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Neuropsychological Performance, Acute Alcohol Intoxication and Aggression in Adult Males

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ABSTRACT

Epidemiological and laboratory research supports a relationship between acute alcohol intoxication and aggression. Recent data suggest that alcohol disrupts cognitive abilities associated with frontal lobe function. Moreover, neuropsychological research provides suggestive evidence that frontal lobe dysfunction may predispose an individual to increased aggression. The research recounted in this thesis was conducted to investigate the role of individual differences of cognitive abilities associated with the dorsolateral frontal cortex in aggressive behaviour and to test the hypothesis that alcohol indirectly potentiates aggression by impairing these cognitive abilities. The three experiments included in this thesis demonstrated that: (1) acute alcohol intoxication interferes with the ability to integrate previously acquired knowledge in the formulation of behavioral strategies; (2) individuals grouped according to performance on two neuropsychological tests of cognitive abilities associated with frontal lobe function differ in degree of aggressive response. Specifically, individuals in the lower versus upper performance quartiles became more aggressive when provoked; and (3) individuals in the upper cognitive performance quartile demonstrate significantly greater reductions of unprovoked aggression in response to monetary reward.

RÉSUMÉ

Les recherches épidémiologiques et de laboratoires supportent un lien entre l'intoxication aiguë à l'alcool et l'agression. Des données récentes suggèrent que l'alcool interfère avec les habilités cognitives associées avec les fonctions du lobe frontal. De plus, les recherches neuropsychologiques fournissent des évidences qui suggèrent que le dysfonctionnement du lobe frontal peut prédisposer un individu à plus d'agression. La recherche présentée dans cette thèse fut conduite afin d'étudier le rôle des différences individuelles au niveau des habilités cognitives associées au cortex frontal dorsolatéral dans le comportement agressif et afin de tester l'hypothèse que l'alcool indirectement potentialise l'agression en altérant ces habilités cognitives. Les trois expériences inclues dans cette thèse démontrent que: (1) l'intoxication aiguë à l'alcool interfère avec l'habilité d'intégrer des connaissances antérieurement acquises dans la formulation de stratégies comportementales; (2) les individus groupés selon la performance sur deux tests neuropsychologiques des habilités cognitives associées aux fonctions du lobe frontal diffèrent en degrés de réponses agressives. Plus spécifiquement, les individus dans le quartile de performance plus bas contrairement au plus haut deviennent plus agressifs lorsque provoqués; et (3) les individus dans le quartile de performance cognitive supérieur démontrent de façon significative plus de réductions d'agression non provoquée en réponse à une récompense monétaire.

NOTE REGARDING MANUSCRIPTS AND AUTHORSHIP

The McGill University Faculty of Graduate Studies and Research requires that item #3 of the "Guidelines Concerning Thesis Preparation" be cited in full in the introductory sections of any thesis to which it applies. In accordance with this requirement, item #3 is cited below:

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The thesis must still conform to all other requirements of the "Guidelines for Thesis Preparation". **The thesis must include:** A Table of Contents, an abstract in English and French, an introduction which clearly states the rationale and objectives of the study, a comprehensive review of the literature, a final conclusion and summary, and a thorough bibliography or reference list.

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PREFACE AND STATEMENT OF ORIGINALITY

This thesis presents information that is unique in a variety of ways. For each of the major studies contained in this dissertation, I will briefly outline my specific contributions, and those of the co-authors. For Study 1 (Lau & Pihl, 1994), I designed the study, performed the data scoring and wrote the final paper, under the direction of Dr. R.O. Pihl. I analyzed the data with the assistance of Dr. Jordan Peterson, a post-doctoral fellow at the time. Peter Ciancola and Robert Roth assisted in data collection. For Study 2 (Lau, Pihl, & Peterson, 1995), I designed the study, analyzed the data, and wrote the final paper under the direction and guidance of Dr. Pihl and Dr. Peterson. Again Peter Giancola and Robert Roth assisted in data collection. For Study 3 (Lau & Pihl, submitted), I designed the study, analyzed the data, and wrote the final paper, under Dr. Pihl's direction. Jean-Marc Assaad, Aviva Greenstein, Nathalie Morrissette, and Shanna Ross assisted in the data collection and scoring. Marc Gross and David Krinigan provided assistance with the data collection system for this study. Two of these studies have been previously published and one has been submitted for publication. While I did receive assistance in completing the research described in this dissertation, this assistance took the form of collaboration; the work described herein can accurately be considered an original, personal contribution to the literature in this area.

ACKNOWLEDGEMENTS

Dr. Robert O. Pihl supervised the research projects described in this dissertation. In addition, he provided the necessary financial resources from his grants and laboratory research space for the testing necessary for completion of the research described in this thesis.

I take this opportunity to express my sincere gratitude for his consistent support. I appreciated his style of supervision which allowed me a great deal of independence in my work, although he always made himself available for direction as needed. I continue to be impressed by his wealth of knowledge of the alcohol and aggression research and his analytical abilities, all of which were of tremendous benefit to me in producing the ideas presented in this thesis.

I would also like to thank Dr. Jordan Peterson who was a post doctoral fellow in the alcohol research laboratory during my time at McGill. I am grateful for his receptivity to discuss practical and theoretical issues with him as well as his solid support and I was impressed with his creative abilities. All of this greatly helped me in my graduate career.

I would like to thank the National Science and Engineering Research Council (NSERC) of Canada, and Fonds pour la Formation de Chercheurs et l'Aide a la Recherche (FCAR) of Quebec for financial support throughout my graduate studies.

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dissertation. In addition, I would like to thank Robert Roth, Peter Giancola, and Patricia Conrod, all research assistants in Dr. Pihl's laboratory at one point, for their assistance with co-ordinating laboratory time and equipment as well as providing a helping hand when needed. I also thank Judy Young, Dr. Pihl's secretary for her support and assistance.

Finally, I wish to express my deep appreciation to my parents who have consistently supported my ambitions in a variety of ways over the years. I thank my sister, brother, close friends, classmates, and fellow students in the alcohol research lab for their provision of support and entertainment throughout my stay at McGill.

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INTRODUCTION

Interpersonal violence presents a serious and growing challenge to North American society. In the United States, more than a million and a half violent crimes were reported in 1991 including more than one million assaults and 20,000 murders (U.S. Bureau of the Census, 1993). Alarmingly, the number of violent crimes has increased by 45% from 1982-91 (U.S. Bureau of the Census, 1993). Furthermore, the toll on public health and the economy that these crimes exacts is huge. Homicide is the second leading cause of death for Americans aged fifteen to thirty four (Hammett, Powell, O'Carroll, & Clanton, 1992). Moreover, violent crime has been estimated to cause 34 billion dollars in health related costs and lost productivity annually for the period from 1987-1990 (Miller, Cohen & Rossman, 1993).

Despite the magnitude of this problem, this carnage continues largely unabated. This failure derives in part from the ineffectiveness of the predominant response to violence, that is, the commitment of resources to deterring and incarcerating violent offenders (Reiss & Roth, 1993). Additional preventive methods are obviously required. Public health policy is now shifting to one that stresses the importance of focusing on the social, behavioral, and environmental factors that cause violence (Mercy, Rosenberg, Powell, Broome & Roper, 1993). As there is a growing body of evidence of an association between alcohol and violent crime, understanding how alcohol influences violence, or aggression, has become particularly germane.

LITERATURE REVIEW OF ALCOHOL AND CRIMINAL BEHAVIOUR

The evidence of a statistical association between alcohol use events and criminal behaviour derives from

three types of studies. These include 1) trend studies, 2) studies of alcoholics in prison populations and 3) individual coincidence estimates.

Trend Studies

Trend studies, which provide the least determinate evidence of an alcohol-violent crime association, examine per capita rates of alcohol consumption and crime longitudinally within and/or across populations. The evidence, limited primarily to Scandinavian studies, has shown a very high correlation between rates of alcohol consumption and violent crime (Lenke 1975, 1976; Lester, 1992).

A second type of analysis is based on data provided from periods where the availability of alcohol is manipulated by naturally occurring factors such as strikes by liquor store employees. Comparison studies of crime rates during periods of changed availability versus before and after these periods, have demonstrated that reductions in the availability of alcohol were associated with clear decreases in violent crime rates (Lenke, 1975; Takala, 1973). However, the possibility that some third variable was responsible for any covariation in the above studies could not be ruled out.

Studies of Alcoholism in Prison Populations

There are a variety of studies which reveal that alcoholics are over-represented among persons convicted of violent crimes (Pernanen, 1976). The rate of alcoholism among convicted felons in the U.S. has been reported to range from 17-48 per cent of cases (Goodwin, 1973; Guze, Tuason, Gatfield, Stewart, & Picken, 1962; Yarvis, 1972), which is much higher than a rate of 5-10% in the general population (American Medical Association, 1968). More recently, however, Collins & Schlenger

(1988) have shown that individuals diagnosed with alcohol abuse/dependence are not more likely to have committed a violent offense than those who did not meet the criteria for these diagnoses.

Individual Coincidence Estimates

The strongest evidence for an association between alcohol and violent crime, however, derives from individual coincidence studies of the co-occurrence of violent crime and alcohol consumption by the perpetrator, victim, or both.

Numerous epidemiological studies of criminality in Western society consistently demonstrate that alcohol intoxication and violence co-occur. Alcohol-intoxicated individuals are involved in a majority of violent crimes, including murders, assaults, sexual assaults and cases of family violence (Collins, 1981; Murdoch, Pihl, & Ross, 1990; Pernanen, 1976; 1981). Murdoch et al. (1990), in a review of 9,304 criminal cases from 26 studies drawn from 11 countries, concluded that over 50% of perpetrators of all homicides and assaults were drinking at the time of, or shortly before, commission of the crime. Alcohol intoxication has been similarly associated with events of forcible rape (Coid, 1986; Ladouceur & Temple, 1985) and spousal violence (Coleman & Strauss, 1983; Kantnor & Strauss, 1987; Leonard and Blane, 1992).

Despite these statistics, a specific relationship between alcohol and violent crime cannot be accurately assessed unless the association of alcohol with nonviolent crime in the same population is known. Of the few studies of the alcohol-crime relationship that provide comparison groups, the incidence of alcohol-related violent crime, ranging from 24-85%, contrasts dramatically with the range reported for alcohol-related

non-violent crimes: 12-35% (Murdoch et al., 1990). Thus, alcohol appears to be associated with violent crime at a higher level than with nonviolent crime.

Taken together, the findings of epidemiological studies comprise an overwhelming body of literature demonstrating a link between alcohol and violent crime. However, it is difficult to draw a firm conclusion about the nature of this association due to complications associated with these data including 1) methodological problems, 2) variations in the strength of this association and 3) the correlational nature of the data.

First, there a number of methodological problems which could affect the reported strength of the association (Evans, 1986; Murdoch et al., 1990). The majority of these problems derive from the tremendous reliance on police records. For example, the possibility that intoxicated individuals are more likely to be apprehended would result in inflated alcohol-crime statistics (Greenberg, 1981; Shupe, 1954). On the other hand, as it is an irregular occurrence for police to record physical measures of alcohol use, Pittman & Handy (1964) argue this could lead to an underrepresentation of the involvement of alcohol. In addition, there is an assumption that information regarding level of intoxication, contained in police reports, is accurate. However, this information is often based on the offenders' self-report which may be exaggerated as an excuse for his behaviour in order to minimize sentencing (Pernanen, 1976; Virkkunen, 1974). Furthermore, the information that does appear in police reports often lacks detail. Typically, police records contain merely a statement that the crime perpetrator was intoxicated. Shupe (1954), in an effort to avoid the problems inherent in relying on verbal reports by directly assessing inebriation from urine samples of individuals arrested

for a felony, found that violent offenders were often intoxicated well above the legal limit. While this finding lends credibility to studies relying on verbal reports of intoxication, individual differences in the time course of blood alcohol levels (BAL) and the fact that the relation between BAL and behavioral change is generally unknown (Brain, 1986) further complicate interpretations of the alcohol-crime relationship.

Second, the reported association between alcohol and violent crime is not invariant. Not everyone who drinks becomes aggressive (Heath, 1983), nor does this relationship necessarily hold across all cultures. While Horton (1943) reported that intoxication was associated with all types of aggressive behaviour ranging from verbal insult to homicide in almost every primitive society, MacAndrew and Edgarton (1969) reported wide cultural differences in the expression of aggression during alcohol intoxication. For example, settlement of disputes by individuals of the Abipone Indians, a warlike tribe living on the great plains of the Paraguayan Chaco, accomplished by verbal negotiation when sober, frequently led to fighting and slaughter when intoxicated (Martin & Dobrixhoffer, 1822). In contrast, individuals of the Yuruna Indians, a war-like, head-hunting tribe living in the Xingu region of South America's tropical forest, have been reported to withdraw and behave much as though no one else exists when they are intoxicated (Nimuendaju, 1948). While anthropological descriptions are not statistical surveys of the rate of intoxicated violence, the existence of inter-individual and inter-cultural variation suggests that any explanations for the alcohol-aggression relationship should not be limited to biological or pharmacological phenomena alone.

Third, and most importantly, epidemiological studies are correlational and do not permit one to discriminate

between theories implicating alcohol as a causal factor or as a covariate of some third factor such as the setting or the personalities of those who drink. For example, the theoretical implications of the relationship between alcohol and violent crime will vary depending on who has been drinking before commission of the crime (Murdoch et al., 1990). A causal hypothesis is supported if the offender alone has been drinking; however, if both the offender and the victim have been drinking, the issue becomes clouded.

A number of studies of homicide and assault cases report the presence of alcohol in the blood of high proportions of homicide victims (Abel, 1986; Goodman, Mercy, Loya, Rosenberg, Smith, Allen, Vargas & Kolts, 1986; Welte & Abel, 1989). For example, of 4,950 Los Angeles homicides in the period 1970-79, alcohol was detected in the blood 46% of the 4,092 victims tested (Goodman et al., 1986). A similar pattern has been reported for assault cases (Cherpitel, 1993; Shepherd, Irish, Scully & Leslie, 1989; Wasikhongo, 1976). The high percentage of both offender and victim intoxication implies that the alcohol-crime relationship may be artifactual describing only when and where the violence takes place (Murdoch et al., 1990).

Closer examination of these statistics, however, reveals two interesting facts that implicate a role of alcohol in the initiation of a violent altercation (Murdoch et al., 1990). First, it appears that it is the precipitator of the incident who is more likely to be intoxicated. While the incidence of victim or offender initiated altercations is approximately equal (Mayfield, 1976; Virkkunen, 1974), the victim has consumed alcohol in 60% of victim-precipitated homicides but in only 47% of offender-precipitated cases (Wolfgang & Strohm, 1956).

Second, the proportion of victims with detectable

blood alcohol varies notably by crime circumstance (Goodman et al., 1986; Welte & Abel, 1989). For example, Goodman et al. (1986) report that 52.5% of homicide victims killed during a physical fight were intoxicated versus only 19.8% of crime-related victims. Furthermore, assault is more frequently precipitated by quarrelling when alcohol is present (Pittman & Handy, 1964; Virkkunen, 1974). For example, of the quarrel-precipitated homicides described by Lindqvist (1986), 100% of the offenders were intoxicated at the time. Thus, it appears that the victim is more likely to be intoxicated when precipitating the altercation and that alcohol consumption along with a physical fight or verbal argument usually precede a violent act.

Although, these data are consistent with hypotheses that alcohol promotes aggressive behaviour and aversive interaction, the nature of the evidence prevents the establishment of a causal relationship. To test for causality, one must turn to the animal and human experimental literature.

EXPERIMENTAL RESEARCH ON ANIMALS

Experimental studies of alcohol's effects on animal aggression have been used extensively in an effort to shed light on the nature of alcohol-related violence in humans. This derives in part from the reduced ethical and methodological problems associated with animal studies. More importantly, however, as physiological processes characteristic of aggression may be common to both humans and other mammals, these processes may be seen in simpler forms in animals due to the lack of interference from social or psychological factors (Lagerspetz, 1981). Thus, animal studies might be expected to provide a clearer picture of the association between alcohol and aggression (Berry & Smoothy, 1986).

A major complicating factor in studying the effect of alcohol on animal aggression, however, is the striking heterogeneity of test methods used to measure this attribute (Brain 1981, 1989; Miczek, Weerts & DeBold, 1993). In rodents, for example, aggression has been incited by pairing pre-isolated males (intermale aggression); arranging for an unfamiliar intruder to enter the nest area of a lactating female with her offspring (maternal aggression) or providing the subject with the opportunity to kill prey (predatory aggression)(Brain, Miras & Berry, 1993). Thus, it is unlikely that the same motivation is measured across the various tests. Rather, these diverse harm-directed activities variously tap offensive, defensive or even predatory motivations (Brain, 1984).

One of the most important shifts in the behavioral analysis of aggression in animal models over the past dozen years has been toward ethoexperimental approaches (Blanchard, Brain, Blanchard, & Parmigiani, 1989; Miczek, Kruk, & Olivier, 1984). This method creates laboratory environments which emulate the requirements of the animal in the wild. More importantly, this method involves detailed inclusive analyses of behaviour which allows for the assessment of formal characteristics of the initiation, execution and termination of each behavioral act in space and time. This permits a cogent basis from which to determine the level at which alcohol may alter aggressive behaviour: by inhibiting motor activity, by distorting potentially aggression-provoking or -inhibiting signals, by fragmenting behavioral sequences, by shortening latencies to initiate aggressive acts, by lengthening aggressive bursts and causing failures to terminate, or by increasing the rate and intensity of aggressive acts (Miczek, Weerts, Haney & Tidey, 1994).

Studies of the impact of alcohol on animal

aggression, however, have failed to provide a consistent picture. Alcohol appears to exert differential effects in terms of either suppressing or stimulating aggressive behaviour (e.g. Ervin, Palmour, Young, Guzman-Flores & Juarez, 1990). Alcohol at high doses generally reduces fighting and attack behaviour due to its broadly sedative actions (Brain, 1986; Brain et al., 1993). In addition, moderate doses of alcohol can also have marked anti-aggression effects that are unassociated with obvious motor incapacitation (Berry & Smoothy, 1986).

In contrast, low doses of alcohol have been shown to increase aggressive behaviour but only under some conditions. Numerous studies in a diverse range of mammalian species including rodents, dogs, cats and primates demonstrate that alcohol, at selected low acute doses, increases aggression (see Berry and Smoothy, 1986 and Miczek et al., 1993, for reviews). In rodents, for example, this dose is about 0.3 to 1.0 g/kg. However, within this dose range, the response to alcohol appears to vary with particular subject characteristics. For example, moderately aggressive, but not highly aggressive or nonaggressive, male rats have shown increases in potentiation of attack toward smaller male intruders at doses under 0.5 g/kg alcohol (Blanchard, Hori, Blanchard, & Hall, 1987). Furthermore, alcohol's effects also appear to be mediated by behavioral subtypes including dominant, subordinate and fearful subjects (Berry & Smoothy, 1986; Winslow & Miczek, 1988), but not in a consistent manner. For example, while alcohol appears to manifest greater increases in aggression for subordinate Rhesus monkeys (Peretti & Lewis, 1969), large increases in aggression following acute alcohol doses (0.1, 0.3, 0.6 g/kg) have been reported in dominant, but not subordinate, socially housed squirrel monkeys (Winslow & Miczek, 1985).

While low doses of alcohol can enhance aggression, many studies fail to report such effects (Berry, 1993). In fact, under some conditions, acute alcohol doses in rodents and primates suppress aggression (Benton & Smoothy, 1984; Smoothy & Berry, 1983; Winslow & Miczek, 1985). Moreover, negative findings may be underrepresented in the literature as they are regarded as disappointing (Winslow, DeBold, & Miczek, 1987).

Taken together, these results suggest that alcohol's effects on animal aggression vary both across and within dose levels. Heterogeneity in test methods, however, does not entirely account for the equivocal results of the animal alcohol aggression literature. For example, within the same study, isolate aggressive mice showed increased aggressiveness at 0.3 g/kg but a decrease at 0.8 g/kg whereas isolate timid/defensive mice showed increases at 0.8 g/kg and sociable isolates showed no increase at any dose tested (Krsiak, 1977). This suggests that the increase in aggression by low alcohol doses is complex and that some additional factor(s), differentially involved in the various tests, mediate the alcohol aggression relationship.

One such factor may be an alcohol induced reduction in defensiveness, fear or anxiety. This notion is based on the patterning of alcohol induced aggression which suggests that this may be most apparent in tests in which defensiveness or anxiety act to reduce aggression. Support for this notion comes from rat studies which measure the effect of alcohol on four behaviours measured in the Anxiety/Defense Test Battery (e.g., Blanchard, Veniegas, Elloran, & Blanchard, 1993). This battery is a series of brief tests involving cat exposure which measure, for example, risk assessment behaviours and inhibition of nondefensive behaviours as well as a variety of locomotor and grooming activities. Alcohol

(0.6 and 1.2 g/kg) altered the four behaviours in a manner consistent with the effects of another anxiolytic, diazepam. Thus, one possibility is that alcohol increases the probability of aggression when an animal encounters provoking stimuli in a situation that also elicits anxiety or defensiveness. Conversely, alcohol would not be expected to release aggression when this aggression is not suppressed by anxiety. Thus, highly aggressive males would show no alcohol induced enhancement of attack when faced with opponents that do not elicit any notable degree of defensiveness.

A second mediating factor may be alcohol's effects on signalling and perception during social conflict. Brain et al. (1993) examined the possibility that alcohol increases the likelihood of aggressive victimization in mice. Mice, administered ethanol (0.5, 1.0 or 2.0 g/kg) or 0.9% saline solution, were introduced either to nonaggressive or aggressive mice. The alcohol-treated mice elicited increased probability of attack from aggressive responders, yet elicited increasingly tentative reactions from non-aggressive responders. The most likely explanation is that alcohol impaired the social signals that the injected mice traditionally employed to reduce attack. Thus, low doses of alcohol may increase aggression in part through its effects on cognitive-perceptual functioning.

In sum, despite an early pattern of seemingly inconsistent findings on alcohol and animal aggression, more recent analyses have begun to identify various determinants which mediate alcohol induced aggression. The impact, however, of this contribution to our current understanding of the alcohol aggression relationship in humans has been minimal.

EXPERIMENTAL RESEARCH ON HUMANS

Experimental studies of human aggression have yielded somewhat clearer results than the animal literature. Before beginning to address this subject, however, the definition and measurement of human aggression will be discussed as these issues are central to research in this area. The difficulty in defining aggression derives from the fact that the term is used to refer to a large variety of actions. Thus, one is faced with a serious and controversial issue, namely, how to define the concept in a meaningful and useful manner.

Aggression Definition

Aggression is a multidimensional concept where notions such as the intent of the aggressor, the form and target of the aggression and the motivation of the recipient to avoid the treatment are central to the definition. It is therefore not surprising that many definitions have been proposed (Berkowitz, 1974, 1981; Buss, 1961; Feshbach, 1970; Zillman, 1979) each of which would qualify distinctly different acts as aggressive. In spite of this controversy, some agreement among psychologists along with other social scientists exists with Baron's (Baron & Richardson, 1994) definition of aggression:

> **Aggression is any form of behaviour directed toward the goal of harming or injuring another living being who is motivated to avoid such treatment (p.7).**

This definition views aggression, not as an emotion, a motive, or an attitude, but as a form of behaviour.

Baron & Richardson (1994) highlight important facets of this definition which underlay some of the issues

involved in the concept of aggression. First, this definition deals with the issue of intent by limiting the definition to acts in which the aggressor intends to harm the victim. While there are difficulties involved in the determination of the presence or absence of intent, this criteria is necessary in order to rule out as aggressive accidental harm, or injury, to others as well as prosocial behaviour. In addition, if intent was excluded from the definition, failed attempts to harm or injure others would not be labelled as aggressive despite the fact that had the attempt been successful, serious injury to the victim would have occurred.

Second, specifying that aggression involves harm to the victim implies that the form of aggression is not restricted to acts resulting in physical damage. Rather it expands the range of acts to include those in which the individual has experienced some type of aversive consequence. Thus, causing others to experience public embarrassment or depriving them of needed objects would be considered aggressive.

Third, by restricting aggressive acts to those that harm living beings, this definition excludes destructive behaviours directed at inanimate objects unless this act indirectly harms an individual.

Finally, as the definition is restricted to behaviours directed at a recipient who is motivated to avoid such treatment, suicide or masochistic acts would not be defined as aggression.

Further classification of the endless variety of specific aggressive behaviours utilized by human beings has relied on the intent of the aggressor and the form of aggression. Early distinctions discriminated between hostile and instrumental aggression (Buss, 1961; Feshbach, 1970). The former is applied to aggressive behaviour where the primary or major goal is to cause the

victim to suffer. Instrumental aggression refers to assault on others primarily as a means of attaining other noninjurious goals rather than out of a strong desire to produce suffering. Bandura (1973), however, has argued that since both forms of aggression are directed toward the attainment of specific, albeit different, goals, they can both be labelled as instrumental.

In response to this criticism, alternative aggression dichotomies have been proposed (Dodge & Coie, 1987; Zillman, 1979). For example, Dodge & Coie (1987) have proposed the terms "reactive" and "proactive" aggression. The former involves retaliation against a perceived threat whereas the latter is behaviour, such as coercion, directed toward attaining a specific positive outcome. Empirical evidence (Dodge & Coie, 1987) supports the validity of this distinction. For example, reactive aggressive elementary school boys are likely to overinterpret peers' behaviours as being hostile resulting in an aggressive response. In contrast, proactive aggressive boys tend not to make the same interpretive errors of other's behaviour as reactive aggressive boys.

Alternatively, Buss (1961) has proposed a framework based on the form of the aggressive behaviour. This framework classifies aggressive acts along three dichotomized dimensions: physical-verbal, active-passive, and direct-indirect. Together, these dimensions yield eight possible categories within which to classify aggressive behaviours.

Aggression Measures

Given the diversity of aggression definitions, it is understandable that experimental alcohol-aggression research has operationalized this concept in a wide variety of ways. The variety of aggression measures

utilized can be categorized as consisting basically of two types. First, there are a variety of measures of non-aggressive behaviours that are assumed to be related to aggressive behaviour. These measures have included methods as diverse as the appreciation of aggressive humour (Hetherington & Wray, 1964), time spent looking at aggressive pictures (George & Marlatt, 1986; George, Dermen, & Nochajski, 1989) or the strength and frequency of power fantasies (McClelland, Davis, Kalin, & Wanner, 1972). As these measures do not conform to the above described aggression definition they will not be reviewed here (for review see Baron & Richardson, 1994; Gustafson, 1993; Pihl, 1983). The second group of measures are comprised of three types of active aggression measures: verbal-indirect or -direct, and physical-direct.

Verbal Aggression Measures

Verbal aggression is considered to be indirect when the victim is absent and is typically measured by means of a questionnaire (for review see Edmunds & Kendrick, 1980). Examples include the Buss-Durkee Hostility Inventory (Buss & Durkee, 1957), the Buss-Perry Aggression Questionnaire (Buss & Perry, 1992) and the Spielberger Anger-Expression Scale (Spielberger, Johnson, Russell, Crane, Jacobs, & Worden, 1985). The use of questionnaires has been criticized in part because correlations of these indirect measures with each other and with other measures have typically been low (Buss, 1961; Taylor, 1967).

Verbal aggression is considered direct when the victim is present and is measured by recording a subject's vocal comments during a social interaction (Boyatzis, 1974, 1975; Graham, LaRocque, Yetman, Ross & Guistra, 1980; Murdoch & Pihl, 1988; Murdoch, Pihl, & Ross, 1988; Takala, Pihkanen & Markkanen, 1957). These studies measure an actual harm-doing behaviour rather

than relying on the accuracy of the subject's memory.

In addition, when these studies are done in naturalist settings, the subjects are not aware they are in an experiment and presumably respond in a natural fashion. Thus, these studies offer the important advantage of providing ecological validity to laboratory research. However, there are ethical issues raised by naturalistic experiments. First, the subject's informed consent is impossible to obtain. Second, there is a danger of possible harm to participants and/or accomplices in studies where the accomplice instigates an altercation (e.g. Murdoch et al., 1988).

Physical Aggression Measures

While verbal aggression measures are a useful tool in alcohol-aggression research, what is really needed in a measure is a response that varies in intensity along quantifiable dimensions (Pihl, 1983). Direct measures of physical aggression fulfil this requirement. Most procedures are variations of the Buss (1961) teacher-learner paradigm (e.g., Gustafson, 1985a) or the Taylor (1967) competitive reaction-time task (e.g., Bond & Lader, 1986; Zeichner & Pihl, 1979), both of which provide a believable context for the administration of a quantifiable noxious stimuli, typically electric shock or uncomfortable sound blasts, to a fictitious opponent. More recently, Cherek (1981) developed an aggression paradigm based on a free-operant point subtraction procedure.

Buss Teacher-Learner Paradigm

Buss (1961) devised the first technique for directly investigating physical aggression. In the original paradigm, a subject and a confederate were told that the study was concerned with the effects of punishment on learning. The subject, assigned the teacher role, was instructed to punish the learner via the administration

of electric shocks of different intensity levels each time the learner committed an error on a discrimination task. The general use of this paradigm has been criticized because of artificiality and the potential demand of altruism (Baron & Eggleston, 1972; Shuck and Pisor, 1974). Because the situation was set up as a learning task and the fictitious partner was unable to retaliate, the partner could be viewed as helpless and the task as altruistic.

Taylor Competitive Reaction-Time Task

Taylor (1967) resolved these problems by refining the Buss paradigm so that both the subject and the fictitious opponent compete on the same reaction-time task. At the beginning of each trial, the subject chooses a level of shock he will administer to the opponent if he wins the reaction-time trial. At the end of each trial, the subject is informed of the shock level selected by the opponent. The loser of the trial then receives the indicated shock. This procedure yields three measures of aggression. Shock intensity is the intensity of shock chosen by the subject to be delivered to the opponent. The subject's first shock choice, which is made before he has received a shock or information regarding the intentions of the opponent, is considered a measure of unprovoked aggression (Hammock & Richardson, 1992). Shock choices during the remaining trials during which the subject is exposed to provocation is thought to be a measure of retaliative behaviour (Hammock & Richardson, 1992). Shock duration is the length of time the subject delivers a selected shock intensity to the opponent. Shock intensity and duration have been interpreted as measures of direct and indirect aggression, respectively (Rogers, 1983).

The Taylor (1967) task has since become the paradigm used most frequently to investigate the alcohol-

aggression relationship in part due to a number of important advantages. In contrast to the Buss paradigm, the opponent is not helpless and can retaliate, thus, providing a more realistic approximation of actual aggression. In addition, one can manipulate provocation of the subject and study the effects of more than one attack on aggressive responding (Bertilson, 1990).

The Taylor task has been shown to be a valid measure of aggression independent of alcohol research. This measure differentiates between groups theoretically expected to differ in aggressive potential including undercontrolled versus overcontrolled individuals (Taylor, 1967), psychopathic individuals (Dengerink, 1971), prejudiced persons (Genthner & Taylor, 1973), prison inmates (Wolfe & Baron, 1971) and subjects low in approval motivation (Taylor, 1970). In addition, it is sensitive to environmental factors theoretically expected to influence aggression (Borden & Taylor, 1973; Hendrick and Taylor, 1971; Pisano & Taylor, 1971). Lastly, there is direct support for convergent and discriminant validity of this task (Bernstein, Richardson, & Hammock, 1987; Giancola & Zeichner, 1994a).

Along with these important advantages, however, come some potential drawbacks. There are concerns related to the ethical questions of deception and the presumed delivery of shocks to the opponent along with the fact that subjects in this paradigm actually receive a series of uncomfortable electric shocks. Pihl, Zacchia & Zeichner (1981), however, report data suggesting that extensive debriefing of subjects following an experiment is sufficient to deal with these questions. In a follow up analysis of 78 subjects who had participated in alcohol experiments using the aggression paradigm, only 14 subjects reported that aspects of the experiment bothered them. Only 3 subjects were mildly distressed by

the deception, 4 by the administration of electric shocks and the remaining 7 were concerned with either boredom or the nature of the drinks. These concerns were neither severe nor long lasting.

