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Substance Use and Acquired Brain Injury

by

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Contents

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Abstract

The first section of this thesis presents a review on substance use and brain injury. The review identifies that a large percentage of individuals following brain injury are likely to use substances, both pre and post injury. This substance use can have an impact on rehabilitation and outcome. Therefore, this issue must be considered by clinicians working in the field of brain injury. There is limited evidence regarding treatment programmes, particularly in the UK. There has been no investigation into known predictors of relapse from the field of addictions such as cognitive flexibility and employment status. The study presented in the second part of the thesis aims to investigate the relationship between post-injury alcohol use and cognitive flexibility, post-injury productivity, pre-injury alcohol use and time since injury. A significant association was found between pre- and post-injury alcohol use, with trends towards significance with cognitive flexibility and post-injury productivity. However, these indices did not vary significantly for those identified as 'at risk' or 'not at risk'. Regression analysis identified that pre-injury drinking and time since injury were significant predictors of post-injury drinking risk. The results indicate that brain injury services need to assess for alcohol use and to consider follow up procedures after discharge. More research is recommended investigating what other factors may contribute to this risk as time increases following the injury. In addition, it is recommended that time is spent examining the efficacy of treatment of alcohol use following brain injury, for example using modified CBT.

Substance Use and Acquired Brain Injury:

What We Know & Where To Go.

A review of the literature

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Abstract

This review explores substance use and brain injury. Individuals aged 16-24 years are more likely to suffer a brain injury and are more likely to participate in substance use than those in other age groups. Substance use is associated with a negative outcome following brain injury and may limit the potential benefits of rehabilitation. It is known that both brain injuries and substance abuse can result in impairment in cognitive function, in particular executive function. Thus, when substances are used following brain injury, they may exacerbate residual deficits from the injury, and hence it is important for clinicians to be able to assess and treat both problems. However, there is only a limited amount of evidence on how to treat individuals with substance use problems following brain injury, particularly within the UK, although treatment programmes from the USA have been discussed. In addition, assessments currently available for substance use problems are not necessarily valid for use within the brain injury population so research is needed to help develop assessments and treatment programmes for this population group. Also, there is limited research investigating if known predictors of relapse within the addiction field are applicable within the brain injury population. Early identification of individuals with a brain injury who are at risk of substance use may provide clinicians with an opportunity to target rehabilitation to address the issue, although there is little evidence to guide clinicians on assessment and intervention.

Substance Use and Acquired Brain Injury: What We Know & Where To Go

This article reviews the current literature investigating substance use and brain injury and incorporates information on treatment programmes for individuals with substance use problems following brain injury. For the purpose of this review, the term substance use will be used as an umbrella term incorporating drug use (including legal, illegal and prescription drugs) and alcohol use. The review will examine the prevalence of acquired brain injury (ABI) and the impact it can have on an individual. The prevalence, and impact of drug and alcohol use will be presented for individuals with and without ABI, as well as for society.

A section will be devoted to a brief review of models of the development and maintenance of substance use and dependency. The impact of substance use and ABI, particularly traumatic brain injury (TBI) on cognitive, emotional and physiological responses will be considered, with particular consideration given to executive functioning.

Pre-injury substance use will be explored with examination of the acute and chronic effects of substance use. The review will then consider the problems of substance use following ABI and discuss issues of assessment and treatment within this population, as well as directions for future research.

Acquired Brain Injury (ABI)

ABI is one of the leading causes of disability in the UK (British Society of Rehabilitation Medicine; BSRM, 1998). ABI can be defined as an acute (rapid onset) brain injury due to: head injury or post surgical damage (e.g. following tumour excision); vascular accident (stroke or subarachnoid haemorrhage); cerebral anoxia; other toxic or metabolic insult (e.g. hypoglycaemia); infection

(e.g. meningitis, encephalitis); or other inflammation (e.g. vasculitis) (National Clinical Guidelines, 2003).

Stroke and TBI are the biggest causes of ABI. The annual incidence of stroke is estimated at approximately 195 per 100 000 for a first episode of stroke, with 220 per 100 000 suffering a stroke each year (Oxford Community Stroke Project, 1983).

It is difficult to find accurate estimates for TBI, due to difficulty in detecting mild brain injury. These individuals are often not admitted to hospital or may have other injuries requiring immediate treatment. One study based on a household census conducted in the United States (Sosin, Sniezek, & Thurman, 1996) indicated that mild TBI occurs in 519 per 100 000 people, with moderate to severe injury in 99 per 100 000.

There are likely to be differences in incidence rates between the United States and the United Kingdom as well as variation within each country according to geographical and socio-economic factors. Thornhill (et al., 2000) reported an incidence of 329 per 100 000 people per year admitted to hospital in Glasgow with a head injury (Thornhill et al., 2000), with about 150 people still being disabled one year post-injury.

Whilst there are likely to be variations in the incidence and severity rates of injury, the cause of the injury tends to be more consistent. The most common reason for injury is due to road traffic accidents (RTA; 45%) followed by falls (~ 30%). A number of injuries are due to occupational or recreational accidents (~ 10% for each), with a small but significant number of injuries resulting from assaults (~ 5%; King & Tyerman, 2003).

Of those individuals who incur TBI in the UK each year, it is estimated that around 150 will acquire a significant disability as a direct result (BSRM, 1998). This disability may be physical, cognitive, emotional or behavioural and is likely to have a significant impact on the daily life of the individual. The more severe the brain injury the more likely there will be residual impairments.

Classification of severity of a brain injury is determined by one or more of the following measures: The Glasgow Coma Scale[1] (GCS; Teasdale & Jennett, 1974): with scores that range from 3 – 15; the lower the score the more severe the injury; or the length of post-traumatic amnesia (PTA), which is the period of time between receiving the injury and regaining continuous memory for day-to-day events; the longer the PTA the more severe the injury, with over 24 hours being considered severe. Length of unconsciousness can also be used as a measure of severity; up to 15 minutes is considered mild, whilst any injury with more than 6 hours of unconsciousness is classified as severe. However, it is important to note that many individuals in extended coma are frequently sedated, to help minimise agitation, making it difficult to determine the actual duration of unconsciousness due to the injury. Finally, the presence of neurological signs following the injury and damage revealed with neuro-imaging techniques are also considered as indicators of severity (King & Tyerman, 2003).

Substance Use

Alcohol Use. The recommended limit for men is 3-4 units of alcohol[2], per day with a maximum of 21 units a week (Institute of Alcohol Studies (IAS), 2001). Whilst women are recommended to consume no more than 2-3 units on

[1] GCS is a recognised measure of injury severity scored across three parameters measuring verbal, motor and eye responsiveness of the individual

[2] One unit of alcohol equates to a small (125ml) glass of wine, a standard measure of spirit or half pint of beer (Alcohol Concern, 2004)

any occasion with a maximum of 14 units a week. Alcohol dependency is characterised by physical and psychological cravings for alcohol, particularly early in the day, with a general preoccupation for alcohol. Withdrawal from alcohol causes physical symptoms such as nausea, sweating and shaking (American Psychiatric Association, 1994). The UK has seen an increase in the number of people with alcohol dependency, with 1 in 13 people being classified as dependent (Alcohol Concern, 2004).

There has also been an increase in the level of binge drinking with 6 million people reporting that they regularly binge drink (Alcohol Concern, 2004). Binge drinking is classified as consuming more than twice the recommended daily allowance.

Indeed, up to 27% of men regularly drink more than the recommended weekly limit (IAS, 2001), with up to 21% regularly consuming at least 56 units a week (Office for National Statistics, 2000). Figures for women have shown an increase from 10 to 14% drinking more than the recommended weekly limit (IAS, 2001), with up to 8% regularly consuming 42 units a week (Office for National Statistics, 2000).

Drug Use. In the UK, 12% of people aged 16-59 years will have taken illicit drugs, whilst up to 3% admit to having used Class A drugs, within the last year (Home Office Research Development and Statistics Directorate, 2004). Cannabis, a class B drug, is the most commonly reported drug, with 11% of 16-59 year olds reporting regular use. Although the percentage of people using drugs has remained stable since 2001, there have been noticeable trends in the type of drugs used. There has been a decrease in the use of amphetamines, LSD and ecstasy and increased use of cocaine. Younger people are more likely to use

drugs, with up to 28% of those aged 16-24 years using at least one drug within the last year (Home Office Research Development and Statistics Directorate, 2004).

Substance Use and Societal Impact. Drug and alcohol dependency are recognised as a burden to society due to their impact on societal cohesion, crime rates and high levels of co-morbidity with neuropsychiatric illness, such as drug-induced psychoses (Everitt, Dickson & Robbins, 2001). In the UK, it has been calculated that the annual cost of health, policing and lost productivity because of alcohol related trauma (general trauma & ABI), is at least £20 billion (Alcohol Concern, 2004).

Substance Use & ABI

In addition to the information on the estimated prevalence of ABI, there is evidence highlighting the contribution that alcohol and drugs make to the likelihood of acquiring a brain injury (Cherner, Temkin, Machamer, Sureyya & Dikmen, 2001; Corrigan et al., 1995).

The largest proportion, of both men (37%) and women (23%), who are drinking over the recommended weekly limit fall within the 16-24 years age group, this age group also have a higher risk of incurring a brain injury (Sorenson & Kraus 1991). Up to 40% of those admitted for treatment following brain injury are aged between 16-24 years (Ragnarsson, Thomas, & Zasler, 1993).

Furthermore, alcohol dependency is higher within rehabilitation populations (16-74%), than the general population (8-10%; Pires, 1989). Substance use rates within brain injury rehabilitation populations appear to be influenced by the type of injury. Injuries that are more violent in nature, such as those related to assaults, are more likely to be associated with past alcohol dependency (Drubach,

Kelly, Winslow, & Flynn, 1993). Indeed, up to 44% of all violent attacks are attributed to alcohol consumption (Alcohol Concern, 2004).

Theories of Addiction

Psychological theory and research have contributed greatly to the knowledge base of addictions and addictive behaviours. In order to be able to begin treating such problems it is necessary to understand what causes an individual to participate in what can be classified as self-destructive behaviour and what maintains that behaviour.

Behavioural Theory

Behavioural theory proposes that pathological substance use is a learned behaviour. There are two main approaches considered applicable to the learning process for addictive behaviour. First, is the idea that the behaviour is classically conditioned (Pavlov, 1927). Any neutral stimulus preceding substance use may become capable of prompting substance use behaviours after repeated exposure, i.e. the antecedents to substance use are important. The second learning approach comes from the work of Skinner (1988), and highlights the reinforcing properties of the consequences. In terms of substance use, this theory suggests that the reinforcing qualities of the substance themselves, such as the 'high' feeling induced, will lead to the maintenance of the behaviour. Social reinforcement associated with the substance use is also seen as a maintenance factor. Behavioural explanations for the aetiology and maintenance of substance use have also allowed interventions for reducing substance use to be developed; these will be discussed later in this review.

One problem with a purely behavioural approach is that no consideration is given to beliefs and feelings regarding substance use. Marlatt, Demming, and Reid, (1973) demonstrated that informing drinkers that the drink they were consuming contained alcohol increased alcohol consumption. This suggests that cognitive processes may act as a mediator for addictive behaviours.

Cognitive Model

The cognitive model of addiction (Liese & Franz, 1996) proposes that an individual will participate in substance use behaviours following the presentation of activating stimuli. These activating stimuli, or high-risk situations (including interpersonal conflict, the presence of other users, or a change in mood state such as anxiety or depression), will activate beliefs about the substance, for instance "drinking helps me to relax". Following the activation of the belief structure, the individual will have automatic thoughts that will lead to the physical urges or cravings for the substance. Accompanying the urge are facilitatory beliefs, i.e. beliefs that grant permission to participate in taking the substance. For example, a drinker may think, "I'll just have one drink, then I'll stop" or "one more drink won't harm me" which then allows them to consume alcohol.

Evidence has indicated that memory structures also have a role in maintaining substance use behaviours (Zack, Poulas, Fragopoulos, & MacLeod, 2003). Women who report they are more likely to drink when in a negative mood state demonstrate a greater priming effect for alcohol related information when in a negative emotional state. Accessibility of these memory structures is highly dependent on the available cues and the situation. However, it is proposed that the activated memory structure leads to a bias in decision-making processes and behaviour in favour of the substance.

This idea is supported by incentive-sensitisation theory (Robinson & Berridge, 1993). Robinson and Berridge argue that, through attribution, drug taking stimuli are more attention grabbing and that this attribution is mediated by changes in dopamine levels within the brain. As a result, this attention bias is an automatic process that results in inflexible behaviours directed towards the goal of procuring and administering the chosen substance. This bias, along with the natural reinforcement from the drug itself, results in an increased preoccupation with the drug and an impaired ability to shift or focus attention to non-drug related activities. Smokers with a history of repeated attempts to quit show a greater vigilance to smoking related cues (Bradley, Mogg, Wright, & Field, 2003). This suggests that the automatic processes are stronger for drug related information, thereby making it harder to combat with intentional behaviours, i.e. the numerous attempts to quit. This may explain why there is such a high rate of attrition and relapse in treatment programmes for abusers (Marlatt & Gordon, 1985).

Neurophysiological Theory

It is known that drugs have an influence on the neurophysiological status of the brain. In particular, cannabis, cocaine and alcohol all cause an increase in dopamine transmission. Dopamine has a role to play in natural reinforcement (Bradshaw, 2001) and dopaminergic neurotransmitters are projected widely throughout the frontal lobes. It has been argued that addiction is the process of gradual adaptation of the brain to chronic drug exposure (Everitt et al., 2001; Lyvers, 2000). Cognitive functioning requiring activation of the prefrontal cortex, such as problem solving, has been shown to become impaired with

excessive release of dopamine, as well as with over stimulation of the dopamine receptors within the prefrontal cortex (Arnsten & Goldman-Rakic, 1998).

This increase in the levels of dopamine within the prefrontal cortex is the likely explanation for the associated reduction in cognition associated with this region (Lyvers, 2000). Chronic alcohol use results in the depletion of dopamine stores, which causes an increase in sensitivity of dopamine receptors, and helps to explain loss of control and craving shown by alcoholics (Modell, Mountz, Glaser, & Lee, 1993). This is further supported by evidence that drugs used to block dopamine receptors can be used to inhibit alcohol cravings (Modell et al., 1993). Parallel to this, there is evidence suggesting that the changes to the dopamine circuits caused by cocaine use leave the individual vulnerable to craving and relapse even after a prolonged period of abstinence (O'Brien, Childress, McEllan, & Ehrman, 1992).

Further evidence for changes at a neurophysiological level is gained from functional imaging studies. Results show that the medial prefrontal cortex and the anterior cingulate cortex become activated in addicts when shown drug related stimuli (Childress, McElgin, Mozley, & O'Brien, 1999). In addition, PET scans have revealed changes in rates of glucose metabolism within these areas in chronic users of alcohol (Volkow & Fowler, 1992). These cortical areas are known to be involved in attention, planning and self-monitoring and may explain why it becomes difficult to redirect attention to non-drug related activities.

Substance Use, Cognition and Emotion

When discussing substance use and cognition, it is important to explore the possible effects (acute & chronic) that different substances may have on cognitive functioning and emotion.

Acute Use of Alcohol

Alcohol intoxication can cause reductions in motor skills and reaction times, as well as increased aggression and difficulties with problem solving (Ihara et al., 2000). In addition, alcohol intoxication has been shown to cause a significant reduction in verbal memory and abstract reasoning skills, as well as increased levels of disinhibition (Horner, Waid, Johnson, Latham, & Anton, 1999). These changes in cognitive skills remain when drinking history and education level are controlled, highlighting that the deficits are linked to the state of acute alcohol intoxication (Horner et al., 1999).

In addition to the effect on cognitive skills, alcohol consumption can also influence mood states. Alcohol is known to help reduce anxiety through its relaxing properties and individuals will often self medicate to manage their mood (Brady & Randall, 1999; Christiansen & Goldman, 1983). However, heavy drinkers are more vulnerable to depression and anxiety, and show a higher risk of suicide (Buono, Daru, Colucci, & Pava, 2004). In addition, individuals are more likely to drink when experiencing elevated levels of hostility and sadness, which in turn leads to increased levels of these negative emotional states (Hussong, Hicks, Levy, & Curran, 2001).

