Executive Cognitive Function, Alcohol Intoxication, and Aggressive Behaviour in Adult Men and Women

Peter Neil Spencer Hoaken

Department of Psychology

McGill University

Montreal, Quebec, Canada

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General Abstract

The present thesis and series of studies explores the underlying cognitive and neuropsychological processes that underlies propensity for aggressive response in adult men and women, both sober and intoxicated. Previous research demonstrated that poor executive functioning, either pre-existing (idiopathic) or induced by alcohol-intoxication, was associated with heightened aggressive responses. The first study demonstrates that although cognitively impaired when alcohol intoxicated, men with above average pre-alcohol Executive Cognitive Functioning (ECF) do not act aggressively if they are properly motivated to remain non-aggressive, suggesting some ability to use residual executive function. The second study directly compares the aggression-eliciting effects of alcohol in both men and women, an under-investigated issue. Results indicate that aggression levels in the women are not significantly less than those of men, and that alcohol-intoxication is not as predictive a factor in women as in men. The third study, a post-hoc analysis of the second, indicates that like for men, executive function level in women is highly related to propensity for aggressive response, in fact far more predictive than acute alcohol-intoxication. The fourth study was intended to investigate a possible behavioural explanation for the ECF-aggression relationship. Specifically, this study was designed to assess whether the aggression manifested by individuals with poor ECF was rapid or impulsive, i.e. due to a disinhibition process. Contrary to this popular

contention, this study demonstrates that when faced with complex, social interactions, low-ECF individuals act aggressively, but only after a somewhat slow period of apparent contemplation. These findings and others conducted by the author are discussed in a speculative model of the ECF-aggression relationship. Means by which to test this model are proposed, as are other theoretical implications of the work.

Résumé général

La présente thèse ainsi qu'une série d'études explorent les processus cognitifs et neuropsychologiques fondamentaux qui sont à la base de la tendance des réactions agressives chez les hommes et femmes adultes, soit sobres, soit intoxiqués. Des études précédentes ont démontré que le faible fonctionnement cognitif-exécutif, préexistant (idiopathique) ou suscité par l'intoxication à l'alcool, était associé à un accroissement des réactions agressives. La première étude démontre que, même lorsque les facultés cognitives sont diminuées par l'intoxication à l'alcool, les hommes, ayant un niveau de fonctionnement cognitif-exécutif (FCE) supérieur à la normale pré-intoxication, n'agissent pas de manière agressive s'ils sont soigneusement motivés à rester dans un état de non-agressivité, suggérant donc une habileté à utiliser les fonctions exécutives résiduelles. La deuxième étude compare directement l'agressivité occasionnée par l'alcool chez les hommes et les femmes, un élément sous-évalué. Les résultats indiquent que les niveaux d'agressivité chez les femmes ne sont pas significativement moindres que ceux des hommes, et l'intoxication à l'alcool n'est pas un élément prévisible aussi bien pour les femmes que pour les hommes. La troisième étude, une analyse post-hoc de la seconde, indique que, comme pour les hommes, le niveau de fonctionnement exécutif chez les femmes est étroitement relié à la tendance de réactions agressives, et ceci est considérablement plus prévisible qu'une intoxication sévère à l'alcool. La quatrième étude a été conçues pour

investiguer la possibilité d'une explication comportementale pour la relation FCE-agressivité. Plus spécifiquement, cette étude avait comme but d'évaluer si l'agressivité manifestée par les individus ayant un faible FCE était, soit rapide, ou impulsive, causée par exemple par un processus d'inhibition. Contrairement à la croyance populaire, cette étude démontre que, lorsque les individus possédant un bas niveau de FCE font face à des interactions sociales complexes, ils agissent avec agressivité, mais seulement après une lente période de contemplation plausible. Ces résultats ainsi que ceux d'autres recherches du même auteur sont étudiés à l'aide d'un modèle spéculatif de la relation FCE-agressivité. Les procédés selon lesquels ce modèle doit être vérifié sont proposés, comme d'autres implications théoriques de cet ouvrage.

Preface and Statement of Originality

This thesis presents research material which, although being original, can be regarded as logical progressions of previous work. Most specifically, this thesis developed out of work conducted by Drs. Jordan Peterson, Mark Lau and David Lemarquand, all of whom were recent graduates (or on the verge of graduating) when I started my program of study in 1994. Drs. Peterson had written on alcohol and aggression; Dr. Lau had focussed on neuropsychological elements of aggression, specifically executive function; and Dr. Lemarquand was predominantly interested in the relationship between serotonergic function and impulsive aggression. All of their work influenced the issues I chose to investigate in my dissertation.

However, for each of the studies in this dissertation, allow me to outline the specific contributions of myself, co-authors, and other assistants. In the first study (Hoaken, Assaad, & Pihl, 1998), the hypotheses and design of the study were wholly my own. The data were collected with the assistance of Jean-Marc Assaad, who also assisted in some of the analyses. Research assistants Jason Coupland and Pascale Malo also assisted in data collection. I completed the analyses and wrote the paper, with some assistance from Jean-Marc Assaad, under the supervision of Dr. R.O. Pihl.