In addition to the ethical concerns, there are three possible confounding influences present with the use of the Taylor task. First, as the paradigm is presented as a competitive reaction-time task, the subject's behaviour could be interpreted as competitive rather than aggressive. Examination of this issue in two studies revealed that neither competitive behaviour nor thinking about the task in competitive terms was related to shock-setting behaviour (Bernstein et al., 1987; Gaebelin & Taylor, 1971).

Second, it is unclear as to whether sober and intoxicated subjects experience the received shocks with equal levels of discomfort (Gustafson, 1993), particularly since intoxicated subjects calibrate the shock scale higher (Gustafson, 1985b; Jeavons & Taylor, 1985). Thus, intoxicated subjects may receive more intense shocks, both objectively and subjectively, and that their increased aggression may be a function of higher provocation rather than a function of alcohol. However, two studies measuring the subjective experience of the received shocks (Gustafson, 1985b; Gustafson & Kallmen, 1988) provided contradictory results. While the former study indicated that intoxicated subjects experienced the received shocks with more discomfort, the latter came to the opposite conclusion. In a third study, which controlled for differences in the calibration of the shock scale, alcohol still increased physical aggression (Gustafson, 1992).

Third, sober and intoxicated subjects of the Taylor paradigm have received different information regarding the state of intoxication of the opponent such that it is

implied that the confederate receives the same alcohol treatment as the real subject. This confounding factor has been shown to influence the results for men (Gustafson, 1986a) but not for women (Gustafson, 1986b). However, a study controlling for this confound (Gustafson, 1992) confirmed the consistent results of the Taylor paradigm.

Cherek Paradigm

Cherek (1981) developed a third, and different, type of paradigm to study human aggressive behaviour. This paradigm uses a free-operant point-subtraction procedure and includes the availability of non-aggressive, as well as, aggressive response options. The subject and a bogus partner have the choice to press one button to earn money or a second button to subtract money from their bogus partner. The subtraction of money from the subject by the partner is used as a form of provocation. The aggression measure consists of the withdrawal of money by the subject from, or the presentation of uncomfortable sounds to, the opponent. The third non-aggressive response alternative, namely, that of giving money to the opponent is also provided to the subject. This option permits the identification of any general excitatory drug actions such as nonspecific increases in button-pressing responses (Cherek, Steinberg & Manno, 1985).

Laboratory Investigations of Alcohol and Aggression

With the exception of a few published negative studies (Bennett, Buss, & Carpenter, 1969; Gustafson, 1984; Rohsenow & Bacharowski, 1984), many studies have demonstrated that subjects consuming more than a very little alcohol are more aggressive than those not given alcohol (Bond & Lader, 1986; Cherek et al., 1985; Cherek, Steinberg & Vines, 1984; Gustafson, 1985a; Pihl, Smith & Farrell, 1984; Richardson, 1981; Shuntich & Taylor,

1972; Taylor & Gammon, 1975; Taylor, Gammon & Capasso, 1976; Zeichner & Pihl, 1979, 1980). High doses of alcohol (1.32 ml 100% alcohol/kg body weight) have typically led to increased aggression in provoked subjects (e.g. Pihl, 1983; Taylor & Leonard, 1983). Doses of alcohol as low as 0.23 ml/kg (Cherek et al., 1985), 0.30 ml/kg (Bond & Lader, 1986) and 0.60 ml/kg (Gustafson, 1985a), levels well below that usually defined as intoxicating, have also been shown to enhance aggression under provocative conditions. Furthermore, aggression is related to the quantity of alcohol ingested (Taylor & Chermack, 1993; Taylor & Gammon, 1975; Taylor, Vardaris, Rawich, Gammon, Cranston, & Lubetkin, 1976).

Moreover, both descriptive summaries and meta-analytic reviews have concluded that alcohol does indeed cause aggression (Bushman & Cooper, 1990; Pihl & Ross, 1987; Taylor & Leonard, 1983). Furthermore, external validity for these findings are provided in studies of individuals in party situations (Boyatzis, 1974, 1975) and in bars (Murdoch & Pihl, 1988).

While these studies suggest that alcohol facilitates the expression of aggressive behaviour, it could be argued that any intoxicant, regardless of its specific pharmacological properties, might increase aggression. This view suggests that it is the altered psychological state that facilitates aggression, and not the particular substance producing the intoxication.

A number of studies have been conducted to explore this issue. For example, Taylor, et al. (1976) compared the shock setting behaviour on the Taylor task of subjects who had ingested high or low doses of alcohol (1.5 vs. 0.5 oz per 40 lbs body weight) or delta-9-tetrahydrocannabinol (THC; 0.3 vs 0.1 mg/kg), the primary psychoactive ingredient in marihuana. Not surprisingly, subjects in the high-dose alcohol condition set

significantly higher shock levels than those in the low-dose condition. In contrast, relative to the low-dose condition, the high dose of THC tended to suppress aggression. These results have been replicated (Myerscough & Taylor, 1985) and are consistent with the literature concerning marihuana and violence (Tinklenberg, 1973). Thus, increased aggression does not appear to be an inevitable result of any intoxicated state.

While the considerable epidemiological and human laboratory evidence consistently reports a relationship between acute alcohol use and increased aggression, any relationship is necessarily complex (Brain, 1986; Pihl, Peterson, & Lau, 1993). This is largely due to ample evidence of a dissociation between alcohol consumption and aggressive behaviour. For example, roughly half of all violent crimes occur in the absence of alcohol use (Murdoch et al. 1990), and substantial amounts of alcohol are consumed in social settings without increases in aggressive behaviour (MacAndrew & Edgerton, 1969). Finally, individuals with histories of becoming violent when intoxicated do not always get aggressive when drunk (Heath, 1983).

A number of parameters including alcohol-related, contextual, and individual factors have been identified which moderate the alcohol aggression relationship in humans. As an understanding of the impact of these factors is essential to evaluating explanations of the alcohol-aggression relationship, these factors are reviewed below.

Pharmacological variables

Alcohol-related factors, that is dose and type of alcohol, significantly affect the expression of aggression. The effect of alcohol dose has been examined in several studies. Studies that employed a between-

subjects design typically have demonstrated that individuals in high dose conditions selected higher shock levels (e.g. Taylor & Gammon, 1975; Taylor et al., 1976). For example, Taylor & Gammon (1975) administered .5 oz or 1.5 oz of 100 proof spirits per 40 lbs. of body weight to male subjects. In comparison to subjects who had not consumed alcohol, the results indicated that the high dose of alcohol instigated, whereas the low dose inhibited, aggressive responding. Thus, aggressive responding appears to be related to the quantity of alcohol consumed at doses up to 1.32 ml/kg of 100% alcohol.

Not all studies, however, have replicated this effect. For example, Bushman & Cooper (1990) concluded from an analysis of four studies using within-subjects manipulations of alcohol dose that the dose effect was not significant. Two studies, however, (Bennett et al, 1969; Gustafson, 1984) used the Buss paradigm as the dependent measure, the use of which has been criticized (Baron & Eggleston, 1972; Shuck & Pisor, 1974). Moreover, the two other studies (Cherek et al., 1985; Cherek et al., 1984) may have failed to demonstrate dose related effects due to a lack of statistical power as only 15 subjects were assessed. Finally, no study controlled for the possible confounding influences of order effects of administration of dose. Thus, methodological problems associated with these studies may explain the failure to demonstrate a dose effect of alcohol on aggressive responding.

Intoxicated aggression also appears to vary as a function of the type of beverage. Greater verbal (Boyatzis, 1974; Takala, Pihkanen, & Markkanen, 1957) and physical (Gustafson, 1988; Murdoch & Pihl, 1988; Pihl et al., 1984) aggression is elicited following consumption of distilled spirits rather than beer. In addition,

vodka elicits greater aggression than bourbon (Taylor & Gammon, 1975; Taylor et al., 1976). Finally, wine has been reported not to increase aggression, although this may have been due to low BAL's (Gustafson, 1990).

The effect of beverage type has been attributed to the different expectancies associated with different types of alcoholic beverages (Lindeman & Lang, 1986; Pihl et al., 1984) and/or to the congener content of the beverage (Greenberg, 1970; Katkin, Hayes, Teger, & Prutt, 1970). Congeners are chemical substances other than ethanol frequently found in alcoholic beverages. Taylor & Leonard (1983) have suggested that higher congener content retards the absorption of ethanol resulting in lower BAL's at the time of testing. Thus, distilled spirits, with the lowest congener content in a standard drink (Greizerstein, 1981), would be absorbed faster leading to higher BAL's and increased aggressive responding.

Situational variables

Alcohol consumption does not invariably elicit aggression. Two situational variables that have proved to interact with alcohol in eliciting aggression are provocation and social pressure. Provocation increases the likelihood of aggression in general, and specifically appears to mediate the alcohol-aggression relationship (Gustafson, 1993; Kelly & Cherek, 1993; Taylor & Chermack, 1993). Evidence supporting the importance of provocation in the alcohol-aggression relationship has been provided by both experimental and naturalistic evidence.

In the absence of provocation, there is little experimental evidence that alcohol increases aggression. Initial studies with the Buss paradigm failed to demonstrate an effect of alcohol on aggression (Bennett et al., 1969; Gustafson, 1984). However, this early

design of the paradigm was faulted in that the subject was not provoked as the bogus partner could not retaliate (Baron & Eggleston, 1972; Shuck & Pisor, 1974). Shuntich and Taylor (1972), using a modified task (Taylor, 1967) that allowed retaliation by the bogus partner, demonstrated that intoxicated subjects behaved more aggressively than those who had consumed a placebo beverage.

One explanation for the discrepancy between the results reported by Bennett et al. (1969) and Shuntich & Taylor (1972) was the greater inherent threat in the latter paradigm. Taylor et al. (1976) specifically tested this hypothesis by having male subjects compete in the Taylor task against either a potentially threatening opponent or a nonthreatening opponent. Intoxicated subjects were more aggressive only in the threatening situation suggesting that alcohol interacts with threat to increase aggression. Intoxicated subjects have also been shown to be more responsive to intense provocation (Taylor, Schmutte, Leonard, & Cranston, 1975) and tend to exaggerate threat as compared to sober subjects (Schmutte, Leonard, & Taylor, 1979). These results generalize to the two other aggression paradigms. Modification of the Buss paradigm to incorporate frustration of the subject prior to the task has yielded results that intoxicated and frustrated subjects were more aggressive than sober frustrated subjects (Gustafson, 1985a). Furthermore, Kelly, Cherek, Steinberg, & Robinson (1988), using the Cherek paradigm, have demonstrated that alcohol facilitates aggressive responding only under provocative conditions.

Gustafson (1993) has argued, however, that alcohol increases aggressive responding under provocative conditions only in situations where the subject is restricted to an aggressive response alternative. When

a variable neutral response alternative is provided along with an aggressive response option alcohol does not increase aggressive responding (Gustafson, 1991a,b). However, there is some question as to whether the subjects in these studies were sufficiently provoked. Frustration constituted the form of provocation in these studies, yet, the subjects reported very low frustration levels and these levels were much lower than the frustration the subject thought others might experience in the same situation. This suggests that the subjects were, in fact, not frustrated. The importance of some form of provocation in the alcohol-aggression relationship and the repeated demonstration that alcohol does increase aggression when subjects are provided with an aggressive and non-aggressive response alternative (for review Kelly & Cherek, 1993) considerably weakens Gustafson's argument.

Provocation appears to be an important mediator of alcohol related violence in the real world as well. For example, real-life assault is more frequently precipitated by quarrelling when alcohol is present (Pittman & Handy, 1964; Virkkunen, 1974).

The corollary that the probability of aggression increases when both individuals in an interacting dyad are intoxicated is borne out in real life and in the lab. Violent crimes occur more often in situations where both the offender and the victim are intoxicated (Murdoch et al., 1990). In addition, intoxicated dyads select significantly higher levels of shock while participating in the Taylor task, while mixed dyads select intermediate shock levels (Leonard, 1984).

A second variable identified as a potent instigator of alcohol-induced aggression is social pressure. Taylor & Sears (1988) used social pressure to persuade subjects to administer a high magnitude shock, twice the value of

the opponent's unpleasantness threshold. Social pressure was applied by the presence of two confederate observers who attempted to convince the subjects to increase their shock settings. Under this condition, intoxicated subjects significantly increased their use of the high magnitude shock as compared to placebo subjects in spite of the fact that the opponent selected the lowest intensity shock for all trials. Thus, it appears that the social context as well as provocation modify alcohol's effects on aggressive behaviour.

Individual variables

While there has been a great deal of attention devoted to the influence of pharmacological and situational factors, little attention has been devoted to investigating the influence of personality or individual difference variables on the alcohol-aggression relationship in an experimental paradigm (Taylor & Chermack, 1993). However, one of the most consistent findings of research in this area is the tremendous amount of intersubject variability (Pihl, 1983). Thus, one of the most important questions in this area may in fact be delineating who will respond aggressively when intoxicated and why.

Individual difference variables that have been investigated include aggressive predisposition, drinking experience and sex of the consumer. Studies specifically examining the role of aggressive disposition in the alcohol-aggression relationship have consistently demonstrated that a predisposition or a history of aggression is a good predictor of intoxicated aggression (Bailey & Taylor, 1991; Boyatzis, 1975; Lindman, Jarvinen, & Vidjeskog, 1987; Pihl, Zacchia, & Zeichner, 1982). For example, Bailey and Taylor (1991) compared the aggressive behaviour of intoxicated and nonintoxicated subjects with self-reported high, moderate

and low aggressive dispositions under provocative conditions. Aggressive disposition and intoxication interacted such that only the high and moderate aggressive disposition groups increased their shock settings as provocation increased when intoxicated. On the other hand, intoxicated subjects with nonaggressive dispositions were no more aggressive than nonaggressive subjects who had consumed a placebo cocktail.

In a meta-analysis of six studies of the effects of heavy drinking habit on alcohol-mediated aggression, Bushman & Cooper (1990) concluded that alcohol and expectancy manipulations have nonsignificant effects on the aggression displayed by male heavy drinkers. However, these results may merely reflect that fact that heavy drinkers were more tolerant of alcohol effects due to habituation to alcohol's pharmacological properties. Thus, using a heavier dose in these studies might have produced different results.

Finally, investigations of gender effects on alcohol induced aggression have produced equivocal results. Most of the few studies of female subjects indicate that women react no differently than do men (Bushman & Cooper, 1990; Gustafson, 1993). In contrast, Giancola & Zeichner (1995) have recently reported gender differences in alcohol related aggression. Men were more aggressive to men than women whereas women showed equal amounts of aggression to both genders. Furthermore, while males were more aggressive overall, there were no gender differences in the sober and placebo conditions.

Taken together, these findings confirm the fact that aggression does not inevitably follow alcohol consumption. Rather alcohol appears to modify the probability of aggressive responding engendered by individual variables, such as aggressive disposition, as well as situational factors such as provocation and the

social context. This is important as these parameters can account for, at least in part, the dissociation between alcohol and aggressive behaviour observed in natural ecology. As a result, it lends validity to an exploration of possible explanations of the alcohol-aggression relationship.

EXPLANATIONS OF THE ALCOHOL-AGGRESSION RELATIONSHIP

Numerous theories have been generated over the years in an effort to explain the alcohol-aggression relationship (for reviews see Graham, 1980; Gustafson, 1993; Pernanen, 1981; Pihl & Ross, 1987). Relevant theories will be reviewed in terms of the causal role assigned to alcohol consumption as follows: 1) the alcohol-aggression relationship is spurious, 2) aggressive behaviour leads to heavy alcohol use and 3) alcohol use increases the likelihood of aggressive behaviour.

Spurious Explanations

The spurious model postulates that both alcohol consumption and aggression are related because they share common causes rather than a direct causal link (White, 1990). On the one hand, alcohol use and aggression may be a part of a constellation of behaviours related to a central personality characteristic such as power concerns or disinhibitory psychopathology. McClelland et al. (1972) have found that alcohol use and aggression are related to concerns of personal power. An individual with an elevated interest in personal power is also more likely to be more aggressive and a heavy drinker. Drinking will further increase power concerns and aggressiveness which would lead to increased drinking.

Both alcoholism and antisocial personality disorder (APD), which includes aggression as one of the Diagnostic

and Statistical Manual of Mental Disorders-IV (1994) diagnostic criteria, have been considered to fall under the general rubric of disinhibitory psychopathology (Gorenstein & Newman, 1980). Alcohol use disorders are highly comorbid with antisocial personality disorder (Helzer & Pryzbeck, 1988; Hesselbrock, Meyer, & Keener, 1985; Lewis, Cloninger, & Pais, 1983). Sher & Trull (1994) conclude from their review of disinhibitory psychopathology and personality that personality variables such as impulsivity/disinhibition are important components of etiological models of these disorders. Thus, the epidemiological evidence of a correlation between alcohol and aggression may simply reflect a high probability of alcohol use and aggressive behaviour among individuals with disinhibitory psychopathology.

On the other hand, the relation between alcohol and aggression may be due to factors associated with the drinking situation. One explanation is the belief that the drinking situation provides an acceptable outlet for aggression (Boyatzis, 1975; MacAndrew & Edgerton, 1969). There is evidence that different expectations are brought to drinking situations (Cavan, 1966; Mass-Observation, 1943). In particular, alcohol consumption is typically associated with the expectation of increased aggressive behaviour (Kreutzer, Schneider, & Myatt, 1984; Rohsenow & Bacharowski, 1984). However, these results must be interpreted cautiously as an attitude does not always lead to a behaviour.

Experimental studies of the role of expectancy effects in alcohol-related aggression typically rely on the "balanced-placebo design" (Marlatt & Rohsenow, 1980) where beverage contents (alcohol or non-alcoholic) are crossed with the beliefs subjects hold regarding the contents of those beverages. This design yields four belief/beverage combinations: 1) told alcoholic

beverage/received alcoholic beverage; 2) told alcoholic beverage/received non-alcoholic beverage; 3) told non-alcoholic beverage/received alcoholic beverage; and 4) told non-alcoholic beverage/ received non-alcoholic beverage. These four conditions allow for the testing of hypotheses about the extent to which pharmacological or expectancy factors, or some combination of the two, account for the alcohol-aggression relationship.

Results of studies investigating the role of expectancies in alcohol related aggression are equivocal (for review see Lang & Sibrel, 1989). On the one hand, aggression has been shown to be related to the belief one had consumed alcohol, regardless of the actual beverages consumed (Lang, Goeckner, Adesso, & Marlatt, 1975). In this study which used heavy drinkers, alcohol expectancies, but not alcohol itself, facilitated aggressive responding. On the other hand, Gustafson (1985c) has shown that only alcohol and not expectancy increased aggression in moderate drinkers. A meta-analytic review of seven studies using a balanced-placebo design to examine the role of expectancy on aggressive behaviour concluded that expectancies alone do not increase aggression (Hull & Bond, 1986). More recently, Bushman & Cooper (1990) concluded from their review of studies using a placebo or balanced-placebo design that neither the pure pharmacological or psychological effects of alcohol increase aggression by themselves. Rather, it is possible that both effects must occur together for alcohol to cause aggression.

A second set of explanations proposes that environmental or situational factors associated with the drinking context induce aggression. Drinking typically occurs in bars or at social gatherings. Bennett et al. (1969) have proposed that as aggression is an interpersonal act, the presence of many other persons

might be enough to increase aggression. However, Graham et al. (1980) found that the mere presence of others in bars was simply not sufficient to increase aggression. Alternatively, physical aspects typically associated with drinking situations may play a role in the incidence of aggression. High ambient temperature, noise and unhealthy air have all been demonstrated to increase aggressive responding (Baron & Richardson, 1994). However, the specific demonstration of an alcohol-aggression relationship under controlled laboratory conditions (Bushman & Cooper, 1990), in parties (Boyatzis, 1974; 1975) and in bars (Murdoch & Pihl, 1988) argues for at most a conjunctive role for any of these external determinants in intoxicated aggression.

Aggressiveness Leads to Heavy Drinking

This model hypothesizes that aggressive behaviour leads to heavy alcohol use. It is predicated on the notion that aggressive individuals are more likely to select situations or peer groups in which heavy drinking is encouraged (Johnston, O'Malley & Eveland, 1978). Two elaborations of this idea are that aggressive individuals may drink heavily to give themselves an excuse to act aggressively (Boyatzis, 1975) or to self-medicate (Khantzian, 1985).

While there is one report that acute anger per se does not contribute to increased drinking (Marlatt, Kosturn, & Lang, 1975), a few studies have demonstrated that a history of aggressive behaviour can predict alcohol related aggression. Data from a recent prospective, longitudinal study of adolescent males indicate that early aggressive behaviour predicts increased alcohol use and alcohol-related aggression, but that levels of alcohol use do not predict later aggressive behaviour (White, Brick & Hansell, 1993). In

addition, Jaffe, Babor, & Fishbein (1988) have shown that aggression under the influence of alcohol correlated with the history of early childhood aggression in recently hospitalized alcoholics.

The above findings do not necessarily refute experimental studies demonstrating that intoxication leads to aggressive behaviour. Instead, the strength of the aggressive response may depend in part on the subject's baseline level of aggression (Bailey & Taylor, 1991; Boyatzis, 1975, Lindman et al., 1987; Pihl et al., 1982). For example, a large amount of the variation in retrospectively reported behavioral distress and aggression following alcohol consumption can be explained by a history of childhood aggression (Fishbein, Jaffe, Snyder, Haertzen, & Hickey, 1993). These authors also demonstrated that behavioral distress and aggression becomes more severe with increasing levels of alcohol consumption in recently hospitalized alcoholics than in non problem drinkers. Thus, alcohol's effects on aggression may interact with an individual's aggressivity such that aggressive as compared to nonaggressive individuals are more likely to drink heavily and more likely to become aggressive when intoxicated (White et al., 1993) possibly by triggering preexisting aggressive mechanisms (Fishbein et al., 1993).

However, there is recent evidence arguing against this notion (Pihl, Lau & Assaad, 1995). In this study, subjects, assigned to high/low aggression groups based on scores derived from a psychiatric status schedule, competed in the Taylor aggression paradigm, sober or intoxicated. While increased aggression was manifest by the high versus low aggressive group when sober, there were no differences in aggression between the two groups when intoxicated. In addition, there were no group differences in self reported beverage consumption. Thus,

these results argue against the idea that aggressiveness causes drinking.

Alcohol Intoxication Increases Aggressive Behaviour

Alcohol has been postulated to cause aggression due to its direct as well as indirect effects. One explanation is that alcohol has an anaesthetizing effect on the brain center that normally inhibits aggressive responding, thereby releasing aggressive behaviour (Pernanen, 1976). However, the diversity of brain mechanisms involved in aggressive behaviour argues against the idea of a single aggression center in the brain (Elliot, 1992). Furthermore, this theory by itself has limited explanatory power as it does not account for variations in alcohol induced aggression across individuals (e.g. Boyatzis, 1975), cultures (Heath, 1983), nor drinking settings (Kalin, 1972). Thus, any theory invoking a direct cause explanation needs to include other mediating factors to account for these variations (Graham, 1980).

The indirect cause explanation suggests that alcohol consumption induces certain physiological, emotional and cognitive changes, which indirectly increase the probability of aggression. Research on the impact of alcohol induced physiological changes on aggression has sought to demonstrate a link between alcohol-induced changes in nervous system, or hormonal, function and increased aggressiveness. However, the available evidence suggests that the pharmacological consequences of alcohol for the functions thought to be most relevant to social behaviour are diffuse, nonspecific, and variable (even reversible) across the dose-response curve (Berry & Brain, 1986). While there is little experimental evidence supporting the idea that alcohol increases aggression via physiological changes, alcohol

may increase aggression via emotional changes. Pihl et al. (1993) have proposed that alcohol may increase aggression, in part due to its anxiolytic effects which can be compared to those produced by barbiturates or benzodiazepines. These effects are thought to be due to alcohol's pharmacological effects on the septal/hippocampal system (Gray, 1982, 1987). As the septal/hippocampal system is thought to be responsible for the inhibition of ongoing behaviour as a consequence of exposure to threat or novelty (Gray, 1982, 1987), the expression of any behaviour normally inhibited by threat, including aggression, would be facilitated by alcohol. A number of experimental studies have produced results consistent with a hypothesis that alcohol potentiates aggression in threatening situations (Cherek et al., 1985; Kelly et al., 1988; Leonard, 1984; Shuntich & Taylor, 1972; Taylor & Gammon, 1975; Taylor et al., 1976; Zeichner & Pihl, 1979; 1980; Zeichner, Pihl, Niaura, & Zacchia, 1982).

In addition, there is good evidence addressing the possibility that alcohol consumption induces cognitive changes, which indirectly increase the probability of aggression. This discussion will be organized around Dodge & Crick's (1990) social information processing model which describes the cognitive processes related to the expression of aggressive behaviour. This model comprises five sequential skills: cue encoding, cue interpretation, response generation, outcome anticipation/response selection and response enactment which are initially set in motion by provocation. It is argued that skilful processing of each step leads to socially competent behaviour whereas impaired processing will lead to deviant, possibly aggressive, social behaviour.

There is evidence from two studies suggesting that

alcohol consumption may impair cue interpretation such that this biased interpretation leads to increased aggressive responding. First, Zeichner & Pihl (1980) have demonstrated that the aggressive behaviour of only intoxicated individuals was immune to inhibition of aggression induced by consideration of the opponent's intent. Second, there is the finding that intoxicated subjects, who typically select higher shocks for their opponents than sober subjects on the Taylor (1967) task, expect to receive higher shocks from an opponent than individuals consuming a placebo beverage (Schmutte et al., 1979). Thus, alcohol may interfere with the process of making causal attributions such that intoxicated individuals are more likely than sober individuals to make attributions of hostility.

Alcohol intoxication may also alter cognitive processing of cues that normally modify the response to provocation (Pihl & Ross, 1987; Steele & Southwick, 1985; Taylor & Leonard, 1983). Alcohol may change an individual's response to external, less immediately contingent stimuli by reducing the ability to process and extract meaning from those stimuli (Steele & Josephs, 1990). Thus, alcohol intoxication theoretically restricts cognition so that less immediately contingent aspects of a situation (such as peripheral cues or embedded meanings) do not command normal levels of attention. Alcohol intoxication may therefore increase the likelihood of an aggressive response in situations where the salient cue is provocation (which unconditionally elicits aggression) and where peripheral, or less contingent cues inhibit such aggression (Steele & Southwick, 1985). Consistent with this theory is the finding that alcohol-intoxicated subjects fail to modify their aggression in response to threat (Zeichner & Pihl, 1979). One possible reason is that knowledge concerning

the consequences of their behaviour no longer served an inhibitory function (Zeichner, Pihl, Niaura, & Zacchia, 1982). In sum, these findings suggest that alcohol may also interfere with the evaluation of the consequences of response.

While the above studies are consistent with the idea that alcohol's effects on specific cognitive skills leads to increased aggression, these studies were not designed to evaluate each skill in the context of the possible contributions of other skills. It remains possible that impairment in other skills may explain the above results. Sayette, Wilson & Elias (1993) have addressed this possibility by exploring the effects of alcohol on the first four social information processing skills. The results showed that alcohol impaired response generation and outcome anticipation/response selection to increase aggression even when the situation was encoded and interpreted in a manner similar to that of sober subjects.

Although the above studies provide good evidence that alcohol's impairment of a number of perceptual and cognitive processes leads to dyscontrol of aggression in provocative situations, a major question in the alcohol-aggression literature is what neural mechanisms mediate alcohol's effects on these processes.

Pihl, Peterson & Lau's (1993) biosocial model of the alcohol-aggression relationship proposes that alcohol modulates the expression of aggression in part by interfering with cognitive abilities associated with frontal lobe function. This notion is based on evidence that alcohol impairs performance on a variety of neuropsychological tests measuring selected aspects of higher order cognitive abilities. Neuropsychological tests are one of the most sensitive methods for detecting subtle variations in cognitive performance and allow

researchers to form hypotheses about the site of a lesion (Lezak, 1983). Furthermore, neuropsychological assessment has been extensively used as a research tool for identifying brain dysfunctions associated with a wide variety of pathological behaviours including antisocial behaviour (e.g. Moffitt, 1990), schizophrenia (Goldstein, 1986) and risk for alcoholism (Tarter, Hegedus, Goldstein, Shelly, & Alterman, 1984).

Acute alcohol intoxication impairs normal performance on neuropsychological tests that measure cognitive abilities associated with the frontal cortex, but does not impair performance on standard intelligence tests (Peterson, Rothfleisch, Zelazo & Pihl, 1990). This impairment can be attributed to the pharmacological effects of alcohol as Peterson et al. (1990), employing a balanced placebo design, demonstrated minimal effects of alcohol-intoxication expectancy upon test performance. One possible explanation is that alcohol intoxication results in a temporary form of minimal frontal lobe dysfunction.

Studies specifically designed to test the idea that alcohol related disruption in frontal lobe functioning leads to increased aggression are lacking. However, there is some neuropsychological evidence that associates frontal lobe deficits with poorer regulation of social behaviour. This work is reviewed below to explore the possibility that frontal lobe dysfunction can lead to increased aggressive behaviour.

THE FRONTAL LOBES AND AGGRESSION

Anatomy of the Frontal Cortex

The frontal lobes consist of the bilateral brain regions anterior to the central sulcus and superior to the sylvian fissure; this area can be further divided into medial, dorsolateral, and orbitofrontal cortex

(Stuss & Benson, 1984). The entire frontal cortex is thought to be responsible for organizing intellectual activity as a whole (Damasio, 1979; Luria, 1980). The cognitive functions attributed to the frontal lobes include abstraction, inhibition of unsuccessful, inappropriate, or impulsive behaviours, with adaptive shifting to alternative behaviours, mental flexibility, sequencing, attention and concentration, and regulation of emotion and affect (Moffitt, 1990; Raine, 1993). In particular, the frontal lobes are thought to synthesize information related to the outside world, the state of the internal milieu and relevant stimuli in the light of past experiences relating to reward or punishment in order to calculate appropriate responses to what is being perceived (Bear, 1991; Damasio, 1979; Luria, 1980; Nauta, 1971).

The frontal cortex is particularly important in the control of social behaviour from both an evolutionary and anatomical perspective. The frontal cortex is the most recently evolved part of the brain in humans (MacLean, 1990). Moreover, phylogenetically, the frontal cortex develops to a maximum in the human brain where it comprises nearly one third of the neocortex (Fuster, 1989) versus 3.5% of the neocortex in cats, 7% in dogs and 17% in chimpanzees (Raine, 1993).

Anatomically, the frontal cortex is the only cortical area which interconnects with all sensory regions, including the olfactory area (Stuss, Gow, & Hetherington, 1992). In addition, it connects to the limbic system, a group of interconnected subcortical brain structures consisting of the hippocampus, amygdala, fornix, septal region, cingulate gyrus, and mammillary bodies along with connections to the thalamus and hypothalamus. These connections are particularly relevant as the limbic system is involved in emotional

and aggressive behaviour as well as learning and memory. The proximity of the frontal cortex to the brain's motor regions and its connections to lower brain centres, make it a "final common pathway" in formulating appropriate motor responses (Bianchi, 1922; Weiger & Bear, 1988). Moreover, Dimond (1980) has proposed that the frontal lobes play a specialized, critical role in the processing of complex, socially relevant peripheral cues. Thus, it is conceivable that impairment in the operation of frontal lobe functions, regardless of origin, might lead to impaired regulation of social behaviour-perhaps to reduced control of aggression.

Evidence associating frontal lobe deficits with poorer regulation of human social behaviour comes from clinical reports of behaviour in patients with frontal lobe lesions and neuropsychological and neuroimaging studies of individuals who manifest deviant social behaviour.

Frontal Lobe Lesion Studies

Large numbers of clinical case studies of the behaviour of patients with frontal lobe damage have demonstrated significant changes in personality and emotions. One of the first and most striking single case studies linking frontal lobe damage to personality change is that of Phineas Gage. Gage, a railway construction foreman, experienced an accident in which a 3-cm-thick, 109-cm-long iron tamping rod was hurled through his head by a dynamite explosion, entering at the lower cheek and exiting from the upper forehead. Miraculously, Gage survived this injury demonstrating good physical recovery and many preserved cognitive abilities. However, his emotional behaviour and personality changed to such an extent that his friends reported that "Gage was no longer Gage" (Harlow, 1868). His respect for social conventions

disappeared, he had taken leave of his sense of responsibility and he became irreverent, impulsive, profane and capricious. Recently, neuroimaging techniques were used to demonstrate that the lesion involved areas of the ventromedial region of both frontal lobes while sparing the dorsolateral (Damasio, Grabowski, Frank, Galaburda & Damasio, 1994).