Chronic Use of Alcohol

It has long been accepted that chronic alcohol abuse has a detrimental effect on cognitive functioning, with links established between prolonged alcohol abuse

and the emergence of Korsakoff type dementia. Prolonged alcohol use appears to have a marked effect on verbal memory for new information, inhibition, control, and abstract reasoning (Horner et al., 1999; Ihara et al., 2000).

Individuals with alcohol dependency frequently exhibit maladaptive behaviours that closely resemble those seen in individuals with lesions in the orbitofrontal cortex, i.e. they exhibit a loss of control over their behaviour and demonstrate difficulties with forward planning. Ihara and colleagues (2000) found that there was a significant difference between alcoholics and controls on an assessment of executive function, even when general intellectual functioning was relatively intact. In particular, Ihara et al. noticed that planning was poor, especially in contrast to the individuals' ability to verbalise what the task required from them.

Kubota and colleagues (Kubota et al., 2001) compared frontal lobe volume in social drinkers with and without dependency. They used magnetic resonance imaging scanning techniques to investigate frontal lobe volume in age groups (30-39, 40-49, 50-59 & 60-69 years) comparing abstainers and individuals whose drinking ranged from light to dependent drinking. They observed a general decrease in volume of the frontal lobes with age, which was accelerated by heavy drinking as well as alcoholism. Heavy drinkers showed twice the rate of loss of volume in the frontal lobes when compared to abstainers and light drinkers within the same age group. This suggests that individuals who are heavy drinkers but who do not meet the criteria for dependency may also be at risk of cognitive impairment.

This decrease in frontal lobe volume may have an impact on associated skills, primarily executive functioning such as cognitive flexibility, and planning.

Comparison of glucose cortical metabolism shows that alcohol produces a significant metabolic depression in the prefrontal cortex, but not in other brain regions (Volkow et al., 1990). PET scans indicate that these abnormalities in metabolism tend to continue even following withdrawal, in spite of an initial improvement (Volkow & Fowler, 1992). It is, therefore, likely that some of the impairments in self-control frequently displayed by alcoholics may be due to these long lasting cortical changes within the prefrontal cortex. Indeed, Wolwer, Burtscheidt, and Gtaebel, (1997) and colleagues have argued that cognitive flexibility is a useful predictor of relapse.

Acute Use of Drugs

Cannabis is the second most common drug used within the brain injury population (Corrigan et al., 1995). Evidence of the effect of cannabis on cognitive functioning is conflicting, with some research highlighting a significant impact on areas such as memory, concentration, cognitive flexibility (Corrigan et al., 1995) and planning (Nahas, 1984). Other research indicates that cannabis has a minimal impact on cognition, but a significant impact on mood causing paranoia, depression, anxiety and depersonalisation (Nahas, 1984; Payne, 2000).

There is, however, agreement in the literature that cannabis has a negative impact on work and family relationships through decreased motivation and suppressed arousal (Corrigan et al., 1995; Payne, 2000).

There is less research available about the effects of cocaine use. Acute use has been shown to reduce blood supply in the frontal and parietal areas of the cortex (Mena, Miller, & Garrett, 1989). Porrino found that following administration of cocaine to animals with intact brains, the prefrontal cortex was the first area of the brain to start showing metabolic changes, even with low doses

(Porrino, Domer, Crane, & Sokoloff, 1988). This change is attributed to the impact that cocaine has on dopaminergic transmission, which it is believed to enhance (London, et el., 1996). This may explain the fact that current users of cocaine often have difficulties in planning, self-monitoring, and impulsivity, difficulties which remain even after an individual has been abstinent of cocaine for at least one month (Volkow et al., 1997).

Chronic Use of Drugs

There is little evidence to suggest that the impact of chronic cannabis use is significantly different to the effects seen with acute use. However, it has been suggested that chronic use of cannabis can lead to the development of psychoses, in particular paranoia (Nahas, 1984).

Brain scanning of chronic cocaine users after a period of abstinence has shown reduced activity within the prefrontal, orbitofrontal and cingulate gyrus areas, associated with poorer executive functions (Volkow et al., 1997). Often, subtle executive functioning deficits are seen in cocaine addicts related to dysfunction within the prefrontal cortex. Bolla, Cadet and London (1998) argue that these deficits are most likely to be due to heavy cocaine use rather than pre-morbid pathology. The deficits arise due to the sensitisation of the mesocortical dopamine system (Volkow et al., 1993). Disinhibition, impulsivity and attentional deficits are commonly seen in individuals addicted to cocaine (Majewska, 1996). Gollub et al., (1998) showed that chronic abusers of cocaine and heroin had significantly lower white matter volume in the frontal lobes; although it may be possible that this is a reflection of a pre-existing condition making the individual more vulnerable to substance use, Liu Matochik, Cadet, and London (1998) report a negative correlation between prefrontal cortex

volume and years of cocaine abuse, interpreting the change in prefrontal volume as a direct effect of chronic drug use.

In summary, it is clear that the use of these substances, on their own, will be likely to impact on cognitive abilities, regardless of whether the individual has suffered a brain injury. From information provided by scanning techniques, it is clear that most of the structural changes occur within the prefrontal cortex, the area of the brain most associated with executive functioning. This would indicate that individuals with a history of chronic substance use might experience difficulties including poor problem solving and poor impulse control. These difficulties, in turn, are likely to act as risk factors for brain injury and the possibility of further cognitive impairment. The prefrontal cortex has been frequently implicated at a neural level for self-control, therefore, it is unsurprising that drug use, both acute and chronic, results in impaired self-control, which has long been associated with addiction (Jellinek, 1960).

Both acute and chronic substance use can have an impact on cognitions and emotions. ABI is also known to have an impact on these functions, and a brief discussion will follow.

ABI, Cognition and Emotion

The cognitive, emotional and behavioural consequences are individually based and linked to both the injury site (e.g. contre-coup or diffuse) and the severity of injury (the more severe the injury the greater length of coma regardless of lesion site (Dikmen, Machamer, Powell, & Temkin, 2003). Knowledge of the type of injury, severity and site of any focal damage can help

clinicians to predict possible cognitive, affective and behavioural deficits and plan rehabilitation (Lezak, Howieson, Loring, Hannay, & Fisher, 2004).

Following mild TBI, individuals often report a cluster of symptoms recognised as post-concussional syndrome (Lezak et al., 2004; Wood, 2004; Smith-Seemiller, Fow, Kant, & Franzen, 2003). Diagnosis of post-concussional syndrome is still regarded as controversial. It includes affective (irritability & anxiety), cognitive (reduced mental speed, memory & attention problems) and somatic symptoms (headaches, dizziness & fatigue) that tend to remit within a few months post-trauma. For a minority, however, symptoms can persist as long as 12 months post-injury (Wood, 2004). For individuals who suffer with residual impairments following mild TBI, the deficits are often subtle and may prevent return to pre-injury levels of work and activities from before the injury (Lezak et al., 2004).

There are often discrete differences in deficits shown by individuals who have suffered a closed head injury (CHI) as opposed to a penetrating head injury (PHI, Lezak et al., 2004). PHI often has a focal point of damage, which may be accompanied by diffuse damage as a result of axonal shearing, whilst CHI often leads to contre-coup damage, frequently involving the frontal and temporal lobes, as well as diffuse damage (Lezak et al., 2004). Other causes of brain damage include brain swelling, bleeding, and lack of oxygen or glucose in the brain, which can arise with stroke, infection and hypoglycaemia, for example.

Knowing that severity and type of injury can have an impact on the types of deficits that may arise, it is important to discuss what symptoms are commonly reported following TBI and ABI.

It is common to see personality and emotional changes following brain injury (Lezak et al., 2004; Milders, Fuchs, & Crawford, 2003). Individuals may display increased irritability, decreased social sensitivity, or they may be emotionally labile or appear to have flattening of emotional responses and often lack motivation (Lezak et al., 2004). With severe injuries, these affective disturbances are often organically based. However, it is also possible that some affective disturbances (depression, anxiety, and irritability) are reactive and associated with functional disability and insight into deficits/changes that are due to the injury (Lezak et al., 2004).

Individuals will commonly report memory deficits following ABI (Ponsford, Sloan & Snow, 1995). However, for some individuals, performance may be within the normal range in testing situations and the difficulties experienced are often found to be due to difficulties with attention including distractibility, and problems with multi-tasking (Ponsford, Sloan, & Snow, 1995). With increasing knowledge of the deficits that can occur following ABI, a number of neuropsychological syndromes have been identified, including amnesic syndromes, syndromes of neglect, and dysexecutive syndrome (Synder & Nussbaum, 2003). Due to the number of deficits that can be seen following ABI, it is beyond the scope of this review to discuss them in detail (see Lezak et al., 2004 for a detailed review). Often the most disabling impairment is linked to a disruption of executive skills, i.e. a person's ability to engage in purposive, self-serving, independent behaviour. If executive skills are intact it is possible to sustain loss of other cognitive skills and still maintain a high level of independence (Lezak et al., 2004). As such, the behavioural problems that are

associated with dysexecutive syndrome serve as an indicator of severity of brain injury.

Executive skills are disrupted by both ABI and substance use so it is important to explore what is meant by executive functions and how we can assess, and treat any deficits.

Executive Functioning

The study of executive functioning, a term first coined by Baddeley in the 1980s, is considered the newest in the field of neuropsychology (Baddeley, 1986; Burgess, 2003). Anatomically, the skills associated with executive functioning (including initiation, sequencing, inhibition, monitoring, planning and adaptation) have been primarily located within the frontal lobes. This area of the brain is particularly vulnerable to damage following brain injury (Stuss, Alexander, & Benson, 1997). Dysexecutive problems often have the largest impact on an individual's ability to engage in and maintain employment as well as being considered as causal in the breakdown of relationships (Burgess, 2003).

In the early development of executive function theory, there was a convergence towards unity, i.e. that there is one common basis underlying these skills as represented by single process or single construct theories (e.g. Cohen, Dunbar & McClelland, 1990; Goldman-Rakic, 1995). However, with the advancement of scanning techniques and research looking at brain activity within non-injured population groups, as well as the dissociations reported between different assessments of executive function (Duncan, Johnson, Swales, & Freer, 1997), there has been a move toward multiple process based theories. Two dominant multiple process theories are Baddeley's Working Memory model

(Baddeley, 1986; Baddeley & Wilson, 1988) and the Supervisory Attentional System, SAS (Shallice & Burgess, 1991). Baddeley suggests that the central executive makes the necessary links between the different areas of the brain allowing a task to be successfully achieved. Whilst Shallice and colleagues state that the SAS has a role in at least eight different processes: working memory, monitoring of performance, schema generation, rejection of schema, processing, goal setting, episodic memory retrieval and intention. Disruption can occur within a process in isolation or across processes, which may explain the variety of presentations clustered under the label 'dysexecutive syndrome'. Despite differences in theoretical approach there is broad acceptance that executive functions encompass a number of core skills which include purposive, self-serving behaviour that requires goal identification, initiation, sequencing, behaviour regulation, problem solving and planning (e.g. Alderman & Burgess, 2004; Evans, 2004; Lezak et al., 2004; Burgess, 1997).

As mentioned, anatomically these skills have been associated with the frontal lobes and animal studies have suggested that there are different roles for the dorsolateral (Goldman-Rakic, 1988) and ventromedial prefrontal cortex (Kowalska, Bachevalier, & Mishkin, 1991). These differences have also been reflected in positron-emission tomography studies, which have shown different patterns of activation in different cognitive tasks (e.g. Paus, Petrides, Evans, & Meyer, 1993). However, there is limited evidence that specific frontal lobe lesions lead to the same specific impairment (Duncan et al., 1997). One reason for inconsistencies in deficits following damage to the frontal cortex may be due to fronto-striatal circuits, which link the frontal cortex to the rest of the cerebral

cortex. These have been identified as having a role within the cluster of symptoms labelled as executive skills (Fuster, 1999).

Although there have been clinical reports of individuals with dysexecutive syndrome, it has only been in the last 20 – 30 years that research has developed clinically meaningful assessments and ideas for methods of rehabilitation of such deficits (Burgess, 2003). Assessments are required not only to help assist with diagnosis, but also to help plan appropriate rehabilitation.

Assessment of Executive Function

There are a number of options available to a clinician wanting to assess an individual's executive skills including: clinical interview; questionnaires; neuropsychological tests; and, assessment of behaviour/function in daily tasks. Individuals with dysexecutive syndrome frequently have problems with insight (Burgess et al., 1996) therefore, it is important that information is also obtained from an informant (usually a close friend or relative). The level of agreement between the individual and the informant can help provide an idea of insight.

Interview

When conducting an interview it is important to ask questions about changes in personality, emotion, motivation, and behaviour as well as cognitive changes. The Dysexecutive Questionnaire (DEX; Burgess et al., 1996) is a 20 item questionnaire that measures cognitive, emotional, and behavioural changes, and it has a version for both the individual and the informant, allowing insight to be assessed. As well as questions, the clinician should note behavioural observations including engagement in the interview, and personal appearance (Lezak et al., 2004)

Psychometric Assessment

A difficulty with psychometric assessment relates to novelty. It is often argued that executive skills are most highly active when the individual is faced with a novel situation (Rabbitt, 1997), which makes it difficult to carry out repeat assessments in this area as, following the initial assessment, it can be argued that the test situation is no longer novel. Another consideration for psychometric assessment is the fact that some dysexecutive problems may be apparent in everyday situations but not show up during formal testing (Manchester, Priestly, & Jackson, 2004). Most neuropsychological tests require the individual to focus on one explicit problem in a short time period, working in a controlled and structured environment, with initiation often being prompted by the examiner (Manchester, Priestley, & Jackson, 2004; Shallice & Burgess, 1991). This can make it difficult to assess executive function, which incorporates initiation, planning and making use of feedback to monitor performance over time.

A summary of some recognised tests of executive functions will be presented. Although it is important to acknowledge that there are a number of assessments available, this review will focus on the assessments most commonly used within ABI research and rehabilitation.

Wisconsin Card Sort Test (WCST; Nelson, 1976)

The aim of this test is the identification of abstract categories and the ability to shift cognitive set making use of external feedback about performance. This task is likely to involve monitoring of performance and inhibition of perseveration (Riccio et al., 1994). The WCST shows promise at identifying individuals with dysexecutive syndrome, with individuals following damage to the frontal lobes completing fewer categories and showing signs of perseveration compared to

non-injured participants. However, there is evidence that individuals who demonstrate significant impairment of executive skill in daily tasks can perform well on the WCST (Eslinger & Damasio, 1985). Another criticism of the WCST is its reliance on memory (Dunbar & Sussman, 1995).

Hayling & Brixton Tests (Burgess & Shallice, 1996)

There are two different tests combined: the Hayling, which looks at initiation, inhibition, and for some individuals highlights strategy generation; and the Brixton, which assesses set attainment and rule detection (Burgess, 2003). This assessment has been shown to differentiate between individuals with frontal lobe lesions and lesions in other areas of the brain (Burgess, 1997).

Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001)

This test battery draws together nine verbal and non-verbal tests that have been widely used to assess executive function (Lezak et al., 2004). Trail Making and the Tower Test assess planning skills. Strategy generation, response monitoring and memory are assessed within Verbal and Design Fluency tasks. The Colour-Word Interference Test is based on the Stroop and looks at an individual's ability to inhibit a dominant response, whilst the Sorting Test is based on the WCST, as discussed above. The Proverb Test can be administered to assess abstract skills. The whole battery does not need to be administered, as there is no composite score. There are norms for both adults and children and the battery incorporates many familiar tests with some modifications; however, the clinical usefulness of these modifications is relatively unknown (Lezak et al., 2004).

Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson, Alderman, Burgess, Emslie, & Evans, 1996).

The BADS was developed with the aim of addressing many of the ecological problems identified with other tests of executive function. It comprises a number of subtasks, drawing on a number of aspects of executive skills. The Modified Six Elements task is based upon the original developed by Shallice and Burgess (1991), assessing planning and self-monitoring skills. The other tasks include the Zoo Map which assesses, firstly, planning with little structure, then planning when more structure is provided. Key Search and Action Programme both assess problem solving. Temporal Judgement assesses the individual's ability to make estimates about time required for completion of tasks, which is important when planning tasks. Finally, the Rule Shift Card Task assesses cognitive flexibility and perseveration.