For the second study (Hoaken & Pihl, 2000), the design of the study was my own. Data collection was assisted by research assistants

Jennifer Finestone, Wendy Strickler, Jamie Mayerovitch, Amber Rowland, and Stephanie Cassie. The writing was once again completed under the supervision of Dr. Pihl.

The third study (Hoaken, Strickler, & Pihl, under review) was a post-hoc analysis of the second, which was proposed in a joint meeting of the three authors. Statistical analyses were conducted in collaboration with Dr. Rhonda Amsel and Dr. James Ramsey. Writing was done by myself and Wendy Strickler under the supervision of Dr. Pihl.

The fourth study (Hoaken, Shaughnessy & Pihl, submitted) was designed wholly by myself, and analysed wholly by myself. The data was collected with the assistance of Carrie Marchionni, Valerie Shaughnessy and Jane Simpson. The writing was done by myself and Valerie Shaughnessy, yet again under the supervision of Dr. Pihl.

Marc Gross and Dave Khernagan provided assistance in all four studies in terms of mechanical and computer aspects of the data collection systems.

Drs. Amsel and Ramsey were consulted in matters of both design and analysis for the final three of the four aforementioned studies.

Acknowledgements

Even when I was ostensibly working on other things, I never gave up reading for leisure during my grad school career. A couple of years ago I slogged through the hundreds and hundreds of pages of Norman Mailer's *The Naked and the Dead.* I was astonished by the sheer magnitude of the minutiae that goes into organising a successful offensive military campaign. In retrospect, I am now amazed at how reminiscent that is of completing a dissertation! Many, many people were involved in this sometimes overwhelming endeavour, and deserve words of acknowledgement:

Dr. Robert O. Pihl supervised all projects contained in this dissertation. Bob deserves my thanks for accepting me into the program, and for giving me the opportunity to succeed. I greatly appreciated his supervisory approach, which allowed my independence and autonomy, but also allowed me to wander in and ask for guidance when I needed it. Bob's breadth of knowledge is astonishing and I always appreciated his novel contributions to matters, especially when I was writing. I thank him for financial support; I thank him for providing space and resources for my studies; I thank him for the many opportunities he gave me to teach. But most of all I thank him for the snide, incisive and often totally accurate insights he provided regarding academia, politics, culture & society at large. I didn't always agree, but it was always interesting...

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Marc Gross and Dave Khernaghan were responsible for all the physical elements of my studies. Marc did all the programming; Dave did all the building. Two terrific guys who were always good-natured about things when my stuff didn't work, and I freaked out.

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I must acknowledge both the Social Sciences and Humanities Research Council of Canada and The Harry Frank Guggenheim Foundation, both of which awarded me Doctoral Fellowships and by virtue of that kept me clothed and fed while I pursued my degree. Without this support I might have done something desperate. Like gone to law school.

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identifying and stimulating my creativity and curiosity. I love what I do, and, to a large extent, have you to thank for that.

Introduction

Definition and Measurement of Human Aggression

The definition and measurement of human aggression is a particularly tricky task. The foremost problem, as has been pointed out previously (Buss, 1961), is that in the English language the term "aggression" is used to refer to a large and varied set of actions. When somebody refers to another as being "aggressive" does that person mean the "aggressor" is physically hurtful, verbally confrontational, simply unfriendly, or particularly willing to stand up for his beliefs? The possible meanings are clearly multitudinous; therefore, we must start the description of the "aggression" literature in humans with definitional consideration.

Defining Aggression

Aggression is clearly a multidimensional construct that defies simple definition. Not surprisingly, then, efforts to define the construct have varied widely over the years. One of the earliest efforts was that of Buss (1961) who contended, simply, that any action that harmed another was aggressive. Despite the apparent simplicity of this definition, however, Buss suggested three dichotomies upon which aggression could be based: physical-verbal, active-passive, and direct-indirect. These three dichotomies produced eight discrete categories of aggression, such as physical-active-direct (punching someone), or verbal-active-indirect

(spreading malicious gossip about someone), into which you can categorise virtually all instances of aggressive behaviour.

The fundamental problem with Buss' definition, that aggression is any act that harms another, is that it does not contain the essential element of intent. Feshbach (1970) and Berkowitz (1974, 1981) both emphasised the necessity of intent, not just consequence, and incorporated it into their definitions. Still others claimed the assault had to produce physical or bodily harm, not simply psychological or emotional (Zillmann, 1979). Clearly, different theorists perceive different actions as aggressive or non-aggressive.

Despite the lack of consensus, Baron (1977; Baron & Richardson, 1994) proposes a definition which deals with several of the important aspects of aggression alluded to above. The authors propose the following definition:

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

This definition deals with the above issues in several ways. It perceives aggression as a behaviour, not an emotion, attitude, strategy, or motive. It contends with the important issue of intent by limiting the definition to acts in which the aggressor intends to harm the victim. This is an important element in a variety of ways. Not only does it rule out cases of accidental harm, but also it includes cases in which the aggressor has attempted to injure another but failed.