Since the 1800's, observed personality changes in individuals with frontal lobe damage have included, for example, decreased concern with social propriety, apathy and indifference, extroversion, social withdrawal, disinhibition, impulsivity, decrease in initiative, automaticity, and outbursts of irritability (Stuss et al., 1992). These changes are characterized by their heterogeneity and the incorporation of opposite extremes in the one syndrome (see Stuss & Benson, 1983, 1986 for a review and extended references).

This heterogeneity may derive, in part, from the contrast in behaviour between patients with orbitofrontal versus dorsolateral frontal lesions (Stuss et al., 1992). Damage to these two areas of the frontal cortex has been associated with two distinct types of personality changes, pseudodepressed versus pseudopsychopathic (Blumer & Benson, 1975). The first syndrome, characterized primarily by an absence of spontaneously generated thought and action, includes, for example, depression, apathy, loss of initiative and indifference. These changes are related to damage of the dorsolateral convexity.

In contrast, the pseudopsychopathic syndrome is associated with damage to the orbitomedial frontal cortex. This syndrome, described as "acquired sociopathy" (Miller, 1987), is characterized by irritability, facetiousness, hyperkinesis or promiscuity and commission of "antisocial" acts (Blumer & Benson,

1975; Damasio et al., 1994; Eslinger & Damasio; 1985; Hecaen & Albert, 1978; Meyers, Berman, Scheibel & Hayman, 1992). This pattern of behaviour has led to the hypothesis that the participation of emotion and its underlying neural machinery in decision making in social situations depends in part on the ventromedial frontal region (Damasio et al, 1994; Eslinger & Damasio, 1985). Furthermore, if such damage occurs early enough in life, it can result in the development of pervasive abnormalities of affective and social behaviour (Eslinger & Damasio, 1985; Price, Daffner, Stowe, & Mesulam, 1990) and the inability to accommodate social impulses into the total personality structure (Ackerly & Benton, 1948).

While the above studies assign frontal regions to different cognitive domains, this idea is compatible with the idea that neurons in these regions might be involved with attention, working memory, and the categorization of contingent relationships regardless of the domain (Goldman-Rakic, 1992).

In sum, lesion studies of noncriminal populations suggest a link between frontal lobe damage and reduced control of social behaviour. However, more direct evidence for frontal dysfunction in violent behaviour comes from neuropsychological studies implicating frontal dysfunction in antisocial or criminal behaviour.

Neuropsychology of Antisocial Behaviour

Several theorists have postulated a role for frontal dysfunction in antisocial or criminal behaviour (e.g. Gorenstein, 1982; Lueger & Gill, 1990; Moffitt, Lynam & Silva, 1994; Raine, 1993; Yeudall, Fedora & Fromm, 1987). Typically, these theories attempt to relate prominent characteristics of deviant social behaviour to individual differences in brain functioning. Behavioral similarities include the failure to inhibit inappropriate

responses, perseveration, low frustration tolerance, difficulty sustaining attention, poor long term planning, and deficits in memory organization (Beaumont, 1983; Stuss & Benson, 1984).

There is a growing body of evidence demonstrating an association between frontal lobe dysfunction and various forms of antisocial behaviour for both adolescent and adult clinical samples.

Delinquency

A sizable literature exists demonstrating that neuropsychological test scores can discriminate between antisocial and non-antisocial delinquents (for review see Moffitt, 1990; Moffitt & Henry, 1991). While these studies employed a wide variety of neuropsychological tests and definitions of delinquency status, the findings consistently cite impairments of executive and verbal functions (Moffitt & Henry, 1989). However, several methodological shortcomings associated with this literature suggested the findings be viewed with caution. Early studies were troubled by problems with subject selection, adequacy of controls, collection of neuropsychological data, data analysis, and failure to evaluate for specificity of effects (Moffitt, 1990).

More recently, however, longitudinal studies of unselected birth cohorts, designed to address the methodological shortcomings of earlier work, have replicated previously reported results (Moffitt, 1990; Seguin, Pihl, Harden, Tremblay, & Boulerice, 1995). The New Zealand study, for example, a prospective, longitudinal study of an unselected birth cohort of 1,037 children born in 1972 from Dunedin, New Zealand has shown that deficits in verbal and "executive" (self-control) functions are associated with delinquency (Moffitt, 1990). More importantly, recent results from this study have produced the first prospective evidence that poor

neuropsychological status, associated with early onset of delinquency, predicted specifically male offending that began prior to age 13 (Moffitt et al., 1994). Furthermore, Seguin et al., (1995) provide evidence consistent with the idea that deficits in executive functions may underlay specifically aggressive behaviour. In this study, the results showed that reduced performance on frontal lobe tests of executive function, predicted fighting in boys aged 13-14.

Taken together, the results of these studies establish a link between frontal dysfunction and delinquent behaviour. Studies of frontal dysfunction in adult antisocial disorders have focused primarily on psychopathy as a prototypic form of antisocial behaviour.

Psychopathy

Psychopathic personalities are described as impulsive, self-centred and aggressively opportunistic; when even slightly frustrated they quickly become furious and vindictive (Miller, 1987). Furthermore, they are easily provoked to attack (Millon, 1981). Theorists have postulated a cortical basis for psychopathy (e.g. Hare & McPherson, 1984). A number of models have specifically implicated septal-hippocampal and frontal dysfunction (e.g. Fowles, 1980; Gorenstein & Newman, 1980; Yeudall et al., 1987).

A small number of studies have tested psychopaths on one or more neuropsychological measures (Hart, Forth & Hare, 1990; Schalling & Rosen, 1968; Smith, Arnett & Newman, 1992) while some studies have specifically focused on frontal lobe functioning in psychopaths (e.g. Gorenstein, 1982; Hare, 1984). The results supporting a neuropsychological basis of psychopathy are equivocal.

Gorenstein (1982), for example, studied adult male patients receiving treatment for substance abuse and/or psychiatric disorders along with normal male college

students. Patients were identified as psychopathic on the basis of the Socialization scale of the California Personality Inventory and a self-report behavioral checklist, which is described as being similar to DSM-III criteria. Psychopathic patients, compared to non-psychopathic patients and normal controls, exhibited the performance pattern of frontal lesion patients on all measures of frontal lobe impairment. Psychopaths made more perseverative errors on the Wisconsin Card Sorting Test (WCST), more total Necker Cube reversals and more errors on the Sequential Matching Memory Task. Moreover, psychopaths did not differ from controls on those measures empirically unrelated to frontal lobe dysfunction including WCST nonperseverative errors and anagrams. Gorenstein concluded that these differences were supportive of a frontal lobe dysfunction explanation of psychopathy.

Further support for this notion comes from studies demonstrating performance deficits in psychopaths on the Porteus Maze (Schalling & Rosen, 1968) and the Necker Cube (Lidberg, Levander, Schalling, & Lidberg, 1978) tests. In addition, criminal psychopaths have demonstrated response perseveration, thought to be a symptom of frontal dysfunction, in a card-playing task incorporating monetary reward (Newman, Patterson, & Kosson, 1987).

Many of these studies, however, have been criticized for a failure to control for potentially confounding variables such as substance abuse, inadequate diagnosis of psychopathy and small sample sizes combined with large test batteries (Hart el al., 1990). Two methodologically rigorous neuropsychological studies of psychopathy did not find group differences in neuropsychological function (Hare, 1984; Hart et al., 1990). Hare (1984), for example, studying a sample of adult male prisoners,

utilized the same neuropsychological tests as Gorenstein (1982), but used the Psychopathy Checklist (Hare, 1980) to determine the presence of psychopathy. When confounding variables such as alcohol, drug use, education, age and IQ, were controlled for, Hare (1984) found no significant difference in perseverative errors on the WCST between psychopaths and the other criminals. These results are consistent with a number of other studies which failed to replicate Gorenstein's findings (Hoffman, Hall, & Bartsch, 1987; Sutker & Allain, 1983).

These latter findings call into question the frontal lobe hypothesis of psychopathic behaviour. However, there are a number of important considerations which might explain these null results. First, controlling for substance abuse may be problematic. ASP and substance abuse disorders are often found together. In particular, some researchers posit that a prefrontal type deficit may underlay both these disinhibitory syndromes (Gorenstein, 1987). Thus, controlling for substance abuse may eliminate individuals with frontal lobe dysfunction.

Second, failures to observe perseveration errors in psychopaths may not be entirely unexpected. Perseverative errors on the WCST have been demonstrated in patients with lesions to the dorsolateral prefrontal cortex. However, it has been speculated that the orbitofrontal region is of potentially greater relevance to antisocial and violent behaviour (Raine, 1993). Thus, psychopaths would not demonstrate deficits on the WCST as damage to the orbitofrontal region does not produce cognitive deficits.

Third, the above studies may have failed to account for the mediating influence of anxiety. There is evidence that low, but not high anxious psychopaths manifest deficits on frontal lobe measures (Smith et al., 1992).

Fourth, if frontal lobe dysfunction is the basis of criminal behaviour, significant differences between psychopathic and nonpsychopathic criminal offenders would not be expected.

Alternatively, frontal lobe dysfunction may be specific to violent criminals. Although psychopaths are more violent than nonpsychopaths, many psychopaths are non-violent. Thus, studies of psychopaths would comprise only an indirect test of the frontal dysfunction-violence hypothesis. A more direct test of this hypothesis consists of neuropsychological as well as brain imaging research of violent individuals.

Neuropsychology of Violence

The idea that brain dysfunction might specifically lead to violent behaviour was popularized by Mark and Ervin (1970). Since then a large body of literature on biology and violence has been accumulated (for review see Mednick, Pollock, Volavka, & Gabrielli, 1982). However, only a few studies have examined a relationship specifically between frontal lobe dysfunction and violence (Bryant, Scott, Golden, and Tori, 1984; Heinrichs, 1989; Yeudall, & Fromm-Auch, 1979)). For example, Yeudall, & Fromm-Auch (1979) examined the relationship between frontal-lobe dysfunction and violent criminal behaviour by comparing violent criminals to normal controls using the Halstead-Reitan Neuropsychological Test Battery (HRTNB). The violent group demonstrated significantly more anterior neuropsychological dysfunction than controls. Kandel & Freed (1989), however, in a recent review of the literature concluded that the evidence supporting this type of relationship is weak at best.

Neuroimaging Studies of Violence

There are four brain imaging studies which have provided evidence for selective frontal dysfunction in

violent offenders, child assaulters, or sex offenders (Hendricks, Fitzpatrick, Hartmann, Quaife, Stratbucker & Graber, 1988; Raine, Buchsbaum, Stanley, Lottenberg, Abel, & Stoddard, 1993; Volkow & Tancredi, 1987; Wright, Nobrega, Langevin & Wortzman, 1990). Raine et al. (1993), for example, reports on a recent brain imaging study using Positron Emission Tomography (PET) of 22 murderers in whom questions regarding mental illness or organic brain injury were raised versus ?2 controls. The results showed selective frontal dysfunction in the murderers. In addition, assessment of 4 violent patients and 4 normal controls using PET demonstrated compromised function of the frontal cortex in 2 out of 4 violent psychiatric patients (Tancredi & Volkow, 1988). Although these studies support the frontal dysfunction-violence hypothesis, the small sample sizes limit their contribution to the suggestion of the potential of brain imaging research for understanding the brain mechanisms that contribute to violent behaviour.

In conclusion, the current neuropsychological evidence linking frontal lobe dysfunction to violence, or increased aggression, is weak. This is due in part to the fact that neuropsychological tests are limited in the extent of their specificity. In particular, distinctions between specific brain regions have not been accurately identified by neuropsychological tests. Thus, one possibility is that violent individuals have deficits in specific regions of the frontal lobes whose functions are not detected by traditional neuropsychological tests thought to reflect frontal functioning.

There are, however, two neuropsychological tests designed by researchers at the Montreal Neurological Institute which have not yet been used to investigate a link between frontal dysfunction and violence that have been mapped to specific brain regions. These two tests,

the spatial conditional associative-learning task (CALT; Petrides, 1985a) and the self-ordered pointing task (SOP; Petrides & Milner, 1982), appear to assess the functions of two distinct areas of the dorsolateral frontal cortex. PET with magnetic resonance imaging of the brains of normal volunteers completing a modified version of the CALT has demonstrated activation of cytoarchitectonic area 8 of the dorsolateral frontal cortex, whereas cytoarchitectonic areas 46 and 9 of the mid-dorsolateral frontal cortex were activated when volunteers completed a modified version of the SOP (Petrides, Alivisatos, Evans, & Meyer, 1993). Individuals with unilateral frontal lobe damage have been shown to perform poorly on both these tasks (Petrides & Milner, 1982; Petrides, 1985a). The impairment on the CALT appears to be due to difficulties in learning to choose from a set the appropriate response to a given stimulus (Petrides, 1985a) whereas the deficits on the SOP can be attributed either to poor monitoring of responses or poor organizational strategies or both (Petrides & Milner, 1982). Finally, the dorsolateral frontal cortex is thought to play a role in the higher-order control of behavioral responses (Petrides, 1985b) and certain aspects of working memory (Petrides, 1991). If this area fails, the brain views the world as a series of disconnected events. As a result, behaviour is excessively dominated by immediate stimulation rather than by a balance of external, internal and past information (Goldman-Rakic, 1992). Thus, deficits in cognitive abilities associated with this brain area may be important in the dyscontrol of aggressive behaviour.

Finally, one caveat must be addressed. The above discussion is not meant to imply that the prefrontal cortex, or any other brain area for that matter, functions in isolation to produce aggressive behaviour.

Our current level of understanding of neuroanatomy is as yet insufficient to support such conclusive causal deductions (Moffitt, 1990). It is more likely that various neural and neurochemical substrates are also involved in the cognitive functions attributed to the dorsolateral prefrontal cortex. Furthermore, these cognitive abilities likely interact with environmental conditions to produce aggressive behaviour.

In sum, the aim of the following experiments was to investigate the role of individual differences in cognitive abilities associated with the dorsolateral frontal cortex in the alcohol-aggression relationship behaviour. As reviewed earlier, the results of previous investigations of individual difference variables were equivocal (e.g., Bailey & Taylor, 1991; Bushman & Cooper, 1990; Giancola & Zeichner, 1995; Pihl et al., 1995). The failure to demonstrate clear cut effects in these studies may derive in part from the use of between-subjects designs. A within-subjects design, however, incorporating reduced error variance, might provide an important advantage in the investigation of influences of individual differences.

STATEMENT OF THE PROBLEM

The aim of the research in this thesis was to investigate the role of individual differences of cognitive functions associated with the prefrontal cortex in alcohol induced aggression. In order to maximize the results of this investigation, a within-subjects design was deemed advantageous. However, the Taylor (1967) competitive reaction time task has not been used previously in a repeated measures design to study the alcohol-aggression relationship. Thus, the validity of using the Taylor aggression paradigm in a repeated measures design was first assessed in Study 1. It was

expected that (1) all individuals would be more aggressive as provocation increased and (2) individuals would become more aggressive when intoxicated than when sober, irrespective of the order in which they completed the task.

Study 2 investigated whether alcohol increased aggression via an impairment of frontal lobe function. Individuals with high or low cognitive performance on the CALT and SOP participated in the aggression paradigm intoxicated or sober. It was specifically hypothesized that (1) all individuals would be more aggressive as provocation increased, (2) intoxicated individuals would be more aggressive than sober individuals, (3) individuals with lower cognitive performance would be more aggressive than those with higher cognitive performance, (4) that provocation would interact with cognitive ability such that individuals with lower cognitive performance would respond to increased provocation with greater increases in aggression, and (5) that intoxicated condition and cognitive ability would interact, such that alcohol would increase aggression more for lower cognitive performers.

STUDY 1

Alcohol and the Taylor Aggression Paradigm: A Repeated Measures Study

Alcohol and the Taylor Aggression Paradigm: A Repeated Measures Study*

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ABSTRACT. Acute alcohol intoxication has been shown to increase physical aggression in the laboratory as measured by the Taylor aggression paradigm with independent groups. Because it would be advantageous to use the Taylor paradigm in a repeated measures design to examine individual differences, the present study compared the performance of subjects on two counterbalanced occasions: when they were intoxicated and when they were sober. Order of testing significantly interacted with drug condition for the aggression measures and pain threshold. However, an analysis of the ratio of pain threshold to shock intensity, and to total aggression, revealed the procedure to be useful in understanding one aspect of how alcohol modulates aggression. The results suggest that acute alcohol intoxication interferes with the ability to integrate previously acquired knowledge in the formulation of behavioral strategies. (*J. Stud. Alcohol* 55: 701-706, 1994)

THERE IS a large body of experimental evidence that intoxicated individuals behave more aggressively, both verbally and physically, than do sober individuals (Bushman and Cooper, 1990). A number of paradigms have been used to measure physical aggression. Most procedures are variations of the Buss (1961) teacher-learner paradigm (e.g., Gustafson, 1985) and the Taylor (1967) competitive reaction-time task (e.g., Zeichner and Pihl, 1979), both of which provide a believable context for the administration of noxious physical stimuli. In the former, a subject, assigned the teacher role, gives electric shocks to punish a learner, a confederate, for making incorrect responses on a visual discrimination task. In the latter, the subject competes in a reaction-time task and exchanges shocks, or other punishing stimuli, typically with a fictitious opponent. The intensity of the voluntarily administered punishment along with the duration constitute the measures of aggression. A third type of paradigm uses a free-operant point-subtraction procedure where the measure of aggression consists of the withdrawal of money from the opponent (Cherek et al., 1984). This paradigm also includes a nonaggressive response alternative, namely, that of giving money to the opponent.

These tasks have been used to confirm the existence of a relationship between alcohol and aggression. However, not everyone who drinks becomes aggressive (Heath, 1983). Thus, one can not conclude that the alcohol-aggression relationship applies to all individuals (Taylor and Leonard, 1983). Specifically, there is a need for systematic investigation of the influences of individual differences. While the moderating role of individual differences can be studied using between subjects designs, within subject designs provide the important advantage of a reduction in error variance. As a result this design is more sensitive to drug effects. For example, several studies using the Cherek paradigm in a repeated measures design have demonstrated increased aggression with very low alcohol doses (Cherek et al., 1984, 1985; Kelly et al., 1988, 1989).

The Buss paradigm has also been used to study the effect of alcohol on aggression in repeated measures designs (e.g., Bennett et al., 1969; Gustafson, 1984). Surprisingly, in neither study did subjects administer a greater intensity of shocks when intoxicated versus when they were sober. However, neither study ruled out possible confounding influences of order effects of administration of dose. Furthermore, the general use of this paradigm has been criticized because of artificiality and the potential demand of altruism in the task (Baron and Eggleston, 1972; Shuck and Pisor, 1974).

According to a review of the literature, the Taylor task does not appear to have been used in a repeated measures design to study the alcohol-aggression relationship. Yet, it is the paradigm used most frequently in this field and has the advantages of being able to manipulate provocation of the subject and study the effects of more than one attack on aggressive responding (Bertilson, 1990). This measure differentiates between groups expected to differ in aggressive potential (Dengerink, 1971; Taylor, 1967, 1970). It is sensitive to environmental factors theoretically expected to influence aggression (Borden and Taylor, 1973; Hendrick and Taylor, 1971; Pisano and Taylor, 1971) and, finally, there is direct support for convergent and discriminant validity of this task (Bernstein et al., 1987).

The present study assessed the possibility of using the Taylor aggression paradigm in a within-subjects design. Individuals competed in a two-provocation condition Taylor paradigm sober and intoxicated on two different occasions. It was expected that (1) all individuals would be more ag-

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gressive as provocation increased and (2) individuals would become more aggressive when intoxicated than when sober, irrespective of the order in which they completed the task.

Method

Subjects

Thirty-six nonalcoholic male social drinkers (who received a score of less than 5 on the short form of the Michigan Alcoholism Screening Test; Pokorny et al., 1972), aged 18–40, in good physical and mental health, recruited through newspaper advertisements, participated in this experiment. Individuals receiving medical treatment that contraindicated alcohol consumption, and who were familiar with psychological experimentation, were excluded from participation.

Apparatus

Aggression was elicited and assessed with the Taylor (1967) competitive reaction-time task. The subject's task board consisted of eight buttons, numbered consecutively from one to eight, where one was the weakest shock and eight was the strongest shock that could be chosen. Above each button was a small red light which when lit indicated the level of shock chosen by the opponent. An Apple computer was used to run the aggression task and record data. Shocks were administered via the Mark I, Behaviour Modifier (Farrall Instruments), connected to an electrode attached to the inner forearm, below the elbow of the nondominant hand. Each subject monitored administration of shock to his fictitious opponent by viewing a DC ammeter provided for that purpose. A Sony television connected to a Quasar VCR was used to play a prerecorded videotape to the subject. This tape displayed the fictitious opponent receiving instructions regarding performance of the aggression task. This tape served to reinforce the subject's belief in the existence of the opponent, and as a review of the instructions of the task for the subject.

An Alco-Sensor III (Thomas, Ltd.) was used to establish the blood alcohol concentration (BAC) of the subjects. In addition, subjects were asked to rate themselves on a 7-point Likert-type "How drunk" scale, with a score of 1 representing sobriety and 7 representing the most intoxicated each subject had ever been.

Procedure

Respondents to newspaper advertisements, who met inclusion criteria, were scheduled for appointments between 1 and 5 PM. They were asked to refrain from the consumption of drugs or alcohol for at least 24 hours prior to testing and the consumption of food for at least 4 hours prior to testing. Upon arrival at the laboratory, all subjects signed an informed consent form and provided demographic data.

Subjects competed in the Taylor aggression task intoxicated and sober on separate days. To test for possible order effects, the order that the subjects were tested, either intoxicated or sober, was counterbalanced. In the alcohol condition, subjects drank 1 ml per kg of body weight of 95% alcohol USP in three drinks of a 1:7 alcohol : orange juice solution. In the sober condition, subjects were administered three drinks of juice of equivalent volume. Subjects were told explicitly what they were drinking in each condition and were given the impression that their opponent was under the same experimental condition.

Drinks were consumed over a 20-minute period. An additional waiting period of 20 minutes followed to allow the subjects in the alcohol condition time to reach near-peak BACs. Following the waiting period, BACs were taken and each subject was asked to rate himself on the "How drunk" Likert scale.

Each subject's pain threshold for electric shock was then determined. A series of shocks which increased stepwise in intensity from zero were delivered at a constant rate. Each subject was instructed to press a button in response to any shock he regarded as painful (1) to stop the administration of the shock and (2) to reduce the level of the next shock by one step. Each shock delivered after pressing the button was therefore one step lower than the shock that induced pressing the button. When a given subject pressed the button upon three presentations of the same shock intensity, delivery of the shocks was stopped. This shock level was defined as the pain threshold.

The aggression task was then introduced as a competitive reaction-time test. Each subject was instructed to select a shock level that he would deliver to his opponent in the event he won the reaction-time trial. The subject would then be informed of the opponent's shock choice. If a given subject lost, he received that shock. If he won, he administered his previously chosen shock to his opponent. The experimenter then left briefly, telling each subject that he was about to verify the readiness of the opponent. Upon his return, the experimenter stated that instructions were about to be delivered to the opponent, and that this delivery could be viewed on the TV monitor, as a review of the instructions. Following the videotape presentation, three practice trials were conducted. These trials were the same as the actual trials except that the experimenter was present to ensure proper performance. Each subject lost two trials and received two shocks ranging from one to four.

The task itself consisted of 26 consecutive trials. The first half consisted of low provocation (shock values administered to the subject rated from 1 to 4—from a very low level up to half a given subject's threshold). The second half consisted of high provocation (shock values administered to the subject rated from 5 to 8—from one-half up to a given subject's full threshold). The outcome of each reaction-time trial and the shock level administered to the subject in the case of a loss was randomly assigned by the computer. All computer-

administered shocks were of the same duration (0.75 sec). Each subject won and lost exactly half of the trials in both provocation conditions.

Subjects returned, usually the next day at the same time, to compete in the aggression paradigm a second time. Following the second session, subjects completed a short questionnaire to verify the success of the deception. Subjects rated their own and their opponent's performance on the aggression task, and described how effective they thought the task was at measuring their reaction time. All subjects were debriefed. Necessity for deception was fully explained. No subject was adversely affected by the deception, according to self-report. Intoxicated subjects were retained in the laboratory until their BAC dropped below 0.03%. All subjects were paid $5 an hour to compensate for lost time.

Two objective measures of aggression were recorded: intensity of the shock each subject selected for each trial and the duration of the shock each subject delivered to the fictitious opponent.

Results

Manipulation checks

Subject measures. Seventeen individuals completed the task while sober for the first session and while intoxicated for the second (Order 1), while the remaining 17 competed in the reverse order (Order 2). Separate two-tailed *t* tests did not reveal any significant differences between the two groups for mean age, years of education, SES, nor beverages per week (Table 1).

Deception success. Of the 36 subjects, two were not deceived and were excluded from the analysis.

Alcohol measures. Subjects were tested on the aggression paradigm sober and intoxicated. In the intoxicated condition, separate two-tailed *t* tests revealed that the mean BAC and mean "How drunk" ratings for subjects of Orders 1 and 2 did not differ significantly (Table 1).

Pain threshold measures. As this variable was positively skewed, a logarithmic transformation was applied. The transformed variable was normal according to a test for departure from normality based on a biweight estimator of scale (Martinez and Iglewicz, 1981). A 2 × 2 (order, drug) mixed-design ANOVA, with drug as the repeated measure, performed on the transformed variable, revealed a significant main effect of drug ($F = 15.02$, 1/32 df, $p < .001$) and a significant interaction between order and drug ($F = 5.76$, 1/32 df, $p < .05$). Simple main effects analysis of drug for Order 1 showed that these subjects exhibited higher mean pain thresholds when intoxicated (mean [±SD] = 89 ± 2.0) than when sober (mean = 78 ± 1.9), but this difference was not significant. However, subjects of Order 2 exhibited significantly higher mean pain thresholds when intoxicated (mean = 101 ± 2.0) than when sober (mean = 57 ± 1.8; $F = 9.96$, 1/32 df, $p < .01$).

Physical aggression

Shock intensity. A 2 × 2 × 2 (order, drug, provocation) mixed-design ANOVA, with drug and provocation as repeated measures, revealed a significant main effect of provocation ($F = 38.32$, 1/32 df, $p < .001$). Subjects selected higher mean shock intensities under high (mean = 4.5) than low (mean = 3.2) provocation. Furthermore, there was a significant interaction between order and drug ($F = 4.80$, 1/32 df, $p < .05$). Simple main effects analysis of order for the sober condition showed that subjects from Order 2 chose significantly higher mean shock levels (mean = 4.3 ± 2.2) than those from Order 1 (mean = 3.4 ± 1.7; $F = 4.97$, 1/32 df, $p < .05$). Thus, subjects competing sober in the second session chose higher mean shock levels than those competing sober in the first. For the intoxicated condition, subjects of Order 1 had similar mean shock intensities (mean = 3.7 ± 1.8) as those of Order 2 (mean = 4.0 ± 2.2); subjects chose similar shock levels when intoxicated regardless of session number (Figure 1).

Shock duration. As this variable was severely positively skewed, a logarithmic transformation was applied. As some of the values were close to zero, a constant (1) was first added to each score. This transformation reduced the skewness so that this variable was normal. A 2 × 2 × 2 (order, drug, provocation) mixed-design ANOVA, with drug and provoca-

TABLE 1. Demographics and alcohol measures

	All subjects ($N = 34$) Mean (±SD)	Order 1 ($n = 17$) Mean (±SD)	Order 2 ($n = 17$) Mean (±SD)
Demographics			
Age	22.2 ± 3.4	22.7 ± 3.4	21.8 ± 3.4
Years of education	14.2 ± 2.3	13.6 ± 2.5	14.8 ± 1.9
SES	1.9 ± 0.9	2.1 ± 1.0	1.8 ± 0.7
Beverages/week	11.1 ± 7.8	12.5 ± 7.5	9.6 ± 8.0
Alcohol measures			
Intoxicated			
"How drunk" scale	4.1 ± 0.9	3.9 ± 0.08	4.3 ± 0.9
BAC (%)	0.11 ± 0.02	0.11 ± 0.02	0.11 ± 0.02
Sober BAC (%)	0.00	0.00	0.00

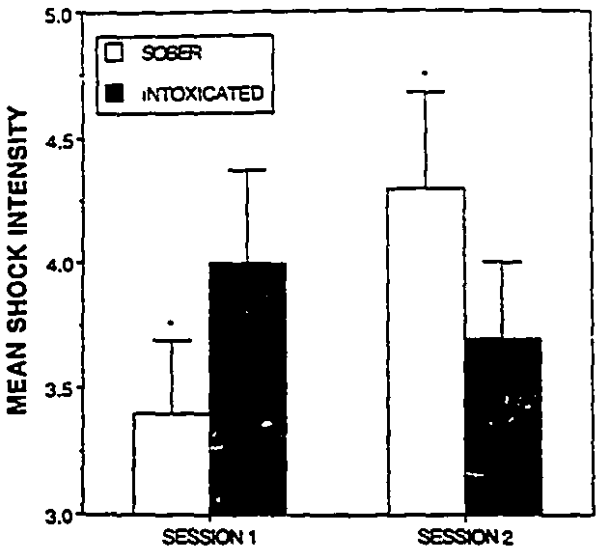

FIGURE 1. Mean shock intensity for the first and second sessions

tion as repeated measures, performed on the transformed variable, revealed a significant main effect of provocation ($F = 14.72$, 1/32 df, $p < .001$). Subjects selected longer mean shock durations under high (mean = 0.82) than under low (mean = 0.67) provocation. In addition, there was a significant interaction between drug and order ($F = 5.54$, 1/32 df, $p < .05$). Simple main effects analysis of order for the sober condition showed that subjects from Order 1 chose similar mean shock durations (mean = 0.71 ± 1.3) as those of Order 2 (mean = 0.70 ± 1.3). For the intoxicated condition, however, subjects of Order 1 chose longer mean shock durations (mean = 0.91 ± 1.3) than those of Order 2 (mean = 0.66 ± 1.3; $F = 10.31$, 1/32 df, $p < .01$). Thus, subjects competing intoxicated in the second session chose longer mean shock durations than those competing intoxicated in the first.

Shock intensity × *shock duration.* This variable provided a measure of total aggression. As this variable was severely positively skewed, a logarithmic transformation was applied. This transformation reduced the skewness so that this variable was normal. A 2 × 2 × 2 (order, drug, provocation) mixed-design ANOVA, with drug and provocation as repeated measures, performed on the transformed variable, revealed a significant main effect of provocation ($F = 45.25$, 1/32 df, $p < .001$). Subjects were more aggressive under high (mean = 4.3) than under low (mean = 3.0) provocation. In addition, there was a significant interaction between drug and order ($F = 6.08$, 1/32 df, $p < .05$). Simple main effects analysis of order for the sober condition showed that subjects from Order 1 showed lower mean total aggression scores (mean = 3.3 ± 1.9) than those of Order 2 (mean = 3.6 ± 2.1), but this difference was not significant. For the intoxicated condition, however, subjects of Order 1 showed significantly higher mean total aggression scores (mean = 4.3 ± 2.0) than those of Order 2 (mean = 3.2 ± 2.1; $F = 6.86$, 1/32 df, $p < .05$). Thus, subjects competing intoxicated in the second session showed higher mean total aggression scores than those competing intoxicated in the first.

Cost/benefit ratio

As mean pain threshold and shock intensity varied according to an interaction between order and drug, a ratio of pain threshold by shock intensity was determined for each subject to assess use of knowledge from Session 1 in Session 2. This ratio represents the amount of pain threshold per mean unit of shock intensity. This ratio was thought to represent a cost/benefit ratio where the cost was the range of shocks received as defined by an individual's pain threshold and the benefit was the mean shock intensity selected for his opponent.

