPTOMATOLOGY AND COGNITIVE VARIABLES

5.4.1 Depressed and Non-depressed Alcoholics

Another question arising from the hypothesis of this study is whether or not depressed alcoholics differ from non-depressed alcoholics on measures of affective symptomatology and cognitive dysfunction. Beck (1967) found that depressed patients reported both depressed mood and cognitive dysfunction, manifested in the pervasive negative content of thought. Beck (1987) has recently stated that negative thinking should be considered as a diagnostic symptom of depression, as thinking of this type is universally found in depressives. Several measures of negative thinking have been developed and scores on these measures have been found to differentiate between depressed and non-depressed subjects, irrespective of the sub-type of depression.

The relationship between these measures of negative thinking and affective symptoms has not been explored in depressed and non-depressed alcoholics. The findings of the present study suggest that depressed alcoholics do not differ from non-depressed alcoholics on baseline measures of depressogenic cognitive style such as dysfunctional attitudes and negative cognitive style. They do differ, however, from non-depressed alcoholics on measures of depression and anxiety and in level of hopelessness and the frequency of negative thinking. As found in studies of depressed populations, scores on measures of affective symptomatology and cognition are significantly correlated with each other in this study, although a weaker association is found between

self-control, state mood measures and dysfunctional attitudes.

The findings of the present study are that depressed alcoholics have higher scores on measures of depression and anxiety than non-depressed alcoholics at admission, but depression and anxiety symptoms improve rapidly with the onset of abstinence.

Self-rated depression and state anxiety and clinician-rated depression discriminated between depressed and non-depressed groups of alcoholics over the duration of admission. The depression scales and a measure of trait anxiety continued to discriminate between major and non-depressed groups at first follow-up.

Although depressed and non-depressed patients were found to differ in the severity of depression and anxiety, there was nonetheless a decrease in scores for all groups across the duration of the study. Scores on each measure indicated less depression and anxiety with time in each diagnostic group. Self-report measures of depression and anxiety decreased in the same way for all groups across admission, whilst clinician-rated depression showed a different pattern of change. Scores on clinician-rated depression were found to decrease between the second and third weeks for the depressed group of alcoholics. The pattern of change on measures of anxiety and depression across the duration of the study showed a similar finding to that of change across admission. Scores on the clinician rating scale were based on the prior seven days and may thus have been sensitive to a wider range of depressive symptomatology than the Beck Depression Inventory, a self-rated

depression scale which assesses depressive symptomatology in a more restricted time period, namely the present.

Of the cognitive measures, scores on the Automatic Thought Questionnaire and Hopelessness Scale discriminated between depressed and non-depressed alcoholics and, as such, reflect the results obtained with measures of depression and anxiety. However, the diagnostic groups did not differ in their scores on the Cognitive Style Test, Dysfunctional Attitude Scale and on the Self-Control Schedule at admission. Although scores on all measures of affective symptomatology and cognition were highly correlated with each other, these three measures of cognitive style did not differentiate between the depressed and non-depressed alcoholics on occasions when measures of depression and anxiety were found to differentiate these groups. Either the depressed group was not scoring highly on these measures or the non-depressed group was showing signs of dysfunctional thinking. The evidence shows that the non-depressed group was scoring higher on all cognitive measures, except on the Cognitive Style Test. These findings indicate that high scores on the Dysfunctional Attitude Scale, Automatic Thought Questionnaire and the Hopelessness Scale and lower scores on the Self-Control Schedule are not specific to a diagnosis of depression. They also indicate that the Cognitive Style Test does not differentiate depressed alcoholics from normal controls. In this sample, the non-depressed alcoholics were reporting dysfunctional thinking usually associated with a diagnosis of depression.

Depressed alcoholics, in comparison, were reporting a similar level of severity of depressed thinking to that

found in samples of unipolar depressed patients. This indicates that dysfunctional cognition is similar in severity in unipolar depressed patients and depressed alcoholics. However, depressed alcoholics suffer from a higher frequency of negative thoughts than unipolar depressed patients. One possible explanation may be that these alcoholics had been recently detoxified from alcohol, and that alcohol itself may produce a high frequency of negative thinking which does not abate quickly.