Evidence suggests that the BADS may be a better predictor of everyday problems than tests such as the WCST or the Stroop (Alderman, Evans, Burgess, & Wilson, 1993). In particular, the Modified Six Elements and the Zoo Map tasks have been shown to be highly sensitive when predicting everyday problems (Alderman et al., 1993). In addition, the BADS appears to have overcome many of the problems encountered when assessing executive function, such as the provision of too much structure and prompting (Shallice & Burgess, 1991). The BADS can provide useful information on problems that may arise due to executive dysfunction and can help to plan rehabilitation (Wilson, Evans, Alderman, Burgess & Emslie, 1997).

All the assessments discussed assess primarily cognitive skills associated with executive functioning. However, other symptoms can be considered, including motor skills (including speech production, e.g. Lezak et al., 2004), which can be disrupted in an individual with dysexcutive syndrome. The DEX questionnaire allows examination of some behavioural changes but, other than behavioural observations, none of the assessments formally assesses motor skills. The Frontal Lobe score is an assessment that assesses aspects of motor skills including alternating pattern copying, rhythm tapping, hand sequences and conflicting demands as well as having a behavioural scale (Ettlin et al., 2000; Wildgruber, Kischka, Fabbender, & Etlin, 2000). This assessment has been shown to detect frontal lobe lesions with 78% sensitivity and discriminates between individuals with non-frontal and frontal lesions (84% specificity).

There are a number of other assessments considered to assess executive functioning, and the reader is referred to writings by Lezak et al. (2004) and Burgess (2003) for detailed discussion of these.

Having completed an assessment and discovered where an individual may have difficulty it is then important, if possible, to offer rehabilitation.

Rehabilitation of Dysexecutive Syndrome

The presentation of executive disorders can be misinterpreted, resulting in misdiagnosis and treatment being offered through psychiatric or forensic services (Worthington, 2003). Even with advances in knowledge of executive dysfunction, there is still no accepted consensus on identification and treatment of such disorders. This results in rehabilitation often being dependent on the

clinician's skills and experiences (Worthington, 2003). However, it is possible to consider the different approaches to rehabilitation.

Rehabilitation is considered either to restore function or to compensate for functional deficit (Evans, 2004). Skills retraining is one way to restore function, based on the idea that 'practice makes perfect', i.e. the function will return. A 'problem solving therapy' approach to help retrain individuals with dysexecutive syndrome was found to improve performance on tests of general intelligence and problem solving, but there was no evidence to indicate generalisation to everyday tasks (von Cramon, Matthes-von Cramon, & Mai, 1991; von Cramon & Matthes-von Cramon, 1992). Based on the concept of 'goal neglect' (Duncan, 1986), Levine et al. (2000) introduced goal management training, which has 5 stages. Levine showed improvement on tasks targeted by the training. Using a similar approach, Evans (2001) described a group for goal management as part of an holistic rehabilitation programme. Clients are provided with a written framework to help guide them through decision making tasks. They are encouraged to practice using the framework to help internalise the strategy.

Alongside these internal strategies, there is also evidence that the use of external strategies, including alarms, pagers, diaries, and checklists, may help people manage dysexecutive symptoms (Manly, Hawkins, Evans, & Robertson, 2002; Wilson, Emslie, Quirk, & Evans, 2000). In addition to both internal and external strategies, behaviour modification can also help individuals to shape and monitor their performance (Alderman, & Burgess, 2003; Alderman, & Ward, 1991).

The previous sections have reviewed the effects of substance use, and of ABI, on emotion and cognition, with particular focus on executive functioning.

Although there may be loss of cognitive skills in other domains, such as memory, the emphasis has been on executive functions due to the disruption that can occur in everyday functioning and the impact that executive dysfunction can have on an individual's life (Burgess, 2004). The next section will explore the possible links between substance use and ABI both prior to and post-injury.

Pre-Injury Substance Use

Most researchers have examined rates of substance abuse by relying on information obtained either from the individual who has suffered a brain injury or from a relative/carer. Studies have also focused on acute (i.e. whether an individual was intoxicated at the time of the injury) versus chronic use (i.e. long term regular use) and how this may affect any injury.

Acute Use

Making use of blood alcohol level readings (BAL), studies have investigated the level of intoxication at the time of injury and its associated prognosis. A positive BAL[3] at the time of injury affects likelihood of survival (Langley, Lindsay, Lam, & Priddy, 1990) and is associated with the increased possibility that the injury is not the first the individual has sustained (Cherner et al., 2001). Many of these prior injuries often go unreported but may have an impact on prognosis. Positive BAL readings on admission to hospital following ABI have been found in 67% of people (Sparadeo & Gill, 1989). Of those involved in pedestrian accidents, up to 30% had consumed alcohol before the injury (Vestrup & Reid, 1989). In addition, regardless of the cause of brain injury, alcohol will

[3] A positive BAL reading is recorded when there is 100mg/dl or more of alcohol in the blood stream.

have been consumed at the time of the injury by over 75% of individuals (Corrigan, 1995).

Furthermore, a positive BAL recorded at the time of injury is linked to a history of alcohol abuse (Corrigan et al., 1995; Sparadeo & Gill, 1989). Similarly, individuals who tested positive for cocaine at the time of their injury were also more likely to have a history of cocaine abuse (Lindenbaum, Carroll, Daskal, & Kapusnick, 1989). Therefore, both chronic and acute use of drugs and alcohol need to be taken into consideration when assessing substance use.

Chronic Use

Individuals with brain injury tend to drink significantly more pre-injury compared to people without a brain injury in the same age group. Indeed, the number of heavy drinkers can be as much as three times that of the general population (Kreutzer, Doherty, Harris, & Zasler, 1990; Taylor et al., 2003). In one study, up to 70% of participants were reported to be moderate or heavy drinkers, whilst only 20% of participants were abstinent before their brain injury (Kolakowsky-Hayner et al., 1999a). Alongside drinking rates, Kolakowsky-Hayner and colleagues also found high rates of drug use. Thirty percent admitted using illicit drugs before their injury, with cannabis being the most common drug. Other drugs reported were heroin (11.5%), cocaine (7.5%), and amphetamines (4%). Several participants reported using multiple drugs. The available evidence supports the idea that although drug use is lower than alcohol use, it must still be regarded as an issue for concern within the brain injury population. Forty percent of a community sample of individuals following brain injury met the diagnostic criteria for substance dependency before their injury (Hibbard et al., 1998). Therefore, it is unsurprising that drug use is seen in a third of people following

brain injury (Taylor et al., 2003). This is consistent with findings from brain injury rehabilitation centres, where up to half of the people in rehabilitation report having pre-morbid substance abuse problems (Corrigan, 1995; Drubach et al., 1993).

Individuals who have chronic alcohol use and alcohol dependency are more vulnerable to many types of injuries, including brain injuries (Solomon & Malloy, 1992) and bone fractures (Ronty, Ahonen, Tolonen, Heikka, & Niemela, 1993). Along with the increased vulnerability to sustaining an injury, alcohol dependency precipitates structural and functional changes within the brain. As has been discussed chronic and heavy alcohol use, both in those with and without alcohol dependency, leads to shrinkage of the frontal lobes, which can create deficits in skills associated with executive functioning (Kubota et al., 2001). These changes make an individual more susceptible to brain damage that can have an impact on their functioning, even following a mild head injury (Ronty et al., 1993). This may explain why the prognosis is worse for individuals with both a history of chronic use and who are intoxicated at the time of the injury (Corrigan, 1995; Langley et al., 1990; Sander, Witol, & Kreutzer, 1997).

Post-Injury Substance Use

It is clear that many individuals who suffer a brain injury have problems linked to substance use before their injury. Therefore, it is important to understand what happens following the injury. Generally, substance use rates tend to drop immediately after the injury (Kreutzer et al., 1990), as is often seen after any major health problem, including a stay in hospital (Tucker, Vuchinich, & Gladsjo, 1984). However, over time, a significant number of individuals return

to pre-morbid levels of use (Bogner, Corrigan, Spafford, & Lamb-Hart, 1997; Edna, 1982; Kreutzer, Wehman, Harris, Burns, & Young, 1991; Kreutzer et al., 1996). Prevalence of substance abuse within rehabilitation units varies from approximately 50% (Bombardier, Rimmele, & Zintel, 2002; Corrigan et al., 1995) to 95% (Bombardier, Ehde, & Kilmer, 1997). This suggests that the presence of brain injury is unlikely to resolve any substance use issues.

Continued use of substances post-injury is an area of concern, as not only can it increase the risk of further injuries (see section on pre-injury substance use), it can also act as a barrier to the potential benefits of rehabilitation (Corrigan, 1995; Ruff et al. 1990). Alcohol is known to have a negative impact on cognitive skills such as memory, planning and self-monitoring, therefore it is likely that continued use post-injury will exacerbate any cognitive deficits that are due to the brain injury. In fact, it has been shown that continued use not only exacerbates cognitive impairment but also leads to social, behavioural and financial problems (Dunlop et al., 1991). Individuals who persist in substance abuse behaviours post-injury show a greater range of psychiatric disorders, increased aggression, higher rates of arrest, lower return to work rates and increased levels of supported employment (Kreutzer et al., 1991; Kreutzer et al., 1995; Sparadeo & Gill, 1989). In addition, these individuals also report having less social support (Cherner et al., 2001) and are likely to report lower levels of life satisfaction compared with non-substance abusing peers (Corrigan, Bogner, Mysiw, Clinchot, & Fugate, 2001).

One of the difficulties when looking at post-injury substance use patterns is that, for many, there may be a period of enforced detoxification immediately after the injury due to limited access to substances whilst in hospital (Delmonico,

Hanley-Peterson, & Englander, 1998). In addition, individuals are often not given the support they may need to achieve the goal of abstinence within a rehabilitation setting. This is because the focus is often on cognitive and work rehabilitation. Yet, it is recognised that individuals following brain injury often have difficulty generalising coping strategies from one situation to another (Delmonico et al., 1998). As such, without explicit guidance on abstinence following brain injury, individuals are unlikely to make progress spontaneously.

Clearly, substance use is an area of concern in terms of rehabilitation and has a significant cost implication for society as a whole. There is a need to identify those at risk of substance abuse and to develop treatment programmes to help minimise the negative impact that the abuse can have on rehabilitation and reintegration (Schmidt & Heinemann, 1999; Bombardier et al., 2002)

Assessment of Substance Use

Although there are measures available, substance use is still relatively under reported within the rehabilitation population (Ashman Schwartz, Cantor, Hibbard, & Gordon, 2004), suggesting a need to identify suitable assessment tools for use within brain injury. It is possible to assess substance use through a variety of means such as biological measures, self-report measures, clinical interview and collateral reports (Arenth, Bogner, Corrigan, & Schmidt, 2001).

Biological Measures

Biological measures of substance use include urine analysis, testing of blood alcohol levels (BAL) and drug levels, liver function and breathalyser tests. These can provide accurate information on current substance use that is unaffected by denial or defensiveness from the individual or their family. However, these tests

are not necessarily able to identify chronic users, who may be substance free at the time of assessment (Skinner, Holt, Schuller, Roy, & Israel, 1984). In addition, the tests can be costly and difficult to administer. There are also ethical issues to be considered when making use of biological measures, in particular issues regarding informed consent for the assessment and the possible impact that taking such measures may have on therapeutic engagement.

Self-Report Measures

Self-report assessments for substance use are usually easier to administer than biological tests. However, one major drawback is a reliance on honesty and insight into the current problem. Alcohol and drug dependency are illnesses of denial, such that those with the problem may defend themselves by not acknowledging that they have a problem (Babor, Kranzler, & Lauerman, 1989). This could undermine the individual's willingness to report accurately their current usage. Any under-reporting may become magnified if sobriety or abstinence is a requirement for service delivery (Babor et al., 1989). Although there is concern with self-report measures, a number is available. Many have been developed and validated for use within the general population (Ashman et al., 2004). However, there is limited evidence on validity within the brain injury population, although there is evidence for effectiveness within the general population (Ashman et al., 2004).

Alcohol Use

CAGE Questionnaire. The CAGE questionnaire (Ewing, 1984) is a brief, much used questionnaire for screening alcohol consumption within clinical settings. It consists of four questions that ask whether an individual has tried to cut down on their drinking, if people have annoyed them by criticizing their

drinking, if they have felt guilty about how much they drink and if they have ever needed to have a drink first thing in the morning. Each question requires a Yes/No response. If an individual answers yes to two or more questions, further investigation is recommended. The CAGE has been shown to identify 97% of heavy drinkers, 92% of those who denied alcoholism and only 4% of those classified as non-alcoholic (Ewing, 1984). Although sensitive for assessing current substance use, sensitivity drops when modified to assess for pre-injury drug or alcohol use (Ashman et al., 2004).

Brief Michigan Alcohol Screening Test (BMAST). The BMAST is a ten-item, shortened version of the Michigan Alcoholism Screening Test (MAST, Selzer, 1971), with which it is strongly correlated (Pokorny, Miller & Kaplan, 1972). It assesses lifetime problems caused by alcohol, encompassing social, family and vocational aspects, and has been reported as useful in identifying individuals abusing alcohol (Fuller, Fishman, Taylor, & Woods, 1994).

Alcohol Use Disorders Identification Test (AUDIT). Like the BMAST, the AUDIT is a ten-item questionnaire. The questions assess alcohol use, alcohol related problems and symptoms of dependency within the previous year. Answers are combined to produce a single score. Scores above the cut off indicate problematic drinking. The AUDIT is intended for the early detection of possibly harmful drinking (Babor, Ramon de la Fuente, Saunders, & Grant, 1992). It can be administered by a range of professions from nurses and social workers through to medics as well supervisors within industrial settings. The AUDIT has been demonstrated to be effective in identifying problematic drinkers when used with individuals following alcohol related trauma, including brain injury (Apodaca & Schermer, 2003).

The Alcohol Expectancies Questionnaire. This questionnaire was developed to assess individuals' beliefs about alcohol, for example, that it has stress reducing properties or helps them in social settings (Brown, Christiansen, & Goldman, 1987). Such beliefs may maintain drinking behaviours.

Alcohol & Drug Use

Substance Abuse Subtle Screening Inventory (SASSI). The SASSI (Miller, 1985) is an empirically based assessment for alcohol and drug use. It does not need to be administered by a professional and can be scored objectively. It allows an individual to be classified according to the amount and frequency of substance use. As an assessment tool, it has been shown to have high sensitivity, 94%, when used in the general population compared with 69-75% sensitivity when used following brain injury (Ashman et al., 2004).

Clinical Interview

Alongside questionnaires, information can be obtained from the individual through interview and observation. Without assessment, services will be unable to offer appropriate treatments to meet the needs of the individuals (Kolakowsky-Hayner et al., 1999b). A recent study found that simply by asking questions it was possible to identify as many abusers as were found using a toxicology screen (Bombardier et al., 2002). In addition, the clinician must also be prepared to ask questions regarding use of prescription drugs, as well as illicit drugs and alcohol (Schmidt & Heineman, 1999).

These questions should address behaviours associated with abuse including frequent job changes, high rates of absenteeism, early morning substance use and guilt surrounding substance use (Taylor et al, 2003).

In addition to using screening assessments and asking questions on drug and alcohol use, there is a need to ascertain an individuals' readiness for change (Bombardier, Edhe, & Kilmer, 1997). For example, there is little point involving an individual in a self-help group such as Alcoholics Anonymous (AA) unless they are in the 'action phase' of readiness for change. Thus, motivational interviewing may be used to help individuals increase their readiness for change (Miller & Rollnick, 2002), a point addressed later in the review.

Collateral-Report Measures

Although there are drawbacks, collateral reports can be used to check the reliability of the information gained from the individual (Sander et al., 1997). For example, relatives may try to minimise the problems because they do not want to give services a bad impression of the individual. They may not be aware of the extent of any drug or alcohol problem, or may have their own problems and, as such, may under report difficulties (Arenth et al., 2001). In addition, some relatives may not be willing to become involved with services, seeing any problem as the individual's and not their own (Arenth et al., 2001).

Encouragingly, research has shown that there can be up to 90% concordance between self-report information and collateral data on measures of alcohol use (Sander et al., 1997). Unsurprisingly, Sander et al. found higher rates of concordance when collateral information was obtained from a spouse as opposed to parents. This research also highlights that more credibility should be given to self-report information, even for sensitive topics such as alcohol use.