The definition is also good inasmuch as specifying that aggression involves harm to the victim does not necessarily imply physical damage. Instead, acts that lead to a wide range of aversive consequences, be they physical, emotional, financial, or otherwise, are all considered aggressive. Furthermore, the aggressive act need not be active, but can be purposefully passive; a mother depriving a child of love, or a supervisor failing to inform a worker of an important meeting, are both aggressive acts under the definition.

The definition suggests that aggressive actions must involve some living being. This distinction is an important one to consider, as actions to inanimate objects can still be either aggressive or non-aggressive. For instance, the definition excludes such actions as hitting a wall out of frustration, but includes vandalising the property of somebody you wish to indirectly harm.

Finally, the definition is restricted to behaviours directed at a recipient who is motivated to avoid such treatment. As such we can disregard several types of actions. Suicide and masochism, for example, fall outside the definition.

As good as the above definition is, it does have one failing, inasmuch as it fails to differentiate between hostile and instrumental aggression, a distinction which goes to the nature of intent (Feshbach, 1970, de Wit & Hartup, 1974). The former term is used to describe aggressive behaviour in which the primary objective is to cause the victim

to suffer. The latter term refers to assault on others primarily as a means of attaining other non-injurious goals, rather than out of a strong desire to produce suffering. This distinction has not always been unanimously agreed upon. Bandura (1973), for example, argued that since both forms of aggression are directed toward the attainment of specific goals, they could both be deemed instrumental, irrespective of the goal.

In response to this observation, several researchers have proposed alternative aggression dichotomies than hostile/instrumental. For example, Zillmann (1979) differentiated "annoyance-motivated" from "incentive-motivated" aggression. In this dichotomy, the former construct represented an action intended to reduce aversive conditions such as anger or mistreatment, whereas the latter was intended to satisfy extrinsic motivations. Dodge and Coie (1987), conversely, differentiated "proactive" from "reactive" aggression. In this distinction, the former construct involves behaviours that are intended to attain a particular profitable outcome. The latter simply involves retaliation against threat or harm.

Measuring Aggression

Given that aggression has been defined in many ways, it is not surprising that there are many methods by which aggression has been investigated. These we will roughly divide into two categories; asking

about aggression and observing aggression. These two large distinctions will be further divided.

Asking about Aggression

Some researchers suggest that the fundamental method of investigating aggressive behaviour is to simply ask about its occurrence. This can be accomplished in a variety of ways. Archival research, for example, refers to examining criminal records, arrest reports, police statistics, riot records, and so on in order to gain understanding of the causes of aggression and violent crime. Archival data has been used, for example, to demonstrate a relationship between atmospheric conditions (temperature, specifically) and reports of violent crime (Rotton & Frey, 1985).

Investigating aggression in the individual clearly requires alternative approaches. Social scientists use verbal reports to investigate aggression, either by asking questions directly of the individual in question, or by asking questions of those who know the individual. In the former case, this can be accomplished either by using self-report measures and/or personality scales such as the Buss-Durkee Hostility Inventory (BDHI; Buss & Durkee, 1957), the Cook-Medley Hostility Scale (Cook & Medley, 1954), the Buss-Perry Aggression Questionnaire (Buss & Perry, 1992), and the State-Trait Anger Scale (Spielberger, Jacobs, Russell & Crane, 1983). In the latter case, those who know and interact with the

individual in question rate his or her behaviour. This is a technique often used with children (Eron, Walder, Huesmann, & Lefowitz, 1978; Seguin, Pihl, Harden, Tremblay, & Boulerice, 1995). However, asking about aggression has a variety of limitations including but not limited to retrospective recall, biased responding, and poor reliability and construct validity of measurements. A better technique is often to observe the behaviour. Observational techniques involve direct recording of ongoing behaviour and by virtue of that avoid the pitfalls of the techniques discussed above. These sorts of techniques are typically of two sorts; either "in the field" or some sort of natural setting, or in the laboratory, when behaviour is observed under circumscribed conditions.

Observing Aggression – In Vivo

Observing aggression in vivo involves observing behaviour when the individuals whose behaviour is being observed are not aware of any investigative efforts. These techniques can take the form of simply watching behaviour as it naturally occurs, or may involve a researcher intrusion, and observation of the concomitant results. The first of these techniques is referred to as naturalistic observation; examples include observing a schoolyard to investigate patterns of childhood deviance (Patterson, 1977), or observing drinking patterns and aggression in a bar so as to discern a link between consumption of different alcoholic

beverage types and likelihood of aggression (Murdoch, Pihl, & Ross, 1988).

The latter of the two naturalistic observation techniques involve some interference of the experimenter. Pragmatically, because aggressive behaviour is relatively infrequent, the researcher may have to provide some form of provocation in order to elicit an observable response. This can be accomplished in a variety of ways. For example, one research group developed a series of methods by which they elicited personal confrontations, including bumping into people from behind, and pushing into line ahead of people waiting in lines (Harris, 1993, 1994; Harris & Samerotte, 1975). Response of the provoked individuals is recorded. Another classic example is the horn-honking scenario developed by Baron (1976), in which a research confederate does not proceed (drive) through a road intersection for some predetermined period of time (example, fifteen seconds) after the traffic light turns green. The behaviour of the individuals behind the confederate (horn honking duration, latency, and frequency) is observed and recorded. Although there are obvious advantages to these techniques, there are just as obvious problems. Foremost of these problems are that of the necessity of operationalising observations (that is, the need for a complex and detailed means of coding), and the corresponding problem of low reliability of observations. Also problematic is the inability to attain informed consent, and the possibility of harm to participants and/or confederates.