Pain threshold/shock intensity. As this variable was severely skewed, a logarithmic transformation was applied. This transformation reduced the skewness so that this variable was normal. A 2 × 2 × 2 (order, drug, provocation) mixed-design ANOVA, with drug as repeated measures, performed on the transformed variable, revealed a significant main effect of provocation ($F = 37.54$, 1/32 df, $p < .001$) and drug ($F = 10.12$, 1/32 df, $p < .01$) as well as a significant interaction between order and drug ($F = 8.12$, 1/32 df, $p < .01$). Subjects demonstrated a smaller cost/benefit ratio under high provocation (mean = 20) than under low provocation (mean = 29). Simple main effects analysis of order for the sober condition showed that subjects from Order 2 had a significantly lower cost/benefit ratio (mean = 16 ± 2.2) than those from Order 1 (mean = 26 ± 1.9; $F = 10.64$, 1/32 df, $p < .01$). Thus, subjects competing sober in the second session had lower mean ratios than those competing sober in the first. For the intoxicated condition, subjects from Order 1 had similar mean ratios (mean = 27 ± 2.2) as those from Order 2 (mean = 31 ± 2.3) (Figure 2).

Total pain threshold/total aggression. In this case, the cost/benefit ratio was determined by dividing the total pain threshold by the total aggression measure. Total pain threshold was defined as the pain threshold multiplied by the duration of the shocks received (0.75 sec). As this variable was severely skewed, a logarithmic transformation was applied. This transformation reduced the skewness so that this variable was normal. A 2 × 2 × 2 (order, drug, provocation) mixed-design ANOVA, with drug as repeated measures, performed on the logarithm of this variable, revealed a significant main effect of provocation ($F = 43.65$, 1/32 df, $p < .001$) and a significant interaction between order and drug ($F = 10.07$, 1/32 df, $p < .01$). Subjects demonstrated a smaller cost/benefit ratio under high provocation (mean = 22) than under low (mean = 38) provocation. In addition, the main effect of drug approached significance ($F = 3.63$,

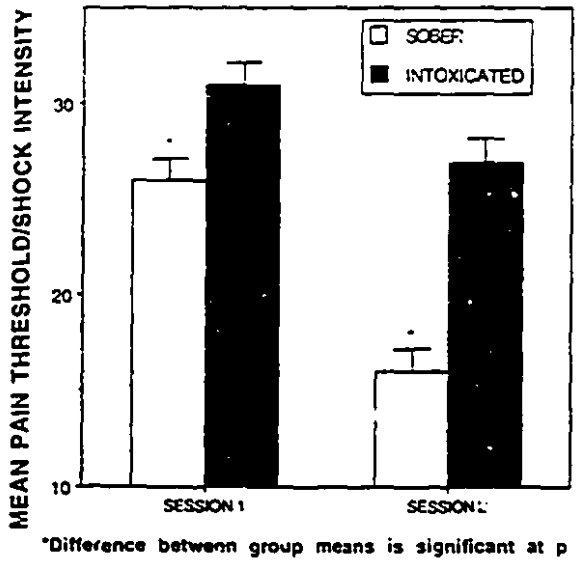

FIGURE 2. Mean logarithm of pain threshold/shock intensity for the first and second sessions

1/32 df, $p = .066$). Simple main effects analysis of order for the intoxicated condition showed that subjects from Order 2 had significantly greater mean cost/benefit ratios (mean = 42 ± 3.3) than those from Order 1 (mean = 26 ± 4.5; $F = 5.15$, 1/32 df, $p < .05$). Thus, subjects competing intoxicated in the first session had greater mean ratios than those competing intoxicated in the second. For the sober condition, subjects from Order 1 had significantly higher mean ratios (mean = 32 ± 3) than those from Order 2 (mean = 20 ± 3.3; $F = 4.90$, 1/32 df, $p < .05$). Therefore, subjects competing sober in the first session had greater mean ratios than those competing sober in the second.

Discussion

The results of this study demonstrate that mean shock intensity, duration and total aggression as well as mean pain threshold varied according to an interaction between order and drug. Further analysis of the results revealed interesting aspects of the alcohol-aggression relationship. Specifically, by creating separate ratios of pain threshold with two measures of aggression, shock intensity and total aggression, it was evident that sober individuals competing in the second session exhibited the most advantageous cost/benefit ratios. This is in part due to the fact that these subjects selected the highest mean shock intensities while receiving the lowest shocks in return. In a previous study, sober individuals who competed sober in the Taylor paradigm on repeated occasions selected higher mean shock intensities during the second session (8.16) than the first session (7.37) (Taylor et al., 1979). However, the effects of repeated sessions on pain threshold, shock duration and total aggression were not examined.

Shock level choice by sober subjects may possibly be understood in terms of provocation effects. In the present study, all subjects responded to an increase in provocation with a significant increase in mean shock intensity. As the high provocation condition was the last condition of Day 1 and subjects believed they faced the same opponent on Day 2, the subjects may have maintained an increased level of aggression when beginning the task on the second day. Prior exposure to provocation may increase aggression by increasing a sober individual's anticipation of becoming a recipient of future attacks (Taylor et al., 1979). This may be important as aggressive behavior is thought to vary according to the degree with which a person attributes aggressive intent to his victim (Epstein and Taylor, 1967; Greenwell and Dengerink, 1973).

Not only did sober individuals participating in the second session increase their aggression, they demonstrated lower pain thresholds. One possibility is that these individuals are trying to minimize the shocks they will receive when competing in the paradigm for the second time. This suggests that sober individuals competing for the second time use the knowledge acquired from their experience in the first session to form a strategy or plan in anticipation of facing an aggressive opponent. Interestingly, individuals participating intoxicated in the second session had similar cost/benefit ratios as those individuals who participated intoxicated in the first session. It appears that the former individuals did not use their previous experience in the paradigm to modify their behavior.

Taken together, these results suggest that acute alcohol intoxication interferes with one's ability to integrate previously acquired knowledge in the formulation of behavioral strategies. The frontal cortex plays a vital role in the formulation of verbal and motor strategies (Luria, 1980) and is thought to be important in the application of previously established knowledge in the regulation of behavior (Pihl et al., 1993). Peterson et al. (1990) have demonstrated that alcohol is pharmacologically capable of interfering with performance on various tests of higher order cognitive ability associated with the frontal cortex, but does not affect performance on standard IQ tests. Thus, the present results are consistent with the idea that specific features of higher order cognitive function associated with the frontal cortex are important in the control of aggression (Pihl et al., 1993).

It might be argued that this study measured degree of aggression under conditions where only an aggressive response was permitted. However, the presence of the lowest level of shock button was clearly explained and its use was not discouraged. Furthermore, it is unlikely that allowing a nonaggressive response option would have resulted in substantially different findings. This has been shown to be true with the Cherek paradigm (Cherek et al., 1985).

Finally, as a placebo group was not included in this study, it may be argued that expectancy (Lang et al., 1975) contributed to the results, although this is difficult to reconcile with numerous other studies with the Taylor paradigm (Taylor and Chermack, 1993).

In conclusion, the use of the Taylor aggression paradigm in a repeated measures design to study the alcohol-aggression relationship revealed some limitations but also some interesting aspects of the alcohol-aggression relationship. There were interactions between order and drug for shock intensity, shock duration, total aggression and pain threshold, which greatly limits the use of this procedure. However, the results suggest that alcohol intoxication interferes with the ability to utilize previously acquired knowledge in the formulation of behavioral strategies.

Acknowledgment

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References

BARON, R.A. AND EGGLESTON, R.J. Performance on the "aggression machine": Motivation to help or harm? Psychon. Sci. 26: 321-322, 1972.

BENNETT, R.M., BUSS, A.H. AND CARPENTER, J.A. Alcohol and human physical aggression. Q. J. Stud. Alcohol 30: 870-876, 1969.

BERNSTEIN, S., RICHARDSON, D. AND HAMMOCK, G. Convergent and discriminant validity of the Taylor and Buss measures of physical aggression. Aggress. Behav. 13: 15-24, 1987.

BERTILSON, H.S. Can aggression be justified in order to study aggression? Amer. Behav. Sci. 33: 595-607, 1990.

BORDEN, R.J. AND TAYLOR, S.P. The social instigation and control of physical aggression. J. Appl. Soc. Psychol. 3: 354-361, 1973

BUSHMAN, B.J. AND COOPER, H.M. Effects of alcohol on human aggression: An integrative research review. Psychol. Bull. 107: 341-354, 1990.

BUSS, A.H. The Psychology of Aggression. New York: John Wiley & Sons, Inc., 1961.

CHEREK, D.R., STEINBERG, J.L. AND MANNO, R.B. Effects of alcohol on human aggressive behavior. J. Stud. Alcohol 46: 321-328, 1985.

CHEREK, D.R., STEINBERG, J.L. AND VINES, R.V. Low doses of alcohol affect human aggressive responses. Biol. Psychiat. 19: 263-267, 1984.

DENGERINK, H.A. Anxiety, aggression and physiological arousal. J. Exp. Res. Pers. 5: 223-232, 1971.

EPSTEIN, S. AND TAYLOR, S. Instigation to aggression as a function of degree of defeat and perceived aggressive intent of the opponent. J. Pers. 35: 265-289, 1967.

GREENWELL, J. AND DENGERINK, H. The role of perceived versus actual attack in human physical aggression. J. Pers. Social Psychol. 26: 66-71, 1973.

GUSTAFSON, R. Alcohol, frustration, and direct physical aggression: A methodological point of view. Psychol. Rep. 55: 959-966, 1984.

GUSTAFSON, R. Alcohol and aggression: Pharmacological versus expectancy effects. Psychol. Rep. 57: 955-966, 1985.

HEATH, D.B. Alcohol and aggression: A "missing link" in worldwide perspective. In: GOTTHEIL, E., DRULEY, K.A., SKOLODA, T.E. AND WAXMAN, H.M. (Eds.) Alcohol, Drug Abuse and Aggression. Springfield, Ill.: Charles C Thomas, Pub., 1983, pp. 89-103.

HENDRICK, C. AND TAYLOR, S.P. Effects of belief similarity and aggression on attraction and counteraggression. J. Pers. Social Psychol. 17: 342-349, 1971.

KELLY, T.H., CHEREK, D.R. AND STEINBERG, J.L. Concurrent reinforcement and alcohol: Interactive effects on human aggressive behavior. J. Stud. Alcohol 50: 399-405, 1989.

KELLY, T.H., CHEREK, D.R., STEINBERG, J.L. AND ROBINSON, D. Effects of provocation and alcohol on human aggressive behavior. Drug Alcohol Depend. 21: 105-112, 1988.

LANG, A.R., GOECKNER, D.J., ADESSO, V.J. AND MARLATT, G.A. Effects of alcohol on aggression in male social drinkers. J. Abnorm. Psychol. 84: 508-518, 1975.

LURIA, A.R. Higher Cortical Functions in Man (Haigh, B., transl.), 2d Edition. New York: Basic Books, Inc., 1980.

MARTINEZ, J. AND IGLEWICZ, B. A test for departure from normality based on a biweight estimator of scale. Biometrika 68: 331-333, 1981.

PETERSON, J.B., ROTHFLEISCH, J., ZELAZO, P.D. AND PIHL, R.O. Acute alcohol intoxication and cognitive functioning. J. Stud. Alcohol 51: 114-122, 1990.

PIHL, R.O., PETERSON, J.B. AND LAU, M.A. A biosocial model of the alcohol-aggression relationship. J. Stud. Alcohol, Supplement No. 11, pp. 128-139, 1993.

PISANO, R. AND TAYLOR, S.P. Reduction of physical aggression: The effects of four strategies. J. Pers. Social Psychol. 19: 237-242, 1971.

POKORNY, A.D., MILLER, B.A. AND KAPLAN, H.B. The Brief MAST: A Shortened Version of the Michigan Alcoholism Screening Test. Amer. J. Psychiat. 129: 342-345, 1972.

SHUCK, J. AND PISOR, K. Evaluating an aggressive experiment by the use of simulating subjects. J. Pers. Social Psychol. 29: 181-186, 1974.

TAYLOR, S.P. Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. J. Pers. 35: 297-310, 1967.

TAYLOR, S.P. Aggressive behavior as a function of approval motivation and physical attack. Psychon. Sci. 18: 195-196, 1970.

TAYLOR, S.P. AND CHERMACK, S.T. Alcohol, drugs and human physical aggression. J. Stud. Alcohol, Supplement No. 11, pp. 78-88, 1993.

TAYLOR, S.P. AND LEONARD, K.E. Alcohol and human physical aggression. In: GEEN, R.G. AND DONNERSTEIN, E.I. (Eds.) Aggression: Theoretical and Empirical Reviews. Vol. 2, San Diego, Calif.: Academic Press, Inc., 1983, pp. 77-101.

TAYLOR, S.P., SHUNTICH, R.J. AND GREENBERG, A. The effects of repeated aggressive encounters on subsequent aggressive behavior. J. Social Psychol. 107: 199-208, 1979.

ZEICHNER, A. AND PIHL, R.O. Effects of alcohol and behavior contingencies on human aggression. J. Abnorm. Psychol. 88: 153-160, 1979.

STUDY 2

Provocation, Acute Alcohol Intoxication, Cognitive Performance, and Aggression

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Provocation, Acute Alcohol Intoxication, Cognitive Performance, and Aggression

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This study investigated the relationships between provocation, acute alcohol intoxication, impaired frontal-lobe function, and aggressive behavior. The authors ranked 114 men according to their performance on two neuropsychological tests associated with frontal-lobe function. Forty-eight men (24 with scores in the upper and 24 with scores in the lower performance quartiles) participated in the full study. Half completed an aggression task while intoxicated, the remainder while sober. Aggression was defined as shock intensity delivered to a sham opponent. Shock intensity significantly increased as a main effect of provocation, alcohol intoxication, and lower cognitive performance. Furthermore, provocation interacted significantly with test performance such that individuals in the lower cognitive performance quartile responded to increased provocation with heightened aggression.

Violence presents a serious and growing challenge to North American society. More than a million and a half individuals are victims of violent crime each year (U.S. Bureau of the Census, 1991); 20,000 are murdered (Prothrow-Stith, 1990). Aggression can be defined as any behavior directed toward the goal of harming another living being who is motivated to avoid such treatment (Baron & Richardson, 1994). Various antecedents to aggressive behavior have been implicated, including social, situational, and individual determinants. A major social determinant of aggression is provocation, in the form of verbal insult, physical attack, or other noxious stimuli (Hammock & Richardson, 1992).

One of the most important situational determinants of aggression is acute alcohol intoxication, which is associated with more than half of all homicides, assaults, rapes, and cases of family violence (Murdoch, Pihl, & Ross, 1990). Bushman and Cooper's (1990) meta-analytic review of over 30 controlled laboratory studies concluded that alcohol-intoxicated individuals are significantly more verbally and physically aggressive. However, every one who drinks does not become aggressive, and the study of characteristics of predispositions to aggressive responses becomes particularly relevant.

Many individual determinants including personality traits, attitudes, genetic factors, and sex differences have been implicated in aggressive behavior (Baron & Richardson, 1994; Wood, Wong, & Chachere, 1991). The occurrence of a violent act in response to external factors is thought to require the interaction of various cognitive processes, including planning of behaviors and consciousness of the act and its consequences (Tancredi & Volkow, 1988). As the frontal cortices are thought to be involved in the highest level of goal-directed activity, including the organization and planning of behavior (Damasio, 1979; Luria, 1980), the present study focused on individual differences in performance on neuropsychological tests of frontal-lobe function.

Some neuropsychological evidence associates frontal-lobe deficits with poorer regulation of human social behavior. Individuals with frontal-lobe damage often exhibit a "disinhibition syndrome" characterized generally by impulsivity and socially inappropriate behavior (Hecaen & Albert, 1978; Miller, 1987). If damage occurs early enough in life, it can result in the development of pervasive abnormalities of affective and social behavior (Eslinger & Damasio, 1986) and the inability to accommodate social impulses into the total personality structure (Ackerly & Benton, 1948).

Furthermore, some research links various forms of antisocial behavior with poor performance on putative frontal-lobe function measures (Buikhuisen, 1987; Lueger & Gill, 1990; Moffitt, 1990). However, one review concluded that despite the evidence supporting a specific relationship between violent criminal behavior and frontal-lobe dysfunction, alternative explanations could not be ruled out (Kandel & Freed, 1989).

A theoretical explanation for how impaired frontal-lobe function might lead to impaired regulation of social behavior is that there is a disturbance of the synthesis of external and internal cues underlying the regulation of complex behavior (Luria, 1980). As a consequence, behavior is governed more by impulse, current focus of attention, or salient stimulus cues than by rules or plans (Luria, 1980). Although this condition would not necessarily lead to increased aggression under normal circumstances, it might in situations where the salient cue is provocation and where peripheral or less contingent cues that inhibit aggression, such as fear of violence-related consequences, are

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lessened. Thus, an individual with impaired frontal-lobe function would be expected to demonstrate increased aggression under provocative conditions. Furthermore, as the behavior of this individual is governed more by the stimulus cue, level of frontal-lobe function would interact with provocation.

The interaction between frontal-lobe function and alcohol intoxication may be important for the following reason. Alcohol interferes with selected aspects of higher order cognition dependant on the intact structure of the prefrontal cortex (Peterson, Rothfleisch, Zelazo, & Pihl, 1990). Furthermore, acute alcohol intoxication interferes with the ability to integrate previously acquired knowledge in the formulation of behavioral strategies in a provocative situation (Lau & Pihl, in press), an ability thought to be influenced in part by the frontal cortex (Pihl, Peterson, & Lau, 1993). As alcohol interferes with cognitive abilities associated with the frontal lobes and impaired frontal-lobe function is thought to lead to increased aggression in provocative situations, individuals with reduced frontal function would be more vulnerable to the aggression-increasing effects of alcohol. This potential interaction may be important in understanding individual differences in alcohol-related aggression.

We designed the present study to investigate the main effects and interactions between provocation, acute alcohol intoxication, cognitive abilities associated with frontal lobe function, and aggression. We hypothesized that (a) all individuals would be more aggressive as provocation increased, (b) intoxicated individuals would be more aggressive than sober individuals, (c) individuals with lower cognitive performance would be more aggressive than those with higher cognitive performance, (d) provocation would interact with cognitive ability such that individuals with lower cognitive performance would respond to increased provocation with greater increases in aggression, and (e) intoxicated condition and cognitive ability would interact, such that alcohol would increase aggression more for lower cognitive performers.

Method

Participants

One hundred and fourteen volunteer nonalcoholic male social drinkers (a score of less than 5 on a short form of the Michigan Alcohol Screening Test; Pokorny, Miller, & Kaplan, 1972), aged 18–40, in good physical and mental health, were recruited through newspaper advertisements. Those receiving medical treatment that contraindicated alcohol consumption, who had sustained a serious injury to the head, or were familiar with psychological experimentation were excluded from participation. Women were excluded due to gender differences in physical aggression (Eagly & Steffen, 1986).

The remaining volunteers were assigned to one of two groups on the basis of their performance on two neuropsychological tests: total number of trials and incorrect guesses on the spatial conditional associative-learning task (Petrides, 1985) and the total number of errors on the self-ordered pointing task (Petrides & Milner, 1982). *Z*-score transformations were calculated for these three scores, which were added to determine a cumulative *z* score for each person. Participants whose cumulative *z* scores fell in the upper and lower performance quartiles (UQ and LQ, respectively) of the distribution were selected to complete the entire protocol.

Apparatus

Aggression was elicited and assessed with the Taylor (1967) competitive reaction-time task. In this study, the task board consisted of eight buttons, numbered consecutively from one to eight. Red lights situated above each button indicated the shock level chosen by the opponent when lit. An Apple II computer was used to run the aggression task and to record data. Shocks were administered through the Mark I Behaviour Modifier (Farrall Instruments), connected to an electrode attached to the inner forearm, below the elbow of the nondominant hand. Each person monitored administrations of shocks to his fictitious opponent by viewing a direct current ammeter provided for that purpose. A prerecorded videotape of the opponent receiving instructions regarding performance of the aggression task was played to each man on a Sony television connected to a Quasar VCR. This tape served to reinforce his belief in the existence of the opponent. The men's blood alcohol level (BAL) was determined using an Alco-sensor III (Thomas Ltd.). The men were also asked to rate themselves on a 7-point Likert-type "how drunk" scale. A score of 1 represented a rating of sobriety; 7 represented a rating of the most intoxicated the man had ever been.

The spatial conditional associative-learning task and self-ordered pointing task were used to separate participants into the two groups. In the former, each one of six randomly placed lamps was paired with one of six white cards. None of the men were informed of the pairings. The lamps were randomly lit, one at a time whereupon each man was to touch the cards one at a time until he touched the one that was paired with the lamp. Each person's task was to learn these associations so that when a given light was presented, the correct card would be chosen. Individuals with unilateral frontal-lobe damage have been shown to perform poorly on this task; the impairment on this task appears to be due to difficulties in learning to choose from a set the appropriate response to a given stimulus (Petrides, 1985).[1]

In the self-ordered pointing task, participants were presented 12 representational drawings of familiar objects arranged in a 3 × 4 matrix on each of 12 pages. The same designs appeared on each page; however, the positions of the drawings were different and randomly determined for each page. Participants were told to point to a different design on each page without choosing any given design more than once. This test theoretically measures organizational ability and sequencing of responses rather than the reproduction of sequences preorganized by the experimenter. Individuals with unilateral frontal-lobe damage are significantly impaired on this task; the deficits can be attributed either to poor monitoring of responses or poor organizational strategies, or both (Petrides & Milner, 1982).[2]

The Information, Block Design, and Vocabulary subtests of the Wechsler Adult Intelligence Scale–Revised (WAIS-R) were administered to provide estimates of full-scale IQ (Brooker & Cyr, 1986) and overall cognitive ability.

Procedure

Respondents to newspaper advertisements, who met inclusion criteria, were asked not to consume drugs or alcohol for at least 24 hr prior to testing. All participants signed an informed consent form and provided demographic data including age, years of education, subjective

[1] Positron emission tomography with magnetic resonance imaging of the brains of normal volunteers completing a modified version of this task demonstrated activation of cytoarchitectonic area 8 of the dorsolateral frontal cortex (Petrides, Alivisatos, Evans, & Meyer, 1993).

[2] Positron emission tomography with magnetic resonance imaging of the brains of normal volunteers completing a modified version of this task demonstrated activation of cytoarchitectonic areas 46 and 9 of the mid-dorsolateral frontal cortex (Petrides et al., 1993).

report of alcoholic beverages consumed per week, and salary code. The latter was defined as annual personal income where each point represented an increment of $5,000. The participants then completed the battery of neuropsychological and intelligence tests.

Men in the UQ or LQ competed in the Taylor aggression task, usually within 7 days of the first testing session. Half of the men in each quartile were randomly assigned to the alcohol condition, the remainder to the sober condition. In the alcohol condition, the men were administered 1 milliliter per kg of body weight of 95% alcohol USP units in three drinks of a 1:7 alcohol:orange juice solution. In the sober condition, the men were administered three drinks of juice of equivalent volume. In each condition, participants were told explicitly what they were drinking.

Drinks were consumed over a 20-min period. A 20-min waiting period followed to allow the men in the alcohol condition time to reach near-peak BALs. BALs were then taken and each person rated himself on the "how drunk" Likert scale.

Each man's pain threshold for electric shock was determined by delivering a series of shocks from 0–255 units (0–5.61 milliamperes [mA], which increased stepwise by 5 units (5 units = 0.11 mA) at a constant rate. Each man was to press a button in response to any shock he regarded as painful (a) to stop the administration of the shock and (b) to reduce the level of the next shock by one step. Therefore, the next shock was one step lower than the shock that induced pressing the button. Pressing the button on 3 consecutive presentations of the same shock intensity stopped shock delivery. This shock intensity was defined as the man's pain threshold.

The aggression task was then introduced as a competitive reaction-time task. Each man was instructed to select a shock level that he would deliver to his opponent after winning a reaction-time trial. Following the reaction-time task, the person would be informed of the opponent's shock choice. The one who lost would receive that shock. Shock levels 1–8 increased linearly from 15% to 100% of the person's given pain threshold. If he won, he administered the previously chosen shock to his opponent. The experimenter then left briefly, telling each man that he was about to verify the readiness of the opponent. On his return, the experimenter stated that instructions were about to be delivered to the opponent, and that this delivery could be viewed on the television monitor. In fact, what was actually presented was a prerecorded videotape of a fictitious opponent. Three practice trials were then conducted.

The task itself consisted of 26 consecutive trials including a block of 12 trials followed by a transition trial, a second block of trials, and a final trial. The opponent's shock choices ranged from 1–4 in the first block and from 5–8 in the second block of trials. The order of wins and losses as well as the opponent's shock choices were randomly assigned by the computer. The opponent's shocks were all of the same duration. All participants received three shocks at each level alternately winning one trial and losing two trials versus winning two trials and losing one trial. All participants lost the transition trial and won the final trial. In both cases the opponent's shock choice was a 5.

Following the aggression task, the men completed a short questionnaire to verify the success of the deception. They rated their own and their opponent's performance on the aggression task and described how effective they thought the task was at measuring their reaction time. All the men were debriefed and the necessity for deception was fully explained. No one was adversely affected by the deception, according to self-report. The experimenter rated each man's deception on a Likert-type scale from 1–6. A score of 1 represented a rating of "not deceived"; 6 represented a rating of "totally deceived." Intoxicated participants were retained in the laboratory until their BAL dropped below one third of 1%. Each person was paid $5 an hour to compensate for lost time.

The objective measures of aggression were the intensity of shock each man selected for each provocation level (1–8) for trials following a loss (receipt of a shock) and a win (receipt of information regarding the opponent's choice of shock level). The first measure reflects an individual's response to physical provocation, whereas the second measure reflects an individual's response only to the opponent's intentions. The first shock choice was made before the first reaction-time test but after three practice trials with the opponent, and so it was not included in the analysis.

Results

Participant Measures

The two tests were completed by 114 men. Fifty-six men with LQ and UQ cumulative test z scores for the entire distribution were selected for further participation in the study; 48 of them (24 UQ and 24 LQ) completed the entire protocol. (Two men from the LQ were not deceived and were excluded from the analysis, and 6 others—2 from the LQ and 4 from the UQ—did not return to complete the testing.) Mean demographics by quartile were as follows: for age, LQ $M = 23.6$, $SD = 5.3$, UQ $M = 24.4$, $SD = 5.1$; for years of education, LQ $M = 12.7$, $SD = 2.2$, UQ $M = 14.9$, $SD = 1.8$; for salary code, LQ $M = 1.9$, $SD = 1.2$, UQ $M = 2.4$, $SD = 1.4$; for beverages per week, LQ $M = 6.6$, $SD = 7.3$, UQ $M = 7.8$, $SD = 7.1$; and for IQ, LQ $M = 97$, $SD = 7$, UQ $M = 111$, $SD = 11$. Separate two-tailed t tests revealed that quartile groups differed in mean years of education and IQ, $t(45) = 3.85$, $p < .0001$, and $t(46) = 5.24$, $p < .0001$, respectively.

Neuropsychological Test Measures

Separate two-tailed t tests revealed significant differences ($ps < .0001$) between each quartile's mean scores for the conditioned associative-learning task trials (CAT: LQ $M = 175$, $SD = 12$; UQ $M = 51$, $SD = 17$; $t[46] = -29.1$) and total number of conditioned associative-learning task errors (CAE: LQ $M = 159$, $SD = 54$; UQ $M = 20$, $SD = 11$; $t[46] = -12.2$), and for self-ordered pointing errors (SOPE: LQ $M = 5.1$, $SD = 2.4$; UQ $M = 1.5$, $SD = 1$; $t[46] = -6.8$). Correlations between CAT and CAE on the conditional-associative learning task and SOPE on the self-ordered pointing task were as follows: CAT and CAE, $r = .83$, $p < .0001$; CAT and SOPE, $r = .45$, $p < .0001$; CAE and SOPE, $r = .39$, $p < .0001$; coefficient $\alpha = .69$.

Alcohol Measures

Participants were tested on the aggression paradigm sober (BAL $M = 0\%$, $SD = 0.00$) or intoxicated (BAL $M =$ < one tenth of 1%, $SD = 0.02$; "how drunk" scale $M = 3.8$, $SD = 1.1$). Separate two-tailed t tests revealed that the mean BAL for the intoxicated subjects of the LQ ($M =$ one tenth of 1%, $SD = 0.01$) and UQ ($M =$ less than one tenth of 1%, $SD = 0.03$) and mean "how drunk" ratings of the LQ ($M = 3.8$, $SD = 1.4$) and UQ ($M = 3.8$, $SD = 0.8$) did not differ significantly.

Deception Measure

Typically, the men did not question the existence of the opponent when completing the short questionnaire to verify the success of the deception. All but two were classified as being deceived. A 2 (quartile) × 2 (drug) analysis of variance (ANOVA) conducted on the 6-point deception scale revealed that the

mean deception rating of the LQ (M = 4.1, SD = 1.3) and UQ (M = 4.3, SD = 1.0) did not differ significantly, nor were there any significant interactions.

Pain Threshold Measures

A 2 (quartile) × 2 (drug) ANOVA conducted on pain threshold revealed a significant main effect of quartile, $F(1, 44) = 9.00$, $p < .01$. Individuals in the UQ had lower pain thresholds for electric shock than those in the LQ, M = 99, SD = 77, and M = 171, SD = 88, respectively.

Shock Intensity Measures

A 2 (quartile) × 2 (drug) × 2 (response condition: loss–win) × 8 (provocation) mixed design ANOVA, with response condition and provocation as repeated measures, conducted on shock intensity was done to compare the men's responses following wins and losses. This analysis revealed a significant main effect of response condition, $F(1, 44) = 26.52$, $p < .0001$, with participants choosing higher mean shock intensities following a loss and receipt of a shock (M = 4.1) than after winning a trial and administering a shock (M = 3.5). In addition, there was a significant interaction between response condition and provocation, $F(7, 308) = 3.27$, $p < .01$). As a result, the data for the two response conditions were analyzed separately.

A 2 (quartile) × 2 (drug) × 8 (provocation) mixed-design ANOVA, with provocation as the repeated measure, conducted on shock intensity chosen after a loss, using Huyn-Feldt conservative degrees of freedom, revealed significant main effects for quartile (Ms = 3.5 and 4.7 for UQ and LQ, respectively; $F[1, 44] = 7.29$, $p < .01$); drug (Ms = 3.7 and 4.5 for UQ and LQ, respectively; $F[1, 44] = 4.04$, $p < .05$); and provocation ($F[7, 308] = 21.20$, $p < .0001$). The effect for provocation was decomposed into a linear and pooled-nonlinear trend. The results indicated a significant linear trend, $F(1, 308) = 136$, $p < .0001$, but no significant nonlinear trend, $F(6, 308) = 2.07$.

Furthermore, there was a significant interaction between provocation and quartile, $F(7, 308) = 2.67$, $p < .05$. The interaction between quartile and the linear trend for provocation was significant, $F(1, 308) = 11.6$, $p < .001$, indicating that the slopes of the two quartiles differed. Thus, individuals of the LQ showed a greater increase in mean shock intensity as provocation increased (Figure 1).[3] Analysis of the pooled nonlinear portion of the interaction did not reveal any additional differential pattern of responding, $F(6, 308) = 1.25$.

A 2 (quartile) × 2 (drug) × 8 (provocation) mixed-design ANOVA, with provocation as the repeated measure, conducted on shock intensity chosen after a win, revealed a significant main effect for provocation, $F(7, 308) = 4.73$, $p < .0001$. The effect for provocation was decomposed into a linear and pooled-nonlinear trend. The results showed a significant linear trend, $F(1, 308) = 30.8$, $p < .0001$, but no significant nonlinear trend, $F(6, 308) = 0.40$. The main effects for drug (Ms = 3.2 and 4.0 for UQ and LQ, respectively), $F(1, 44) = 3.29$, $p = .0764$, and quartile (Ms = 3.1 and 4.0 for UQ and LQ respectively), $F(1, 44) = 4.03$, $p = .051$, were marginally significant.