Summary

There are a number of ways that substance abuse can be assessed. These include biological measures, self-report and collateral questionnaires as well as

clinical interview. As mentioned above, Blood Alcohol Level tests can be expensive and invasive as there is a need for blood to be taken and analysed. If, as Arenth et al., (2001) have argued, self-report questionnaires are as accurate as BAL readings, it should be possible and more cost effective to start the assessment process with questionnaires and then use further assessment, as needed. This may also help to circumvent the difficulty that biological measures have in identifying those who are not over the limit at the time of assessment, but who do have a problem with substance use. However, difficulties are still found in gaining accurate information of pre-injury substance use, yet this is likely to be a strong predictor of post-injury use (Bombardier, Temkin, Machamer, & Dikmen, 2003). The evidence presented indicates that it is possible to assess substance use. Given that rates of substance use within rehabilitation populations can be as high as 95% (Bombardier et al., 1997) it is necessary to conduct an assessment. Following on from an assessment, decisions must be made on the most appropriate treatment to be offered.

Treatment of Substance Use

Cognitive and behaviour theory have had a strong influence on the development of treatments for substance use. Application of behaviour theory has led to the development of treatment using cue exposure and contingency management. Based on classical conditioning, cue exposure predicts that substance use can be ended by extinguishing conditioned responses (e.g. Havermans & Jansen, 2003). Evidence for cue exposure has been mixed. Although it offers promise, a greater understanding is required of any factors (including cognitive and emotional factors) that may mediate or moderate cue reactivity (Kadden, 2001). Making use

of reinforcement contingencies through the application of behavioural principles has been shown to have some effect in treating cocaine and opiate dependency (Iguchi, Belding, Moral, Lamb, & Husband, 1997). Individuals attending outpatient clinics were provided with vouchers when they provided clean urine tests or completed sections of their treatment programme. These vouchers could then be exchanged for items or activities considered in line with treatment goals. This approach is designed to help increase the ratio of reinforcement from substance free sources, as well as providing an opportunity to try alternative substance free activities. This is an important aspect to be considered, as many individuals who engage in substance use will frequently have a limited social repertoire.

Many approaches based on traditional cognitive behaviour therapy consider substance dependency as a maladaptive coping strategy and intervention is focused on skill development in cognitions and emotions (e.g. Ritvo et al., 2003). Although CBT has demonstrated efficacy, it is not superior to other treatments including motivational enhancement (Project MATCH Research Group, 1997).

With the increased recognition of motivational aspects there has been a drive in the addictions field to develop interventions that allow a clinician to help increase motivation towards cessation of substance use and maintained abstinence. One such approach, linking cognitive and behavioural aspects, is Motivational Interviewing (MI; Miller & Rollnick, 2002). MI is a method hypothesised to increase motivation towards making change through the application of Prochaska and DiClemente's (1982) transtheoretical model of change. Five stages of change are identified: precontemplation, contemplation, preparation, action, and maintenance, and treatment should fit the stage of

change. For example, an individual identified in the precontemplation stage may be classed as 'in denial' due to their lack of reporting any problems with their behaviour. It would be inappropriate to work with this individual on planning alternative activities; the focus of treatment needs to be on helping move the person towards acknowledging that there is a problem.

MI is "a directive client centred counselling style for eliciting behaviour change by helping clients explore and resolve ambivalence" (Rollnick & Miller, 1995, p326). Movement through the stages of change is created with the application of five basic principles: avoiding arguing; rolling with resistance; supporting self-efficacy; expressing empathy; and, developing discrepancy. Although the empirical validation of this model is mixed (Blanchard, Morgensten, Morgan, Labouvie, & Bux, 2003), it is still considered a helpful framework with good face validity. However, there are concerns about how a clinician can assess and place an individual within a stage that can then offer predictive utility for future behaviour. Alongside this, there may be difficulties in working with people following brain injury who have an organic insight deficit, rather than a problem of denial as they may be misclassified as being in precontemplation and thus not be seen as ready to shift with regard to alcohol and drug use behaviours.

One major difficulty of working within substance use rehabilitation in the general population is the recognised high attrition rate (Baekeland & Lundwall, 1975; Marlatt & Gordon, 1985). Younger people are more likely to drop out, especially if they have a family history of substance abuse (Baekeland & Lundwall, 1975). This may have implications when working with people following brain injury, as those aged 16-24 years have the greatest risk of incurring a brain injury (Ragnarsson et al., 1993; Sorenson and Kraus 1991).

Completion of any treatment for substance use has a significant effect on the likelihood of maintaining abstinence. In one study, 73% of those who completed therapy remained abstinent whilst only 24% of those who did not were able to maintain abstinence (Baekeland & Lundwall, 1975). Although these figures are quite old, there is little evidence that there has been a significant change in attrition rates. For example, from a sample of 117 people who requested treatment, only 11% were still engaged in treatment after one month (Stark, Campbell, & Binkerhoff, 1990).

Another issue to be considered when working with individuals following brain injury is the impact that any cognitive deficits will have on their ability to comprehend and retain information (Langley et al., 1990). Treatment programmes need to be tailored to an individual's cognitive status. For example, information may need to be presented verbally and/or visually. The language used may need to be concrete with situational specific examples, since generalisation of skills is often difficult following brain injury (e.g. von Cramon et al., 1991). This concept has been applied to the treatment of substance use in brain injury, particularly in America, and some of the available literature on these programmes will now be discussed.

Traumatic Brain Injury Network. The Traumatic Brain Injury Network (TBI Network) is a community based intervention programme established in the US (Corrigan et al, 1997). The focus is on the coordination of resources to ensure access to support within the individual's own environment. Evidence shows that after a year of coordinating community resources within the TBI Network, participants displayed improved levels of productivity with higher rates of abstinence from substance taking compared to baseline (Corrigan et al., 1997).

Higher rates of abstinence were also reported among individuals who entered the TBI Network programme shortly after their injury than those who were longer post-injury. This has led Corrigan and colleagues to suggest that there is a "window of opportunity" for intervention to be most successful.

This concept is supported by evidence that drinking rates have a tendency to increase with time after injury (Kreutzer et al., 1996). Research has shown that, following a brain injury, people are unlikely to be "light" social drinkers; the pattern appears to be either abstinence or frequent heavy drinking (Kolakowsky-Hayner, et al, 1999a). Thus, early identification may help prevent problem drinking from appearing by enabling the individual to develop alternative coping strategies to use in situations that are likely to lead to substance use.

Skills Based Substance Abuse Prevention Counselling. Another treatment approach is Skills Based Substance Abuse Prevention Counselling (SBSAPC, Langley et al., 1990). SBSAPC consists of four stages and hopes to circumvent cognitive deficits, such as difficulties in new learning and generalisation, by working slowly through the stages. The first stage is a comprehensive educational phase about substance use and its impact on daily living. Individuals are taught about the benefits of conscious self-monitoring to help them understand their own substance use behaviours. Time is spent enhancing motivation to change through increasing dissonance and focusing on reasons for change. Then, the individual will spend time on learning and developing alternative coping skills. Development of these skills is enhanced through role-play and modelling, i.e. the work is multi-modal and not just didactic in nature. Once the individual has developed a number of alternative coping skills for risky situations, there is a process of structured generalisation where specific situations

are targeted. Those who have participated in this intervention have reported that the preparatory section of the intervention is the most helpful. Goal setting, completing cost-benefit analysis and the use of role-plays were more beneficial than the actual skill rehearsal. It is possible that these stages allowed the individual not only to learn the alternate skills but also to develop confidence in their ability to use them in vivo (Langley et al., 1990).

Summary

Much of the evidence presented has come from work carried out in North America. It has shown that it is possible to treat substance use problems in individuals following brain injury. What becomes clear is that there is a need for time to be spent ensuring that programmes are individually based, making allowances for cognitive deficits (Langley et al., 1990). Any educational component should focus on the impact of continued substance use on functional and independent status, i.e. a return to normality, as this is frequently the ultimate goal of an individual in rehabilitation following brain injury (Schmidt & Heinemann, 1999).

Areas for Future Research

Many of the studies cited in this review suggest that substance use may be under reported within the brain injury population (e.g. Corrigan et al., 1995). A large-scale prevalence study is needed to help clarify the rates of substance use as well as the types of substances used. In addition, research is needed to explore outcome following brain injury in relation to different substances.

There is a strong association between brain injury and substance use (Cherner et al., 2001; Corrigan et al., 1995), and, therefore, a need for brain injury

rehabilitation services to assess for substance use post-injury (Bombardier et al., 2002; Kolakowsky-Hayner et al., 1999b; Schmidt & Heinemann, 1999). In addition, it has been recommended that services promote early identification of individuals who may be vulnerable to post-injury substance abuse (Kolakowsky-Hayner et al., 1999b). Unfortunately, no clear advice is provided on how to identify those who are vulnerable. Poor cognitive flexibility and poor planning have been found to be predictive of relapse within the addictions field (Wolwer et al., 1997), yet there is no evidence examining the role that these skills may play in post-injury substance abuse. Exploration of these cognitive skills may help to develop, and enhance, a screening process that would allow clinicians to identify those who are at risk of post-injury substance abuse early on in their recovery.

In addition, it would be beneficial to develop an assessment tool that is sensitive for both pre- and post-injury substance use. Although there are assessments available, the majority have been standardised for use within the general population and assess recent substance use. It could be argued that if people are assessed early enough post-injury, the assessment will cover any substance use from before the injury. However, from experience, many individuals may not seek help in terms of rehabilitation, particularly cognitive rehabilitation, until they have experienced significant difficulty in their day-to-day life. In these cases, there may be a need specifically to assess pre-injury substance use, which might be beyond the scope of many of the available assessments.

Any assessment process should help to guide treatment plans. Unfortunately, as well as concerns about the assessments available, most of the evidence on treatment programmes has been established from research conducted

in North America. Evidence for cross-cultural validity for these programmes is not yet available. Investigations are needed to establish the efficacy of such treatments within the UK, which may have different service structures as well as cultural influences, such as religion and ethnicity. When working with individuals with substance use post-injury, it is important to acknowledge theories that have enhanced understanding of the development and maintenance of substance use behaviours. In particular, cognitive theory has highlighted an attentional bias towards stimuli associated with the substance (Bradley et al., 2003; Robinson & Berridge, 1993). Following brain injury, there are often difficulties with cognitive flexibility and attentional skills. Such organic deficits may have an impact on prognosis and treatment outcome. Research investigating the application of modified cognitive behaviour therapy may help to develop treatment programmes within this area.

This review highlights that there are links between substance use, ABI and executive functioning that may have important clinical and social implications. Although knowledge is growing, there are still questions that need to be addressed and services, both for addictions and brain injury rehabilitation, may need to work together to provide the best support to individuals who cross over between the services.

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Predicting Alcohol Use Following Acquired Brain Injury

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Adhering to APA guidelines

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Abstract

There is a need to help clinician's identify individuals who may be vulnerable to alcohol use following a brain injury. This study aimed to identify possible predictors of post injury alcohol abuse risk, by exploring cognitive flexibility, planning, problem solving, post-injury productivity, time since injury, and pre-injury alcohol use. Data from 33 of 38 participants recruited from a local brain injury service were analysed. Current (post-injury) alcohol use was assessed using the AUDIT and a modified version was administered to assess pre-injury alcohol use. Cognitive flexibility, planning and problem solving were assessed using the Behavioural Assessment of the Dysexecutive Syndrome (BADS). Post-injury productivity was assessed with the Brain Injury Community Rehabilitation Outcome Scale (BICRO-39). Results identified a positive association between pre and post-injury drinking. Trends were found for poor cognitive flexibility and increased productivity to be associated with higher alcohol use post-injury. However, these indices did not vary significantly for those identified as 'at risk' or 'not at risk'. Regression analysis identified that pre-injury drinking and time since injury were significant predictors of post-injury drinking risk. There are service delivery implications, as fewer people are likely to be involved actively in rehabilitation services as time increases post-injury. More research is needed to help explain the complex relationship between brain injury and post-injury drinking.

Predicting Alcohol Use Following Acquired Brain Injury

There is a growing recognition that alcohol intoxication can increase the risk of acquired brain injury, (ABI; Langley, Lindsay, Laum, & Priddy, 1990; Rosenbaum & Hoge, 1989), as well as having a negative impact on outcome following ABI (e.g. Corrigan, 1995; Sander, Witol, & Kreutzer, 1997). However, there is limited research on the risk and impact of alcohol use following ABI. To be able to address this, it is important to have some understanding of both ABI and alcohol use.

ABI can be defined as an acute (rapid onset) brain injury due to: head injury or post surgical damage (e.g. following tumour excision), vascular accident (stroke or subarachnoid haemorrhage), cerebral anoxia, other toxic or metabolic insult (e.g. hypoglycaemia), infection (e.g. meningitis, encephalitis) or other inflammation (e.g. vasculitis) (National Clinical Guidelines, 2003).

Traumatic brain injury (TBI), is a particularly common cause of ABI, occurring in 609 per 100 000 people, in a US household census (Sosin, Sniezek, & Thurman, 1996). UK figures based on hospital admissions indicate that 329 per 100 000 adults (14 years or more) sustained a TBI per year (Thornhill et al., 2000). In this Glasgow study, 90% of the sample had a minor injury, and most had had either a fall (43%) or been assaulted (34%), whilst 61% had consumed alcohol at the time of injury. The most common reason for moderate to severe brain injury is road traffic accidents (RTA; 45%) followed by falls (~30%). A number of injuries are due to occupational or recreational accidents (~10% for each), with a small but significant number of injuries resulting from assaults (~5%; King, & Tyerman, 2003). Furthermore, there is a known association between both RTAs and assaults and alcohol intoxication (Corrigan, Rust, Lamb-Hart, 1995; Movig et al., 2004).

High levels of alcohol consumption can increase the risk of ABI. There is a threefold increase in the risk of intracerebral and subarachnoid haemorrhage, when alcohol consumption is at least 30g daily (approximately 3 units[1] of alcohol; Edwards, Marshall & Cook, 1997). In addition to the increased risk of haemorrhage, chronic, excessive use of alcohol is also associated with coronary heart disease and hypoglycaemia (Edwards et al., 1997). These problems can lead to loss of consciousness, and in some cases, brain damage through lack of oxygen and glucose in the brain (Edwards, et al., 1997).

Due to their location, the frontal lobes are particularly vulnerable to damage following RTAs, fights and falls (Stuss, Alexander, & Benson, 1997). This area of the brain has long been associated with executive functioning (Benton, 1991). Although there has been some debate regarding the theoretical basis of executive functions (Goldman-Rakic, 1995; Baddeley & Wilson, 1988; Shallice & Burgess, 1991), it is commonly agreed that the skills include purposive, self-serving behaviour that requires goal identification, initiation, sequencing, behaviour regulation, problem solving and planning (e.g. Alderman & Burgess, 2004; Evans, 2004; Lezak, Howieson, Loring, Hannay, & Fisher, 2004; Burgess, 1997). These functions can be disrupted following TBI (e.g. Della Sala, Gray, Spinnler & Trivelli, 1998; Tranel, Anderson, & Benton, 1994). Disorder of executive function, sometimes called dysexecutive syndrome is particularly important in the context of brain injury rehabilitation, as it can be a barrier to progress (Evans, 2004). As a result, rehabilitation often focuses on both internal and external strategies to help overcome difficulties in such skills as initiation and self monitoring (Levine et al., 2000; Manly, Hawkins, Evans, & Robertson, 2002;

[1] One unit of alcohol equates to a small (125ml) glass of wine, a standard measure of spirit or half pint of beer (Alcohol Concern, 2004)

Wilson, Emslie, Quirk, & Evans, 2000). Additional difficulties may arise due to motivational deficits or lack of insight, which are commonly associated with dysexecutive syndrome (Al-Adawi, Powell & Greenwood, 1998).

In addition to the significant impact of TBI on executive functions, alcohol intoxication is also associated with a disruption in executive functioning and a reduction in problem-solving skills (Chermack & Giancola, 1997). However, it is unclear whether alcohol causes this disruption or if poor executive skills lead to heavy drinking in later life (Klinteberg, Andersson, Magnusson, & Stattin, 1993; McMurran, et al., 2002). Individuals with chronic alcohol dependency exhibit similar deficits to those seen with dysexecutive syndrome (Block, Erwin, & Ghoneim, 2002; Ihara, Berrios, & London, 2000; Horner, Waid, Johnson, Latham, & Anton, 1999). This pattern of deficits is consistent with evidence that frontal lobe shrinkage is seen in heavy drinkers as well as those with alcohol dependency (Kubota et al., 2001), and that this shrinkage is greater than would be expected with normal ageing.