Observing Aggression – In Vitro

In light of the above problems, many researchers attempt to observe aggression in the laboratory. In an attempt to gain insight into aggressive behaviour, a variety of experimental approaches have been developed. One approach has been to examine rates of non-aggressive behaviour, which were, in theory, thought to reflect underlying aggression. For example, researchers have interpreted the appreciation of aggressive humour (Hetherington & Wray, 1964), time participants spent looking at aggressive pictures (George & Martlett, 1986; George, Dermen, & Nochajski, 1989), and the intensity and frequency of power fantasies (McClelland, Davis, Kalin, & Wanner, 1972) as indirect measures of aggression. However, these approaches have been justifiably criticised for not attempting to validate the "measure" of aggression utilised (Gustafson, 1993).

More methodologically sound approaches to the laboratory-based investigations of aggression include three types of active aggression measures: measures of verbal-indirect and direct aggression, and measures of physical-direct aggression.

Measures of Verbal Aggression

Much of the early laboratory work on aggression used methods focussed on verbal rather than physical aggression. In these studies the practice was to frustrate, irritate or otherwise invoke the ire of the

participant, and then allow him or her to retaliate against the target of their ire through some sort of spoken or written assessment. While less frequent, these types of techniques are still conducted, and are generally conducted in one of two ways. In order to look at indirect verbal aggression, in which the subject/recipient of the aggression is absent, aggression is typically measured by means of a questionnaire (typically the self-report questionnaires mentioned above; for review see Edmunds & Kendrick, 1980). However, the use of questionnaires has been criticised due of the fact that the correlations between these indirect measures, as well as correlation between these indirect measures and measures thought to report associated constructs, have generally been very low (Buss, 1961; Taylor, 1967). In order to study direct verbal aggression, on the other hand, the "victim" must be present. Usually this is achieved by recording, coding and scoring the verbal comments of the participant during some sort of social interaction. This approach is clearly suited to the naturalistic environment (Murdoch, Pihl, & Ross, 1988), but can also be performed in the laboratory. For example, a participant might be allowed to judge or verbally interact with a confederate who had previously expressed provocative and confrontational views (such as "I think my religion is the best and I don't think others are worth a damn"; Wheeler & Caggiula, 1966).

Measures of Physical Aggression

Measures of verbal aggression are clearly useful. But just as clearly, if we are to understand violent crime we must have some manner of assessing physical aggression. The most commonly used means of assessing physical aggression in the laboratory involve a direct, physical assault against another human being. Although several techniques have been developed, which vary considerably, they are all alike in that they all rely on deception; the participant is led to believe that he or she can physically harm another person, when in fact that is not the case. The most commonly utilised means of assessing physical aggression in the laboratory are detailed below.

The Buss Paradigm

The first technique for directly investigating physical aggression was devised by Buss (1961). In what has become known as the Buss Teacher-Learner paradigm, the participant was told that the study he was to participate in concerned the effects of punishment on learning. The participant was introduced to a confederate, and the two were "randomly assigned" roles; the participant always received the teacher role, and the confederate the learner role. The teacher's task was to present some sort of material to the learner, who was to develop some sort of mastery over it. If the learner made a correct response, he or she was to be rewarded by the teacher; however, when an error was made, the teacher punishes the

learner via the administration of electric shocks. The intensity and duration of these shocks were at the discretion of the teacher. The general use of this paradigm has been criticised because of artificiality, demand characteristics, and because aggression is confounded with altruistic or helping behaviour (Tedeschi & Quigley, 1996, 2000). For these reasons, the Buss paradigm is for the most part no longer in common usage. The two laboratory paradigms most used in contemporary studies are the "Point Subtraction Aggression Paradigm" (Cherek, 1981) and the "Competitive Reaction Time Task" (Taylor, 1967).

Point Subtraction Aggression Paradigm

The Point Subtraction Aggression Paradigm (PSAP) has its origins in experimental behavioural pharmacology research (Kelly & Cherek, 1993), in that in its development individual participants were exposed to experimental contingencies across several sessions until stable patterns of aggression emerged. At this point, a drug (usually alcohol) was administered to examine the differential effects.

The PSAP requires participants to sit in front of a response panel, which typically displays two buttons. The participant's task is to accumulate as many points as possible, as these points will later be converted into monetary reward. There are two ways in which the participant can gain points: pressing the first button approximately one hundred times earns a point; conversely, pressing the second button

approximately ten times deducts a point from a fictitious "opponent". The "opponent" at a predetermined basis deducts points from the participant, so as to provide a source of provocation. The dependent measure of aggression is the number of times the button, which presumably subtracts points from the opponent, is pressed. The developers of this paradigm assert that the availability of non-aggressive responses is an advantage of this paradigm over others, and further that the PSAP is well-suited to repeated measures designs.