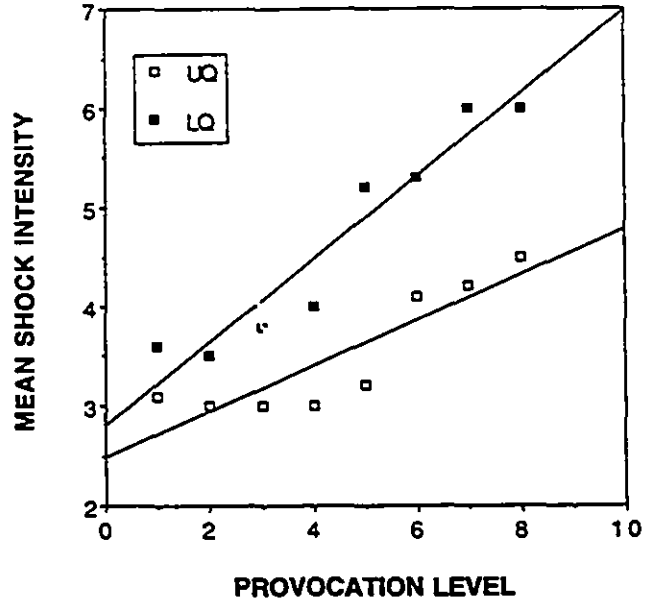

Figure 1. Mean shock intensity chosen after a loss, by provocation level, for cognitive performances in the upper and lower quartile (UQ and LQ, respectively) groups. See text for details.

Discussion

The results of this study support the previously reviewed work that has shown that provocation heightens aggression during a competitive task and that alcohol intoxication increases aggression. In addition, it is significant that the results also show that individuals grouped according to performance on two neuropsychological tests of cognitive abilities associated with frontal-lobe function differ in degree of aggressive response. Specifically, individuals in the lower performance quartile become even more aggressive when provoked.

The notion that reduced frontal-lobe function may partially disinhibit aggressive behavior is one explanation for these results. If decreased frontal-lobe function impairs an individual's ability to use socially relevant information (Dimond, 1980),

[3] Separate correlations of IQ, years of education, and pain threshold on individual mean shock intensity revealed that IQ was significantly correlated with mean shock intensity ($r = -.32$, $p < .05$). However, neither years of education nor pain threshold were significantly correlated with shock intensity. In addition, IQ was not significantly correlated with scores on the two neuropsychological tests. To control for the possible confounding effect of IQ, this variable was included as a covariate in the above ANOVA. However, there was a significant interaction between quartile and IQ, $F(1, 40) = 6.18$, $p < .05$, which violates the assumption of homogeneity of slopes on which the analysis of covariance rests. Therefore, IQ was included as a continuous independent variable in a fully saturated model as a main effect and in interaction with the categorical independent variables. These analyses revealed that the main effect of quartile was not affected, $F(1, 40) = 5.32$, $p < .05$. Furthermore, the main effect of IQ was not significant. Comparable analyses conducted on years of education and pain threshold did not substantially affect the results.

specifically through deficits in the internalization of inhibitory influences, these individuals should respond more aggressively when presented with provocation or punishment. This idea is consistent with observations that monkeys with frontal cortical ablations are more labile in social interactions and substantially more aggressive (Dimond, 1980). Furthermore, reduced performance on tasks reflecting abilities associated with frontal-lobe functioning has been shown to predict fighting in young boys (Seguin, Harden, Pihl, & Tremblay, 1993).

Contrary to expectations, there was no significant interaction between quartile and alcohol intoxication. The lack of an interaction may be due to a ceiling effect on shock intensity choice for LQ individuals. However, the mean shock intensity of approximately 5 chosen by both LQ groups is well below the maximum potential intensity of 8. Alternatively, the absence of an interaction may have been due to low statistical power.

The possibility that the results of this study were due to group differences in IQ is unlikely. Controlling for IQ in the analysis did not substantially affect the results. Thus, group differences in neuropsychological test scores predicted aggression beyond any prediction provided by IQ. Furthermore, IQ and neuropsychological test scores were not significantly correlated. A more likely explanation is that some IQ deficit is to be expected with a lifelong frontal deficit. Although the two neuropsychological tests used in this study are not thought to be particularly sensitive to variations in IQ (Petrides, 1985; Petrides & Milner, 1982), these tests were validated on individuals who had sustained frontal-lobe damage later in life, when IQ is not necessarily affected by such insult (Black, 1976).

It might be argued that this study measured degree of aggression in conditions where only an aggressive response was permitted. However, the presence of the lowest level shock button was clearly explained, and its use was not discouraged. In addition, there is research using paradigms that include a nonaggressive response option where alcohol has been shown to specifically affect aggressive responding (Cherek, Steinberg, & Manno, 1985).

In conclusion, the present study demonstrates that determinants such as provocation, drug effects, and the preexistent cognitive abilities of an individual increase the likelihood of aggression. More importantly, preexisting cognitive abilities and provocation have been shown to interact to predict aggressive behavior.

References

Ackerly, S. S., & Benton, A. L. (1948). Report of a case of bilateral frontal lobe defect. *Research on Nervous and Mental Diseases, 27,* 479–504.

Baron, R. A., & Richardson, D. R. (1994). *Human aggression* (2nd ed.). New York: Plenum Press.

Black, F. W. (1976). Cognitive deficits in patients with unilateral war-related frontal lobe lesions. *Journal of Clinical Psychology, 32,* 366–372.

Brooker, B. H., & Cyr, J. J. (1986). Tables for clinicians to use to convert WAIS-R Short Forms. *Journal of Clinical Psychology, 42,* 983–984.

Buikhuisen, W. (1987). Cerebral dysfunctions and persistent juvenile delinquency. In S. A. Mednick, T. E. Moffitt, & S. A. Stack (Eds.), *The causes of crime: New biological approaches* (pp. 168–184). Cambridge: Cambridge University Press.

Bushman, B. J., & Cooper, H. M. (1990). Effects of alcohol on human aggression: An integrative research review. *Psychological Bulletin, 107,* 341–354.

Cherek, D. R., Steinberg, J. L., & Manno, B. R. (1985). Effects of alcohol on human aggressive behaviour. *Journal of Studies on Alcohol, 46,* 321–328.

Damasio, A. (1979). The frontal lobes. In K. M. Heilman & E. Valenstein (Eds.), *Clinical neuropsychology* (pp. 360–412). New York: Oxford University Press.

Dimond, S. J. (1980). *Neuropsychology: A textbook of systems and psychological functions of the human brain.* London: Butterworth.

Eagly, A. H., & Steffen, V. J. (1986). Gender and aggressive behaviour: A meta-analytic review of the social psychological literature. *Psychological Bulletin, 100,* 309–330.

Eslinger, P. J., & Damasio, A. R. (1986). Severe disturbances of higher cognition after bilateral frontal lobe ablation: Patient EVR. *Neurology, 35,* 1731–1741.

Hammock, G. S., & Richardson, D. R. (1992). Predictors of aggressive behaviour. *Aggressive Behaviour, 18,* 219–229.

Hecaen, H., & Albert, M. L. (1978). *Human neuropsychology.* New York: Wiley.

Kandel, E., & Freed, D. (1989). Frontal-lobe dysfunction and antisocial behaviour: A review. *Journal of Clinical Psychology, 45,* 404–413.

Lau, M. A., & Pihl, R. O. (in press). Alcohol and the Taylor Aggression Paradigm: A repeated measures study. *Journal of Studies on Alcohol.*

Lueger, R. J., & Gill, K. J. (1990). Frontal-lobe cognitive dysfunction in conduct disorder adolescents. *Journal of Clinical Psychology, 46,* 696–706.

Luria, A. R. (1980). *Higher cortical functions in man* (2nd ed.). New York: Basic Books.

Miller, L. (1987). Neuropsychology of the aggressive psychopath: An integrative review. *Aggressive Behaviour, 13,* 119–140.

Moffitt, T. E. (1990). The neuropsychology of juvenile delinquency: A critical review. In M. Tonry & N. Morris (Eds.), *Crime and justice: A review of research* (pp. 99–169). Chicago: University of Chicago Press.

Murdoch, D., Pihl, R. O., & Ross, D. (1990). Alcohol and crimes of violence: Present issues. *International Journal of Addiction, 25,* 1059–1075.

Peterson, J. B., Rothfleisch, J., Zelazo, P. D., & Pihl, R. O. (1990). Acute alcohol intoxication and cognitive functioning. *Journal of Studies on Alcohol, 51,* 114–122.

Petrides, M. (1985). Deficits on conditional associative-learning tasks after frontal- and temporal-lobe lesions in man. *Neuropsychologia, 23,* 601–614.

Petrides, M., Alivisatos, B., Evans, A. C., & Meyer, E. (1993). Dissociation of human mid-dorsolateral from posterior dorsolateral frontal cortex in memory processing. *Proceedings of the National Academy of Sciences, 90,* 873–877.

Petrides, M., & Milner, B. (1982). Deficits on subject-ordered tasks after frontal- and temporal-lobe lesions in man. *Neuropsychologia, 20,* 249–262.

Pihl, R. O., Peterson, J. B., & Lau, M. A. (1993). A biosocial model of the alcohol-aggression relationship. *Journal of Studies on Alcohol, Supplement 11,* 128–139.

Pokorny, A. D., Miller, B. A., & Kaplan, H. B. (1972). The brief MAST: A shortened version of the Michigan Alcoholism Screening Test. *American Journal of Psychiatry, 129,* 342–345.

Prothrow-Stith, D. (1990). The epidemic of violence and its impact on the health care system. *Henry Ford Hospital Medical Journal, 38,* 175–177.

Seguin, J. R., Harden, P., Pihl, R. O., & Tremblay, R. E. (1993). Prediction of fighting in children: The contribution of frontal lobe "type" deficits. *Canadian Psychology, 34,* 227.

Tancredi, L. R., & Volkow, N. (1988). Neural substrates of violent behaviour: Implications for law and public policy. *International Journal of Law and Psychiatry, 11*, 13–49.

Taylor, S. P. (1967). Aggressive behaviour and physiological arousal as a function of provocation and the tendency to inhibit aggression. *Journal of Personality, 35*, 297–310.

U.S. Bureau of the Census (1991). *Statistical abstract of the United States 1991* (111th ed.). Washington, DC: U.S. Government Printing Office.

Wood, W., Wong, F. Y., & Chachere, J. G. (1991). Effects of media violence on viewers' aggression in unconstrained social interaction. *Psychological Bulletin, 109*, 371–383.

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BRIDGE TO STUDY 3

The most significant result of this study is the demonstration that impaired neuropsychological function was associated with increased aggression. This result is important because it is the first demonstration of this relationship under tightly controlled conditions. These include the use of an appropriate comparison group, a clear operational definition of aggression, and neuropsychological tests shown to be associated with specific areas of the frontal cortex. In addition, the study design allowed investigation of the interaction between individual and situational factors.

This is meaningful because previous investigations of this relationship are plagued by a number of methodological issues which have severely limited the strength of the conclusions that can be drawn (Kandel & Freed, 1989; Raine, 1993). These studies have been criticised for a lack of appropriate control groups and a reliance on official court records. In addition, it is a rare occurrence that the neuropsychological measures used have been shown to be associated with specific brain areas. Finally, these studies tend to ignore, with a few exceptions (e.g. Moffitt, 1990; Denno, 1989) the interaction between environmental and biological factors. Thus, these results provide important convergent validity to the results of naturalistic surveys of neuropsychological deficits and aggressive or violent behaviour (Kandel & Freed, 1989; Raine, 1993; Seguin et al., 1995).

A major question in this research area that these studies have not yet addressed is the causal direction of this association. Evidence from prospective studies have demonstrated that poor neuropsychological status predict specifically male delinquency that begins before age 13 (e.g. Moffitt et al., 1994). However, these results do

not specifically address the issue of neuropsychological dysfunction and aggressive or violent behaviour. Thus, further investigation as to how impaired neuropsychological test performance is related to increased aggression under laboratory conditions is warranted.

One theoretical explanation for the increased aggressive behaviour of individuals with poor neuropsychological performance is that the cognitive deficits represented an impairment in the ability to inhibit inappropriate or impulsive behaviours. This is predicated on the notion that the cognitive functions assessed by the neuropsychological tests employed in Study 2 are thought to depend on the intact function of the frontal lobes (Petrides, 1985a; Petrides et al., 1993; Petrides & Milner, 1982). The frontal lobes, in turn, are thought to be the neural substrate for a variety of functions including inhibitory behaviour (Fuster, 1989; Milner & Petrides, 1984). Thus, Study 3 was performed to investigate the above hypothesis by manipulating the presence of inhibitory cues during the aggression task for individuals with high versus low neuropsychological performance. Specifically, this hypothesis was tested by offering high and low cognitive performers monetary reward to inhibit their aggression. It was hypothesized that high versus low cognitive performers would show greater reductions of aggression in response to the monetary reward.

STUDY 3

Cognitive Performance, Inhibition and Aggression

COGNITIVE PERFORMANCE, INHIBITION AND AGGRESSION

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Running Head: COGNITIVE - LAU

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Abstract

Individuals with impaired cognitive abilities associated with frontal lobe function have recently been shown to manifest increased aggression. This study investigated the effect of contingent money to inhibit aggression in these individuals. 140 males, aged 18-40, were ranked according to their performance on the spatial conditional associative-learning task. Forty-eight males (24 with scores in each of the upper and lower performance quartiles) participated in the full study. Half of the subjects competed in a Taylor aggression task where they received greater monetary reward for choosing lower shocks; the remainder competed without any monetary contingency. Aggression was defined as shock intensity delivered to a sham opponent. Shock intensity significantly increased as a main effect of lower cognitive performance, absence of monetary reward and provocation. In addition, provocation interacted significantly with test performance. Finally, individuals in the upper cognitive performance quartile showed significantly greater reductions of unprovoked aggression in response to monetary reward. The results are discussed in terms of impairments in the ability to integrate inhibitory influences in the formulation of behavioral strategies in aggressive situations.

Introduction

Interpersonal violence presents a serious challenge to North American society with more than a million and a half violent crime victims annually [U.S. Bureau of the Census, 1993]. In response to the magnitude of this problem, public policy is now shifting to one that stresses the importance of focusing on causative factors underlying violent behaviour [Mercy, Rosenberg, Powell, Broome & Roper, 1993].

While mechanisms underlying aggression are clearly multifactorial [Baron & Richardson, 1994], cognitive processes including control, planning of behaviours, and consciousness of an act and its consequences have been increasingly implicated in importance [Dodge & Crick, 1990; Huesmann, 1988; Tancredi & Volkow, 1988]. Specifically, neuropsychological studies of adult criminal and juvenile delinquent populations commonly find a suggestion of frontal lobe dysfunction [Moffitt, 1990, Moffitt, Lynam & Silva, 1994]. These deficits are often taken as underlying the antisocial behaviour [Buikhuisen, 1987; Lueger & Gill, 1990; Moffitt & Henry, 1989] and specifically, in some cases, violent criminal behaviour [Raine, 1993; Tarter, Hegedus, Winsten, & Alterman, 1984; Yeudall & Fromm-Auch, 1979; Yeudall, Fromm-Auch, & Davies, 1982]. However, one review concluded that a general lack of methodological rigor must limit conclusions [Kandel & Freed, 1989].

Increased aggression in the laboratory, however, has recently been associated with impaired performance on two neuropsychological tests putatively assessing frontal lobe function [Lau, Pihl & Peterson, 1995]. Although, increased aggression has been associated with poor performance on only one of these tests, the spatial conditional associative learning task [CALT; Petrides, 1985] has been associated with increased laboratory

aggression in young males [Giancola & Zeichner, 1994] as well as fighting in young boys [Seguin, Pihl, Harden, Tremblay & Boulerice, 1995].

A theoretical explanation for how frontal lobe dysfunction may lead to problematic regulation of aggression is found in the suggestion of an impairment in the ability to inhibit inappropriate or impulsive behaviours. The frontal lobes, considered essential for context dependant behaviour [Dimond, 1980; Pribram, 1973], are thought to calculate appropriate behavioral responses, in part, by integrating information about the state of the external and internal worlds [Fuster, 1989; Luria, 1980, Nauta, 1971]. Impaired frontal function might result in a failure to inhibit inappropriate responses possibly due to a restricted evaluation of personal and social consequences. As a result of an impairment in this ability, behaviour would be governed more by salient stimulus cues than by rules or plans [Luria, 1980]. With respect to aggression, impaired frontal function might interfere with the ability to process the contingency cues that normally inhibit aggressive behaviour. Thus, increased likelihood of aggression would result in situations where the probability of aggression is ambiguous or the salient cue is provocation and the salience of contingent cues which inhibit aggression are lessened.

As reduced performance on the CALT appears to reflect difficulties in learning to choose from a set the appropriate response to a given stimulus, one possibility for why individuals who performed poorly on the CALT were more aggressive is that they were impaired in their ability to learn to associate inhibitory cues with provocative stimuli.

To test this hypothesis, this study used money as a contingency to inhibit aggressive responding. Contingent

money has been shown previously to influence aggressive behaviour on the Taylor [1967] competitive reaction-time task. Specifically, subjects given money for choosing more intense shocks became more aggressive [Borden, Bowen, & Taylor, 1971]. This finding raised the possibility that aggressive responding can also be inhibited by providing greater monetary reward for choosing lower shocks. As individuals demonstrating poor performance on the CALT are hypothesized to be less able to utilize inhibitory cues to constrain their aggression, these individuals would be expected to be less affected by contingent monetary reward. Thus, this study compared the effect of contingent monetary reward to inhibit aggressive responding for two groups of individuals differing in performance on the spatial conditional associative-learning task. Half of the subjects in each group were offered greater amounts of money for choosing lower shocks while competing with their opponents on the Taylor task. It was specifically hypothesized that high versus low cognitive performers would show greater reductions of their aggression in response to the monetary reward.

Method

Subjects

One hundred and forty volunteer non-alcoholic male social drinkers (who received a score of less than 5 on a short form of the Michigan Alcohol Screening Test [Pokorny, Miller, & Kaplan, 1972]), aged 18-40, in good physical and mental health (based on self-report), recruited through newspaper advertisements, participated in this experiment. Individuals who had sustained a serious head injury (defined as any head injury resulting in loss consciousness) and who had familiarity with psychological experimentation were excluded from participation. Females were excluded from participation

as men are more aggressive than women, particularly with respect to physical aggression [Eagly & Steffen, 1986].

Subjects were assigned to one of two groups based on the total number of trials and incorrect guesses on the spatial conditional associative-learning task [Petrides, 1985]. Z-score transformations were completed on these two scores which were added to determine a cumulative z-score for each subject. Subjects whose cumulative z-scores fell in the upper (UQ) and lower (LQ) performance quartiles of the distribution were selected to complete the entire protocol.

Apparatus

Aggression was elicited and assessed with a modified form of the Taylor [1967] competitive reaction-time task [Lau et al., 1995]. In this study, the task board consisted of eight consecutively numbered buttons. In the monetary reward condition, a cardboard display of monetary values was placed above the buttons and a counter was provided for the subject to monitor the amount of money corresponding to his button choice, and upon the completion of each trial, the total amount of money earned up to that trial.

The spatial CALT consisted of six randomly placed lamps each paired with one of six white cards. The subject was not informed of the pairings. The lamps were randomly lit, one at a time whereupon the subject was to touch the cards one at a time until he touched the one that was paired with the lamp. The subject's task was to learn these associations so that when a given light was presented, the correct card would be chosen. Individuals with unilateral frontal lobe damage have been shown to perform poorly on this task; the impairment on this task appears to be due to difficulties in learning to choose from a set the appropriate response to a given stimulus [Petrides, 1985].[1]

The SOP task [Petrides & Milner, 1982] was included in the test battery to determine whether this task predicted aggression scores over and above the prediction provided by the CALT. This test theoretically measures organizational ability and sequencing of responses rather than the reproduction of sequences pre-organized by the experimenter. Individuals with unilateral frontal lobe damage are significantly impaired on this task; the deficits can be attributed either to poor monitoring of responses or poor organizational strategies or both [Petrides & Milner, 1982].[2]

The block-tapping task [Corsi, 1972] was administered to assess cognitive abilities associated with right temporal lobe function. Individuals with unilateral right temporal-lobe damage have been shown to be impaired on this task [Corsi, 1972]. As individuals with lesions of the right temporal lobe with radical involvement of the hippocampal region also exhibit impaired performance on the CALT [Petrides, 1985], this task was included to rule out the possibility that the impaired performance on the CALT of individuals of the lower quartile was due to impaired right temporal lobe function. Briefly, the subject was required to tap out on 9 fixed blocks exactly the same pattern tapped out by the examiner. First, the subject's immediate span, that is, the longest pattern that the subject can successfully repeat, was determined. Following this, a series of 24 patterns, one block in excess of the patient's immediate span in length, were presented. Every third block sequence was repeated with the intervening sequences occurring only once. Two scores consisting of the number of 1) recurring block sequences (7 maximum) and, 2) the number of non-recurring sequences tapped in the correct order, were obtained. Finally, the Block Design and Vocabulary subtests of the WAIS-R were administered to

provide estimates of full scale IQ [Brooker & Cyr, 1986] and overall cognitive ability.

Procedure

Respondents to newspaper advertisements, who met inclusion criteria, were asked not to consume drugs or alcohol for at least 24 hours prior to testing[3]. All subjects signed an informed consent form and provided demographic data including age, the subject's and his parents years of education, self report of alcoholic beverages drank per week, salary code[4] and neighbourhood code (defined as an ordinal scale of the type of housing and the economic status of the people who live in it). The subjects then completed the battery of neuropsychological and intelligence tests. These tests were administered by undergraduate honours psychology students under the supervision of a pre-doctoral graduate student. Subjects of the upper or lower performance quartiles competed in the Taylor aggression task during a second session anywhere from 3-45 days (M = 9.24) following the screening session. First, each subject's pain threshold for electric shock was determined by delivering a series of shocks which increased stepwise at a constant rate [Lau et al., 1995]. The shock intensity which the subject indicated as painful upon three consecutive presentations was defined as the subject's pain threshold.

Second, the aggression task was introduced as a competitive reaction-time task [Lau et al., 1995]. Briefly, each subject was instructed to select a shock level that he would deliver to his opponent upon winning a reaction-time trial. If he lost he received a shock ostensibly chosen by the opponent. The task itself consisted of 26 consecutive trials including a block of 12 trials followed by a transition trial, a second block of trials and a final trial. The opponent's shock

choices ranged from 1-4 in the first block and from 5-8 in the second block of trials. The order of wins and losses as well as the opponent's shock choices were randomly assigned by the computer. The opponent's shocks were all of the same duration.

Half of the subjects in each quartile were randomly assigned to the control condition, the remainder to the inhibition condition. The experimenter who ran the aggression task, a pre-doctoral graduate student, was blind to the subject's grouping. In the control condition, there was no monetary reward associated with the subject's shock choice. In the inhibition condition, subjects received forty cents for choosing shock level one, with the amounts decreasing by five cents for each level to a value of 5 cents for selecting shock level eight. Subjects were told that they would receive the amount of money appearing above their shock choice regardless of whether they won or lost the reaction time trial. The subject would receive the total amount of money displayed on the counter at the end of the all the trials.

Following the aggression task, subjects were interviewed to verify the success of the deception. Each subject was asked a series of 6 questions designed to solicit comments concerning the task. These questions inquired as to the subject's; 1) shock selection strategy, 2) description of his opponent, 3) understanding of the experimental procedures, 4) feelings and reactions, as well as 5) whether the subject found any aspect of the task odd, confusing or disturbing and 6) if there might have been more to the experiment than meets the eye. Both the experimenter and the subject rated the subject's deception on a Likert type scale from 1-5 based on the subject's response to the battery of questions. A score of one represented a rating of "not

deceived". Five represented a rating of "totally deceived". Following this, each subject was debriefed and the necessity for deception was fully explained. No subject was adversely affected by the deception, according to self-report. All subjects were paid $5.00 an hour to compensate for lost time.

The objective measures of aggression were the intensity of shock the subject selected: 1) for the first trial, 2) for each provocation level (1-8) following a loss (receipt of a shock) and, 3) for each provocation level (1-8) following a win (receipt of information regarding the opponent's choice of shock level). The first measure reflects unprovoked aggression as it is selected before the subject receives any information regarding the opponent's shock choices. The second measure reflects an individual's response to physical provocation whereas the third measure reflects an individual's response only to the opponent's intentions.

Results

Subject Measures

140 subjects completed the spatial conditional associative-learning task. 70 subjects with cumulative test z-scores in either the lower (LQ) or upper (UQ) performance quartiles for the entire distribution were selected for further participation in the study. Twelve subjects (6 from the LQ and 6 from the UQ) were not deceived and were excluded from the analysis. Ten additional subjects (5 from each quartile) did not return for complete testing. Thus, the following analysis is based on 48 subjects (UQ/LQ; N=24/24).

Mean age, years of education, father's years of education, mother's years of education, beverages per week, salary code, neighbourhood code and IQ are presented in Table 1. Separate 2 (quartile) x 2 (condition) ANOVAs conducted on all of these variables

(except for beverages per week) revealed that quartile groups differed in mean age ($F(1,44) = 4.75$, $p < .01$), years of education ($F(1,44) = 4.75$, $p < .05$) and IQ ($F(1,44) = 10.00$, $p < .01$). As the data for beverages per week was severely skewed, a nonparametric analysis was done which revealed no significant group differences.

Neuropsychological Test Measures

Mean scores on the spatial conditional associative-learning task, self ordered pointing task and block-tapping task are also presented in Table 1. Separate 2 (quartile) x 2 (condition) ANOVAs revealed that the mean error scores on the spatial conditional associative-learning task (total number of trials (CALTT) and total number of errors (CALTE)), and the self ordered pointing task (total number of errors (SOPE)) for each quartile were significantly different ($p < .0001$). CALTT and CALTE scores on the CALT were significantly correlated ($r = .89$, $p < .0001$). In addition, SOPE scores were significantly correlated with CALTT ($r = .48$, $p < .001$) and CALTE ($r = .40$, $p < .01$).

INSERT TABLE 1 ABOUT HERE

Deception Measures

Typically, the subjects did not question the existence of the opponent during the debriefing interview. Twelve subjects with a combined experimenter and self rating of 6 or less were classified as not deceived. Separate 2 (quartile) x 2 (condition) ANOVAs conducted on the 5-point deception scale as rated by the experimenter, and the subject, revealed no significant main effects nor interactions. Thus, neither mean deception ratings differed significantly between quartiles (see Table 1). Furthermore, a correlational analysis revealed that neither deception rating was significantly correlated with mean shock intensity.

Pain Threshold Measures

A 2 (quartile) x 2 (condition) ANOVA conducted on pain threshold revealed no significant main effects or interactions (see Table 1).

Initial Shock Setting

A 2 (quartile) x 2 (condition) ANOVA conducted on the initial shock choice revealed a significant interaction between quartile and condition ($F(1,44) = 5.34$, $p < .05$). Simple main effects analysis showed that for the UQ, individuals in the inhibition condition selected significantly lower mean initial shock settings ($M = 1.6$) than those of the control condition ($M = 3.3$; $F(1,44) = 7.77$, $p < .05$). However, for the LQ, mean initial shock intensities chosen by individuals of the inhibition ($M = 3.3$) and control ($M = 3.1$) conditions were quite similar (Figure 1).[5]

INSERT FIGURE 1 ABOUT HERE

Shock Intensity Measures

A 2 (quartile) x 2 (condition) x 2 (response condition: loss/win) x 8 (provocation) mixed design ANOVA, with response condition and provocation as repeated measures, conducted on shock intensity was done to compare the subjects' responses following wins and losses. This analysis revealed, using Huyn-Feldt conservative degrees of freedom, a significant main effect of response condition ($F(1,44) = 3.99$, $p = .05$), a significant interaction of response condition with provocation ($F(7,308) = 3.52$, $p < .01$) and a significant interaction between quartile, response condition and provocation ($F(7,308) = 2.17$, $p < .05$). Due to the interactions of response condition with other factors in the design, the data for the two response conditions were analyzed separately.

A 2 (quartile) x 2 (condition) x 8 (provocation) mixed-design ANOVA, with provocation as the repeated measure, conducted on shock intensity chosen after a

loss, using Huyn-Feldt conservative degrees of freedom, revealed significant main effects for quartile ($F(1,44) = 6.28$, $p < .05$), condition ($F(1,44) = 6.03$, $p < .05$) and provocation ($F(7,308) = 17.36$, $p < .0001$). Furthermore, there were significant interactions of provocation with quartile ($F(7,308) = 2.20$, $p < .05$) and provocation with condition ($F(7,308) = 2.35$, $p < .05$).

The interaction between quartile and the linear trend for provocation was significant ($F(1,308) = 11.13$, $p < .01$) indicating that the slopes of the two quartiles differed. Individuals of the LQ showed a greater increase in mean shock intensity as provocation increased (Figure 2). Analysis of the pooled non-linear portion of the interaction did not reveal any additional differential pattern of responding.

INSERT FIGURE 2 ABOUT HERE

The interaction of condition and the linear trend for provocation was significant ($F(1,308) = 6.33$, $p < .05$) indicating that the slopes of the two conditions differed. Individuals in the control condition showed a greater increase in mean shock intensity as provocation increased. Deviations from linearity were non-significant.

Planned comparisons of mean shock intensity of the control versus the inhibition condition for each quartile were done to test the hypothesis that individuals of the UQ would reduce their aggression more than those of the LQ in the inhibited condition. This analyses demonstrated that individuals of the UQ selected significantly lower mean shock intensities in the inhibited ($M = 2.4$) versus control conditions ($M = 3.7$; $p < .05$). The mean shock intensities selected by individuals in the LQ did not differ significantly between the inhibition ($M = 3.7$) and control conditions ($M = 4.4$).

A 2 (quartile) x 2 (condition) x 8 (provocation) mixed-design ANOVA, with provocation as the repeated measure, conducted on shock intensity chosen after a win, revealed significant main effects for quartile ($F(1,44) = 5.01$, $p < .05$), condition ($F(1,44) = 4.45$, $p < .05$) and provocation ($F(7,308) = 3.85$, $p < .001$). Individuals of the LQ chose significantly higher mean shock intensities ($M = 3.8$) than those of the UQ ($M = 3.0$). Individuals of the control condition ($M = 3.7$) chose higher mean shock intensities than those of the inhibition condition ($M = 3.0$). Decomposition of the means for each level of provocation into a linear trend and a pooled non-linear trend revealed that deviations from linearity were non-significant. Subjects selected greater shocks as provocation level increased.

Like the analysis of mean shock intensity following a loss, planned comparisons of mean shock intensity following a win of the control (Ms = 3.9 and 3.5 for LQ and UQ, respectively) versus inhibition (Ms = 3.6 and 2.4 for LQ and UQ, respectively) conditions for each quartile revealed that only individuals of the UQ selected significantly lower mean shock intensities in the inhibition condition ($p < .05$).

Discussion

The results of this study support previous work which shows that individuals who perform poorly on neuropsychological tests putatively assessing frontal lobe function are more aggressive under provocative conditions [Giancola & Zeichner, 1994; Lau et al., 1995]. Furthermore, these findings are consistent with naturalistic studies of neuropsychological deficits and aggressive or violent behavior [e.g. Raine, 1993; Tarter et al., 1984]. This convergence of findings across complementary methods and measures provides strong support for neurobehavioral theories implicating higher

order cognitive abilities, associated with the function of the prefrontal cortex, in the control of evoked aggression [e.g. Pihl, Peterson, & Lau, 1993].

More importantly, however, this study demonstrates that UQ individuals were more responsive to the inhibition manipulation than LQ individuals. Specifically, the UQ group selected significantly lower mean initial shock settings than the LQ group in the inhibition condition.

These findings support the hypothesis that an impairment in cognitive abilities associated with the frontal lobes is a proximal cause of specific acts of aggression. Inhibition of inappropriate or impulsive behaviours, with adaptive shifting to alternative behaviours are functions attributed to the frontal cortex [Moffitt & Henry, 1989]. If reduced frontal lobe function partially disinhibits aggressive behaviour specifically through deficits in the internalization of inhibitory influences [Dimond, 1980], these individuals should inhibit their aggressive behaviour less when presented with contingent monetary reward. This idea is consistent with observations that frontal-lobe damaged patients often demonstrate an inability to rapidly adjust behaviour in response to external cues, despite the apparent verbal processing of these cues [Milner & Petrides, 1984] and that monkeys with frontal resections sluggishly process reinforcement such as reward or punishment [Jacobsen & Nissen, 1937].