Alcohol use is associated with an increase in dopamine transmission, which is important for natural reinforcement (Bradshaw, 2001). Dopaminergic neurotransmitters are projected throughout the frontal lobes and animal studies have found that excessive release of dopamine within the prefrontal cortex results in an impairment of cognitive skills theoretically reliant on prefrontal cortex activation (Arnsten & Goldman-Rakic, 1998). This alcohol related increase in dopamine levels in the prefrontal cortex is a possible explanation for the deficits in executive functions seen in alcoholics (Lyvers, 2000).

The role of alcohol in brain injuries varies. Alcohol is identified as a causative factor when the injury is the result of an assault (Corrigan, Rust, Lamb-

Hart, 1995), or with self-inflicted trauma (Kreutzer et al., 1996; Taylor, Kreutzer, Demm, & Meade, 2003). Blood alcohol readings taken upon admission to hospital show that up to 67% of those with brain injury will register a positive reading[2] (Sparadeo & Gill, 1989). Positive BAL readings are found in up to 30% of pedestrian accidents (Vestrup & Reid, 1989) and as many as 78% of those who incur a brain injury will have consumed some alcohol at the time of their injury (Corrigan, 1995). In addition, individuals who have a positive BAL at admission to hospital are ten times more likely to have a history of alcohol abuse than those with a negative reading (Sparadeo & Gill, 1989).

The association between alcohol use and acquired brain injury is reflected by more people being classified as heavy drinkers within brain injury populations compared to the general population (Kolakowsky-Hayner et al., 1999). In addition, abstinence rates are significantly lower for those with brain injury than in the general population (Kolakowsky-Hayner et al., 1999).

Alcohol use prior to any brain injury is a cause of concern as there is a higher risk of mortality and further complications including: prolonged bleeding, increased hospital stay, increased levels of agitation, and poorer cognitive status at discharge, associated with positive blood alcohol readings (Edna, 1984; Kelly, Johnson, Knoller, Drubach, & Winslow, 1997; Kreutzer, Harris-Marwitz, & Witol, 1995; Sparedo & Gill, 1989). Alcohol use before brain injury is also associated with poorer outcome following rehabilitation, with decreased life satisfaction following the injury and increased likelihood of depression and further injuries (Corrigan, 1995; Sander, Witol, & Kreutzer, 1997). If an individual continues to use alcohol post-injury, they are likely to exacerbate any

[2] A positive reading is recorded when there is 100mg/dl or more of alcohol in the blood stream.

residual impairments and it frequently causes financial problems (Dunlop et al., 1991; Jones, 1989; Meek, Clark, & Solana, 1989) as well as increases the risk of further injury (Barnfield & Leathem, 1998).

As alcohol use can be a barrier to rehabilitation (Evans, 2004; Heinemann, 1986), there is a need for clinicians working in the field of brain injury to be able to identify and address alcohol use (Arenth et al., 2001). However, there is limited information available, not only on how to identify, but also how to treat these individuals (Ashman Schwartz, Cantor, Hibbard & Gordon, 2004). There is a need to be able to identify those vulnerable to post-injury alcohol use, as the literature reports that alcohol use is likely to increase with time post injury (Bogner et al., 2001; Kreuzter et al., 1996; Sparadeo & Gill, 1989). A number of reasons for this increase in alcohol use post-injury have been suggested including increased availability and contact with situations that may be associated with alcohol use (Delmonico, Hanley-Peterson, & Englander, 1998). In addition, survivors of brain injury often report feeling that there is pressure to return to 'normal' (Corrigan, 1995) and for those who engaged in drinking before the injury drinking may be associated with that return to 'normal'.

In summary, alcohol abuse has been shown to be a risk factor for both acquired brain injury, including TBI, subarachnoid haemorrhage, hypoglycaemia ((Edwards, et al., 1997) and for frontal damage in its own right (Kubota et al., 2001). Alcohol use before brain injury can have an impact on the severity and outcome of the injury, whilst alcohol use post-injury can impede rehabilitation. Pre-injury alcohol use and unemployment, which is common following ABI, have been identified as possible risk factors for post-injury alcohol use. The frontal damage caused by ABI may also provide an additional risk for post-injury alcohol

use, as dysexecutive symptoms including impulsivity and poor planning have been identified as risk factors for alcohol use and relapse within the general population (Edwards et al., 1997; Klinteberg et al., 1993; Wolwer, Burtscheidt, & Gtaebel, 1997). Taken together, it is therefore essential that clinicians are able to identify those individuals who are vulnerable to developing alcohol problems post-injury and that rehabilitation incorporates intervention for alcohol use when this is appropriate.

Study Aims

This study was designed to explore the association between post-injury alcohol use and pre-injury alcohol use, executive functioning skills (planning & cognitive flexibility), productivity levels (paid employment, voluntary work, training & child care) and time since the injury. The aim was to identify factors that might help clinicians to predict those individuals at risk of alcohol use post-injury.

Question 1: Is pre-injury alcohol use correlated with post-injury alcohol use?

Previous research has highlighted that pre-injury drinking has a positive association with post-injury drinking levels (Taylor et al., 2003). Therefore, it was expected that pre-injury alcohol use would be associated with current alcohol use within this study.

Question 2: Are cognitive flexibility, and planning skills associated with post-injury alcohol use?

Research from the addictions field has indicated that inflexible thinking, poor planning skills and impulsive behaviours are risk factors for relapse (Wolwer et al., 1997). This suggests that cognitive flexibility and planning skills may act as additional risk factors for those likely to use alcohol post-injury.

Question 3: Are levels of post-injury productivity associated with post-injury alcohol use?

Research has suggested that there may be a link between reduced employment levels and alcohol use (Corrigan et al., 2001). Post-injury productivity includes paid employment, voluntary work, childcare and training/studying (Powell, Beckers & Greenwood, 1998). Powell and colleagues have argued that productivity measures are actually more meaningful than simply assessing whether an individual has returned to paid employment following brain injury. It was expected that there would be an association between productivity levels and current alcohol use.

Question 4: Does post-injury alcohol use increase with increased length of time since the injury?

Corrigan (1995) has suggested that as length of time increases post-injury so does the risk that people will return to pre-morbid alcohol consumption levels. As such, it was expected that as time increases following the injury, alcohol use would also increase.

Question 5: Can post-injury alcohol be predicted from pre-injury alcohol use, executive function measures, productivity or time since injury?

By exploring which variables showed significant associations with post-injury alcohol use in answer to Questions 1-4, the aim was to identify possible predictors of post-injury alcohol use.

Method

Participants

Participants were recruited through a local Community Brain Injury Rehabilitation Service in the South of England. Prospective participants were identified from both the active and discharged case files in the Psychology Department (N = 327); thirty-eight participants were recruited for this study. Referrals to the Psychology Department were received from the local Neurology Department as well as the Brain Injury Team.

Inclusion & Exclusion Criteria

For the purpose of this study, and in line with the local service, ABI included adults aged 18-65 years who had a rapid onset of brain injury of any type, but excluded people with vascular accident. All participants had incurred the injury at least one year before the study. All Neurology referrals were excluded, as these referrals generally consisted of degenerative illnesses, including multiple sclerosis and early on-set dementia (n = 115). Individuals were excluded if they had acquired their brain injury before the age of 18 (n = 57) or because they were less than one year post-injury (n = 58). Individuals with memory impairment, more than two standard deviations below the age normed mean (Wechsler Memory Scale – Third Edition; Wechsler, 1997), were excluded as there may have been concerns regarding the reliability of information regarding alcohol use.

Measures

Memory. The Logical Memory subtest from the Wechsler Memory Scale – Third Edition (WMS-III; Wechsler, 1997) was used to assess participants' memory ability. The scale is popular in both clinical and research settings, and

has been updated in line with theoretical advances (Tulsky & Ledbetter, 2000) with standardisation conducted on a larger, more culturally, representative sample within the UK (Wycherley & Benjamin, 1999). The Logical Memory test has good test-retest reliability (from .75 to .99; Iverson, 2001).

Executive Function. Three tasks from the Behavioural Assessment of the Dysexecutive Syndrome (BADS; Wilson, Alderman, Burgess, Emslie, & Evans, 1996) were administered to assess participants' executive skills. The Rule-Shift Card Task was used to assess cognitive flexibility, whilst the Zoo Map and the Six Elements tests assessed planning skills. Scoring on all the tasks ranged from 0 (impaired) to 4 (superior). The BADS is standardised for use with people with brain injury and has good sensitivity to executive dysfunction (Norris & Tate, 2000). Evidence suggests that the BADS is a better predictor of everyday problems, within the brain injury population, than tests such as the Wisconsin Card Sort Task or the Stroop (Alderman, Evans, Burgess, & Wilson, 1993). Indeed, the Six Elements and the Zoo Map tasks have been shown to be particularly sensitive to day-to-day problems associated with dysexecutive syndrome (Alderman, et al., 1993).

Productive Employment. The productivity subscale from the Brain Injury Community Rehabilitation (BICRO-39: Powell, Beckers, & Greenwood, 1998) questionnaire was administered to assess post-injury productivity. This subscale assesses participation in a number of productive roles: paid employment, voluntary work, training/studying and childcare. The BICRO-39 has been validated for use with people with brain injury (Powell et al., 1998). In addition to the patient questionnaires, there is a post-injury informant version for relatives or other carers to complete. The authors have demonstrated that this has high

agreement with the patient questionnaire ($r > .60$ for all subscales). The questionnaires have good test-retest reliability with reliability coefficients greater than .70 for all the subscales (Powell et al., 1998). Scoring on the productivity scale can produce scores ranging from 0 (where an individual is engaging in more than 20 hours of paid work, voluntary work, studying and/or childcare) to 20 (where an individual is not engaging in any productive employment activities).

Alcohol Use. The Alcohol Use Disorders Identification Test (AUDIT; Babor, Ramon de la Fuente, Saunders & Grant 1992) was administered to assess current levels of alcohol consumption. The AUDIT is a 10-item screening questionnaire that assesses the amount and frequency of drinking, alcohol dependence and problems that are caused by alcohol within the last year. Each question is scored from 0-4, with a maximum score of 40. The higher the score the greater the alcohol use and problems associated with alcohol use. Using a cut off of 8 points, the scale has been shown to have excellent sensitivity to hazardous consumption (95%) and alcohol dependency (93%). The AUDIT also has very high specificity (93%; Babor et al., 1992). Hazardous consumption is defined as a level of alcohol intake or pattern of drinking which, if it continues, is likely to result in harm (Piccinelli et al., 1997). As such, men regularly drinking more than 3 units/day (21 units/week) and women regularly drinking more than 2 units/day (14 units/ week) can be regarded as hazardous drinkers.

In order to assess pre-injury alcohol consumption, a pre-injury version of the AUDIT questionnaire was developed (see Appendix F). The wording in questions was amended to ask about alcohol consumption before the injury. For example, participants were asked 'how often did you consume alcohol?', rather than 'how often do you consume alcohol?'. In addition, some of the questions on

the AUDIT provide the participant with a choice of three answers 'No' (scoring 0), 'Yes, but not within the last year' (scoring 1) and ' Yes, within the last year' (scoring 2). These options were altered to 'No' (scoring 0) and 'Yes' (scoring 2).

Mood. The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) was used as a brief assessment of current mood state. This assessment has been shown to have good internal consistency; Cronbach's alpha of .89 for both the anxiety and depression subscales, and significant, $r = .72$, test-retest reliability for both the anxiety and depression subscales (Savard, Laberge, Gauthier, Ivers, & Bergeron, 1998). The British Society for Rehabilitation Medicine has recommended the use of the HADS within rehabilitation fields (BSRM, 2005). The scale was designed for use within hospital settings with medically ill individuals, although it is recognised that more work is needed to gain information on validity and reliability with brain injury (BSRM, 2005). The advantage of the HADS is that it is quick to administer and score, overcoming the bias of somatic complaints found in many other inventories (BSRM, 2005). The HADS has been shown to be sensitive to change in individuals following subarachnoid haemorrhage (Jarvis & Talbot, 2004).

Procedure

Ethics Committee approval from the local NHS REC was obtained for this study (see Appendix A). Following approval, invitation letters were sent out to 97 prospective participants. Thirty-eight (39%) replied and the researcher contacted each participant to arrange a mutually convenient time to complete the assessments. The assessment session lasted up to one and a half hours and all participants were reimbursed for any travel costs.

Participants received an information sheet and signed a consent form at the start of the session (see Appendix B). Following this, the cognitive assessment was conducted, starting with the memory assessment using the Logical Memory subtest from the WMS–III (Wechsler, 1997). The Rule Shift Cards, Zoo Map and Six Elements tests from the BADS were then administered between immediate and delayed recall of the short stories. Any remaining time was filled with questions about the participant's injury. Participants with scores two standard deviations below the mean for their age group were thanked for their participation and informed that the session was complete. They were informed that, because of the difficulties that they had with the memory assessment, some of the questionnaires might be stressful for them to complete, due to their reliance on memory. The AUDIT and pre-injury drinking questionnaire were administered within a semi-structured interview format (see Appendix D). For ease of administration and to encourage the participant to reflect on any changes in their drinking, the AUDIT and pre-injury drinking questionnaire were presented alongside each other. For example, participants were asked how often they would have a drink containing alcohol now, and then asked how often they would have had a drink containing alcohol before their injury.

During the interview, participants were encouraged to talk freely about their injury, and their perceptions about alcohol. Any comments regarding changes in the effect of alcohol were noted down (see Appendix E). On completion of the assessment, participants were asked for consent to contact a family member or close friend who knew them both before and after the injury, in order to complete an informant version of the BICRO-39 and the pre and post-injury AUDIT scales. Following this, a consent form and information sheets were sent to the informant

(see Appendix C), requesting a contact number, if they were willing to participate in the study. Upon receipt of a signed consent form, the informant was contacted by telephone and the three questionnaires (BICRO-39, Pre-injury drinking & post-injury AUDIT) were administered over the telephone.

Analysis

One-sample Kolmogorov-Smirnov tests were used to check for normality. Correlations were conducted using Pearson's Product Moment and Spearman's Rho, to investigate whether there were any significant relationship between the independent variables and post-injury AUDIT. Comparisons between those classified 'At risk' and 'Not at risk' on their post-injury AUDIT responses were conducted using t-tests and Mann Whitney U tests. Hierarchical logistic regression analysis was used to investigate the prediction of post-injury AUDIT group ('At risk'/'Not at risk'). Where data were strongly skewed, log transformation was carried out in order that the variables could be entered into the regression model.

Results

Thirty-eight participants were recruited. Three (7%) participants were excluded following poor memory test performance. One was excluded as the length of their hospital stay was more than three standard deviations from the mean of all the other participants, at > 1000 days. Of the 34 remaining participants, 28 (82%) were male and 6 (18%) were female. Examination of participants' age at the time of the injury was carried out to ascertain how representative the sample was of the brain injury population. Although the population in the area where the study was conducted was reflective of the

general population (Dorset Health Commission, 1993), the study sample may not be reflective of Local and National figures for brain injury. The study group showed a peak in the numbers acquiring brain injury between 31 and 40 years of age, compared with a peak seen in the National population for injuries between the ages of 21-30 years (Dorset Health Commission, 1993).

Men are considered more at risk of alcohol use than women. In this sample, there was a greater number of males (n = 28) than females (n = 6). However, there were no significant differences between male and female participants in their age at assessment, $t(32) = 0.78$, $p = .441$, age at injury, $t(32) = 0.09$, $p = .926$, time since injury, $t(32) = .58$, $p = .569$, anxiety, $t(32) = -1.47$, $p = .151$, depression, $t(32) = -1.37$, $p = .181$, or pre-injury AUDIT $t(32) = -0.62$, $p = .539$, scores. In addition, there were no significant differences in length of stay in hospital ($U = 39.5$, $N1 = 28$, $N2 = 6$, p = .150) or time unconscious ($U = 33$, $N1 = 28$, $N2 = 6$, p = .074), see Table 1. Subsequent analysis was conducted looking at the group as a whole.