There is considerable data to demonstrate validity of the PSAP. For example, violent parolees have been demonstrated to be more aggressive on the PSAP than non-violent parolees (Cherek, Schnapp, Moeller, & Dougherty, 1996; Cherek, Moeller, Schnapp, & Dougherty, 1997). As well, adolescents rated as aggressive by teacher (Pelham, Millich, Cummings, Murphey, Schaughency, & Greiner, 1991) and psychologist (Murphey, Pelham, & Lang, 1992) ratings have been shown to act aggressively on the PSAP.

However, this paradigm has limitations. First of all, some might question its utility for investigating physical aggression due to its lack of a physical provocation (or retaliation option). Second, testing sessions tend to be long, and factors such as fatigue, boredom and lapses in concentration may affect results. Third, and perhaps most important, this paradigm can be criticised inasmuch as it appears to reward participants for behaviour operationalised as "aggressive". Fourth, extended time is

necessary to establish performance baselines. Fifth, frequency and intensity of aggression are confounded. For reasons such as these, many researchers continue to prefer the other predominant aggression paradigm, the Taylor (1967) reaction-time paradigm.

Taylor Competitive Reaction-Time Task

The Taylor (1967) task is popular because of the above reasons, as well as the fact that it resolves many of the problems inherent to the Buss paradigm. In this paradigm, the participant is told that he will be competing against another participant on a reaction-time task. Again, in reality, the reaction time "opponent" is fictitious. Each reaction-time trial occurs as a series of steps. At the beginning of each trial, the participant chooses a level of shock he will administer to the opponent if he wins the reaction-time trial. Then there is a signal of some kind that the assessment of reaction time is to begin. For instance, a light indicates that the participant should press down on a button; that light turning off indicates that the subject should release the button as fast as possible, so as to determine reaction time. At this point, the participant is informed of the shock level selected by the opponent. Then, depending on whether the participant "won" or "lost", he gives or receives the indicated shock.

This behaviour of the participant is quantified in a variety of ways, and different researchers report different aspects of the paradigm as their dependant measures. Virtually all researchers report "shock intensity" as

an important measure; this is simply the intensity of the shocks chosen by the participant to be delivered to the opponent. The initial shock selected by the participant, a decision made before anything regarding the intention of the opponent is known, is considered a measure of unprovoked aggression; conversely, shock choices during subsequent trials, when the participant is provoked, is thought to indicate retaliative aggression (Hammock & Richardson, 1992). Shock duration is the length of time the subject delivers the shock he has selected to the opponent; whereas shock intensity is clearly a measure of direct aggression, shock duration has been interpreted as a measure of indirect aggression (Rogers, 1983; Zeichner, Giancola & Allen 1995), although there is no uniform agreement regarding this distinction.

This reaction-time paradigm has a number of important advantages over the Buss paradigm. First, the participant is physically provoked, as opposed to the non-physical provocation inherent to the Buss paradigm; the participant is provoked, and can retaliate, thus providing a more realistic approximation of an actual aggressive interaction. Second, the behaviour of the "opponent" can be systematically varied so as to determine the influence of varied interactive "styles". For example, most researchers divide the trials into "low-" and "high-provocation" blocks that represent the shock settings "chosen" by the opponent. Alternatively, the opponent may appear consistently provocative (high intensity shocks) or deferential (low intensity shocks). Third, this paradigm allows for the

ready investigation of the influence of a wide variety of third-variables, such as physical environment, presence of an audience (Richardson, Bernstein & Taylor, 1979), and perhaps most commonly, drugs (Taylor & Chermack, 1993).

The Taylor reaction-time aggression paradigm, and minor alterations thereof, is perhaps the most popularly utilised laboratory measures of aggression. However, the paradigm has been the target of recent criticism, along with several other aggression paradigms (Tedeschi & Quigley, 1996). These authors argued that all aggression paradigms are essentially invalid. A response to this position from Giancola and Chermack (1998), while agreeing that many aggression paradigms have significant theoretical shortcomings, did not agree with the specific criticisms of the Taylor paradigm. They argued that the Taylor paradigm has been shown several times to have good construct validity (Giancola & Zeichner, 1995c; Bernstein, Richardson, & Hammock, 1987). The Taylor paradigm also has demonstrated convergent validity, in that it correlates significantly and positively with other measures of aggression, such as the Buss-Durkee Hostility Inventory. Furthermore, it has established discriminant validity, in that it does not correlate with other measures thought to be theoretically unrelated to aggression, such as competition, suspicion, or guilt (Gaebelein & Taylor, 1971). It also has been demonstrated to have group discrimination ability. Individuals thought to be aggressive by nature appear so on this paradigm, while non-aggressive individuals do not (Giancola & Chermack, 1998), and it reliably

differentiates between groups theoretically expected to differ in aggressive potential including psychopaths (Dengerink, 1971), and prison inmates (Wolfe & Baron, 1971). Lastly, the paradigm appears sensitive to environmental factors thought likely to influence aggression (Borden & Taylor, 1973; Hendrick and Taylor, 1971; Pisano & Taylor, 1971). Clearly, the paradigm offers researchers a powerful tool with which to investigate physical aggression