Dodge & Crick's [1990] cognitive process model of aggression provides a context within which to integrate the results of this study. They have proposed a 5 step sequential process for competent social performance and argue that deficient processing of provocative cues may lead to aggressive behaviour. The five steps include 1) social cue encoding, 2) cue interpretation, 3) response

search, 4) response evaluation and 5) response enactment. The fourth step, response evaluation, involves a choice by the individual of one of a number of accessed responses. One of the criteria for choosing a particular response is consideration of the potential consequences of the response. Specifically, Dollard and his colleagues have stated, and Berkowitz [1962] concurs that: "the strength of inhibition of any act of aggression varies positively with the amount of punishment anticipated to be a consequence of that act" [1939, p.33]. Given the results of the present study, frontal lobe impairment provides a neuropsychological mechanism to explain the proposed disruptions of Dodge & Crick's [1990] cognitive process model.

This model also suggests that frontal lobe dysfunction may increase the probability of aggression by playing a role in the development and acquisition of aggressive behaviour. The third step of this model postulates that when an individual searches memory for a behavioral response, available responses would be accessed relatively easily if there are limited responses available. As the aggressive behaviour of individuals with frontal lobe dysfunction is difficult to modify, these individuals might experience and learn fewer alternative behaviours. As a result, they would have access to fewer responses and available ones would probably be aggressive. Thus, an impaired individual in a provocative situation, with a restricted range of responses, would be more likely to access an aggressive response. This is consistent with the finding that reduced performance on tasks reflecting abilities associated with executive function have been shown to predict fighting in young boys [Seguin et al., 1995]. Furthermore, prefrontal-type deficits have been implicated as a predisposing factor common to other

disinhibitory syndromes such as psychopathy and antisocial personality disorder [Gorenstein, 1987; Moffitt et al., 1994; Newman, 1987] which are notable for their recidivistic criminal, and specifically violent behaviours. Thus, impairments in cognitive abilities associated with frontal lobe function may indirectly contribute to the maintenance of aggressive behaviour as well as directly increasing the probability of an aggressive response.

An alternative hypothesis is that the two groups in this study processed the inhibitory cue similarly and that the group differences in shock intensities were due to differential reinforcement value from retaliating. LQ individuals may have gained more reinforcement from the act of retaliating and found the modest sum of money as an incentive to inhibit aggression to be insufficient. This, however, is unlikely as LQ individuals did not reduce their initial shock settings, considered a measure of unprovoked aggression [Hammock & Richardson, 1992], in the monetary reward condition. The initial shock selection can be assumed to have been made in the absence of arousal from provocation and thus not part of retaliation.

The findings of this study cannot be attributed readily to a global cognitive impairment of individuals in the low performance quartile. Although there were group differences in IQ, controlling for IQ in the analysis did not substantially affect the results. Furthermore, there were no group differences in performance on the block-tapping task which assesses right hippocampal function strengthening the conclusion that the cognitive impairment of the LQ individuals is specific to the frontal lobes.

It is unlikely that non-neuropsychological factors were responsible for group differences in aggression.

Controlling for both age and education through covariance did not substantially affect the results. As individuals of the LQ manifest slightly higher mean pain thresholds, one can not argue that the LQ group was more sensitive to the shocks and therefore more provoked. The absence of group differences for salary and neighbourhood codes argues against an explanation that the group differences in aggression reduction was due to differences in financial motivation.

One might argue that the LQ group did not understand the reward contingency. This is unlikely as they demonstrated mastery of all other elements of the task. Furthermore, the values for the monetary contingency were clearly visible immediately above the shock level buttons and a counter prominently displayed the amount of money corresponding to the subject's shock choice.

In conclusion, the present study demonstrates that reduced cognitive abilities are associated with a lack of response to inhibitory cues, in this case a monetary contingency. These findings support the notion that the increased aggression of individuals with impaired cognitive abilities may be due to an inability to internalize and integrate inhibitory influences. Further research is required to determine whether these findings generalize to other inhibitory cues, such as response cost or contingent unpleasant stimuli.

Table 1

Mean Demographics and Test Scores by Quartile and Condition

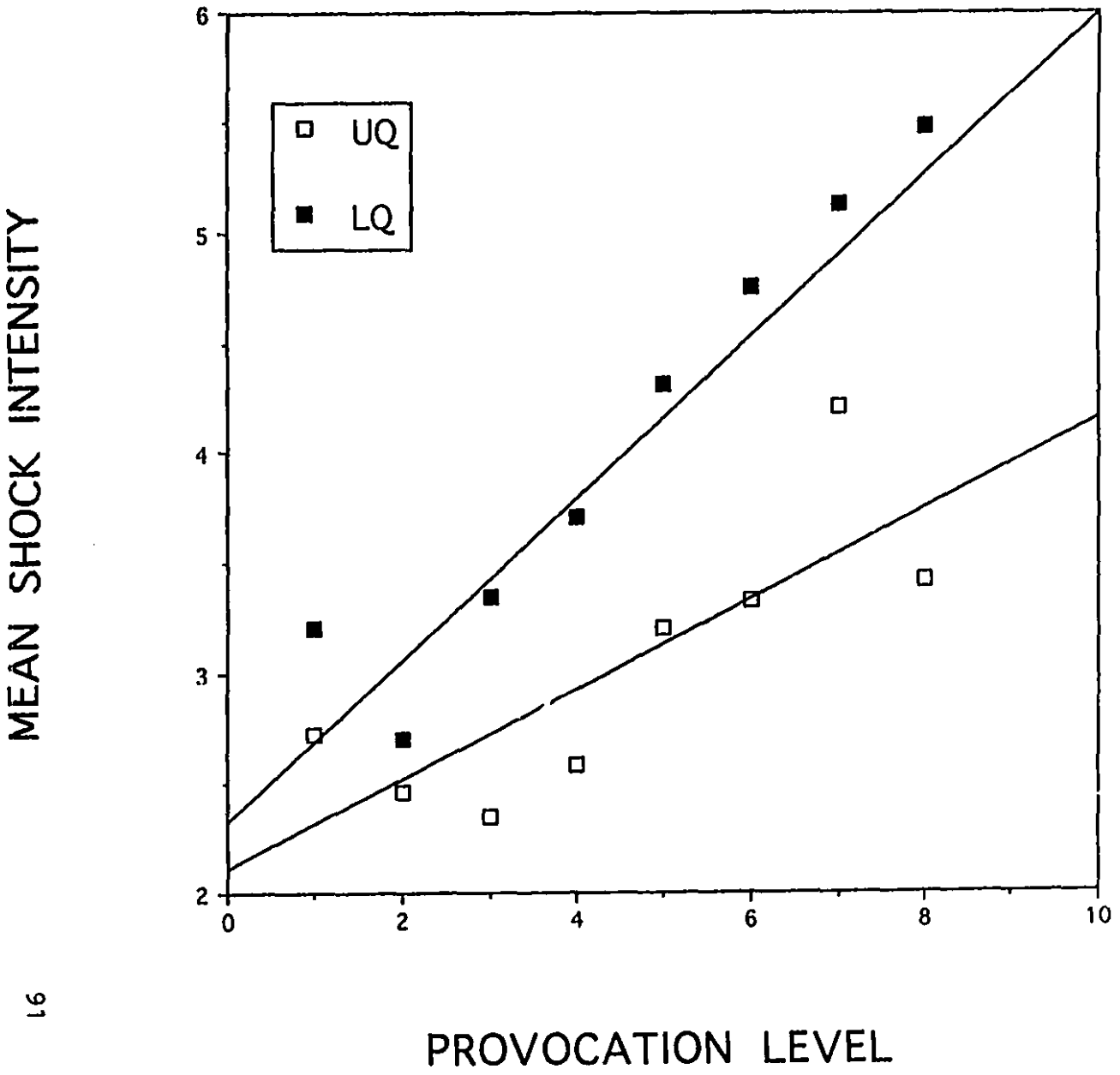
	LQ	UQ	LQ		UQ	
			Control	Inhibition	Control	Inhibition
	(n=24)	(n=24)	(n=12)	(n=12)	(n=12)	(n=12)
	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)
DEMOGRAPHIC DATA						
Age	27 (7.0)*	23 (3.3)*	26 (7.0)	29 (6.8)	24 (2.8)	22 (3.6)
Years of Education	14 (3.0)*	15 (2.8)*	14 (3.6)	13 (2.1)	17 (2.6)	14 (2.4)
Father's years of education	14 (4.1)	14 (4.5)	12 (3.8)	15 (4.0)	14 (5.0)	14 (4.1)
Mother's years of education	12 (3.3)	13 (4.1)	12 (3.7)	13 (3.0)	12 (4.2)	14 (4.1)
Salary Code	2.5 (1.7)	2.2 (1.7)	2.6 (2.1)	2.4 (1.2)	2.2 (1.8)	2.2 (1.6)
Beverage per week	3.6 (4.1)	7.3 (9.6)	2.5 (3.2)	4.7 (4.9)	7 (11.5)	7.6 (7.7)
Neighbourhood code	3.3 (1.4)	3.7 (1.1)	3.3 (1.4)	3.2 (1.5)	3.8 (.94)	3.6 (1.3)
TEST SCORES						
Conditional Associative learning (CALT)						
Total No. of Trials	179 (3)*	52 (14)*	180 (1.4)	179 (4.6)	48 (16)	55 (12)
Total No. of Errors	165 (49)*	23 (10)*	175 (63)	154 (29)	21 (10)	26 (8.9)
Block-tapping task						
Correct recurrent sequences	3.0 (2.0)	3.3 (2.1)	2.9 (1.7)	3.1 (2.3)	4.5 (1.3)	2.1 (2.2)
Correct non-recurrent sequences	3.5 (2.1)	4.8 (3.0)	3.6 (2.7)	3.3 (1.5)	6.0 (3.9)	3.7 (1.2)
Self-ordered pointing (SOP)	4.5 (2.7)*	2.3 (0.8)*	4.3 (2.6)	4.8 (2.9)	2.4 (.67)	2.2 (.83)
IQ	98 (14)*	111 (13)*	100 (13)	97 (15)	112 (12)	110 (15)
DECEPTION RATINGS						
Experimenter	4.9 (.26)	4.7 (.58)	4.9 (.26)	4.9 (.27)	4.7 (.59)	4.8 (.57)
Subject	4.5 (.78)	4.3 (.78)	4.4 (.91)	4.6 (.63)	4.5 (.64)	4.0 (.87)
PAIN THRESHOLD MEASURES	147 (94)	116 (76)	148 (84)	145 (88)	108 (57)	124 (75)

*$p < 0.01$

Figure 1: Mean initial shock intensity by upper and lower cognitive performance quartile groups for the control and monetary reward condition. See results for complete interpretation.

MEAN INITIAL SHOCK INTENSITY
4.0
3.0
2.0
1.0
CONTROL
INHIBITION
UQ
LQ

Figure 2: Mean shock intensity chosen after a loss, by provocation level, for the upper and lower cognitive performance quartile groups. See Results for complete interpretation.


MEAN SHOCK INTENSITY
6
5
4
3
2
UQ
LQ
0
2
4
6
8
10
PROVOCATION LEVEL

References

Baron RA, Richardson DR (1994): "Human Aggression (2nd ed.)." New York: Plenum Press.

Berkowitz L (1962): "Aggression: A social psychological analysis." New York: McGraw-Hill.

Borden RJ, Bowen R, Taylor SP (1971): Shock setting behaviour as a function of physical attack and extrinsic reward. Perceptual and Motor Skills 33: 563-568.

Brooker BH, Cyr JJ (1986): Tables for clinicians to use to convert WAIS-R Short Forms. Journal of Clinical Psychology 42: 983-984.

Buikhuisen W (1987): Cerebral dysfunctions and persistent juvenile delinquency. In Mednick SA, Moffitt TE, Stack SA (eds): "The Causes of Crime: New Biological Approaches (pp. 168-184)." Cambridge: Cambridge University Press.

Corsi PM (1972): Human Memory and the medial temporal region of the brain. Unpublished doctoral dissertation, McGill University, Montreal.

Dimond SJ (1980): "Neuropsychology: A Textbook of Systems and Psychological Functions of the Human Brain." London: Butterworth.

Dodge KA, Crick NR (1990): Social information-processing bases of aggressive behaviour in children. Personality and Social Psychology Bulletin 16: 8-22.

Dollard J, Doob L, Miller N, Mowrer OH, Sears RR (1939): "Frustration and aggression." New Haven, CT: Yale University Press.

Eagly AH, Steffen VJ (1986): Gender and aggressive behaviour: A meta-analytic review of the social psychological literature. Psychological Bulletin 100: 309-330.

Fuster JM (1989): "The Prefrontal Cortex: Anatomy,

Physiology, and Neuropsychology of the Frontal Lobe." New York: Raven Press.

Giancola PR, Zeichner A (1994): Neuropsychological performance on tests of frontal-lobe functioning and aggression in human males. Journal of Abnormal Psychology 103: 832-835.

Gorenstein EE (1987): Cognitive-perceptual deficit in an alcoholism spectrum disorder. Journal of Studies on Alcohol 48: 310-318.

Hammock GS, Richardson DR (1992): Predictors of aggressive behaviour. Aggressive Behaviour 18: 219-229.

Huesmann LR (1988): An information processing model for the development of aggression. Aggressive Behaviour 14: 13-24.

Jacobsen Nissen (1937): Studies of cerebral function in primates: IV The effects of frontal lobe lesions in the delayed alternation habit in monkeys. Journal of Comprehensive Physical Psychology 23: 101-112.

Kandel E, Freed D (1989): Frontal-lobe dysfunction and antisocial behaviour: A review. Journal of Clinical Psychology 45: 404-413.

Lau MA, Pihl RO, Peterson JB (1995): Provocation, acute alcohol intoxication, cognitive performance, and aggression. Journal of Abnormal Psychology 104: 150-155.

Lueger RJ, Gill KJ (1990): Frontal-lobe cognitive dysfunction in conduct disorder adolescents. Journal of Clinical Psychology 46: 696-706.

Luria AR (1980): "Higher Cortical Functions in Man (2nd ed.)." New York: Basic Books, Inc.

Mercy JA, Rosenberg ML, Powell KE, Broome CV, Roper WL (1993): Public health policy for preventing violence. Health Affairs, Winter: 7-29.

Milner B, Petrides M (1984): Behavioral effects of

frontal-lobe lesions in man. Trends in Neurosciences November: 403-407.

Moffitt TE (1990): The neuropsychology of juvenile delinquency: A critical review. In Tonry M, Morris N (eds): "Crime and Justice: A Review of Research (pp. 99-169)." Chicago: University of Chicago Press, Vol. 12.

Moffitt TE, Henry B (1989): Neuropsychological assessment of executive functions in self-reported delinquents. Development and Psychopathology 1: 105-118.

Moffitt TE, Lynam DR, Silva PA (1994): Neuropsychological tests predicting persistent male delinquency. Criminology 32: 277-300.

Nauta WJ (1971): The problem of the frontal lobe: A reinterpretation. Journal of Psychiatric Research 8: 167-187.

Newman JP (1987): Reaction to punishment in extraverts and psychopaths: Implications for the impulsive behaviour of disinhibited individuals. Journal of Research in Personality 21: 464-480.

Petrides M (1985): Deficits on conditional associative-learning tasks after frontal- and temporal-lobe lesions in man. Neuropsychologia 23: 601-614.

Petrides M, Alivisatos B, Evans AC, Meyer E (1993): Dissociation of human mid-dorsolateral from posterior dorsolateral frontal cortex in memory processing. Proceedings of the National Academy of Sciences 90: 873-877.

Petrides M, Milner B (1982): Deficits on subject-ordered tasks after frontal- and temporal-lobe lesions in man. Neuropsychologia 20: 249-262.

Pihl RO, Peterson JB, Lau MA (1993): A biosocial model of the alcohol-aggression relationship. Journal of Studies on Alcohol Suppl. 11: 128-139.

Pokorny AD, Miller BA, Kaplan HB (1972): The brief MAST: A shortened version of the Michigan Alcoholism Screening Test. American Journal of Psychiatry 129: 342-345.

Pribram KH (1973): The primate frontal cortex-Executive of the brain. In Pribram KH, Luria AR (eds): "Psychophysiology of the Frontal Lobes." New York: Academic Press.

Raine A (1993): "The psychopathology of crime." New York: Academic Press.

Seguin JR, Pihl RO, Harden P, Tremblay RE, Boulerice B (1995): Cognitive and neuropsychological characteristics of physically aggressive boys. Journal of Abnormal Psychology, in press.

Tancredi LR, Volkow N (1988): Neural substrates of violent behaviour: Implications for law and public policy. International Journal of Law and Psychiatry 11: 13-49.

Tarter RE, Hegedus AM, Winsten NE, Alterman AI (1984): Neuropsychological, personality, and familial characteristics of physically abused delinquents. Journal of the American Academy of Child Psychiatry 23: 668-674.

Taylor SP (1967): Aggressive behaviour and physiological arousal as a function of provocation and the tendency to inhibit aggression. Journal of Personality 35: 297-310.

U.S. Bureau of the Census (1993): "Statistical Abstract of the United States: 1993 (113th edition)." Washington D.C.; U.S. Government Printing Office.

Yeudall LT, Fromm-Auch D (1979): Neuro-psychological impairments in various psychopathological populations. In Gruzelier J, Flor-Henry P (eds): "Hemisphere Asymmetrics of Function and Psychopathology (pp. 5-13)." New York:

Elsevier/North Holland.

Yeudall LT, Fromm-Auch D, Davies P (1982): Neuropsychological impairment of persistent delinquency. Journal of Nervous & Mental Disease 170: 257-265.

Footnotes

[1]Positron emission tomography with magnetic resonance imaging of the brains of normal volunteers completing a modified version of this task demonstrated activation of cytoarchitectonic area 8 of the dorsolateral frontal cortex (Petrides, Alivisatos, Evans & Meyer, 1993).

[2]Positron emission tomography with magnetic resonance imaging of the brains of normal volunteers completing a modified version of this task demonstrated activation of cytoarchitectonic areas 46 and 9 of the mid-dorsolateral frontal cortex (Petrides et al., 1993).

[3]Breathalysers or urine toxicology were not used in this study to verify that subjects had not used alcohol/drugs. However, in a previous study (Lau, Pihl, & Peterson, 1995) where the same request was made, testing with breathalysers did not reveal any evidence that any subject had drank alcohol prior to the experiment. Thus, it is reasonable to conclude from this that participants in general comply with the request. In addition, the experimenter who conducted the aggression task, a graduate student with prior experience in testing intoxicated subjects, would most likely have detected any gross violations of this request.

[4]The Salary Code variable is a 7 point Likert type scale measuring annual financial earnings: 1 = $0-$5,000; 2 = $5,001-$10,000; 3 = $10,001-$15,000; 4 = $15,001-$20,000; 5 = $20,001-$25,000; 6 = $25,001 - $30,000; 7 = $30,001+.

[5]To control for the possible confounding effect of IQ, this variable was included separately as a covariate in the ANOVAs for initial shock setting, mean shock intensity following a loss and a win. As a covariate, IQ failed to reach significance in any case and its inclusion did not substantially affect the results. The

within-class regression coefficients for IQ were homogenous in all cases. Comparable analyses conducted on self-ordered pointing score, years of education, age and pain threshold also did not affect the results.

GENERAL DISCUSSION AND CONCLUSION

The initial objective of this work was to explore the potential role of individual differences of cognitive abilities associated with the prefrontal cortex in the alcohol-aggression relationship. The first two studies were designed to determine: (a) the validity of using the Taylor aggression paradigm in a within-subjects design to investigate the alcohol-aggression relationship; and (b) the effects of acute alcohol intoxication on the aggressive response pattern of individuals differing with respect to cognitive abilities associated with the prefrontal cortex.

Study 1 compared the performance of subjects on the Taylor task under two counterbalanced drug conditions, intoxicated and sober. The results of this study demonstrated that mean shock intensity, duration and total aggression as well as mean pain threshold varied according to an interaction between order and intoxication condition. Consequently, ensuing investigations in this research were restricted to using the Taylor task in a between-subjects design.

Study 2 considered the level of aggressive behaviour of two groups differing in cognitive performance (high vs. low) across two drug conditions (sober vs. intoxicated) under provocative conditions. Contrary to expectations, no significant interaction between drug condition and cognitive performance was found. Hence, this study's results did not support the hypothesis that acute alcohol intoxication impairs cognitive abilities of the prefrontal cortex to increase provoked aggression (Pihl et al., 1993). Cognitive performance, however, interacted significantly with provocation. Specifically, low cognitive performance was associated with greater aggression as provocation increased. The demonstration

of this relationship under tightly controlled experimental conditions was particularly important. Previous epidemiological and neuroimaging studies had done little to address the mechanisms underlying the nature of this relationship in part due to the methodological limitations inherent in the study designs (Kandel & Freed, 1989; Raine, 1993). A laboratory analogue design, however, with good internal validity offered the potential advantages of contributing to the understanding of these mechanisms in a manner that previous investigations could not.

Study 3 tested the hypothesis that the cognitive deficit associated with increased aggression represented an impairment in the ability to inhibit inappropriate or impulsive behaviours. This study compared the effect of contingent monetary reward to inhibit aggressive responding across cognitive performance groups. In this study, CALT performance alone determined group assignment. In Study 2, individual differences in neuropsychological function were determined from the combined performance on the spatial conditional associative-learning task (Petrides, 1985a) and the self-ordered pointing task (Petrides & Milner, 1982). However, a subsequent report demonstrated that increased aggression was associated with poor performance only on the former task (Giancola & Zeichner, 1994b)[1].

In Study 3, as in Study 2, individuals in the lower cognitive performance quartile showed greater increases in aggression as provocation increased. More importantly, however, individuals of the lower performance quartile demonstrated significantly smaller reductions of unprovoked aggression in response to

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[1] Preliminary results of Study 2 were reported by Lau, Pihl, & Peterson (1992).

monetary reward.

INTEGRATION OF FINDINGS

In sum, CALT performance proved to be a meaningful variable in discriminating response to environmental stimuli in sober young males. Specifically, impaired CALT performance was associated with heightened aggression to stimuli that elicited aggression (provocation) and diminished response to stimuli thought to inhibit aggression (contingent monetary reward).

One explanation is that the ability to retrieve appropriate, and inhibit inappropriate responses to environmental stimuli is important in aggression control. The CALT was designed to assess the ability to retrieve appropriate behavioral responses upon presentation of previously encountered environmental stimuli (Petrides, 1985a). As mentioned previously, the CALT is thought to assess cognitive abilities associated with the intact function of the posterior dorsolateral prefrontal cortex (Petrides, 1985a; Petrides et al., 1993). Moreover, the performance deficits on the CALT: 1) were not generalized to a variety of demographic variables (e.g. SES) nor other cognitive functions (i.e. Corsi Block Tapping task), and 2) predicted level of aggression beyond any prediction provided by IQ. Thus, the results of this research implicate a role for cognitive abilities of the dorsolateral prefrontal cortex in aggression control.

INTEGRATION WITH PREVIOUS RESEARCH

These findings are consistent with a small but growing body of evidence from human and animal studies which have specifically investigated the role of the dorsolateral prefrontal cortex in the control of aggression in humans and animals.

Human research

Recent investigations have considered the specific relationship between impaired CALT performance and increased laboratory aggression in young males (Giancola & Zeichner, 1994b) as well as fighting in young boys (Seguin et al., 1995). Combined results suggest that impaired CALT performance is associated with increased aggressive behaviour across laboratory and naturalistic aggressive measures. Similarly, Harden & Pihl (1995) have reported an association between impaired CALT performance and disruptive behaviour in young boys.

Furthermore, Grafman, Vance, Weingartner, Salazar, & Amin (1986) compared war veterans with wounds to either the dorso-, orbito-, or non-frontal cortex on self-report and observed measures of mood state. The results suggested that only dorsofrontal cortical injury led to disinhibition of mood states reflecting anger and hostility.

Animal Research

There exist a few, and typically not recent, animal studies which have also considered the possible role of the dorsolateral prefrontal cortex in the control of physical aggression. This is due to a scarcity of contemporary animal studies investigating this relationship (Giancola, 1995). The following discussion is limited to monkey and cat studies. While there are lesions studies using rats, there appears to be no homolog for the dorsolateral region in the rat (Preuss, 1995).

Lesion studies have demonstrated increased aggression following bilateral ablation of the dorsolateral area of the prefrontal cortex in pigtail (Kamback & Rogal, 1973), stumptail (Mass & Kling, 1975), and rhesus macaques (Kling, 1976; Miller, 1976). Studies

comparing the effects of dorsolateral and orbital lesions in macaques have demonstrated that dorsolateral lesions led to increased aggression whereas orbital lesions led to reduced aggression (Butter, Mishkin, & Mirsky, 1968; Kamback, 1973). Furthermore, hypothalamically-elicited attack behaviour in cats has been suppressed via electrical stimulation of the lateral aspect of the prefrontal cortex, but not adjacent sites (Siegel, Edinger, & Dotto, 1975; Siegel, Edinger, & Koo, 1977). Thus, the dorsolateral region may be a part of a neural circuit responsible for exerting inhibitory influences on aggressive impulses arising from various subcortical structures (Brutus, Shaikh, Siegel, & Siegel, 1984; Siegel & Edinger, 1983; Watson, Edinger, & Siegel, 1983).

Furthermore, these findings are consistent with a larger body of research associating neuropsychological dysfunction of the frontal lobes with a predisposition to violence (e.g. Kandel & Freed, 1989; Raine, 1993).

IMPLICATIONS FOR THEORETICAL ACCOUNTS OF VIOLENCE

A variety of contrasting theoretical approaches to understanding violence have been proposed. Baron & Richardson (1994) have grouped these perspectives into four categories where aggression is attributed primarily to 1) instinctive behaviour, 2) an elicited drive 3) cognitive and emotional processes; or 4) learned social behaviour. The latter perspective suggests that an understanding of aggressive behaviour necessitates attention to these factors including how aggression is acquired, instigated and maintained. This perspective acknowledges the contribution of biological factors in the acquisition of aggressive behaviour. A biological predisposition to aggression or violence may be expressed through disruption of the neural mechanisms that typically moderate and control behaviour. A variety of

brain deficits may predispose to violence, including left hemisphere dysfunction, temporal dysfunction or lateralization abnormalities (Raine, 1993). However, the findings of this research are consistent with the hypothesis that frontal lobe dysfunction may primarily predispose an individual to violent behaviour.

This brain area is thought to be the neural substrate that subserves a number of executive functions necessary to produce context-appropriate, goal-oriented self-directed behaviour (Goldman-Rakic, 1988). These functions include attention, social/self monitoring, modification of behaviour as conditions change, selection and generation of responses, temporal ordering, associative learning and inhibitory behaviour (Fuster, 1989; Milner & Petrides, 1984; Passingham, 1993). Perecman (1987) notes that these functions refer to the regulation of a behaviour or to the integration of a specific behaviour into a larger strategy of behaviour rather than to a specific behaviour per se.

Impaired frontal function might result in a failure of any of the above listed abilities. Regardless, the findings of this research are consistent with the notion that impaired frontal function might result in a failure to inhibit inappropriate responses possibly due to a restricted evaluation of personal and social consequences.

One neuropsychological explanation for how this might happen is based on the idea that the prefrontal cortex plays a pivotal role in working memory (Goldman-Rakic, 1992). In this model, this brain area acts as a neural comparator to simultaneously evaluate and weigh a variety of internal and external priorities in order to develop an appropriate and optimal plan of action. In this way, an individual is able to behave on the basis of previous experience rather than simply responding to the

provoking environmental stimulus.

From a neurophysiological perspective, impaired prefrontal function could result in an inability to inhibit subcortical structures thought to facilitate aggression (Brutus et al., 1984; Weiger & Bear, 1988).

At a personality level, frontal damage is associated with impulsivity and disinhibitory psychopathology (Luria, 1980; Gorenstein & Newman, 1980). The findings of this research are consistent with the notion of impaired reflectivity in disinhibition syndromes (Patterson & Newman, 1993). These authors propose that disinhibited and nondisinhibited individuals may adopt a dominant response set when given an opportunity for reward. Further, the occurrence of unexpected punishment, omission of reward or delayed gratification while that set is dominant is seen as leading to an increase in arousal. This arousal is thought to facilitate the dominant response set (e.g. aggression) of disinhibited individuals but arrest the behaviour of nondisinhibited individuals which allows them time to reflect on possible alternative behaviours. The facilitated responding typified by disinhibited individuals leads to a modulated response bias. Disinhibited individuals modulate the overt goal-directed behaviour rather than altering the response set in accordance with changing environmental events and contingencies. As such these individuals do not learn alternative ways to avoid aversive events. Moreover, the failure to reflect could interfere with the processing of punishment or nonreward cues which might result in reduced formation of causal associations between behaviours and their consequences. In sum, a lack of retrospective reflection due to facilitation of a dominant response set, therefore, may contribute to an enduring impulsive style. As impulsivity plays an

important role in aggressive behaviour (Farrington, Loeber, & Van Kammen, 1990; Hurt & Naglieri, 1992; Moffitt & Henry, 1989), an individual's nonreflective reaction to punishment or non-reward may contribute to an aggressive style.

In the context of the above discussion, the findings of this research suggest a possible neuropsychological basis for a variety of theoretical accounts of aggression including biosocial (Pihl et al., 1993), cognitive (e.g. Dodge & Crick, 1990) and social interactionist theories on aggression (Felson & Tedeschi, 1993).

First, the ability to learn culturally acceptable responses to provocative situations and to perceive cues associated with the threat of punishment are thought to be important components of the General Expectancy Set (GES; Pihl et al., 1993). The GES, a theoretical construct, is comprised of all extant expectancies. Expectancy states, dynamic models of what will happen in the future as a result of current behaviours (Luria, 1980), appear to arise as an emergent property of acquired knowledge (Pihl & Peterson, 1992). More specifically, the GES is thought to perform three functions which determine the elicitation, construction and modification of aggression. First, the GES determines the context within which "objective" stimuli are subjectively interpreted. Second, the GES represents the repertoire of culturally determined behaviours internalized during the course of socialization from which to draw aggressive responses. Third, the GES determines whether aggression is subject to the threat of punishment and therefore inhibited or to the hope of reward leading to its potentiation. As discussed above in relation to impulsivity, prefrontal dysfunction might result in deficits in the ability to acquire culturally acceptable behavioral response to provocation and an

impairment in the ability to detect cues for punishment.

Second, the cognitive abilities thought to be associated with intact prefrontal cortical function are similar to steps 3 and 4, i.e., response search and response evaluation, of Dodge & Crick's (1990) 5 step cognitive process model of aggression. Thus, impaired prefrontal function might result in deficient response search and evaluation which might lead to an increased probability of aggressive responding.

Third, social-interactionist theoreticians suggest for example that aggressive individuals are more likely to break rules and hence are more likely to elicit punishment thereby increasing the likelihood of a provocation-induced aggressive conflict. Moreover, there is a greater likelihood of aggression by those unable to use other forms of influence. Impaired frontal function might be the biological substrate underlying these individual differences in behaviour.

Alternatively, prefrontal dysfunction may underlie a more global disorder that is associated with aggressive behaviour. Prefrontal dysfunction has been associated with a variety of disorders including hyperactivity (Shue & Douglas, 1992), antisocial behaviour and primary alcoholism (Gorenstein & Newman, 1980) as well as with adolescent and adult non-alcoholic sons of male alcoholics (Giancola, Peterson, & Pihl, 1993; Peterson, Finn, & Pihl, 1992).

The above discussion of the possible role of the prefrontal cortex in the regulation of aggressive behaviour is of a heuristic nature. The goal of this research was not to identify a specific anatomical lesion site in the brain that causes aggressive behaviour. Functional neuroanatomy is as yet insufficiently understood to support conclusive causal deductions (Moffitt, 1990). For example, the dorsolateral

prefrontal cortex forms only a part of a hierarchical network of cortical and subcortical connections. Thus, it is likely that numerous other structures are also involved in the cognitive functions attributed to this brain area. It is possible other brain areas can also independently perform the same functions. For example, competing neurobehavioral theories have proposed that cognitive deficits have been attributed to the projecting thalamic or reticular activating system processes rather than frontal-lobe impairment (Mattes, 1980). Thus, impaired regulation of aggression might be attributed to disruptions of brain areas distant from the cortical mantle, or from a disturbance at a biochemical level with no observable structural change.