Twenty two (65%) participants were involved in a road traffic accident (RTA). An RTA is defined as any injury that involves a motor vehicle, including situations when the participant was a pedestrian knocked down by a vehicle. Five (15%) of the participants were assaulted, of whom 3 (60%) admitted that alcohol had been consumed at the time of the assault. One (3%) participant was unable to identify the cause of injury. The remaining 6 (17%) received a rapid onset brain injury from other sources, such as infection.

Table 1.

Descriptive Characteristics for Male and Female Participants

	Male			Female		
	M	SD	range	M	SD	range
Age at Assessment (years)	45.2	12.5	19-65	40.8	11.9	24-54
Age at Injury (years)	37.5	12.8	18-61	37	11.4	22-50
Time since Injury (years)	7.6	8.6	1-30	3.8	2.7	2-9
Hospital Stay (days)	60.6	107.7	0-504	13.2	17.7	1-41
Time Unconscious (hours)	136.7	208.3	0-672	4.2	9.4	0-21
Anxiety (HADS score)	7.5	4.4	0-18	10.5	5.2	2-17
Depression (HADS score)	5.6	4.6	0-16	8.5	5.3	0-15

Note HADS = Hospital Anxiety and Depression Scale

Medical records were accessed for all participants who provided consent (*n* = 31, 91%) unfortunately, not all participants had a Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974) or length of post traumatic amnesia recording (Ponsford et al., 2004). In addition, it was not possible to obtain scan results as some of the participants were treated, during the acute phase of recovery, in other areas. Length of unconsciousness can be used as a measure of severity (King & Tyerman, 2003), but a number of participants (n = 5, 15%) reported that they were sedated whilst in hospital and some data were missing (n = 3, 9%). Thus, to

explore severity, participants were grouped according to length of hospital stay, which can be an indication of severity of injury (Dorset Health Commission, 1993). In this sample, 21 (62%) participants stayed in hospital for more than ten days. The fact that 14 (41%) participants were in hospital for less than ten days may explain why a large number of participants had no record of a GCS score.

Pre-injury alcohol use scores (pre-injury AUDIT), as rated by participants, ranged from 1 to 17 (n = 34, M = 7.2, SD = 4.4), whilst ratings for carer/relative pre-injury drinking ranged from 1 to 12 (n = 12, M = 5.3, SD = 3.4). Participants' ratings of post-injury drinking (post-injury AUDIT) ranged from 0 to 15 (n = 34, M = 6.4, SD = 5.0) with carer/relative ratings ranging from 0 to 14 (n = 12, M = 5.8, SD = 4.7).

Participants were divided according to the cut off (at risk $\geq$8) on the participant post-injury AUDIT into those 'At risk' and 'Not at risk' of alcohol dependency. There were more participants within the 'Not at risk' category both pre-injury (n = 21, 62%) and post-injury (n = 21, 62%).

There were no significant differences between the means for participants grouped by AUDIT as 'At risk' and 'Not at risk', in terms of age at injury, $t(32)$ = 0.90, p = .378, age at assessment, $t(32)$ = -0.76, p = .454, anxiety, $t(32)$ = -0.97, p = .342 or depression scores, $t(32)$ = -0.03, p = .973. In addition, no significant differences were found for time unconscious (U = 95, $N1$ = 13, $N2$ = 21, p = .687), or length of stay in hospital (U = 109, $N1$ = 13, $N2$ = 21, p = .555). There was a significant difference for time since injury, $t(32)$ = -2.20, p = .040, see Table 2. Those falling into the 'At risk' category had experienced their injury a greater amount of time in the past.

Using Fishers Exact Test indicated that there was no significant difference in distribution of male and female participants according to risk category, (1) = 0.43, $p = .653$.

Table 2.

Descriptive characteristics for those classified as At Risk and Not at Risk according to scores from the post-injury AUDIT

	At Risk			Not at Risk		
	M	*SD*	range	*M*	*SD*	range
Age at Assessment (years)	46.5	10.8	20-61	43.1	13.3	19-65
Age at Injury (years)	35	12.6	18-55	39	12.4	18-61
Time since Injury (years)	11.5	10.4	1-30	4.2	4.3	1-20
Hospital Stay (days)	46.5	79.5	1-280	57.3	112.9	0-504
Time Unconscious (hours)	119.1	237.5	0-672	113.3	176.9	0-504
Anxiety (HADS score)	9	4.5	2-17	7.4	4.7	0-18
Depression (HADS score)	6.2	5	1-16	6.1	4.7	0-14

Note. At Risk = AUDIT ≥8; HADS = Hospital Anxiety and Depression Scale

Concordance of Participant and Informant Data

Where consent was obtained, relatives or close friends/carers were approached to complete the AUDIT (pre- and post-injury) questionnaires. Three (9%) participants declined, 4 (12%) could not identify someone who knew them well enough, leaving 27 (79%) who consented. Of these, responses were received

from 12 (44%) of informants approached. Comparison of these data with those of the corresponding participant revealed significant correlations for both pre-injury, $r(12) = .64$, $p = .025$, and post-injury alcohol use, $r(12) = .83$, $p = .001$. When comparing categorisation of risk status ('At risk' or 'Not at risk'), participant data were 87.5% sensitive and 75.0% specific in predicting carers/informant categorisations. Sensitivity for post-injury risk status was 75.0% and specificity was 100%.

Taken together, these results suggest that participant data provided good estimates of alcohol use levels by comparison with carer scores both pre and post-injury. Scores for participants and carers' data on the BICRO-39 were also significantly correlated $r(12) = .60$, $p = .040$. Further analysis was conducted using participant data alone, for which there was a larger sample available.

Question 1: Is pre-injury alcohol use correlated with post-injury alcohol use?

There was a significant positive relationship between pre- and post-injury AUDIT scores, $r(34) = .49$, $p = .003$, suggesting that post-injury drinking may be partly related to pre-injury drinking levels.

Question 2: Are cognitive flexibility, and planning skills associated with post-injury alcohol use?

The relationship between planning skills (Six Elements and Zoo Map tasks), cognitive flexibility (Rule Shift Card task) and post-injury alcohol use was investigated. No significant relationship was found between post-injury alcohol use (AUDIT) and performance on the Six Elements, $r(34) = .30$, $p = .086$, although there was a trend towards a significant relationship. No significant relationship was found with the Zoo Map, $r(34) = .09$, $p = .634$.

Performance on the Rule Shift Card task was significantly correlated with post-injury alcohol use, $r(34) = .38, p = .028$.

Question 3: Are levels of post-injury productivity associated with post-injury alcohol use?

A significant negative correlation was found between post-injury alcohol (AUDIT) use and post-injury productivity scores (BICRO-39 productivity subscale), $r(34) = -.34, p = .048$. This suggests that as current (i.e. post-injury) productivity increased, (i.e. participants were engaged in more productive hours) post-injury alcohol use also increased.

Question 4: Is post-injury alcohol use associated with the length of time since the injury?

A significant relationship was found between time since injury and participant post-injury AUDIT scores $r(34) = .34, p = .048$. This indicates that as time since injury increased so did post-injury AUDIT scores.

Question 5: Can post-injury alcohol be predicted from pre-injury alcohol use, executive function measures, productivity or time since injury?

Pre-injury alcohol use, post-injury productivity, cognitive flexibility and time since injury were found to have significant associations with post-injury alcohol use levels. However, prediction of whether an individual is 'At risk' or 'Not at risk' is clinically more useful. Accordingly comparisons were made between the 'At risk' and 'Not at risk' groups determined from their post-injury AUDIT scores.

Comparison of those classified as 'At risk' and 'Not at risk' post-injury on their pre-injury AUDIT scores identified that there was a significant difference, $t(32) = -2.42, p = .022$. Comparison of scores revealed no significant differences

for the Rule-Shift Card task ($U = 114.5, N1 = 13, N2 = 21, p = .441$), or the Six Elements task ($U = 109, N1 = 13, N2 = 21, p = .344$). In addition, no differences were found in scores for pre-injury productivity ($U = 127, N1 = 13, N2 = 21, p = .753$) and post-injury productivity $t(32) = 1.27, p = .229$ (see Table 3).

Since group comparison of 'At risk' and 'Not at risk' participants revealed that there were no significant difference for post-injury productivity, planning skills or cognitive flexibility these variables were not included in subsequent regression analysis.

Table 3.

Comparison of scores for those 'At risk' and 'Not at risk' post injury.

	At Risk (n = 13)			Not at Risk (n = 21)		
	M	*SD*	range	*M*	*SD*	range
Rule Shift Card task (BADS)	3.38	0.65	2-4	3.05	1.02	0-4
Six Elements (BADS)	3.23	1.17	1-4	2.90	1.09	1-4
Pre-injury productive employment (BICRO-39)	12.31	3.04	7-15	12.95	3.31	7-20
Post-injury productive employment (BICRO-39)	14.38	4.21	7-20	15.90	3.02	9-20

Note. At Risk = AUDIT ≥ 8; BADS = Behavioural Assessment of the Dysexecutive Syndrome; BICRO-39 = Brain Injury Community Rehabilitation Outcome Scale.

Regression analysis was conducted in order to identify whether pre-injury AUDIT scores and time since injury were predictive of 'At risk' or 'Not at risk' post-injury alcohol use measured on the AUDIT. Hierarchical logistic regression

analysis was selected as it allows for a categorical dependent variable. This was considered more helpful from a clinical perspective where the aim would be to identify those likely to be at risk of alcohol misuse post-injury. Previous research has indicated that pre-injury drinking is associated with post-injury drinking; thus pre-injury AUDIT scores were entered into the regression analysis as the first continuous predictor. Apart from the suggestion that drinking has a tendency to return to pre-morbid levels within a year post-injury (Corrigan, 1995), there has been no research investigating the relationship between time since the injury and post-injury alcohol use. Therefore, time since injury was entered as the next continuous predictor.

Pre-injury AUDIT scores were found to be a significant predictor of post-injury AUDIT risk, B(1) = 0.20, *Exp(B)* = 1.23, p = .035. Sensitivity was 38.5% in correctly identifying those currently at risk of alcohol abuse. The model demonstrated good specificity (85.7%), indicating that predictive accuracy for individuals who were 'Not at risk' was good; that is low levels of alcohol use before injury were a strong predictor of alcohol consumption within safe limits post-injury.

Adding time since injury to the model improved the predictive power, B(1) = 2.57, *Exp (B)* = 13.04, p = .015; R^2 change = .314. Along with increasing the predictive power of the model, the addition of this variable also resulted in improvement in sensitivity and specificity of the model, to 69.2% and 90.5% respectively. These results indicated that as time increases following injury those with high pre-injury alcohol use are at increased risk of being 'At risk' of alcohol abuse post-injury.

Anecdotal Information

During the course of the interview, many participants spoke spontaneously about their perceptions of changes in how alcohol affected them. The researcher made note of these comments. Given that participants were not interviewed using a formal qualitative interview process, full qualitative analysis was not felt to be appropriate. However, this anecdotal information is worthy of mention as it suggests that individuals perceive that alcohol affects them differently following acquired brain injury. Twenty participants (59%) commented that less alcohol was required to feel intoxicated post-injury compared to pre-injury. For example, 'nobody tells you that it [alcohol] works quicker following an injury' and 'alcohol seems to work differently now, and a lot quicker'. One participant highlighted the change in their relationship with alcohol saying 'alcohol has a very different effect now, I don't enjoy it like before, it's more like an enemy than a friend', others said 'I don't really drink since my injury, as after one beer I feel very drunk' and 'I can't tolerate as much alcohol as before'.

Discussion

This study aimed to investigate the relationship between post-injury drinking and pre-injury drinking, post-injury productivity, cognitive flexibility, planning skills and length of time since the injury. The results of the present study are consistent with the findings from previous research, but they also raise new questions that would benefit from further investigation.

Concordance of Participant & Informant Data

A strong positive association was identified between participants' reports of pre- and post-injury drinking with those of a chosen informant. High sensitivity

and specificity were found for participant ratings of both pre- and post-injury drinking. This significant relationship between participant and informant data has been demonstrated in previous research (Sander et al., 1997) confirming that individuals can provide accurate information regarding their drinking behaviour, if they are asked appropriate questions.

Question 1: Is pre-injury alcohol use correlated with post-injury alcohol use?

A significant positive correlation was shown between pre-injury and post-injury drinking. This suggests that individuals who reported high AUDIT scores for pre-injury were more likely to report high AUDIT scores for post-injury alcohol use. This adds support to previous research showing a strong association between pre- and post-injury drinking (e.g. Taylor et al., 2003; Bogner et al., 1997; Edna, 1982; Kreutzer et al., 1991; Kreutzer et al., 1996). It is possible that this reflects a return to pre-morbid behaviours, however the association accounts for less than 50% of the variance, suggesting that other factors are also likely to contribute to risk of alcohol difficulties post injury.

Question 2: Are cognitive flexibility, and planning skills associated with post-injury alcohol use?

A significant relationship was found between an individual's cognitive flexibility, as measured by the Rule Shift Card task from the BADS and post-injury AUDIT scores. In addition, a trend towards significance was found with planning skills, as measured by the Six Elements test. Cognitive flexibility has been found to be predictive of those who relapse, in studies investigating addictive behaviours (Wolwer et al., 1997). The significant correlation suggests that cognitive flexibility may be a predictor of relapse for individuals following brain injury. However, when the data were examined looking for differences

between those classified, as 'At risk' and 'Not at risk' there were no significant differences in scores on the Rule Shift Card task and the Six Elements Test.

Question 3: Are levels of post-injury productivity associated with post-injury alcohol use?

A trend was identified toward those individuals who scored lower on the BICRO-39, consuming more alcohol. This suggests that those people who have a greater number of roles, in terms of paid employment, childcare, studying and training, may also report consuming more alcohol post-injury. However, as was seen with the cognitive skills, this effect was not demonstrated when analysis was conducted comparing the two groups by risk status. In a larger sample, the correlation or risk group difference might have proven significant. Certainly, greater levels of productivity might be likely to increase opportunities for alcohol consumption both socially and in terms of the finances to support it.

Question 4: Is post-injury alcohol use associated with the length of time since the injury?

Corrigan (1995) reported that drinking rates are likely to return to levels similar to pre-injury levels within a year of the acquired brain injury. The findings from this study show that the longer the time since injury, the greater the likelihood that individuals will score in the 'At risk' category for post-injury alcohol use. Importantly, those 'At risk' were, on average, 11 years post-injury. Rehabilitation services often provide support and advice for individuals in the early stages of recovery following a brain injury. This support is likely to reduce with time post injury, as the individual finds that they are able to return to employment and activities they engaged in before their injury. With reduced support and possible increased demands, alcohol may be used to help manage

stress. For those individuals who have successfully integrated back into employment and social activities post-injury, this increased socialisation may also provide more opportunities for the individual to consume alcohol. Therefore, support may need to be available for longer than the one or two years post injury.

Question 5: Can post-injury alcohol be predicted from pre-injury alcohol use, executive function measures, productivity or time since injury?

The results from the correlation analysis identified that pre-injury AUDIT scores, post-injury productivity scores, cognitive flexibility and time since injury were associated with post-injury drinking. The group comparisons identified that two of those variables also distinguished between those 'At risk' and 'Not at risk' of alcohol abuse post injury: pre-injury AUDIT scores and time since injury. Hierarchical logistic regression identified that both pre-injury drinking and time since the injury were significantly predictive of post-injury drinking risk. This suggests that, for those who previously used alcohol heavily, only a small number were at a low risk of alcohol abuse post-injury. However, even with these two variables combined, the model would misidentify around one third of people. Indeed the model was better at identifying those who were unlikely to have problems with alcohol use post-injury than those who were likely to have high risk. This demonstrates the complex nature of problematic drinking behaviour and why it will continue to be a challenge for those working in brain injury rehabilitation.

Anecdotal Information

Information provided by participants during the session indicates that there may be a change in tolerance, or at least in perceived tolerance, for alcohol following a brain injury. Research investigating the effects of alcohol on the

brain following injury is very limited. Alcohol is known to cause a reduction in processing speed, and cause individuals to become more aggressive as well as to have difficulties with problem solving (Ihara et al., 2000). If an individual has additional problems with processing speed and problem solving due to their injury, it may be that this reduced capacity results in them being more vulnerable to further loss of skills, which may be reflected in perceived loss of tolerance. An alternative view is that individuals who attribute their injury to alcohol consumption may be more aware of changes induced by alcohol consumption. In addition, there may be some psychological avoidance of alcohol that is justified by the reported reduced tolerance.