Some cognitive measures did not show the same pattern of change as mood measures. Scores on the Dysfunctional Attitude Scale, the Cognitive Style Test, and the Hopelessness Scale did not change over time. However, scores on the Automatic Thoughts Questionnaire and the Self-Control Schedule did show change over the duration of the study.

These findings indicate that alcoholics, regardless of diagnosis of depression, are not only dysfunctional on some measures of cognitive style at admission, but remain so throughout the study. These signs of cognitive dysfunction may be explained by residual depression, as reflected by the relatively high scores on measures of anxiety and depression after detoxification, where even the non-depressed group scored in the mild to moderate depressed range of the BDI (Beck et al., 1988). As the majority of patients had a diagnosis of depression in the pre-admission episode, the non-depressed diagnostic group (after detoxification) may therefore be in remission from depression. Cognitive measures might be expected to show elevations beyond the normal range as a result of mild

depression or, alternatively, the effects of chronic alcohol intoxication.

When the scores on cognitive measures and self-control of those who were not depressed in the episode leading to admission are compared to those of normal control groups, non-depressed alcoholics score significantly higher on some cognitive measures than normal controls. Specifically, those alcoholics who were not depressed in the episode leading to admission were found to score significantly higher at admission on measures of dysfunctional attitudes and frequency of negative thoughts than normal control groups, but their scores do not differ from normal controls on measures of hopelessness, negative cognitive style and self-control. This strengthens the argument that alcoholics, regardless of diagnosis of depression, show at least some elements of cognitive dysfunction. Dysfunctional cognitive style can however be induced by chronic alcohol abuse rather than depression per se. Thus cognitive dysfunction may not be specific to a diagnosis of depression.

The correlation between the frequency of negative thinking and the Severity of Alcohol Dependence Questionnaire (SADQ) ($r=.32$, $p< .01$) and the frequency of negative thinking with the total number of units of alcohol consumed in the week before admission ($r=.27$, $p< .05$) suggest that there is an association between recent alcohol consumption, the severity of dependence and the frequency of negative thinking in alcoholics. The lack of significant correlation between the measure of dysfunctional attitudes and drinking measures at admission, indicates that dysfunctional attitudes are not related to recent alcohol consumption or the severity of

dependence on alcohol. The hypothesis that alcoholics show dysfunctional thinking remains a possibility as scores on the Dysfunctional Attitude Scale, the Cognitive Style Test and the Hopelessness Scale remained stable over the duration of the study.

Measures of affective symptomatology and cognition were found to correlate highly with each other on each occasion of testing. A principal component analysis of baseline affective symptoms and cognition was carried out and two factors were extracted. The first factor contains those measures of affective symptomatology and cognition which were rated by subjects as being representative of how they feel or think in relation to the very recent past, the present or future. The second factor was representative of more "stable" aspects of mood and cognition, as measured by the Dysfunctional Attitude Scale, the Cognitive Style Test and the Self-Control Schedule. These measures are designed to gauge more permanent aspects of affect and cognition, such as beliefs or attitudes, self-description of self control, general level of anxiety and how one views positive and negative situations. As mentioned previously, Beck et al (1983) have suggested that measures of depressogenic cognitive style can be divided according to their level of stability. Consequently, the first of these factors was called a "labile" factor, and the second a "stable" factor.

Those measures on which the depressed alcoholics did not differ from the non-depressed, the Dysfunctional Attitude Scale, the Cognitive Style Test and the Self-Control Schedule, loaded highly on the "stable" factor. This factor did not differentiate the diagnostic groups.

However, the depressed group scored more highly on the "labile" factor.

Some of these measures, the Dysfunctional Attitude Scale, the Automatic Thoughts Questionnaire and the Hopelessness Scale have been classified according to their level of stability in depression. Using a state-trait concept, Beck et al (1983) have suggested that the Dysfunctional Attitude Scale may represent more a trait measure, and the Automatic Thoughts Questionnaire, more a state measure with low stability. The Hopelessness Scale was regarded as being intermediate in terms of stability as it reflects an individual's current cognition and more stable underlying assumptions about the future.