Thus, this work was not meant to attribute the control of aggression to specific brain areas per se. Administration of a test battery employing a selection of tests with demonstrated validity of other anterior and posterior brain areas would help in this regard. In the present study, functional assessment of other prefrontal areas, such as the orbito-medial frontal cortex, was not performed in part because the appropriate assessment instruments do not yet exist. Furthermore, a complete test battery was not performed so as to minimize the time required for subject participation. Thus, the results of these studies do not necessarily invalidate theories that the control of aggressive behaviour involves areas such as the orbito-medial frontal cortex (Bear, 1991). Although, Giancola (1995) argues, based on a review of the human and animal literature investigating the role of the dorsolateral and orbital prefrontal regions in aggression control, that while disinhibited, non-aggressive behaviours are typically associated with orbito-medial lesions, physical aggression itself appears to be a correlate of dorsolateral, rather than orbito-

medial, damage. Instead, orbito-medial injury might serve to increase the propensity for aggression in dorsolateral individuals by disturbing emotional regulation/modulation.

This discussion does not and would not assume that severe violence is due to biological factors alone, and that social processes are irrelevant in explaining violence. These factors likely interact to explain violence. For example, a history of an abusive childhood environment may interact with neuropsychological deficits to produce a violently reactive individual. Widom (1989) has shown that males, neglected and abused as children, manifest an increased likelihood of arrests for delinquency, adult criminality, and violent criminal behaviour. Widom (1989) notes, however, that while childhood victimization may contribute to the later development of criminal violence, other factors which interact with victimization may also be involved. One of these factors might be individual differences in neuropsychological function. There is evidence that intrinsic neuropsychiatric vulnerabilities, and a history of a violent abusive environments is a better predictor of adult violent crime than early violence alone (Lewis, Lovely, Yeager, Della Femina, 1989). One possibility is that children with neuropsychiatric impairments may be less able resist aggressive models and choose alternative, more appropriate behaviours.

POSSIBLE CAUSES OF FRONTAL LOBE DYSFUNCTION

A major question is what underlies the neuropsychological performance deficits observed in this research. Individuals with a history of head injury, substance abuse and mental or physical illness were excluded from participation. Thus, it is unlikely that the impaired performance of participants in these studies

was due to a gross neuroanatomical deficit.

Poor neuropsychological test performance does not necessarily indicate a brain lesion (Lueger & Gill, 1990). Rather, other mechanisms might be involved including fluctuating neurochemical states, heritable individual variation in brains, disruptions in fetal brain development, childhood exposure to neurotoxins and early environmental deprivation (Moffitt, 1990). For example, the observed neuropsychological deficits might derive from a "biochemical lesion", for instance, from dysregulation of the serotonin system. This neurotransmitter may play an important role in the regulation of aggressive behaviour (Pihl & Peterson, 1993). In particular, reduced brain serotonin is associated with an increased probability of aggressive or impulsive behaviour (see Virkkunen & Linnoila, 1993 for review). Furthermore, antisocial populations have been shown to manifest reduced central serotonin levels, with the lowest levels found in antisocials with a history of violence, borderline personality disorder and alcohol abuse (Raine, 1993). Conversely, experimental manipulations elevating serotonin levels generally lead to decreases in aggression (Brizer, 1988).

Reduced serotonin is thought to underlie, along with low norepinephrine, underactivation of the behavioral inhibition system (BIS; Gray, 1975; Fowles, 1988). According to Gray (1975), the BIS is thought to inhibit behaviour in response to cues of punishment or frustrative nonreward. Fowles (1988) has argued that reduced BIS functioning may underlie the disinhibitory, impulsive behaviours of antisocial individuals. Scerbo & Raine (1992) propose that reductions in serotonin levels might contribute to social information-processing deficits which increase the likelihood of aggression (Dodge & Crick, 1990). Further research, however, is

required to validate this hypothesis as well as whether brain serotonin deficiencies might be specifically associated with poor performance on the CALT.

Alternatively, individual variation in neuropsychological function may be attributable to heritable variation. For example, inherited decrements in neuropsychological performance have been reported for Sons of Male Alcoholics (SOMAs; Peterson & Pihl, 1990). Specifically, SOMAs are characterised by deficits in cognitive abilities thought to depend on the intact functioning of the prefrontal cortex (Gorenstein, 1987; Pihl, Peterson, & Finn, 1987; Tarter et al., 1984). Interestingly, pre-adolescent SOMAs have been shown to perform poorly on the CALT, react impulsively during objective testing and demonstrate disruptive behaviour by parent rating when compared to a matched control group (Harden & Pihl, 1995). Differently, genetic defects in the metabolism of neurotransmitters such as serotonin or noradrenaline might affect aggressive behaviour. Until recently such mutations had not been reported. However, in a recent study of a large kindred in which many males were affected by a syndrome of abnormal behaviour including impulsive aggression, this behaviour was associated with a selective deficiency of enzymatic activity of monoamine oxidase A (MAOA) attributed to a specific gene mutation (Brunner, Nelen, Breakefield, Ropers, & van Oost, 1993). According to these authors, reduced central serotonin levels might result from absent MAOA activity in these subjects.

Finally, neurotransmitter levels may be adversely affected by a deviant rearing environment including parental substance abuse and psychiatric hospitalizations or otherwise poor family functioning. A deviant rearing environment has been associated with enduring serotonin abnormalities in nonhuman primates (Higley, Suomi, &

Linnoila, 1990).

IMPLICATIONS FOR THE ALCOHOL-AGGRESSION RELATIONSHIP

The initial focus of this research was to determine the role of individual differences of cognitive abilities associated with the frontal lobes in the alcohol-aggression relationship. That alcohol disrupts higher order cognitive abilities involved in the control of aggressive behaviour is suggested by the results of Study 1. In Study 2, it was postulated that drug condition would interact with decreased cognitive performance, such that alcohol would increase aggression more for lower cognitive performers. Counter to expectations, the results did not reveal a significant interaction. Given the results of study 1, the failure to find an interaction in Study 2 is likely a function of the study design. It was not possible, however, to determine the extent to which ceiling effects and limited statistical power impacted on these results. It remains possible, therefore, that alcohol facilitates aggression more for individuals whose aggression is under inhibitory control. The results of Study 3 are consistent with the idea that low cognitive performers were impaired in their ability to inhibit aggressive behaviour when sober. Thus, alcohol may selectively impair cognitive abilities for individuals without pre-existing cognitive deficits.

Alternatively, deficits in frontal function may interact with alcohol's anxiolytic effects. These effects are thought to reduce the inhibitory effect of threat by impairing the threat detection system dependant on intact hippocampal function (Pihl et al., 1993). When the expression of aggression was under the inhibitory influence of threat, then this expression would be facilitated by alcohol. Thus, an impairment in the ability to integrate inhibitory influences combined with

a reduction in the strength of the inhibitory influence of threat might result in an even greater probability of an aggressive response under provocative conditions.

Finally, it remains possible that individuals who manifest increased aggressive behaviour tend to consume more alcohol. The results of this research demonstrate an association between impaired cognitive function, increased aggression and disinhibition. Moreover, alcoholism is also thought to derive from a disinhibitory syndrome (Gorenstein & Newman, 1980). This hypothesis might be tested using a repeated measures design with another paradigm (e.g. Cherek et al., 1985).

LIMITATIONS

Aggressive behaviour was measured in these studies using the Taylor competitive reaction time task. The paradigm's success, however, depends on convincing the participants that they are delivering mild electric shocks to an opponent. With the wide publicization of the Milgram experiments (e.g., Milgram, 1963), there is the possibility that participants will not be deceived. As a result, their reactions may be quite different from those that would occur in natural settings. While the pre-experimental interview excluded a few subjects who cited knowledge of Milgram's experiments, a large majority of subjects who participated in these studies were very surprised to find out that there was no opponent. Moreover, no subject guessed the purpose of the experiment. Thus, it appears that valid results were obtained using the Taylor paradigm.

It has been argued that aggression as measured in the laboratory is "in fact, research on defensive coercion compelled by norms of reciprocity" (Gottfredson & Hirschi, 1993, p. 52). Thus, those who manifest increased "aggression" are in fact unusually sensitive to

normative expectations. As such, these individuals would be the least likely to engage in assaultive and violent acts. A history of aggressive acts was not solicited in these studies. This information, if collected, might provide the necessary information to definitively refute this criticism. Nevertheless, the Taylor task differentiates between groups theoretically expected to differ in aggressive potential including psychopathic individuals (Dengerink, 1971) and prison inmates (Wolfe & Baron, 1971). Furthermore, there is direct support for the convergent and discriminant validity of this task (e.g. Giancola & Zeichner, 1994a). Thus, it is unlikely that the Taylor task measures defensive coercion. A more likely explanation is that the above criticisms were directed at the Buss paradigm.

Additionally, there are three possible threats to internal validity in these studies: (1) difficulty in ruling out possible third variables, (2) selection bias, and (3) experimenter bias. Firstly, perhaps the greatest threat to the validity of these studies lies in the possibility that the groups differed with respect to an unidentified third variable. This threat arises from the fact that subject assignment to high and low cognitive performance groups was not random. Thus, an unlimited number of possible third variables might explain the results of these studies. The most obvious third variables (i.e. IQ, non-verbal memory associated with right hippocampal function, demographics, history of head injury, psychopathology, etc.) have been ruled out. However, a number of potentially important variables were not specifically investigated. These include, for example, a history of neglect and abuse (Widom, 1989), or a deviant rearing environment (Volavka, Martell & Convit, 1992). Further studies would benefit from measurement of these variables.

Second, participants in this research were solicited with advertisements placed in the Help Wanted section of a newspaper. As a result, the sample consisted predominantly of unemployed individuals and students. Performance quartiles significantly differed with respect to years of education in Study 2 & 3. Controlling for this potential confound did not affect the results. It remains possible, however, that other factors associated with student status might have contributed to group differences in aggression. However, the results of these studies have been replicated elsewhere using primarily a student population (Giancola & Zeichner, 1994b). Thus, it appears that CALT performance predicts aggression over and above any contribution arising from third factors associated with being a student.

Third, in Study 2, the experimenter operating the aggression task was not blind to the subject's grouping when testing the first half of the subject cohort due to manpower limitations. An analysis performed on the results derived from the first and second halves of the study did not reveal any differences. Furthermore, the experimenter was blinded to the subject's grouping in Study 3. The result of a quartile by provocation interaction was present in both studies. These results discount the possibility that experimenter bias affected the results of Study 2.

Finally, it is important to recognize that the results of this research relate specifically to young males sampled from the normal population. Thus, the results cannot be generalized to violent offenders nor women at this stage.

RECOMMENDATIONS FOR FUTURE RESEARCH

First, and perhaps most importantly, is to replicate the findings of Study 3 to establish or disconfirm the

reliability and generalizability of the finding that poor performance on the CALT is associated with an impairment in the ability to inhibit aggression in response to reward. Future research should be designed to correct the limitations of this study. For example, to increase reliability, other inhibitory influences such as response cost or contingent unpleasant stimuli might be used. Furthermore, it would be useful to determine whether individuals with inferior CALT performance derive differential reinforcement from retaliation.

To increase generalizability, use of the CALT should be expanded to assess other populations such as violent criminals. This measure may prove useful in the prediction of who is likely to engage in violent behaviour. This is important for efforts to prevent or control such behaviour. Future research might also be directed at investigating the possible relationship between antisociality and impaired CALT performance.

These studies emphasized the advantage of considering the interaction between individual and situational variables in understanding aggression. Thus, future investigations should consider the advantages of studying the interaction between social, situational and individual determinants of aggression.

Finally, modifications of this analogue design might be used to further investigate alcohol related aggression. For example, future research might selectively focus on the effects of alcohol on the aggressive behaviour of individuals with intact cognitive functioning.

CONCLUSION

The results of these studies suggest that higher order cognitive abilities associated with the prefrontal cortex are important in the regulation of aggressive

behaviour under provocative conditions. Specifically, these results corroborate the theory that frontal lobe dysfunction causes increased aggression under provocative conditions. Furthermore, increased aggression may be due to an impairment in the ability to integrate inhibitory influences to aggressive responding. Moreover, the results are consistent with the notion that acute alcohol intoxication impairs higher order cognitive abilities involved in the formulation of behavioral strategies under provocative conditions. However, the results did not support the hypothesis that acute alcohol intoxication increases aggressions by temporarily impairing cognitive abilities previously demonstrated to be associated with the frontal lobes. Thus, further research is required to elucidate the neural mechanisms underlying alcohol induced aggression.

BIBLIOGRAPHY

Abel, E.L. (1986). Guns and blood alcohol levels among homicide victims. Drug and Alcohol Dependence, 18, 253-257.

Ackerly, S.S., & Benton, A.L. (1948). Report of a case of bilateral frontal lobe defect. Research on Nervous and Mental Diseases, 27, 479-504.

American Medical Association (1968). Manual on Alcoholism. Chicago: American Medical Association.

Bailey, D.S., & Taylor, S.P. (1991). Effects of alcohol and aggressive disposition on human physical aggression. Journal of Research on Personality, 25, 334-342.

Bandura, A. (1973). Aggression: A Social Learning Analysis. Englewood Cliffs, NJ: Prentice-Hall.

Baron, R.A., & Eggleston, R.J. (1972). Performance on the "aggression machine": Motivation to help or harm? Psychonomic Science, 26, 321-322.

Baron, R.A., & Richardson, D.R. (1994). Human Aggression, 2nd ed. New York: Plenum Press.

Bear, D. (1991). Neurological perspectives on aggressive behaviour. Neurology of Aggression, 3, S3-S7.

Beaumont, J.G. (1983). Introduction to neuropsychology. New York: Guilford Press.

Bennett, R.M., Buss, A.H., & Carpenter, J.A. (1969). Alcohol and human physical aggression. Quarterly Journal of Studies on Alcohol, 30, 870-877.

Benton, D., & Smoothy, R. (1984). The relationship between blood alcohol levels and aggression in mice. Physiology and Behaviour, 33, 757-760.

Berkowitz, L. (1974). Some determinants of impulsive aggression: The role of mediated associations with reinforcements for aggression. Psychological Review, 81, 165-176.

Berkowitz, L. (1981). The concept of aggression. In P.F. Brain, & D. Benton (Eds.), Multidisciplinary Approaches to Aggression Research. Amsterdam: Elsevier/North-Holland and Biomedical Press, pp. 3-15.

Bernstein, S., Richardson, D., & Hammock, G. (1987). Convergent and discriminant validity of the Taylor and Buss measures of physical aggression. Aggressive Behaviour, 13, 15-24.

Berry, M.S. (1993). Ethanol-induced enhancement of defensive behaviour in different models of murine aggression. Journal of Studies on Alcohol, Suppl. 11, 156-162.

Berry, M.S., & Brain, P.F. (1986). Neurophysiological and endocrinological consequences of alcohol. In P.F. Brain (Ed.), Alcohol and Aggression. Dover, NH: Croom Helm, pp. 19-54.

Berry, M.S., & Smoothy, R. (1986). A critical evaluation of claimed relationships between alcohol intake and aggression in infra-human animals. In P.F. Brain (Ed.), Alcohol and Aggression. Dover, N.H.: Croom Helm, pp. 84-137.

Bertilson, H.S. (1990). Can aggression be justified in order to study aggression? American Behavioral Scientist, 33, 595-607.

Bianchi, L. (1922). The mechanism of the brain and the functions of the frontal lobes. New York: William Wood.

Blanchard, R.J., Brain, P.F., Blanchard, D.C., & Parmigiani, S. (Eds.), Ethoexperimental Approaches to the Study of Behaviour. Boston: Kluwer Academic Publishers.

Blanchard, R.J., Hori, K., Blanchard, D.C., & Hall, J. (1987). Ethanol effects on aggression of rats selected for different levels of aggressiveness. Pharmacology, Biochemistry and Behaviour, 27, 641-644.

Blanchard, D.C., Veniegas, R., Elloran, I., & Blanchard, R.J. (1993). Alcohol and anxiety: Effects on offensive and defensive aggression. Journal of Studies on Alcohol, Suppl. 11, 9-19.

Blumer, D., & Benson, D.F. (1975). Personality changes with frontal and temporal lobe lesions. In D.F. Benson & D. Blumer (Eds.), Psychiatric aspects of neurologic disease. New York: Grune & Stratton, pp. 151-170.

Bond, A., & Lader, M. (1986). A method to elicit

aggressive feelings and behaviour via provocation. Biological Psychology, 22, 69-79.

Borden, R.J., & Taylor, S.P. (1973). The social instigation and control of physical aggression. Journal of Applied Social Psychology, 3, 354-361.

Boyatzis, R.E. (1974). The effect of alcohol consumption on the aggressive behaviour of men. Quarterly Journal of Studies on Alcohol, 35, 959-972.

Boyatzis, R.E. (1975). The predisposition toward alcohol-related interpersonal aggression in men. Journal of Studies on Alcohol, 36, 1196-1208.

Brain, P.F. (1981). Differentiating types of attack and defense in rodents. In P.F. Brain, & D. Benton (Eds.), Multidisciplinary Approaches to Aggression Research. Amsterdam: Elsevier/North-Holland and Biomedical Press, pp. 53-78.

Brain, P.F. (1984). Biological explanations of human aggression and the resulting therapies offered by such approaches: A critical evaluation. In R.J. Blanchard & D.C. Blanchard (Eds.), Advances in the Study of Aggression, 1. San Diego, Calif.: Academic Press, Inc., pp.63-102.

Brain, P.F. (1986). Multidisciplinary examinations of the "causes" of crime: The case of the link between alcohol and violence. Alcohol and Alcoholism, 21, 237-240.

Brain, P.F. (1989). The adaptiveness of house mouse aggression. In P.F. Brain, D. Mainardi, & S. Parmigiani (Eds.), House Mouse Aggression: A Model for Understanding the Evolution of Social Behaviour. New York: Harwood Academic Publishers, pp. 1-21.

Brain, P.F., Miras, R.L., & Berry, M.S. (1993). Diversity of animal models of aggression: Their impact on the putative alcohol/aggression link. Journal of Studies on Alcohol, Suppl. 11, 140-145.

Brizer, D.A. (1988). Psychopharmacology and the management of violent patients. Psychiatric Clinics of North America, 11, 551-568.

Brunner, H.G., Nelen, M., Breakefield, X.O., Ropers, H.H., & van Oost, B.A. (1993). Abnormal behaviour associated with a point mutation in the structural

gene for monoamine oxidase A. Science, 262, 578-580.

Brutus, M., Shaikh, M., Siegel, H., & Siegel, A. (1984). An analysis of the mechanisms underlying septal area control of hypothalamically elicited aggression in the cat. Brain Research, 310, 235-248.

Bryant, E.T., Scott, M.L., Golden, C.J., & Tori, C.D. (1984). Neuropsychological deficits, learning disability, and violent behaviour. Journal of Consulting and Clinical Psychology, 52, 323-324.

Bushman, B.J., & Cooper, H.M. (1990). Effects of alcohol on human aggression: An integrative research review. Psychological Bulletin, 107, 341-354.

Buss, A.H. (1961). The Psychology of Aggression. New York: Wiley.

Buss, A.H., & Durkee, A. (1957). An inventory for assessing different kinds of hostility. Journal of Consulting Psychology, 21, 343-349.

Buss, A.H., & Perry, P. (1992). The aggression questionnaire. Journal of Personality and Social Psychology, 63, 452-459.

Butter, C., Mishkin, M., & Mirsky, A. (1968). Emotional responses toward humans in monkeys with selective frontal lesions. Physiology and Behaviour, 3, 213-215.

Cavan, S. (1966). Liquor license: An ethnography of bar behaviour. Chicago: Aldine.

Cherek, D.R. (1981). Effects of smoking different doses of nicotine on human aggressive behaviour. Psychopharmacology, 75, 339-345.

Cherek, D.R., Steinberg, J.L., & Manno, R.B. (1985). Effects of alcohol on human aggressive behaviour. Journal of Studies on Alcohol, 46, 321-328.

Cherek, D.R., Steinberg, J.L., & Vines, R.V. (1984). Low doses of alcohol affect human aggressive responses. Journal of Studies on Alcohol, 46, 321-328.

Cherpitel, C.J. (1993). What emergency room studies reveal about alcohol involvement in violence-related injuries. Alcohol Health & Research World, 17, 162-

166.

Coid, J. (1986). Alcohol, rape and sexual assault. In P.F. Brain (Ed.), Alcohol and Aggression. Dover, N.H.: Croom Helm, pp. 161-183.

Collins, J.J., Jr. (Ed.)(1981). Drinking and Crime. New York: Guilford Press.

Collins, J.J., & Schlenger, W.E. (1988). Acute and chronic effects of alcohol use on violence. Journal of Studies on Alcohol, 49, 516-521.

Damasio, A. (1979). The frontal lobes. In K.M. Heilman, & E. Valenstein (Eds.), Clinical Neuropsychology. New York: Oxford University Press, pp. 360-412.

Damasio, H., Grabowski, T., Frank, R., Galaburda, A.M., & Damasio, A.R. (1994). The return of Phineas Gage: Clues about the brain from the skull of a famous patient. Science, 264, 1102-1105.

Dengerink, H.A. (1971). Anxiety, aggression and physiological arousal. Journal of Experimental Research in Personality, 5, 223-232.

Denno, D. (1990). Biology and Violence: From Birth to Adulthood. Cambridge: Cambridge University Press.

Dimond, S.J. (1980). Neuropsychology: A Textbook of Systems and Psychological Functions of the Human Brain. London: Butterworth.

Dodge, K.A., & Coie, J.D. (1987). Social information-processing factors in reactive and proactive aggression in children's peer groups. Journal of Personality and Social Psychology, 53, 1146-1158.

Dodge, K.A., & Crick, N.R. (1990). Social information-processing bases of aggressive behaviour in children. Personality and Social Psychology Bulletin, 16, 8-22.

Edmunds, G., & Kendrick, D.C., (1980) The Measurement of Human Aggressiveness. New York: John Wiley & Sons, pp. 39-66.

Elliot, F.A. (1992). Violence: The neurologic contribution: An overview. Archives of Neurology, 49, 595-603.

Ervin, F.R., Palmour, R.M., Young, S.N., Guzman-Flores,

C., & Juarez, J. (1990). Voluntary consumption of beverage alcohol by vervet monkeys: Population screening, descriptive behavior and biochemical measures. Pharmacology, Biochemistry, & Behaviour, 36, 367-373.

Eslinger, P.J., & Damasio, A.R. (1985). Severe disturbances of higher cognition after bilateral frontal lobe ablation: patient EVR. Neurology, 35, 1731-1741.

Evans, C.M. (1986). Alcohol and violence: Problems relating to methodology, statistics and causation. In P.F. Brain (Ed.), Alcohol and Aggression. Dover, N.H.: Croom Helm, pp. 138-160.

Farrington, D.P., Loeber, R. & Van Kammen, W.B. (1990). Long-term criminal outcomes of hyperactivity-impulsivity-attention deficit and conduct problems in childhood. In L.N. Robins & M. Rutter (Eds.) Straight and devious pathways from childhood to adulthood. Cambridge, Mass.: Cambridge University Press, pp. 62-81.

Felson, R.B., & Tedeschi, J.T. (1993). Aggression and Violence: Social Interactionist Perspectives. Washington, D.C.: American Psychological Association.

Feshbach, S. (1970). Aggression. In P.H. Mussen (Ed.), Carmichael's manual of child psychology. New York: Wiley, pp. 159-259.

Fishbein, D.H., Jaffe, J.H., Snyder, F.R., Haertzen, C.A., & Hickey, J.E. (1993). Drug users' self-reports of behaviours and affective states under the influence of alcohol. The International Journal of the Addictions, 28, 1565-1585.

Fowles, D.C. (1980). The three arousal model: Implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. Psychophysiology, 17, 87-104.

Fowles, D.C. (1988). Psychophysiology and psychopathology: A motivational approach. Psychophysiology, 25, 373-391.

Fuster, J.M. (1989). The Prefrontal Cortex: Anatomy, Physiology, and Neuropsychology of the Frontal Lobe. New York: Raven Press.

Gaebelin, J.W., & Taylor, S.P. (1971). The effects of competition and attack on physical aggression. Psychonomic Science, 24, 65-67.

Genthner, R.W., & Taylor, S.P. (1973). Physical aggression as a function of racial prejudice and the race of the target. Journal of Personality and Social Psychology, 27, 207-210.

George, W.H., Dermen, K.H., & Nochajski, T.H. (1989). Expectancy set, self-reported expectancies and predispositional traits: Predicting interest in violence and erotica. Journal of Studies on Alcohol, 50, 541-551.

George, W.H., & Marlatt, G.A. (1986). The effects of alcohol and anger on interest in violence, erotica, and deviance. Journal of Abnormal Psychology, 95, 150-158.

Giancola, P.R. (1995). Evidence for dorsolateral and orbital prefrontal cortical involvement in the expression of aggressive behaviour. Aggressive Behaviour, in press.

Giancola, P.R., Peterson, J.B., & Pihl, R.O. (1993). Risk for alcoholism, antisocial behaviour, and response perseveration. Journal of Clinical Psychology, 49, 423-428.

Giancola, P.R., & Zeichner, A. (1994a). Construct validity of a competitive reaction-time aggression paradigm. Unpublished manuscript.

Giancola, P.R., & Zeichner, A. (1994b). Neuropsychological performance on tests of frontal-lobe functioning and aggression in human males. Journal of Abnormal Psychology, 103, 832-835.

Giancola, P.R., & Zeichner, A. (1995). An investigation of gender differences in alcohol-related aggression. Journal of Studies on Alcohol, in press.

Goldman-Rakic, P.S. (1988). Topography of cognition: Parallel distributed networks in primate association cortex. Annual Review of Neuroscience, 11, 137-156.

Goldman-Rakic, P.S. (1992). Working memory and the mind. Scientific American, 267, 111-117.

Goldstein, G. (1986). The neuropsychology of

schizophrenia. In I. Grant, & K.M. Adams (Eds.), Neuropsychological assessment of neuropsychiatric disorders. New York: Oxford University Press.

Goodman, R.A., Mercy, J.A., Loya, F., Rosenberg, M.L., Smith, J.S., Allen, N.H., Vargas, L., & Kolts, R. (1986). Alcohol use and interpersonal violence: Alcohol detected in homicide victims. American Journal of Public Health, 76, 144-149.

Goodwin, D.W. (1973). Alcohol in suicide and homicide. Quarterly Journal of Studies on Alcohol, 34, 144-156.

Gorenstein, E.E. (1982). Frontal lobe functions in psychopaths. Journal of Abnormal Psychology, 91, 368-379.

Gorenstein, E.E. (1987). Cognitive-perceptual deficit in an alcoholism spectrum disorder. Journal of Studies on Alcohol, 48, 310-318.

Gorenstein, E.E., & Newman, J.P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. Psychological Review, 87, 301-315.

Gottfredson, M.R. & Hirschi, T. (1993). A control theory interpretation of psychological research on aggression. In R.B. Felson & J.T. Tedeschi (Eds.) Aggression and Violence: Social Interactionist Perspectives. Washington, D.C.: American Psychological Association.

Grafman, J., Vance, S., Weingartner, H., Salazar, A., & Amin, D. (1986). The effects of lateralized frontal lesions on mood regulation. Brain, 109, 1127-1148.

Graham, K. (1980). Theories of intoxicated aggression. Canadian Journal of Behavioral Science, 12, 141-158.

Graham, K., LaRocque, L., Yetman, R., Ross, T.J., & Guistra, E. (1980). Aggression and barroom environments. Journal of Studies on Alcohol, 41, 277-292.

Gray, J.A. (1975). Elements of a two-process theory of learning. New York: Academic Press.

Gray, J.A. (1982). The Neuropsychology of Anxiety: An Enquiry into the Function of the Septo-Hippocampal System. New York: Oxford Univ. Press.

Gray, J.A. (1987). The Psychology of Fear and Stress, 2nd Edition. New York: Cambridge Univ. Press.

Greenberg, L.A. (1970). The appearance of some congeners of alcoholic beverages and their metabolites in blood. Quarterly Journal of Studies on Alcohol, Suppl. 5, 20-25.

Greenberg, S.W. (1981). Alcohol and crime: A methodological critique of the literature. In J.J. Collins (Ed.), Drinking and Crime. New York: Guilford Press.

Greizerstein, H.B. (1981). Congener contents of alcoholic beverages. Journal of Studies on Alcohol, 42, 1030-1037.

Gustafson, R. (1984). Alcohol, frustration, and direct physical aggression: A methodological point of view. Psychological Reports, 55, 959-966.

Gustafson, R. (1985a). Frustration as an important determinant of alcohol-related aggression. Psychological Reports, 57, 3-14.

Gustafson, R. (1985b). Alcohol and aggression: A validation study of the Taylor aggression paradigm. Psychological Reports, 57, 667-676.

Gustafson, R. (1985c). Alcohol and aggression - Pharmacological versus expectancy effects. Psychological Reports, 57, 955-966.

Gustafson, R. (1986a). A possible confounding variable in different versions of the "aggression machine" when used in research on alcohol. Psychological Reports, 58, 303-308.

Gustafson, R. (1986b). Alcohol, aggression and the validity of experimental paradigms with women. Psychological Reports, 59, 51-56.

Gustafson, R. (1988). Beer intoxication and physical aggression in males. Drug and Alcohol Dependence, 21, 237-242.

Gustafson, R. (1990). Wine and male physical aggression. Journal of Drug Issues, 20, 75-86.

Gustafson, R. (1991a). Male physical aggression as a function of alcohol intoxication and frustration: Experimental results and methodological

considerations. Alcoholism: Clinical and Experimental Research, 15, 158-164.

Gustafson, R. (1991b). Aggressive and nonaggressive behaviour as a function of alcohol intoxication and frustration in women. Alcoholism: Clinical and Experimental Research, 15, 886-892.

Gustafson, R. (1992). Alcohol and aggression: A replication study controlling for potential confounding variables. Aggressive Behaviour, 18, 21-28.

Gustafson, R. (1993). What do experimental paradigms tell us about alcohol-related aggressive responding? Journal of Studies on Alcohol, Suppl. 11, 20-29.

Gustafson, R., & Kallmen, H. (1988). Alcohol and unpleasant stimulation: Subjective shock calibrations and pain and discomfort perception. Perception & Motor Skills, 66, 739-742.

Guze, S.B., Tuason, V.B., Gatfield, P.D., Stewart, M.A., & Picken, B. (1962). Psychiatric illness and crime with particular reference to alcoholism; studies of 233 criminals. Journal of Nervous and Mental Diseases, 134, 512-521.

Hammett, M., Powell, K.E., O'Carroll, P.W., & Clanton, S.T. (1992). Homicide surveillance, 1979-1988. In Centers for Disease Control and Prevention Surveillance Summaries, 29 May 1992, Morbidity and Mortality Weekly Report 41, no. SS-3, 1-33.

Hammock, G.S., & Richardson, D.R. (1992). Predictors of aggressive behaviour. Aggressive Behaviour, 18, 219-229.

Harden, P.W. & Pihl, R.O. (1995). Cognitive function, cardiovascular reactivity, and behaviour in boys at high risk for alcoholism. Journal of Abnormal Psychology, 104, 94-103.

Hare, R.D. (1980). A research scale for the assessment of psychopathy in criminal populations. Personality and Individual Differences, 1, 111-119.

Hare, R.D. (1984). Performance of psychopaths on cognitive tasks related to frontal lobe function. Journal of abnormal Psychology, 93, 133-140.

Hare, R.D., & McPherson, L.M. (1984). Psychopathy and

perceptual asymmetry during verbal dichotic listening. Journal of Abnormal Psychology, 93, 141-149.

Harlow, J.M. (1868). Recovery after severe injury to the head. Publication of the Massachusetts Medical Society, 2, 327-346.

Hart, S.D., Forth, A.H., & Hare, R.D. (1990). Performance of criminal psychopaths on selected neuropsychological tests. Journal of Abnormal Psychology, 99, 374-379.

Heath, D.B. (1983). Alcohol and aggression: A "missing link" in worldwide perspective. In E. Gottheil, K.A. Druley, T.E. Skoloda, & H.M. Waxman (Eds.), Alcohol, Drug Abuse and Aggression. Springfield, Ill: Charles C. Thomas, Publishers, pp. 89-103.

Hecaen, H., & Albert, M.L. (1978). Human Neuropsychology. New York: Wiley.

Heinrichs, R.W. (1989). Frontal cerebral lesions and violent incidents in chronic neuropsychiatric patients. Biological Psychiatry, 25, 174-178.

Helzer, J.E., & Pryzbeck, T.R. (1988). The co-occurrence of alcoholism with other psychiatric disorders in the general population and its impact on treatment. Journal of Studies on Alcohol, 49, 219-224.