Methodological Considerations

Before discussing the clinical implications of this study, it is important to note any methodological considerations.

Administration of the alcohol use questionnaires. There is a possibility that asking participants to respond to each question by considering pre- and then post-injury alcohol use may have contaminated the answers provided by the participants. That is, participants may have answered both questions as if they were being asked about current drinking. It could be argued, however, that this method of administration encouraged participants to reflect on any changes in alcohol use, and reduced the risk of priming. That is, participants were helped to be aware, at all stages during questioning, that they were being asked to think about current drinking as well as about previous drinking. If the two questionnaires had been separated, there was a risk that participants might vaguely recall, due to their memory difficulties, having answered similar

questions before and repeated their previous responses. The anecdotal comments recorded during the study suggested that the method used helped to focus participants' on the distinctions between now and in the past. Many participants were acutely aware of differences in their experience of alcohol use. Furthermore, the high levels of sensitivity and specificity between the carer and participant data also suggest minimal contamination within the participant responses.

Modification of the AUDIT. It is possible that the adaptations of the AUDIT influenced the reliability and validity of the questionnaire. However, the wording of the questions was kept as close to the original, with only the time frame (within the last year vs. before your injury) being changed. With limited assessments available, especially for pre-injury use, and the time constraints of the study it was considered the most appropriate action. Assessment of test-retest reliability was beyond the scope of this study.

Insight deficits. No assessment was conducted to assess insight. Difficulties with insight may affect an individual's ability to report accurately on their drinking behaviour, causing them to either under- or over-estimate their alcohol use. However, good agreement between carers/relatives and participants would suggest that the participants were able to reflect reasonably accurately on their alcohol use. Indeed, in clinical practice, it is useful to gather information from an informant to be able to examine insight and there is no reason why this will not apply for gathering information about alcohol use.

Selection bias. There is often a selection bias in recruitment of participants from a clinical setting. The sample available consisted of individuals who had either been referred for support or who had actively sought support themselves

when noticing difficulties in everyday life following brain injury. As such, it is possible that those individuals who wanted to give something back to services responded positively to the invitation letter. It is also possible that individuals with very high post-injury drinking did not respond to the invitation letter, which may explain the high proportion of participants in the 'Not at risk' group. Thus, this sample may not be reflective of individuals who are drinking at harmful levels as well as having ABI. This sort of selection issue is not unique to this study.

Sample Size. Given the trends in some of the analyses found, additional significant relationships (for example with the Six Elements and Bicro-39) might have been found. Recruitment of health service populations such as those with brain injury or dementia is often difficult, recruitment of a brain injury population with alcohol problems even more so. A good number of participants were approached. Only a larger catchment and more time for the study could have offered the possibility of a larger sample.

Taking into account these considerations, the study still presents some interesting findings that may need to be considered within clinical settings.

Clinical Implications

Findings presented here offer further support to previous research that has shown it is possible to obtain reliable information on both pre-injury and post-injury drinking from the individual, without involving an informant (Sander et al., 1997). However, it will only be possible to gather such information if individuals are provided with the opportunity to share it. Clinicians need to be aware of

possible concerns about alcohol use post-injury and have the confidence not only to ask the question, but to be able to deal with any concerns.

Thinking about what information is needed post-injury to help clinicians work with individuals who may be at risk of alcohol abuse, it is clear that some form of assessment of pre-injury drinking habits will be helpful. This becomes more important for individuals who come to the attention of services at a later stage of their recovery, i.e. more than one-year post injury. Not only has pre-injury drinking been shown to have a strong association with post-injury drinking, it has also been identified, in this study, as a possible predictor for post-injury drinking risk. However, clinical experience indicates that not all rehabilitation units formally assess pre-injury or post-injury drinking. Yet, it is clear that high rates of drinking can have a significant impact on the rehabilitation process (Heinemann, 1986). This study adds further support to the idea that formal assessment of alcohol use needs to be encouraged.

In addition, the findings indicate that as time increases since the injury the individual is more likely to report a higher level of alcohol use. This is clinically important, as those scoring higher on the AUDIT were on average eleven years post-injury. This means that they were more likely to have completed treatment programmes within brain injury services and therefore, have reduced support from services. Thinking about this finding in a clinical setting, it could have an impact on service provision, especially follow-up procedures and contact after discharge.

Future Research

This study suggests that individuals may provide reliable information about how much they are drinking. However, it must be acknowledged that the drinking levels within this sample were more likely to result in individuals being classified as 'Not at risk' of alcohol abuse (60% pre-injury, 63% post-injury). In addition, a number of those falling within the 'At risk' category were scoring towards the low end of the range for 'At risk'. It would be useful to assess the reliability of information from participants who are exhibiting higher levels of drinking. It may be possible to reduce any potential sampling bias in this study related to drinking levels by recruiting across both addiction and brain injury services, thus increasing the chances of recruiting from a wider range of drinking levels. In addition, it may be important to screen within addictions services for signs of ABI. A study across the different services may also help to identify where there is a need for joint working, for example an individual who has suffered a severe ABI and is demonstrating alcohol dependence. Joint working can inform clinicians how best to work with such an individual.

The AUDIT is generally considered a screening assessment, with those individuals with high scores requiring further assessment. In order to help clinicians conduct the assessment, further research is needed to develop and validate assessment tools to help ensure that those individuals following brain injury who may be vulnerable to alcohol use post-injury are identified and appropriate rehabilitation is provided.

Time since injury was shown to be predictive in the regression model, producing questions for service development and delivery, especially regarding follow up after discharge. However, it is not possible to discern how increased

time since injury may lead to individuals becoming more vulnerable to risky alcohol use. It would be helpful clinically to be able to identify what is happening to create this increased risk. A number of possibilities require exploration including changing cognitive demands and changing levels of social support. If future studies were able to identify what factors were related to this increased vulnerability it could help inform clinical practice and rehabilitation to guide follow-up protocol and procedures.

Future research investigating the identified theme of perceived change in tolerance to alcohol could also clarify whether there is a physiological basis for these perceptions. A rigorous, qualitative study exploring the individuals' perceptions of the role that alcohol had in their injury and their perceptions of the effects of alcohol pre and post-injury would be important. Those who blame alcohol may also be more aware of how alcohol affects them post-injury. Alternatively, individuals whose injury resulted from alcohol consumption, may justify psychological avoidance of alcohol post-injury as a perceived reduction in tolerance of alcohol, particularly within a society where it is socially acceptable to drink to excess (Alcohol Concern, 2004). Alternatively, there may be a physiological reason for the perceived reduction in tolerance. If there is the possibility of physiological changes there may be implications in terms of sharing this information with individuals post-injury.

Conclusion

This study has provided some interesting and important information that may help to explain the role of a number of factors in relation to post-injury drinking in individuals following brain injury. In particular, the results indicate that

high levels of pre-injury alcohol use combined with increased length of time since the injury increase the risk of alcohol abuse post-injury. This study suggests possible implications for service provision, as well as providing a starting point for future research.

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Appendices

APPENDIX A

Our Ref: 71/03/B

1 September 2003

Dorset Local Research Ethics Committee (LREC)
Poole Hospital NHS Trust
Longfleet Road
Poole
Dorset
BH15 2JB

Tel: 01202 448 201
Fax: 01202 442 954

Miss Corinne Pearson,
Trainee Clinical Psychologist
Poole Community Health Clinic
Shaftesbury Road
Poole
Dorset BH15 2NT

Dear Miss Pearson

LREC 71/03/B
Alcohol Use and Brain Injury

The East Dorset Local Research Ethics Committee has considered the amendments submitted in response to the Committee's earlier review of your application on 31 July 2003 as set out in our letter dated 31 July 2003. The documents reviewed were as follows:

Letter dated 20 August 2003
Participant Information Sheet, Version 2 dated August 2003
Consent Form Version 2 dated August 2003
Participant Letter version 2 dated August 2003
Reply Slip, version 2 dated August 2003
Relatives Letter version 1 dated August 2003
Relative Information Sheet, version 1 dated August 2003
Consent Form version 2 dated August 2003
Section 1 with Clinical Directors signature

The members of the Committee present agreed that there is no objection on ethical grounds to the proposed study. I am therefore, happy to give you the favourable opinion of the Committee on the understanding that you will follow the conditions set out below:

- You do not recruit any research subjects within a research site unless favourable opinion has been obtained from the relevant East Dorset Local Research Ethics Committee.

- You do not undertake this research in an NHS organisation until the relevant NHS management approval has been gained as set out in the Framework for Research Governance in Health and Social Care.

- You do not deviate from, or make changes to, the protocol without prior written approval of the East Dorset Local Research Ethics Committee, except where this is necessary to eliminate immediate hazards to research participants or when the change involves only logistical or administrative aspects of the research. In such

cases the East Dorset Local Research Ethics Committee should be informed within seven days of the implementation of the change.

- You complete and return the standard progress report form to the East Dorset Local Research Ethics Committee one-year from the date on this letter and thereafter on an annual basis. This form should also be used to notify the East Dorset Local Research Ethics Committee when your research is completed and in this care should be sent to this REC within three months of completion.

- If you decided to terminate this research prematurely you send a report to this East Dorset Local Research Ethics Committee within 15 days, indicating the reason for the early termination.

- You advise the East Dorset Local Research Ethics Committee of any unusual or unexpected results that raise questions about the safety of the research.

The project must be started within three years of the date on which Dorset Local Research Ethics Committee approval is given.

Present at the meeting :

D Jones, Acting Chair	P Leigh	T Howard
M Leggett	F Cowdell	B J Waltho
D Tory	S Elliott	L A Wareing

In attendance: R Hanson, Administrator
M Smits - Observer

Yours sincerely

D JONES
VICE CHAIR
EAST DORSET LOCAL RESEARCH ETHICS COMMITTEE

APPENDIX B

Dorset HealthCare NHS
NHS Trust

POOLE COMMUNITY HEALTH CLINIC
PSYCHOLOGY SERVICE
Shaftesbury Road, Poole, Dorset, BH15 2NT
Telephone: (01202) 683363/684035 Fax: 684036

Community Brain Injury
Rehabilitation Service

Dear

My name is Corinne Pearson and I am a Trainee Clinical Psychologist conducting a research project with Dr Nick Moffat at the Community Brain Injury Rehabilitation Service in Poole. We are conducting a study exploring alcohol use and brain injury. The study involves answering a number of questions and completing a brief assessment. This study is being completed as part of the Doctoral Programme in Clinical Psychology at Southampton University.

You have been approached as a result of your contact with the Community Brain Injury Rehabilitation Service. Your decision to participate or not will have no effect on the current service that you receive. If you agree to participate it would involve attending a meeting, with myself, which will last between one and a half and two hours. This meeting will take place either at Poole Community Health Centre or at a venue in Dorchester; whichever is easiest for you. All the information that will be collected will be anonymous and confidential. You can withdraw from this study at any time without having to provide a reason.

If you have any questions regarding this study, or would like further information please do not hesitate to contact me at Poole Community Health Clinic on 01202 683363.

For the purpose of this study I intend to meet with 40 participants. If you are willing to participate I will contact you upon receipt of your reply, to inform you whether you are within the first 40 responses.

I look forward to hearing from you.

Yours sincerely

Corinne Pearson
Trainee Clinical Psychologist

Dorset HealthCare NHS
NHS Trust

POOLE COMMUNITY HEALTH CLINIC
PSYCHOLOGY SERVICE
Shaftesbury Road, Poole, Dorset, BH15 2NT
Telephone: (01202) 683363/684035 Fax: 684036

Community Brain Injury
Rehabilitation Service

ALCOHOL USE AND BRAIN INJURY – REPLY SLIP

Date: Name:

I am interested in taking part in the study exploring alcohol and brain injury YES [ ] NO [ ]

I can be contacted on (tel) [ ]* between the hours of [ ] and [ ]

* If you would rather call us to arrange an appointment please leave the telephone number blank

Dorset HealthCare NHS
NHS Trust

POOLE COMMUNITY HEALTH CLINIC
PSYCHOLOGY SERVICE
Shaftesbury Road, Poole, Dorset, BH15 2NT
Telephone: (01202) 683363/684035 Fax: 684036

Community Brain Injury
Rehabilitation Service

PARTICIPANT INFORMATION SHEET

ALCOHOL USE AND BRAIN INJURY?

You are being invited to participate in a research project investigating alcohol use and traumatic brain injury. It is important that you understand what is being carried out and what would be involved before making any decision about your participation. The information presented here should help you to understand the research project. Please read this information carefully and discuss it with others if you wish, before making a decision.

What is the purpose of the study?

This study aims to investigate alcohol use of individuals who have suffered a traumatic brain injury. It is hoped that the information obtained through this project will help to improve the services that are available for people who have experienced traumatic brain injury.

Why have I been chosen?

The study is being carried out in Dorset through the Neuropsychological Service based in Poole. You have been selected and invited to participate in this study as you have made use of the Neuropsychological Service. This information sheet has been sent to 120 people who have all accessed the Neuropsychological Service in Poole as a result of having experienced a traumatic bran injury.

Do I have to take part?

It is up to you to decide whether to take part or not. If you decide that you would like to participate in this study then you will be asked to sign a consent form. You can keep this information sheet and will be provided with a copy of your signed consent form. Even if you sign the consent form you are able to withdraw from the study at any time, without having to give a reason and without it affecting your medical care.

What will happen if I take part?

If you decided to participate Corinne Pearson will contact you to arrange a time and date to meet either at the Poole Community Health Clinic or a venue in Dorchester, whichever is easiest for you. This meeting will last between one and a half and two hours. Refreshments will be available.

During this meeting you will undertake a brief assessment looking at the impact of the traumatic brain injury. In addition a short interview will be conducted including questions about alcohol consumption, employment status, age at the time of your injury, medication and current age. You will also be asked for written permission to access your medical records to obtain information about the severity of your injury. At the end of the meeting you will also be asked for permission to contact a member of your family in order for the investigator to ask them some of the questions that you will have been asked. Any travel costs to attend the meeting at the Community Health Clinic will be reimbursed at public transport rates; details of mileage will be collected at the meeting.

What are the possible disadvantages and risks of taking part?

For some people talking about their injury and any changes that have occurred as a result of the injury can be distressing. A few people may also find it stressful completing the brief assessment. If at any time during the meeting you find it too distressing then the investigator will stop the questions/testing and the research meeting will come to a close.

What are the possible benefits of taking part?

It is hoped that the information we get from this study will help improve the service that we are able to offer to people who suffer a traumatic brain injury. You may be reassured to know that your participation in this study will not adversely affect or change the service that you currently receive or have received from the Neuropsychological service.

Will my taking part in this study be kept confidential?

Any information collected about you during the course of this study will be kept strictly confidential. Any information about you that is taken from your medical notes will have your name, address, and other personal details removed from it to ensure that you cannot be recognised from it.

As a Trainee Clinical Psychologist, my research is supervised by an NHS Clinical Psychologist (Dr Nick Moffat) and by Dr Romola Bucks, (Senior Lecturer at the University of Southampton). Both these supervisors will keep any information that is discussed in relation to the study entirely confidential.

What will happen to the results of the research study?

The results of the study will be written up as part of the course requirements for the Doctoral Programme in Clinical Psychology. None of the participants will be identifiable in any written report.

Who is organising and funding the research?

This study is being completed as part of the Doctoral Programme in Clinical Psychology at Southampton University.

Who has reviewed the study?

The East Dorset Local Research Ethics Committee has reviewed the study.

Thank you for your help with this study.