The evidence suggests that a diagnosis of depression does not influence, nor is it reflected in the intensity of drinking, at least after heavy drinking is established. However, this does not preclude a relationship between the severity of depression and past drinking. The evidence from this study indicates that scores at admission on measures of affective symptomatology, and on two measures of cognitive style, the Automatic Thoughts Questionnaire and the Hopelessness Scale, are associated with severity of dependence on alcohol. Further exploration with partial correlation analysis reveals that the association between these measures are not independent from each other in their relationship to the severity of dependence on alcohol. The relationship of the severity of alcohol dependence with the frequency of negative thinking (or hopelessness) disappears when depressive symptomatology is held constant.

5.4.2 Hopelessness and suicidal behaviour

Alcoholism has previously been associated with suicide (Roy and Linnoila, 1986) and there is evidence that the rate of both suicide attempts and suicide is greater in alcoholics than in the general population (Sainsbury, 1978). Other studies have also reported a relationship between suicide, alcoholism and depression (Murphy et al., 1979; Chynoweth et al., 1980) and between hopelessness and suicidal behaviour in parasuicides (Dyer and Kreitman, 1984).

High scores on the Hopelessness Scale are found to have a significant relationship with past suicidal behaviour in the present study. This would suggest that hopelessness, as measured by this scale, is to some extent a stable construct. In addition, two of the subjects in this study committed suicide. These subjects had been interviewed at follow-up (within a month in one case and two days in another, of committing suicide) and their scores on the Hopelessness Scale indicated increasing levels of hopelessness (18 in both cases), one standard deviation above the mean for depressed patients (Nekanda-Trepka et al., 1983). These findings suggest that the Hopelessness Scale reflects both stable and changeable characteristics of cognitive style in that high scores on the Hopelessness Scale were related to past suicidal behaviour for the sample as a whole, and to the completion of suicide in the two subjects mentioned above.

5.5 DRINKING AT OUTCOME: MEASURES OF AFFECTIVE SYMPTOMATOLOGY AND COGNITION

5.5.1 Prediction of outcome

The extent to which cognitive and mood dysfunction in alcoholics were associated with drinking at follow-up was examined. The findings indicate that composite scores (from principal components analysis) of affective symptomatology and cognition measured after detoxification (baseline), when all subjects are abstinent, reflect the prior severity of alcohol dependence, but do not predict drinking at outcome. The measures did not predict whether the patients consumed alcohol or not, nor did they predict the amount of alcohol consumed.

However, drinking at first follow-up is associated with increases in affective symptoms, hopelessness and the frequency of negative thinking. At second follow-up, there is no difference between those abstinent and those drinking on measures of affective symptomatology and cognition but there is an association, for those drinking, between the amount of alcohol consumed and increases in affective symptoms and dysfunctional cognition.

5.5.2 Self-Control

One individual measure of cognitive style, the Self-Control Schedule, deserves particular attention, as it has originated from outwith Beck's cognitive model of depression. Rehm's self-control model of depression (1977), is an attempt to integrate empirical findings from behavioural and cognitive psychology. The model proposes that depressive symptoms can be accounted for by deficits in three inter-related processes of self-evaluation, self-monitoring and self-reinforcement.

Depressed individuals have been shown to monitor fewer positive and more negative behaviours than non-depressed subjects (Roth and Rehm, 1980) and to evaluate their performance by more stringent criteria than non-depressed individuals (Golin and Terrell, 1977). Depressed subjects have also been found to administer lower rates of self-reward (Nelson and Craighead, 1977) and higher rates of self-punishment (Rozensky et al., 1977) than non-depressed subjects.

Rosenbaum's (1980) Self-Control Schedule (SCS) was designed to assess an individual's tendency to apply self-control methods to solving behavioural problems which are regarded as being caused by "internal" events. The assumption behind the SCS therefore is that individuals will vary in their use of self-control strategies in response to "internal" cues or events such as discomforting thoughts or emotions which reduce or disrupt effective problem-solving and thus reduce the likelihood of the individual meeting his or her desired goal. Self-controlling responses are those responses or behaviours which reduce the disruption or interference caused by such "internal" events. In addition, Rosenbaum (1980) assumed that self-control behaviours are learnt behaviours and therefore, it can be assumed, unlikely to vary within individuals over a short period of time. The effect of depression in alcoholics on self-control is relatively unexplored.