Naturally, a paradigm of this sort raises relevant questions of ethicality. First of all, the use of deception is a concern. The apparent delivery of electric shocks to the opponent is also possibly discomforting to the participant. And perhaps most importantly, the paradigm involves receipt of electric shocks by the participant himself. The argument could be made that these concerns outweigh the benefits offered by the paradigm. However, the results of one study suggest these concerns are not warranted (Pihl, Zacchia & Zeichner, 1981). These researchers interviewed 78 participants from experiments using the reaction-time aggression paradigm. Of these participants, only 14 reported that some aspect of the experiment bothered them, and of those, seven were concerned with either boredom or aspects of the study not related to the aggression paradigm (e.g. consumption of beverages). Only four of the participants, or approximately 5%, were bothered by the administration of electric shocks. Only three participants, or approximately 4%, were

distressed by the deception. Therefore, it appears likely that the aversive consequences to the participant are minimal.

There are several means by which researchers can assess aggression in the laboratory. One similarity that these paradigms have is that irrespective of measure, alcohol has been demonstrated to increase aggressive responding. The role of alcohol in aggression will now be considered.

Alcohol and Aggression

Conventional wisdom tells us that individuals who are under the influence of alcohol will act aggressively. This is a situation in which conventional wisdom is likely correct - the vast majority of laboratory studies have demonstrated that participants who consume even a moderate dose of alcohol act more aggressively than those not given alcohol. Several reviews of the experimental literature (Pihl, 1983; Taylor & Leonard, 1983), as well as a number of meta-analyses (Steele & Southwick, 1985; Hull & Bond, 1986; Bushman & Cooper, 1990; Bushman, 1993; Ito, Miller & Pollock, 1996) all conclude that there is a causal role of alcohol in increasing aggression. Epidemiological data demonstrate that more than half of perpetrators of violent crimes have recently consumed alcohol (Collins & Messerschmidt, 1993; Miczek, Weerts, & DeBold, 1993; Roizen, 1993). Alcohol consumption has also been associated with sexual aggression (Parks & Zetes-Zanatta, 1999;

Testa & Parks, 1996; Seto & Barbaree, 1995), family and marital violence (Leonard & Senchack, 1996; Leonard & Jacob, 1988), and suicide (Brent, Perper, & Allman, 1987).

That this relationship exists appears incontrovertible. What are less consistently agreed-upon are the reasons for the relationship, and why only some individuals respond aggressively subsequent to alcohol consumption. Because of the enormous individual variability in terms of the aggression-eliciting properties of alcohol, and because of the inconsistencies in the literature, most researchers now view the relationship between alcohol consumption and aggression as particularly complex and likely the product of several interactive factors. Pharmacological, contextual, situational and, perhaps most importantly, individual factors have been identified which moderate the alcohol aggression relationship in humans.

Alcohol & Aggression – Direct Explanations

The first two explanations of the alcohol-aggression model discussed below are categorically different from each other in that one relies wholly on a pharmacological effect and the other exclusively on beliefs, but they are similar inasmuch as they are both considered "direct cause" explanations. That is, neither relies on third-factors in their explanation of the alcohol-aggression relationship.

Physiological Disinhibition Models

The disinhibition model of the alcohol-aggression relationship is one of the earliest theories of the relationship, but ultimately one of the most simplistic. Essentially, the model suggests that alcohol has a deleterious effect on a brain area that normally acts to sustain inhibitory control over behaviour (Pernanen, 1976; Graham, 1980). Although intuitive, this model taken alone ultimately is untenable simply because not all people become aggressive even when acutely alcohol-intoxicated, and even when profoundly provoked (Jeavons & Taylor, 1985; Hoaken, Assaad, & Pihl, 1998). Moreover, this model fails to accounts for the voluminous literature that clearly demonstrates that there are several factors that can either heighten or diminish alcohol-related aggression. For example, manipulating provocation (Taylor, Schmutte, Leonard, & Cranston, 1979), applying social pressure (Taylor & Sears), and distracting the participant from or focussing the participant to his aggressive responses (Zeichner, Pihl, Niaura, & Zacchia, 1982) have all been shown to alter level of intoxicated behavior. Nor is this model able to account for the demonstrable differences in alcohol-induced aggression across different drinking environments (Kalin, 1972), not to mention different cultures (Heath, 1983). Although disinhibition models are still popular in terms of discussing that alcohol-aggression relationship (for example Fillmore & Vogel-Sprott, 1999, 2000), these explanations tend to

include mediating third factors such as emotion, frustration, cognition, and motivation.