Contact Details

Corinne Pearson,

C/O Poole Community Health Clinic

Poole

Dorset

Tel: 01202 683363 e-mail: cp301@soton.ac.uk

Dorset HealthCare NHS
NHS Trust

POOLE COMMUNITY HEALTH CLINIC
PSYCHOLOGY SERVICE
Shaftesbury Road, Poole, Dorset, BH15 2NT
Telephone: (01202) 683363/684035 Fax: 684036

Community Brain Injury
Rehabilitation Service

Centre Number:
Study Number:
Participant Identification Number for this Study:

CONSENT FORM

Title of Project: Alcohol Use and Brain Injury

Name of Researcher: Corinne Pearson

1. I confirm that I have read and understand the information sheet dated..... (version....) for the above study and have had the opportunity to ask questions. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐
3. I understand that sections of my medical notes relating to my brain injury may be looked at by the researcher. I give permission for Corinne Pearson to have access to my records. ☐
4. I agree to take part in the above study. ☐

Name of Participant	Date	Signature
Researcher	Date	Signature

1 for participant; 1 for researcher

APPENDIX C

Dorset HealthCare NHS
NHS Trust

POOLE COMMUNITY HEALTH CLINIC
PSYCHOLOGY SERVICE
Shaftesbury Road, Poole, Dorset, BH15 2NT
Telephone: (01202) 683363/684035 Fax: 684036

Community Brain Injury
Rehabilitation Service

Dear

My name is Corinne Pearson and I am a Trainee Clinical Psychologist conducting a research project with Dr Nick Moffat at the Community Brain Injury Rehabilitation Service in Poole. We are conducting a study exploring alcohol use and brain injury. The study involves completing three brief questionnaires.

You have been approached following a meeting with who has attended a meeting with myself. They provided consent for me to contact you to see if you would be willing to complete three questionnaires that they have already seen. Your decision to participate or not will have no effect on the current service that your relative receives. If you agree to participate it would involve a phone call with myself, which will last approximately 15 minutes. This call will be conducted at a time that is convenient for you. All the information that will be collected will be anonymous and confidential. You can withdraw from this study at any time without having to provide a reason.

Enclosed is an information sheet about the study for you to keep, and a consent form that I would ask you to sign and return if you are willing to participate in this study. You will receive a copy of the consent form to keep for your records.

If you have any questions regarding this study, or would like further information please do not hesitate to contact me at Poole Community Health Centre on 01202 683363.

I Look forward to hearing from you.

Yours sincerely

Corinne Pearson
Trainee Clinical Psychologist

Dorset HealthCare NHS
NHS Trust

POOLE COMMUNITY HEALTH CLINIC
PSYCHOLOGY SERVICE
Shaftesbury Road, Poole, Dorset, BH15 2NT
Telephone: (01202) 683363/684035 Fax: 684036

Community Brain Injury
Rehabilitation Service

RELATIVE INFORMATION SHEET

ALCOHOL USE AND BRAIN INJURY?

You have been contacted following consent from who has volunteered to participate in a research project investigating alcohol use and traumatic brain injury. It is important that you understand what is being carried out and what would be involved before making any decision about your own participation. The information presented here should help you to understand the research project. Please read this information carefully and discuss it with others if you wish, before making a decision.

What is the purpose of the study?

This study aims to investigate alcohol use of individuals who have suffered a traumatic brain injury. It is hoped that the information obtained through this project will help to improve the services that are available for people who have experienced traumatic brain injury.

Why have I been chosen?

The study is being carried out in Dorset through the Community Brain Injury Service based in Poole. (Relative's name) has already consented to participate in this study and has attended a meeting with the researcher and provided your name and address as a family member that they would like us to contact in order to complete some questionnaires.

Do I have to take part?

It is up to you to decide whether to take part or not. If you decide that you would like to participate in this study then you will be asked to sign a consent form. You can keep this information sheet and one copy of your signed consent form. Even if you sign the consent form you are able to withdraw from the study at any time, without having to give a reason and without it affecting your medical care.

What will happen if I take part?

If you decide to participate Corinne Pearson will contact you by telephone to arrange a convenient time to complete 3 short questionnaires over the phone. The questionnaires are aimed at gathering information pertaining to your relative and their injury. The questionnaires will look at areas including alcohol consumption and employment status. All these questionnaires can be completed over the telephone and will take approximately 15 minutes to complete.

What are the possible disadvantages and risks of taking part?

For a few people it can be distressing to talk about the changes that they have noticed in their relative since their injury. If you find during the course of the telephone interview that it is too distressing to answer the questions then the researcher will be happy to end the call at that time and arrange another convenient time if you wanted to complete the questionnaires in the future.

What are the possible benefits of taking part?

It is hoped that the information we get from this study will help improve the service that we are able to offer to people who suffer a traumatic brain injury. You may be reassured to know that your participation in this study will not adversely affect or change the service that your relative currently receives or has received from the Community Brain Injury Service.

Will my taking part in this study be kept confidential?

Any information collected during the course of this study will be kept strictly confidential. Any information that you provide about your relative will have all personal details removed from it to ensure that your relative cannot be recognised from it.

As a Trainee Clinical Psychologist, my research is supervised by an NHS Clinical Psychologist (Dr Nick Moffat) and by Dr Romola Bucks, (Senior Lecturer at the University of Southampton). Both these supervisors will keep any information that is discussed in relation to the study entirely confidential.

What will happen to the results of the research study?

The results of the study will be written up as part of the course requirements for the Doctoral Programme in Clinical Psychology. None of the participants will be identifiable in any written report.

Who is organising and funding the research?

This study is being completed as part of the Doctoral Programme in Clinical Psychology at Southampton University.

Who has reviewed the study?

The East Dorset Local Research Ethics Committee for East Dorset has reviewed the study.

Thank you for your help with this study.

Contact Details

Corinne Pearson, Poole Community Health Clinic, Poole, Dorset.

Tel: 01202 683363 email: cp301@soton.ac.uk

Dorset HealthCare NHS
NHS Trust

POOLE COMMUNITY HEALTH CLINIC
PSYCHOLOGY SERVICE
Shaftesbury Road, Poole, Dorset, BH15 2NT
Telephone: (01202) 683363/684035 Fax: 684036

Community Brain Injury
Rehabilitation Service

Centre Number:
Study Number:
Participant Identification Number for this Study:

CONSENT FORM

Title of Project: Alcohol Use and Brain Injury

Name of Researcher: Corinne Pearson

1. I confirm that I have read and understand the information sheet dated..... (version....) for the above study and have had the opportunity to ask questions. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐
3. I agree to take part in the above study. ☐

The researcher, Corinne Pearson, can contact me on (tel:_____) between the hours_____ on these days_____

Name of Participant	Date	Signature
Researcher	Date	Signature

1 for participant; 1 for researcher

APPENDIX D

Interview Schedule

Thank you for coming along today. My name is Corinne Pearson and I am conducting in collaboration with Dr Nick Moffatt from the Neuropsychological Service in Poole and Dr Romola Bucks from the University of Southampton.

Talk through information sheet and ask them to sign consent form.

I also need to ask you if you would be willing for me to be able to access your medical notes from the time of your injury. I am interested in looking to see if there is a record of a particular scale used to measure how severe your injury was. This is called the Glasgow Coma Scale. If it was used at the time of your admission, a score will be recorded in your medical notes.

Some of the questions that I will be asking during the interview require you to think back to the time before your injury. To help ensure that this is not too difficult I would like to read a story to you that I will ask you to tell me back as much as you can remember once I have finished telling it.

Story immediate recall
I will ask you a little later on to tell me as much of that story as you can remember.

Filler Tasks:

Rule shift
Zoo map
Six Elements
HADS

Delayed recall of story

Before we begin the interview I'd like to ask you some more general questions about your health.

Firstly have you noticed any difficulties in remembering things that happened at the time of your injury or before your injury?

Do you notice any difficulties with your memory since the injury?

(If significant difficulty with assessment, scores fall below 2 SD from mean, and reported memory problems will be thanked for coming and informed that they can leave now, explaining that as they have reported difficulties with their memory and with the assessment they are likely to find it difficult to answer some of the questions that need to be asked. The investigator will then check if they have any questions and thank them again for their time, taking down any travel cost details.)

Have you ever received treatment for nervous or mental health problems?

How old were you when you had your injury?

How long did you stay in hospital for after the injury?

Were you in a coma at all after the injury? If yes, for how long?

Are you on any medications as a result of your injury, for instance epileptic medication? If yes, what type & dose?

There are often many changes that happen after suffering a TBI, I am interested in finding out about the impact that your injury has had.

Are you currently working? If yes, what job do you have? Is it part-time or full-time?

Did you work before your injury? If yes, what job did you have? Was it part-time or full-time?

What age did you leave school?

What qualifications have you got, e.g. GCSE, A Level, degree etc.?

Administer adapted AUDIT for pre-injury drinking levels
Administer BICRO-39 to assess levels of productivity (pre-injury)
Complete remainder of BADS assessment
Administer AUDIT for current drinking levels
Administer BICRO-39 to assess levels of productivity (post-injury)

I have asked you a lot of questions today about information from the past. We all have difficulty, at times, remembering everything accurately. I wonder if you would mind me contacting a member of your family or a close friend who may be able to help fill in any blanks for you.

If agreed then the investigator will ask them to sign the necessary consent form and provide the relevant contact details. If not they will be reassured that the information they have provided today will still be used for the purpose of the study.

The participant will be thanked for attending and provided with an opportunity to ask any questions about the study. The investigator will collect details about travel for reimbursement and the session will draw to a close.

APPENDIX E

Participant code:
M / F

Date of Assessment	
Date of Birth	
Date of injury	
Description of injury e.g. location/TBI/Stroke	
Length of hospital stay	
Coma? Length?	
GCS	

	Pre-injury	Post-injury	Relative
BICRO Productivity			
AUDIT			

BADS ZM - 6E - RSC -
HADS A – D –

Participant Comments:

APPENDIX F

ASSESSMENT OF PRE-INJURY ALCOHOL CONSUMPTION

Before your Injury:

1. How often did you have a drink containing alcohol?

Never **Monthly or less** **2-4 times/month** **2-3 times/week**

4+ times/week

2. How many drinks containing alcohol did you have on a typical day when you were drinking?

1-2 **3-4** **5-6** **7-9** **10+**

3. How often did you have 6 or more drinks on one occasion?

Never **Less than monthly** **Monthly** **Weekly**

Daily/Almost Daily

4. How often did you find that you were not able to stop drinking once you had started?

Never **Less than monthly** **Monthly** **Weekly**

Daily/Almost Daily

5. How often did you fail to do what was normally expected from you because of drinking?

Never **Less than monthly** **Monthly** **Weekly**

Daily/Almost Daily

6. How often did you need a drink first thing in the morning to get yourself going after a heavy drinking session?

Never **Less than monthly** **Monthly** **Weekly**

Daily/Almost Daily

7. Did you ever feel guilty about the amount you were drinking?

Yes **No**

8. Were you ever unable to remember what had happened the night before because you had been drinking?

Yes **No**

9. Were you or someone else ever injured as a result of your drinking?

Yes **No**

10. Did anyone express their concern about your drinking or suggested that you cut down?

Yes **No**

APPENDIX G

Instructions for Authors

Click here to check your article status

Note to Authors:** please make sure your contact address information is clearly visible on the **outside** of all packages you are sending to Editors.

Please note that the publisher would **actively encourage** authors to submit papers electronically to expedite the peer review process. Please email your paper saved in a standard document format type such as Word, Rich Text Format, or PDF to: reviews@psypress.co.uk

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Journal Production Editor: **kirsten.buchanan@psypress.co.uk**

FORMAT

Typescripts. The style and format of the typescripts should conform to the specifications given in the *Publication Manual of the American Psychological Association* (5th ed.). Typescripts should be **double spaced** on **one side** only of A4 paper, with adequate margins, and numbered throughout. The title page of an article should contain only:
(1) the title of the paper, the name(s) and address(es) of the author(s);
(2) a short title not exceeding 40 letters and spaces, which will be used for page headlines;
(3) name and address of the author to whom correspondence and proofs should be sent;
(4) your telephone, fax and e-mail numbers, as this helps speed of processing considerably.

Abstract. An abstract of 50-200 words should follow the title page on a separate sheet.

Headings. Indicate headings and subheadings for different sections of the paper clearly. Do not number headings.

Acknowledgements. These should be as brief as possible and typed on a separate sheet at the beginning of the text.

Permission to quote. Any direct quotation, regardless of length, must be accompanied by a reference citation that includes a page number. Any quote over six manuscript lines should have formal written permission to quote from the copyright owner. It is the author's responsibility to determine whether permission is required from the copyright owner and, if so, to obtain it. (See the bottom of the page for a template of a letter seeking copyright permission.)

Footnotes. These should be avoided unless absolutely necessary. Essential footnotes should be indicated by superscript figures in the text and collected on a separate sheet at the end of the manuscript.

Reference citations within the text. Use authors' last names, with the year of publication in parentheses after the last author's name, e.g., "Jones and Smith (1987)"; alternatively, "(Brown, 1982; Jones & Smith, 1987; White, Johnson, & Thomas, 1990)". On first citation of references with three to six authors, give all names in full, thereafter use first author "et al.". If more than one article by the same author(s) in the same year is cited, the letters a, b, c etc. should follow the year.

Reference list. A full list of references quoted in the text should be given at the end of the paper in alphabetical order of authors' surnames (or chronologically for a group of references by the same authors), commencing as a new sheet, typed double spaced. Titles of journals and books should be given in full, e.g.:

Books:

Baddeley, A. D. (1999). *Essentials of human memory*. Hove, UK: Psychology Press.

Chapter in an edited book:

> Plomin, R., & Dale, P. S. (2000). Genetics and early language development: A UK study of twins. In D. V. M. Bishop & L. B. Leonard (Eds.), *Speech and language impairments in children: Causes, characteristics, intervention and outcome* (pp. 35-51). Hove, UK: Psychology Press.

Journal article:

> Schwartz, M. F., & Hodgson, C. (2002). A new multiword naming deficit: Evidence and interpretation. *Cognitive Neuropsychology, 19*, 263-288.

Tables. These should be kept to the minimum. Each table should be typed double spaced on a separate sheet, giving the heading, e.g., "Table 2", in Arabic numerals, followed by the legend, followed by the table. Make sure that appropriate units are given. Instructions for placing the table should be given in parentheses in the text, e.g., "(Table 2 about here)".

Figures. Figures should only be used when essential. The same data should not be presented both as a figure and in a table. Where possible, related diagrams should be grouped together to form a single figure. Figures should be drawn to professional standards and it is recommended that the linear dimensions of figures be approximately twice those intended for the final printed version. Each of these should be on a separate page, not integrated with the text. Figures will be reproduced directly from originals supplied by the author(s). These must be of good quality, clearly and completely lettered. Make sure that axes of graphs are properly labelled, and that appropriate units are given. Photocopies will reproduce poorly, as will pale or broken originals. Dense tones should be avoided, and never combined with lettering. Half-tone figures should be clear, highly-contrasted black and white glossy prints.

Black and white figures are included free of charge. Colour figures are not normally acceptable for publication in print -- however, it may be possible both to **print** in black and white and to **publish online** in colour. Colour figures will only be printed by prior arrangement between the editor(s), publisher and author(s); and authors may be asked to share the costs of inclusion of such figures.

The figure captions should be typed in a separate section, headed, e.g., "Figure 2", in Arabic numerals. Instructions for placing the figure should be given in parentheses in the text, e.g., "(Figure 2 about here)". More detailed *Guidelines for the Preparation of Figure Artwork* are available from the publisher: Psychology Press Ltd, 27 Church Road, Hove, East Sussex BN3 2FA, UK (Email: **kirsten.buchanan@psypress.co.uk**).

Statistics. Results of statistical tests should be given in the following form:

"... results showed an effect of group, $F(2, 21) = 13.74$, $MSE = 451.98$, $p < .001$, but there was no effect of repeated trials, $F(5, 105) = 1.44$, $MSE = 17.70$, and no interaction, $F(10, 105) = 1.34$, $MSE = 17.70$."

Other tests should be reported in a similar manner to the above example of an *F* -ratio. For a fuller explanation of statistical presentation, see pages 136-147 of the *APA Publication Manual* (5th ed.). For guidelines on presenting statistical significance, see pages 24-25.

Abbreviations. Abbreviations that are specific to a particular manuscript or to a very specific area of research should be avoided, and authors will be asked to spell out in full any such abbreviations throughout the text. Standard abbreviations such as RT for reaction time, SOA for stimulus onset asynchrony or other standard abbreviations that will be readily understood by readers of the journal are acceptable. Experimental conditions should be named in full, except in tables and figures.

AFTER ACCEPTANCE OF PUBLICATION IN THE JOURNAL

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The following form of words can be used in seeking permission:

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>
> I/we are preparing for publication an article entitled
> [STATE TITLE]
> to be published by Psychology Press Ltd in ***Neuropsychological Rehabilitation.***
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