The design of the present study did not allow the random allocation of subjects with low and high levels of self-control to different treatment groups, such as treatment with antabuse or antidepressant medication. However, it was predicted that individuals with higher

scores on the Self-Control Schedule at admission would remain abstinent at follow-up as they would have enhanced self-control compared to those with low scores. This prediction is not borne out by the findings of this study, nor is it found that clinicians prescribed antabuse to those with low self-control more frequently than those with high self-control. Furthermore, although the sample as a whole, regardless of diagnosis of depression, was found to show an increase in self-control over the period of the study, there were no significant differences in self-control, as measured by the SCS, between those who were abstinent and those who were drinking at follow-up. Although this study was not designed to examine the relationship between clinician's and patient's perceptions of self-control and treatment, nonetheless the above finding may be worthy of future research. It may be that alcoholics who are low in self-control would benefit from antabuse more than those higher in self-control.

5.5.3 Association of drinking with measures of affective symptomatology and cognition

Neither measures of affective symptomatology and cognition at admission, nor the composite scores resulting from principal components analysis, predicted drinking outcome at follow-up. However measures of affective symptomatology and cognition at follow-up were associated with drinking outcome. Comparing those who were drinking with those who were abstinent at the six weeks follow-up, those subjects who had been consuming alcohol had higher scores on those measures which have been described as more labile (all measures of affective symptoms, in addition to measures of the frequency of negative thoughts and hopelessness). These were also the

measures that had differentiated the depressed and non-depressed groups at admission. Scores on measures of dysfunctional attitudes, negative cognitive style and scores on self-control did not differentiate abstainers from drinkers at follow-up. Neither did they differentiate depressed alcoholics from the non-depressed alcoholics at admission. Again, a principal components analysis of mood and cognitive scores obtained at the first follow-up extracted two factors which confirmed this finding: a labile and stable factor structure emerged. Those who had been drinking during the first follow-up period had significantly higher scores on the "labile" factor (BDI, MADRS, STAIS, STAIT, ATQ and HS) compared to those who had remained abstinent from alcohol. In addition, the amount of alcohol consumed over the follow-up period showed a significant association with the scores on the "labile" factor. Alcohol consumption is therefore associated with an increase in affective symptoms, hopelessness and the frequency of negative thoughts. In contrast, no significant difference was found between those drinking and those who remained abstinent on the "stable" factor, which reflects more stable aspects of functioning: underlying attitudes, the degree of negative interpretation of events relating to the self, world and future and perception of self-control behaviours.

At second follow-up, those who had remained abstinent over the second follow-up did not score differently from those who were drinking on any of the measures of affective symptomatology and cognition. In addition, scores on these measures did not factor into two components, but rather all loaded highly on a single

component. The most likely explanation for these results is the greater duration of the second follow-up. Over the period between first and second follow-up, the majority of sample (82%) had consumed alcohol during this follow-up period and drinking could have taken place at any point within that time frame. If drinking had taken place proximal to the second follow-up, it is likely that a stronger association with dysfunctional thinking and affective symptoms would have been found. This assumption is borne out by the significant association, for those who had consumed alcohol, between the number of units of alcohol consumed per day of follow-up and scores on the affective symptom and cognitive factor at second follow-up. This indicated that the greater the consumption of alcohol, the greater the increase in affective symptoms and cognition during the second follow-up period.

One of the strengths of the present study was in the short length of the follow-up periods, especially the first (six weeks). The short duration of this period allowed a close examination of the relationship between the consumption of alcohol and measures of affective symptomatology and cognition associated with a diagnosis of depression. Those measures of affective symptoms and cognitive style which associate with the consumption of alcohol are labile, and their association with alcohol consumption is not obvious over the course of a longer follow-up, such as the second follow-up in the present study.

Summary

A diagnosis of depression, and the depressive symptoms and dysfunctional thinking which are associated

with this diagnosis, do not predict drinking in alcoholics. Of the many factors which influence the consumption of alcohol in alcoholics, depression is not likely to be one of them. It appears that alcohol consumption does induce symptoms of depression and dysfunctional thinking in alcoholics, sometimes to a degree to reach criteria for a diagnosis of depression. Alcoholics who were drinking at follow-up, regardless of diagnosis of depression, scored significantly higher on measures of depression and anxiety, frequency of negative thoughts and hopelessness. In addition, some aspects of dysfunctional thinking, commonly thought to be specific to depression, (hopelessness, dysfunctional attitudes and the frequency of negative thoughts) do occur in alcoholics, regardless of diagnosis of depression, and regardless of alcohol consumption.