Alcohol Expectancies

Some researchers have suggested that alcohol leads to heightened aggression not because of any pharmacological effect, but instead simply because people believe that it will (Lang, Goeckner, Adesso, & Marlatt, 1975; MacAndrew & Edgerton, 1969; Marlatt & Rohsenhow, 1980). This is typically referred to as an alcohol expectancy. Expectancy has been defined as a dynamic model of what is going to happen in the future as the consequence of a set of behaviours (Luria, 1976). The general framework of the expectancy model suggests that expectancies are complex products of memory and knowledge accrued though development and/or socialisation. Recent research has focussed on developmental factors, suggesting expectancies arise as a developmental product of parallel processing memory networks (Rather, Goldman, Roehrich & Brannick, 1992; Dunn & Goldman, 1996, 1998). These culture-specific expectancies modify event appraisal, govern emotional regulation, and by virtue of this determine behavioural repertoires (Mesquita & Frijda, 1992).

There is no question that people expect alcohol to lead to aggression; this belief has been demonstrated consistently in adults (Kidder & Cohn, 1979; Southwick, Steele, Marlatt, & Lindell, 1981) and in children as young as five years old (Pelham & Lang, 1993). More

controversially, recent research has suggested that children's alcohol-expectancies vary somewhat by ethnicity, with black children more likely to expect an alcohol-aggression relationship than white children (Corvo, 2000), a result which would support the premise of culture-specificity of expectancies alluded to above. Alcohol expectancies also appear to vary depending on beverage type (Lang, Kaas & Barnes, 1983) and alcohol dose (Southwick, Steele, Marlatt & Lindell, 1981).

The relative influence of alcohol expectancies versus pharmacological effects has been studied using the balanced placebo design (Chermack & Taylor, 1995). Although psychological expectancy effects regarding alcohol have been demonstrated in some experimental studies to be an important determinant of propensity for violence (Lang, et al., 1975; Lang, 1993), many researchers have failed to demonstrate this relationship. In fact, there are now four large meta-analytic studies that agree that expectancies play an insignificant role in affecting aggression (Bushman, 1993; Bushman & Cooper, 1990; Hull & Bond, 1986; Steele & Southwick, 1985). Specifically, in the Bushman & Cooper (1990) meta-analysis, the effect size of placebo versus non-alcohol was only .10, far less than the considerable alcohol versus non-alcohol effect size of .61. Moreover, Giancola (1997) recently demonstrated that while alcohol was associated with increased aggression while tested on the ascending limb of the blood-alcohol concentration (BAC) curve, there was no corresponding

relationship at the same BAC while tested on the descending limb. Again, this result would argue against an expectancy effect.

However, while it is true that evidence for the alcohol-expectancy effect is not consistent, there may be one mitigating factor that is under-appreciated. The extent to which people believe alcohol elicits aggression appears to have large individual differences. The few studies which have taken into account these individual differences in expectancy have demonstrated moderate support for the theory that alcohol expectancy interacts with alcohol consumption to determine intensity of aggressive response (Chermack & Taylor, 1995; Bjork & Dougherty, 1998).

Alcohol & Aggression – Indirect Explanations

Most current theorising on the alcohol-aggression relationship is in the form of indirect-cause models. That is, most researchers currently hold that alcohol elicits aggression as a result of interfering with some other process, be that psychological, physiological, cognitive, or perceptual. The following is a brief review of several of these interactive third-factor explanations.

Alcohol-Aggression Relationship as a Function of Reward or Psychomotor Stimulant Effects

Another theory regarding the manner in which alcohol elicits aggression relates to the rewarding properties of alcohol (Pihl & Peterson,

1995; Pihl, Peterson & Lau, 1993). It is a given that alcohol has rewarding properties, especially soon after consumption, while alcohol is still being absorbed (the ascending limb of the blood-alcohol curve). These rewarding properties are simplistically analogous to those of stimulants such as cocaine and amphetamine, and appear pharmacologically mediated by the dopaminergic system (Gessa, Muntone, Collu, Vargiu, & Mereu, 1985). Also in common with these other drugs, low and moderate doses of alcohol produce stimulation, manifested in a variety of ways, including increased heart rate (Rush, Higgins, Hughes, & Bickel, 1993; Conrod, Peterson, Pihl, & Mankowski, 1997). These psychomotor stimulant effects may increase the likelihood of aggression in that there is an increase in sensation seeking and impulsivity, as well as increases in novelty seeking and-or approach behaviours. These alterations may lead to increases in confrontational and provocative behaviours on the part of the inebriate, which may lead to either proactive or reactive aggression (Pihl and Peterson, 1995).

Alcohol-Aggression Relationship as a Function of Interrupted Threat-Detection

That alcohol consumption can alleviate subjective feelings of stress is well known. This effect, not unlike that of benzodiazepines or barbiturates, appears related to alcohol's effects on gamma aminobutric acid (GABA), the main inhibitory neurotransmitter. Specifically, theorists

have suggested that alcohol increases firing of GABAergic neurons, increasing their inhibitory influence, particularly in prefrontal and limbic structures which mediate perception of threat (Gray 1981, 1987).