Hendrick, C., & Taylor, S.P. (1971). Effects of belief similarity and aggression on attraction and counteraggression. Journal of Personality and Social Psychology, 17, 342-349.

Hendricks, S.E., Fitzpatrick, D.F., Hartmann, K., Quaife, M.A., Stratbucker, R.A., & Graber, B. (1988). Brain structure and function in sexual molesters of children and adolescents. Journal of Clinical Psychiatry, 49, 108-112.

Hesselbrock, M., Meyer, R.E., & Keener, J.J. (1985). Psychopathology in hospitalized alcoholics. Archives of General Psychiatry, 42, 1050-1055.

Hetherington, E.M., & Wray, N.P. (1964). Aggression, need for social approval, and humor preferences. Journal of Abnormal Social Psychology, 68, 685-689.

Higley, J.D., Suomi, S.J., & Linnoila, M. (1990). Parallels in aggression and serotonin:

Consideration of development, rearing history, and sex differences. In H.M. Van Praag, R. Plutchik, & A. Apter (Eds.), Violence and Suicidality: Perspectives in Clinical and Psychobiological Research. New York: Brunner/Mazel, pp. 245-246.

Hoffman, J.J., Hall, R.W., & Bartsch, T.W. (1987). On the relative importance of "Psychopathic" personality and alcoholism on neuropsychological measures of frontal lobe dysfunction. Journal of Abnormal Psychology, 96, 158-160.

Horton, D. (1943). The functions of alcohol in primitive societies: A cross-cultural study. Quarterly Journal of Studies on Alcohol, 4, 199-320.

Hull, J., & Bond, C. (1986). Social and behavioral consequences of alcohol consumption and expectancy: A meta-analysis. Psychological Bulletin, 99, 347-360.

Hurt, J., & Naglieri, J.A. (1992). Performance of delinquent and nondelinquent males on planning, attention, simultaneous, and successive cognitive processing tasks. Journal of Clinical Psychology, 48, 120-128.

Jaffe, J., Babor, T., & Fishbein, D. (1988). Alcoholics, aggression and antisocial personality. Journal of Studies on Alcohol, 49, 211-218.

Jeavons, C.M., & Taylor, S.P. (1985). The control of alcohol-related aggression: Redirecting the inebriate's attention to socially appropriate conduct. Aggressive Behaviour, 11, 93-101.

Johnston, L.D., O'Malley, P.M., & Eveland, L.K. (1978). Drugs and delinquency: A search for causal connections. In D.B. Kandel (Ed.), Longitudinal Research on Drug Use: Empirical Findings and Methodological Issues. New York: John Wiley & Sons, Inc., pp. 137-156.

Kalin, R. (1972). Social drinking in different settings. In D.C. McClelland, W.N. Davis, R. Kalin, & E. Wanner (Eds.), The Drinking Man. New York: Free Press.

Kamback, M. (1973). The effects of orbital and dorsolateral frontal cortical ablations on ethanol self-selection and emotional behaviours in monkeys (macaca nemestrina). Neuropsychologia, 11, 331-335.

Kamback, M. & Rogal, R. (1973). The effects of frontal cortical ablations on alcohol selection and emotionality in pigtail monkeys (Macaca nemestrina). Biological Psychiatry, 7, 173-177.

Kandel, E., & Freed, D. (1989). Frontal-lobe dysfunction and antisocial behaviour: A review. Journal of Clinical Psychology, 45, 404-413.

Kantnor, G., & Strauss, M.A. (1987). The "Drunken Bum" theory of wife beating. Social Problems, 34, 213-230.

Katkin, E.S., Hayes, W.N., Teger, A.I., & Prutt, D.G. (1970). Effects of alcoholic beverages differing in congener content on psychomotor tasks and risk taking. Quarterly Journal of Studies on Alcohol, Suppl. 5, 101-114.

Kelly, T.H., & Cherek, D.R. (1993). The effects of alcohol on free-operant aggressive behaviour. Journal of Studies on Alcohol, Suppl. 11, 40-52.

Kelly, T.H., Cherek, D.R., Steinberg, J.L., & Robinson, D. (1988). Effects of provocation and alcohol on human aggressive behaviour. Drug and Alcohol Dependence, 21, 105-112.

Khantzian, E.J. (1985). The self-medication hypothesis of addictive disorders. Focus on heroin and cocaine dependence. American Journal of Psychiatry, 142, 1259-1264.

Kling, A. (1976). Frontal and temporal lobe lesions and aggressive behaviour. In W. Smith & A. Kling (eds.) Issues in brain/behaviour control. New York: Spectrum, pp. 11-22.

Kreutzer, J., Schneider, H., & Myatt, C. (1984). Alcohol, aggression, and assertiveness in men: Dosage and expectancy effects. Journal of Studies on Alcohol, 45, 275-278.

Krsiak, M. (1977). Effect of ethanol on aggression and timidity in mice. Psychopharmacology, 51, 75-80.

Ladouceur, P., & Temple, M. (1985). Substance use among rapists: A comparison with other serious felons. Crime & Delinquency, 31, 269-294.

Lagerspetz, K.M.J. (1981). Combining aggression studies in infra-humans and man. In P.F. Brain, & D. Benton

(Eds.), Multidisciplinary Approaches to Aggression Research. Amsterdam: Elsevier/North-Holland Biomedical Press, pp. 390-400.

Lang, A.R., Goeckner, D., Adesso, U., & Marlatt, G. (1975). Effects of alcohol on aggression in male social drinkers. Journal of Abnormal Psychology, 84, 508-518.

Lang, A.R., & Sibrel, P.A. (1989). Psychological perspectives on alcohol consumption and interpersonal aggression: The potential role of individual differences in alcohol-related criminal violence. Criminal Justice and Behaviour, 16, 299-324.

Lau, M.A., Pihl, R.O., & Peterson, J.B. (1992). Alcohol, frontal lobe functioning, and aggression. Pharmacology, Biochemistry, and Behaviour, 42, 376.

Lenke, L. (1975). Valdbrott och alkohol: En studie i misshandelsbrottslighetens utveckling (Violent crime and alcohol: A study of the developments in assaultive crime). Stockholm: Department of Criminology, University of Stockholm.

Lenke, L. (1976). Alkohol och valdbrottslighet (Alcohol and criminal violence). Alkohol och Naarkotika, 70, 8-17.

Leonard, K.E. (1984). Alcohol consumption and escalatory aggression in intoxicated and sober dyads. Journal of Studies on Alcohol, 45, 75-80.

Leonard, K.E., & Blane, H.T. (1992). Alcohol and marital aggression in a national sample of young men. Journal of Interpersonal Violence, 7, 19-30.

Lester, D. (1992). Alcohol consumption and rates of personal violence in Australia. Drug and Alcohol Dependence, 31, 15-17.

Lewis, C.E., Cloninger, C.R., & Pais, J. (1983). Alcoholism, antisocial personality and drug use in a criminal population. Alcohol and Alcoholism, 18, 53-60.

Lewis, D.O., Lovely, R., Yeager, C., & Della Femina, D. (1989). Toward a theory of the genesis of violence: A follow-up study of delinquents. Journal of the American Academy of Child Psychiatry, 28, 431-436.

Lezak, M.D. (1983). Neuropsychological assessment. New York: Oxford University Press.

Lidberg, L., Levander, S.E., Schalling, D., & Lidberg, Y. (1978). Necker cube reversals, arousal, and psychopathy. British Journal of Social and Clinical Psychology, 17, 355-361.

Lindeman, R., & Lang, A.R. (1986). Anticipated effects of alcohol consumption as a function of beverage type: A cross-cultural replication. International Journal of Psychology, 21, 671-678.

Lindman, R., Jarvinen, P., & Vidjeskog, J. (1987). Verbal interactions of aggressively and nonaggressively predisposed males in a drinking situation. Aggressive Behaviour, 13, 187-196.

Lindqvist, P. (1986). Criminal homicide in northern Sweden 1970-1981: Alcohol intoxication, alcohol abuse, and mental disease. International Journal of Law and Psychiatry, 8, 19-37.

Lueger, R.J., & Gill, K.J. (1990). Frontal-lobe cognitive dysfunction in conduct disorder adolescents. Journal of Clinical Psychology, 46, 696-706.

Luria, A. R. (1980). Higher Cortical Functions in Man, 2nd edition. New York: Basic Books, Inc.

MacAndrew, C., & Edgarton, R.B. (1969). Drunken Comportment: A Social Explanation. Chicago: Aldine.

MacLean, P.D. (1990). The Triune Brain in Evolution: Role in Paleocerebral Functions. New York: Plenum Press.

Mark, V., & Ervin, F.R. (1970). Violence and the Brain. New York: Harper & Row.

Marlatt, G.A., Kosturn, C.F., & Lang, A.R. (1975). Provocation to anger and opportunity for retaliation as determinants of alcohol consumption in social drinkers. Journal of Abnormal Psychology, 84, 652-659.

Marlatt, G., & Rohsenow, D. (1980). Cognitive processes in alcohol use: Expectancy and the balanced placebo design. In N. Mello (Ed.), Advances in substance abuse: Behavioral and biological research. Greenwich, CT: JAI Press.

Martin & Dobrixhoffer (1822). As referenced in C.

MacAndrew, & R.B. Edgarton (1969). Drunken Comportment: A Social Explanation. Chicago: Aldine.

Mass, R. & Kling, A. (1975). Social behaviour in stump-tailed macaques (macaca speciosa) after lesions of the dorsolateral frontal cortex. Primates, 16, 239-252.

Mass-Observation. (1943). The Pub and the People: A Worktown Study. London: Victor Gollancz.

Mattes, J.A. (1980). The role of frontal-lobe dysfunction in childhood hyperkinesis. Comprehensive Psychiatry, 21, 358-369.

Mayfield, D. (1976). Alcoholism, alcohol intoxication, and assaultive behaviour. Diseases of the Nervous System, 37, 288-291.

McClelland, D.C., Davis, W.N., Wanner, E., & Kalin, R. (1972). A cross-cultural study of folk-tale content and drinking. In D.C. McClelland, W.N. Davis, R. Kalin, & E. Wanner (Eds.), The Drinking Man. New York: Free Press.

Mednick, S.A., Pollock, V., Volavka, J., & Gabrielli, W.F. (1982). Biology and violence. In M.E. Wolfgang, & N.A. Weiner (Eds.), Criminal Violence. Beverly Hills, Calif.: Sage.

Mercy, J.A., Rosenberg, M.L., Powell, K.E., Broome, C.V., & Roper, W.L. (1993). Public health policy for preventing violence. Health Affairs, Winter, 7-29.

Meyers, C.A., Berman, S.A., Scheibel, R.S., & Hayman, A. (1992). Case Report: Acquired antisocial personality disorder associated with unilateral left orbital frontal lobe damage. Journal of psychiatry and Neuroscience, 17, 121-125.

Miczek, K.A., Kruk, M.R., & Olivier, B. (1984). (Eds.) Ethopharmacological aggression research. New York: Alan R. Liss, Inc.

Miczek, K.A., Weerts, E.M., & DeBold, J.F. (1993). Alcohol, benzodiazepine-GABAA receptor complex and aggression: Ethological analysis of individual differences in rodents and primates. Journal of Studies on Alcohol, Suppl. 11, 170-179.

Miczek, K.A., Weerts, E.M., Haney, M., & Tidey, J. (1994). Neurobiological mechanisms controlling

aggression: Preclinical developments for pharmacotherapeutic interventions. Neuroscience and Biobehavioral Reviews, 18, 97-110.

Milgram, S. (1963). Behavioral study of obedience. Journal of Abnormal and Social Psychology, 67, 137-143.

Miller, L. (1987). Neuropsychology of the aggressive psychopath: An integrative review. Aggressive Behaviour, 13, 119-140.

Miller, M. (1976). Dorsolateral frontal lobe lesions and behaviour in the macaque: Dissociation of threat and aggression. Physiology and Behaviour, 17, 209-213.

Miller, T.R., Cohen, M.A., & Rossman, S.B. (1993). Victim costs of violent crime and resulting injuries. Health Affairs, Winter, 187-198.

Millon, T. (1981). Disorders of personality: DSM-III, Axis II. New York: John Wiley & Sons.

Milner, B. & Petrides, M. (1984). Behavioral effects of frontal-lobe lesions in man. Trends in Neurosciences, November, 403-407.

Moffitt, T.E. (1990). The neuropsychology of juvenile delinquency: A critical review. In M. Tonry, & N. Morris, (Eds.), Crime and Justice: A Review of Research. Chicago: University of Chicago Press.

Moffitt, T.E., & Henry, B. (1989). Neuropsychological assessment of executive functions in self-reported delinquents. Development and Psychopathology, 1, 105-118.

Moffitt, T.E., & Henry, B. (1991). Neuropsychological studies of juvenile delinquency and violence: A review. In J. Milner (Ed.), The neuropsychology of aggression. Norwell, MA: Kluwer Academic Publishers, pp. 67-91.

Moffitt, T.E., Lynam, D.R., & Silva, P.A. (1994). Neuropsychological tests predicting persistent male delinquency. Criminology, 32, 277-300.

Murdoch, D.D., & Pihl, R.O. (1988). The influence of beverage type on aggression in males in the natural setting. Aggressive Behaviour, 14, 325-335.

Murdoch, D.D., Pihl, R.O., & Ross, D. (1988). The influence of dose, beverage type, and sex of interactor on female bar patrons' verbal aggression. The International Journal of the Addictions, 23, 953-966.

Murdoch, D., Pihl, R.O., & Ross, D. (1990). Alcohol and crimes of violence: Present issues. The International Journal of the Addictions, 25, 1059-1075.

Myerscough, R., & Taylor, S.P. (1985). The effects of marijuana on human physical aggression. Journal of Personality and Social Psychology, 49, 1541-1546.

Nauta, W.J. (1971). The problem of the frontal lobe: A reinterpretation. Journal of Psychiatric Research, 8, 167-187.

Newman, J.P., Patterson, C.M., & Kosson, D.S. (1987). Response perseveration in psychopaths. Journal of Abnormal Psychology, 96, 145-148.

Nimuendaju (1948). As referenced in C. MacAndrew, & R.B. Edgarton (1969). Drunken Comportment: A Social Explanation. Chicago: Aldine.

Passingham, R.E. (1993). The Frontal Lobes and Voluntary Action. New York: The Clarendon Press.

Patterson, C.M., & Newman, J.P. (1993). Reflectivity and learning from aversive events: Toward a psychological mechanism for the syndromes of disinhibition. Psychological Review, 100, 716-736.

Perecman, E. (1987). Consciousness and the meta-functions of the frontal lobes: Setting the stage. In E. Perecman (Ed.), The frontal lobes revisited. New York: IRBN Press, pp. 1-10.

Peretti, P.O., & Lewis, B.R. (1969). Effects of alcoholic consumption on the activity patterns of individual rhesus monkeys and their behaviour in a social group. Primates, 10, 181-188.

Pernanen, K. (1976). Alcohol and crimes of violence. In B. Kissin & H. Begleiter (Eds.), The Biology of Alcoholism, 4. New York: Plenum Press.

Pernanen, K. (1981). Theoretical aspects of the relationship between alcohol use and crime. In J. Collins (Ed.), Drinking and Crime. New York:

Guilford Press, pp. 1-69.

Peterson, J.B., Finn, P.R., & Pihl, R.O. (1992). Cognitive dysfunction and the inherited predisposition to alcoholism. Journal of Studies on Alcohol, 53, 154-160.

Peterson, J.B., & Pihl, R.O. (1990). Information processing, neuropsychological function, and the inherited predisposition to alcoholism. Neuropsychology Review, 1, 343-369.

Peterson, J.B., Rothfleisch, J., Zelazo, P.D., & Pihl, R.O. (1990). Acute alcohol intoxication and cognitive functioning. Journal of Studies on Alcohol, 51, 114-122.

Petrides, M. (1985a). Deficits on conditional associative-learning tasks after frontal- and temporal-lobe lesions in man. Neuropsychologia, 23, 601-614.

Petrides, M. (1985b). Deficits in non-spatial conditional associative learning after periarcuate lesions in the monkey. Behavioral Brain Research, 16, 95-101.

Petrides, M. (1991). Proc. R. Soc. London B 246, 293-298.

Petrides, M., Alivisatos, B., Evans, A.C., & Meyer, E. (1993). Dissociation of human mid-dorsolateral from posterior dorsolateral frontal cortex in memory processing. Proceedings of the National Academy of Sciences, 90, 873-877.

Petrides, M., & Milner, B. (1982). Deficits on subject-ordered tasks after frontal- and temporal-lobe lesions in man. Neuropsychologia, 20, 249-262.

Pihl, R.O. (1983). Alcohol and Aggression: A psychological perspective. In E. Gottheil, K.A. Druley, T.E. Skoloda, & H.M. Waxman (Eds.), Alcohol, Drug Abuse and Aggression. Springfield, Ill: Charles C. Thomas.

Pihl, R.O., Lau, M.A., & Assaad, J.M. (1995). Aggressive disposition, alcohol and aggression. Manuscript under review.

Pihl, R.O., & Peterson, J.B. (1992). The etiology of drug abuse. In P.E. Nathan, J.W. Langenbucher, B.S. McCrady, & W. Frankenstein (Eds.), Annual Review of Addictions Research and Treatment. Elmsford, N.Y.:

Pergamon Press, Inc, pp. 153-175.

Pihl, R.O., & Peterson, J.B. (1993). Alcohol, Serotonin, and Aggression. Alcohol Health and Research World, 17, 113-116.

Pihl, R.O., Peterson, J.B., & Finn, P.R. (1987). Autonomic reactivity and neuropsychological deficits in men at high risk for alcoholism. Paper presented at the Fourth Annual Conference on the Treatment of Addictive Behaviours, Bergen, Norway.

Pihl, R.O., Peterson, J.B., & Lau, M.A. (1993). A biosocial model of the alcohol-aggression relationship. Journal of Studies on Alcohol, Suppl. 11, 128-139.

Pihl, R.O., & Ross, D. (1987). Research on alcohol related aggression: A review and implications for understanding aggression. Drugs and Society, 1, 105-126.

Pihl, R.O., Smith, M., & Farrell, B. (1984). Alcohol and aggression in men: A comparison of brewed and distilled beverages. Journal of Studies on Alcohol, 45, 278-282.

Pihl, R.O., Zacchia, C., & Zeichner, A. (1981). Follow-up analysis of the use of deception and aversive contingencies in psychological experiments. Psychological Reports, 48, 927-930.

Pihl, R.O., Zacchia, C., & Zeichner, A. (1982). Predicting levels of aggression after alcohol intake in men social drinkers: A preliminary investigation. Journal of Studies on Alcohol, 43, 599-602.

Pisano, R., & Taylor, S.P. (1971). Reduction of physical aggression: The effects of four strategies. Journal of Personality and Social Psychology, 19, 237-242.

Pittman, D.J., & Handy, W. (1964). Patterns in criminal aggravated assault. Journal of Criminal Law, Criminology, and Police Science, 55, 462-470.

Preuss, T. (1995). Do rats have prefrontal cortex? The Rose-Woolsey-Akert program reconsidered. Journal of Cognitive Neuroscience, 7, 1-24.

Price, B.H., Daffner, K.R., Stowe, R.M., & Mesulam, M.M. (1990). The compartmental learning disabilities of

early frontal lobe damage. Brain, 113, 1383-1393.

Raine, A. (1993). The Psychopathology of Crime: Criminal Behaviour as a Clinical Disorder. San Diego: Academic Press Inc.

Raine, A., Buchsbaum, M.S., Stanley, J., Lottenberg, S., Abel, L., & Stoddard, J. (1993). Selective reductions in pre-frontal glucose metabolism in murders assessed with positron emission tomography. Manuscript under review.

Reiss Jr., A.J., & Roth, J.A. (1993). Understanding and Preventing Violence. Washington: National Academy Press.

Richardson, D. (1981). The effect of alcohol on male aggression toward female targets. Motivation and Emotion, 5, 333-344.

Rogers, R.W. (1983). Race variables in aggression. In R.G. Geen & E.I. Donnerstein (Eds.), Aggression: Theoretical and empirical reviews, Vol. 2. New York: Academic Press, pp. 27-50.

Rohsenow, D.J., & Bacharowski, J.A. (1984). Effects of alcohol and expectancies on verbal aggression in men and women. Journal of Abnormal Psychology, 93, 418-432.

Sayette, M.A., Wilson, G.T., & Elias, M.J. (1993). Alcohol and aggression: A social information processing analysis. Journal of Studies on Alcohol, 54, 399-407.

Scerbo, A., & Raine, A. (1992). Neurotransmitters and antisocial behavior: A meta-analysis. Manuscript under review.

Schalling, D., & Rosen, A. (1968). Porteus maze differences between psychopathic and non-psychopathic criminals. British Journal of Social and Clinical Psychology, 7, 224-228.

Schmutte, G.T., Leonard, K.E., & Taylor, S.P. (1979). Alcohol and expectations of attack. Psychological Reports, 45, 163-167.

Seguin, J.R., Pihl, R.O., Harden, P.W., Tremblay, R.E., & Boulerice, B. (1995). Cognitive and neuropsychological characteristics of physically aggressive boys. Journal of Abnormal Psychology, in

press.

Shepherd, J., Irish, M., Scully, C., & Leslie, I. (1989). Alcohol consumption among victims of violence and among comparable U.K. populations. British Journal of Addictions, 84, 1045-1051.

Sher, K.J., & Trull, T.J. (1994). Personality and disinhibitory psychopathology: Alcoholism and antisocial personality disorder. Journal of Abnormal Psychology, 103, 92-102.

Shuck, J., & Pisor, K. (1974). Evaluating an aggressive experiment by the use of simulating subjects. Journal of Personality and Social Psychology, 29, 181-196.

Shue, K.L. & Douglas, V.I. (1992). Attention deficit hyperactivity disorder and the frontal lobe syndrome. Brain and Cognition, 20, 104-124.

Shuntich, R.J., & Taylor, S.P. (1972). The effects of alcohol and human physical aggression. Journal of Experimental Research in Personality, 6, 34-38.

Shupe, L.M. (1954). Alcohol and crime: A study of the urine alcohol concentration found in 882 persons arrested during or immediately after the commission of a felony. Journal of Criminal Law, Criminology, and Police Science, 44, 661-664.

Siegel, A. & Edinger, H. (1983). Role of the limbic system in hypothalamically elicited attack behaviour. Neuroscience and Biobehavioral Reviews, 7, 395-407.

Siegel, A., Edinger, H., & Dotto, M. (1975). Effects of electrical stimulation of the lateral aspect of the prefrontal cortex upon attack behaviours in cats. Brain Research, 93, 473-484.

Siegel, A., Edinger, H., & Koo, A. (1977). Suppression of attack behaviour in the cat by the prefrontal cortex: Role of the mediodorsal thalamic nucleus. Brain Research, 127, 185-190.

Smith, S.S., Arnett, P.A., & Newman, J.P. (1992). Neuropsychological differentiation of psychopathic and nonpsychopathic criminal offenders. Personality and Individual Differences, 13, 1233-1243.

Smoothy, R., & Berry, M.S. (1983). Effects of ethanol on

behaviour of aggressive mice from two different strains: A comparison of simple and complex behavioral assessments. Pharmacology and Biochemistry of Behaviour, 19, 645-653.

Spielberger, C.D., Johnson, E.H., Russell, S.F., Crane, R.J., Jacobs, G.A., & Worden, T.J. (1985). The experience and expression of anger: Construction and validation of an anger expression scale. In M.A. Chesney, R.H. Rosenman (Eds.) Anger and Hostility in Cardiovascular and Behavioral Disorders. Washington: Hemisphere, pp. 5-30.

Steele, C.M, & Josephs, R.A. (1990). Alcohol myopia: Its prized and dangerous effects. American Psychologist, 45, 921-933.

Steele, C.M., & Southwick, L. (1985). Alcohol and social behaviour I: The psychology of drunken excess. Journal of Personality and Social Psychology, 48, 18-34.

Stuss, D.T., & Benson, D.F. (1983). Emotional concomitants of psychosurgery. In K.M. Heilman, & P. Satz (Eds.), Advances in neuropsychology and behavioral neurology: Vol. 1. Neuropsychology of human emotion. New York: Guilford Press, pp. 111-140.

Stuss, D.T., & Benson, D.F. (1984). Neuropsychological studies of the frontal lobes. Psychological Bulletin, 95, 3-28.

Stuss, D.T., & Benson, D.F. (1986). The frontal lobes. New York: Raven Press.

Stuss, D.T., Gow, C.A., & Hetherington, C.R. (1992). "No longer Gage": Frontal lobe dysfunction and emotional changes. Journal of Consulting and Clinical Psychology, 60, 349-359.

Sutker, P.B., & Allain, A.N. (1983). Behaviour and personality assessment in men labelled adaptive sociopaths. Journal of Behavioral Assessment, 5, 65-79.

Takala, M. (1973). Alkoholstrejkens inverkan pa uppdagad brottslighet (The effect of the alcohol strike on reported crime). Alkoholpolitik, 36, 14-16.

Takala, M., Pihkanen, T.A., & Markkanen, T. (1957). The effects of distilled and brewed beverages:

Physiological, neurological and psychological study. Helsinki: The Finnish Foundation for Alcohol Studies, Publ, No. 4.

Tancredi, L.R., & Volkow, N. (1988). Neural substrates of violent behaviour: Implications for law and public policy. International Journal of Law and Psychiatry, 11, 13-49.

Tarter, R.E., Hegedus, A.M., Goldstein, B., Shelly, C., & Alterman, L. (1984). Adolescent sons of alcoholics: Neuropsychological and personality characteristics. Alcoholism: Clinical and Experimental Research, 8, 330-33.

Taylor, S.P. (1967). Aggressive behaviour and physiological arousal as a function of provocation and the tendency to inhibit aggression. Journal of Personality, 35, 297-310.

Taylor, S.P. (1970). Aggressive behaviour as a function of approval motivation and physical attack. Psychonomic Science, 18, 195-196.

Taylor, S.P., & Chermack, S.T. (1993). Alcohol, drugs and human physical aggression. Journal of Studies on Alcohol, Suppl. 11, 78-88.

Taylor, S.P., & Gammon, C.B. (1975). Effects of type and dose of alcohol on human physical aggression. Journal of Personality and Social Psychology, 32, 169-175.

Taylor, S.P., Gammon, C.B., & Capasso, D.P. (1976). Aggression as a function of the interaction of alcohol and threat. Journal of Personality and Social Psychology, 34, 938-941.

Taylor, S.P., & Leonard, K.E. (1983). Alcohol and human physical aggression. In R.G. Geen, & E.I. Donnerstein (Eds.), Aggression: Theoretical and Empirical Reviews, Vol. 2. San Diego, Calif.: Academic Press, Inc., pp 77-101.

Taylor, S.P., Schmutte, G.T., Leonard, K.E., & Cranston, J.W. (1979). The effects of alcohol and extreme provocation on the use of a highly noxious electric shock. Motivation and Emotion, 3, 73-81.

Taylor, S.P., & Sears, J.D. (1988). The effects of alcohol and persuasive social pressure on human physical aggression. Aggressive Behaviour, 14, 237-

243.

Taylor, S.P., Vardaris, R.M., Rawich, A.B., Gammon, C.B., Cranston, J.W., & Lubetkin, A.I. (1976). The effects of alcohol and delta-9-tetrahydrocannabinol on human physical aggression. Aggressive Behaviour, 2, 215-219.

Tinklenberg, J. (1973). Alcohol and violence. In P. Bourne & R. Fox (Eds.), Alcoholism: Progress in research and treatment. New York: Academic Press.

U.S. Bureau of the Census (1993). Statistical Abstract of the United States: 1993 (113th edition). Washington D.C.; U.S. Government Printing Office.

Virkkunen, M. (1974). Alcohol as a factor precipitating aggression and conflict of behaviour leading to homicide. British Journal of Addiction, 69, 149-154.

Virkkunen, M., & Linnoila, M. (1993). Brain serotonin, Type II alcoholism and impulsive violence. Journal of Studies on Alcohol, Suppl. 11, 163-169.

Volavka, J., Martell, D., & Convit, A. (1992). Psychobiology of the violent offender. Journal of Forensic Sciences, 37, 237-251.

Volkow, N.D., & Tancredi, L. (1987). Neural substrates of violent behaviour: A preliminary study with positron emission tomography. British Journal of Psychiatry, 151, 668-673.

Wasikhongo, J.M. (1976). Uniformities in aggravated assaults in St. Louis (Missouri) and Mombasa (Kenya): A cross-cultural replication. International Journal of Criminology and Penology, 4, 9-24.

Watson, R., Edinger, H., & Siegel, A. (1983). An analysis of the mechanisms underlying hippocampal control of hypothalamically-elicited aggression in the cat. Brain Research, 269, 327-345.

Weiger, W.A., & Bear, D.M. (1988). An approach to the neurology of aggression. Journal of Psychiatric Research, 22, 85-98.

Welte, J.W., & Abel, E.L. (1989). Homicide: Drinking by the victim. Journal of Studies on Alcohol, 50, 197-201.

White, H.R. (1990). The drug use delinquency connection in adolescence. In R. Weisheit (Ed.). Drugs, Crime and the Criminal Justice System. Cincinnati, Ohio: Anderson Pub. Co., Criminal Justice Division, pp. 215-256.

White, H.R., Brick, J., & Hansell, S. (1993). A longitudinal investigation of alcohol use and aggression in adolescence. Journal of Studies on Alcohol, Suppl. 11, 62-77.

Widom, C.S. (1989). The cycle of violence. Science, 244, 160-166.

Winslow, J.T., DeBold, J.G., & Miczek, K.A. (1987). Alcohol effects on the aggressive behaviour of squirrel monkeys and mice are modulated by testosterone. In B. Olivier, J. Mos, & P.F. Brain (Eds.), Ethopharmacology of Agonistic Behaviour in Animals and Humans. Boston: Martinus Nijoff, pp. 223-244.

Winslow, J.T., & Miczek, K.A. (1985). Social status as determinant of alcohol effects on aggressive behaviour in squirrel monkeys (Saimiri sciureus). Psychopharmacology, 85, 167-172.

Winslow, J.T., & Miczek, K.A. (1988). Androgen dependency of alcohol effects on aggressive behaviour: A seasonal rhythm in high-ranking squirrel monkeys. Psychopharmacology, 95, 92-98.

Wolfe, B.M., & Baron, R.A. (1971). Laboratory aggression related to aggression in naturalistic social situations: Effects of an aggressive model on the behaviour of college student and prisoner observers. Psychonomic Science, 24, 193-194.

Wolfgang, M.E., & Strohm, R.B. (1956). The relationships between alcohol and criminal homicide. Quarterly Journal of Studies on Alcohol, 17, 411-425.

Wright, P., Nobrega. J., Langevin, R., & Wortzman, G. (1990). Brain density and symmetry in pedophilic and sexually aggressive offenders. Annals of Sex Research, 3, 319-328.

Yarvis, R.M. (1972). Psychiatric pathology and social deviance in 25 incarcerated offenders. Archives of General Psychiatry, 26, 79-84.

Yeudall, L.T., Fedora, O., & Fromm, D. (1987). A

neuropsychological theory of persistent criminality: Implications for assessment and treatment. In R.W. Rieber (Ed.), Advances in forensic psychology and psychiatry, Vol. 2. Norwood, NJ: Ablex Publishing Corp., pp. 119-191.

Yeudall, L.T., & Fromm-Auch, D. (1979). Neuropsychological impairments in various psychopathological populations. In J. Gruzelier & P. Flor-Henry (Eds.), Hemisphere Asymmetric of Function and Psychopathology. New York: Elsevier/North Holland, pp. 5-13.

Zeichner, A., & Pihl, R.O. (1979). The effects of alcohol and behaviour contingencies on human aggression. Journal of Abnormal Psychology, 88, 153-160.

Zeichner, A., & Pihl, R.O. (1980). Effects of alcohol and instigator intent on human aggression. Journal of Studies on Alcohol, 41, 265-276.

Zeichner, A., Pihl, R.O., Niaura, R., & Zacchia, C. (1982). Attentional processes in alcohol-mediated aggression. Journal of Studies on Alcohol, 43, 714-724.

Zillman, D. (1979). Hostility and aggression. Hillsdale, NJ: Erlbaum.