5.6 IMPLICATIONS OF THE PRESENT FINDINGS

The findings of the present study suggest that abstinence from alcohol is the most important goal in the treatment of depression in alcoholics. Once abstinent from alcohol, depressed alcoholics show a decrease in affective symptoms and, over a period of time, become less dysfunctional in cognitive style. The impact of affective symptoms and cognitive style on treatment was not assessed by the present study but it is likely that negative mood states and dysfunctional cognitions may hamper the degree to which an individual is receptive to treatment, particularly in the first week or two of treatment. As such, treatment which involves more abstract and problem solving strategies would be better delayed until depressive mood has decreased in patients.

In depressed patients, affective symptomatology and cognitive dysfunction change with cognitive therapy and/or pharmacotherapy (Simons et al., 1984; Blackburn and Bishop, 1983). In alcoholics with depression, affective symptoms improve rapidly with abstinence from alcohol. It remains a possibility that cognitive therapy may hasten recovery from depression in depressed alcoholics and may alleviate the feelings of hopelessness which are so prevalent in this population.

5.7 SHORTCOMINGS OF THE PRESENT STUDY.

There are several reasons to be cautious about the findings of the present study. The sample in the present study consisted of 82 alcoholics and as such, the sample size is small. It is possible that the small sample size in the present study is not representative of the population of alcoholics from which the patients were selected. However, as discussed above, the characteristics of the sample suggest that the group selected are reasonably representative of heavy and problem drinkers found in the general population and in clinical samples of alcoholics.

Clarifying the diagnostic status of individuals at follow-up would have provided additional valuable information on the stability of diagnosis across time and how diagnosis of depression alters with changing drinking status at outcome. This information would also have been helpful in determining if the measures of affective symptomatology and cognition over the course of the study were specific to a diagnosis of depression.

Additional information in changes in cognition during the three week in-patient programme would have provided valuable information on the effect of abstinence from alcohol, not only on affective symptomatology but also on cognition. Of special interest would have been aspects of cognition which are more labile, such as that measured by the Automatic Thoughts Questionnaire.

The hypothesis that a proportion of alcoholics drink as a prodromal sign of depression cannot be excluded. The Schedule for Affective Disorders contains a section on the course of the presenting illness. Although this information was obtained for subjects in this study, it

was difficult to ascertain the accuracy of statements about the occurrence of initial symptoms as this involved recalling symptoms which had occurred in the distant past. As it was often not clear to what extent alcohol was being consumed at that time, this information was not analysed. A longitudinal study following individuals who are depressed and individuals who are problem drinkers would be useful in exploring this connection.

5.8 FUTURE RESEARCH

One implication of the findings of the present study is that depressed mood and symptoms follow, rather than precede, excessive alcohol consumption. This hypothesis could be tested more accurately under more controlled conditions, with non-problematic drinkers as a control group and abstinent alcoholics (if ethically acceptable). It also appears that depression may be a non-specific accompanying condition to many psychiatric disorders. This possibility also deserves further investigation as it may clarify the nature of depression.

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APPENDIX I

Alcohol Problems Clinic

I am carrying out a study which concerns the way people feel and how these feelings may influence their drinking.

Taking part in the study involves meeting with me on several occasions during your time in the Alcohol Problems Clinic. On two of these occasions, I will be asking you questions about how you are feeling now and how you have been feeling in the recent past. I will also be asking you details about your drinking. These interviews will take between 1 to 1 1/2 hours and will be arranged at a time to suit you. You will also be asked to complete some questionnaires about how you think and feel, and about your drinking. After these two interviews, I will meet with you briefly during the next two weeks of the programme to find out how you are feeling.

After you leave hospital, I shall be asking you to return to see me twice. The first time, one month after you leave and the second time, three months after that. I shall be asking you about how you feel and about your drinking.

I would be grateful if you would take part in this study and will be pleased to answer any questions which you may have after reading this short explanation of the study. I hope that you will not feel under any pressure to take part in this study.

Thank you for showing an interest in the study.

Kate Davidson, Clinical Psychologist.