The capacity of alcohol to produce stress-response dampening has been implicated in the alcohol-aggression relationship by several researchers (Sayette, 1993; Pihl & Peterson, 1995; Ito, Miller & Pollock, 1996). According to this hypothesis, if alcohol is consumed prior to a provocative or anxiety-eliciting event, its anxiolytic properties will reduce the likelihood of an appropriate appraisal of that event. Therefore, this is essentially a disinhibition model; fear is diminished, and as a result the inhibitory effects that fear usually exerts on aggressive behaviour is itself inhibited. Support for this hypothesis is strong, but is either indirect or derives from the animal literature.

In terms of the literature with humans, several studies have demonstrated the ability of alcohol to disrupt threat-detection (see Sayette, 1993b, for a review), and, as previously noted, a voluminous literature demonstrates the alcohol-aggression relationship. Taylor and colleagues have demonstrated that drugs other than alcohol which are thought to have "anxiolytic" effects also heighten aggression (Gantner & Taylor, 1988; Chermack & Taylor, 1993). Experimental work assessing the differential psychophysiological concomitants of provocative and/or aggressive behaviour in intoxicated versus sober participants is somewhat meagre. One study (Bond & Lader, 1987) suggests that alcohol heightens

aggression on a modified version of the Taylor aggression paradigm, while at the same time dampening cardiac arousal. Another unpublished study (conducted by myself and colleagues, please see Appendix B) also supports this hypothesis; alcohol leads to heightened aggression, but is associated with muted heart rate and blood pressure increase in response to provocation.

The animal literature also supports this model. A large literature demonstrates that alcohol relatively infrequently heightens aggression in laboratory animals, especially rodents (Berry & Smooth, 1986). However, in the specific circumstances in which these animals do become aggressive in response to alcohol (Blanchard, Hori, Blanchard, & Hail, 1987), they demonstrate an "anxiolytic profile" characterised by decreased proxemic avoidance, decreased risk assessment, and reduced suppression of non-defensive behaviours such as eating and drinking (Blanchard, Blanchard, & Rodgers, 1991). Moreover, alcohol administration greatly increases rodent locomotion towards a threat (for example, a compartment containing a cat; see Blanchard, 1993, for a review).

Therefore, the threat-detection interruption model is a powerful one in terms of its explanatory power, but direct human evidence is still relatively scarce.

Alcohol-Aggression Relationship as a Function of Alterations of the Pain System

Pihl, Peterson & Lau (1993) have suggested that alcohol's effects on pain sensitivity may also be consequential in the elicitation of aggression. The theorised relationship between alcohol, the pain system and aggression, is a complex one. On one hand, it is widely accepted that alcohol has sedative and analgesic effects; in fact, at one time it was used as a surgical anaesthetic (Mullin & Lockhart, 1934; Wolff, Hardy & Goodell, 1942). In light of this, it could be surmised that alcohol's relationship with heightened aggression is simply that the analgesic properties block the normally punishing painful aspects of an aggressive altercation.

However, this theory is likely incorrect. Alcohol's effects on pain sensitivity are largely dose and individual dependant, and, moreover, differ greatly during absorption and elimination of alcohol. At moderate dosages, and on the rising limb of the blood-alcohol concentration curve, alcohol has been shown to actually increase ratings of pain sensitivity in humans (Gustafson 1985b). This is consistent with animal work which has demonstrated lowered pain thresholds in alcohol intoxicated rats (Gray, 1982). Thus, the relationship between alcohol, pain and aggression may be paradoxical in nature. Alcohol may contribute to aggression simply by increasing reactivity to pain, and correspondingly the significance of provocation. Because defensive aggression is an unconditioned response, alcohol's heightening of pain sensitivity may therefore increase likelihood of defensive aggression (Pihl et al., 1993). Moreover, one study

demonstrated that subjective experience of pain heightens self-reported irritation, annoyance and anger, and objectively increases aggression towards another, even if the source of pain cannot be attributed to this other person (Berkowitz & Thome, 1987).

(Please see Appendix D for a more comprehensive discussion of alcohol's effects on the reward, threat, and pain systems, and the possible relevance to the alcohol-aggression relationship.)

Alcohol-Aggression Relationship as a Function of Poor Social-Information Processing

Another hypothesis suggests that alcohol interferes with elements of social information processing. The most comprehensive discussion of social information processing and its relationship to the moderation of behaviour comes from Dodge (1986), who used it to explain adjustment and behaviour in children. This model suggests that four interrelated tasks must be accomplished for proper interpretation of a social event: encoding social cues in the environment; representing and interpreting that information mentally; generating behavioural responses; and selecting the most appropriate response on the basis of possible consequences. The social-information processing model of the alcohol-aggression relationship postulates an interference at one or more of these tasks. This theory suggests that alcohol somehow interferes with the ability to correctly and

adaptively interpret social cues, and moreover, to govern behaviour on the basis of these interpretations.

Sayette and colleagues conducted a study which was intended to directly test the hypothesis that alcohol impairs social information processing, and that this impairment elicits aggressive responses (Sayette, Wilson, & Elias, 1993). The results of the study suggest that although intoxicated men did not appear to have problems encoding and-or interpreting social cues, they were less able to generate competent (non-aggressive) solutions, and were less likely than controls to select adaptive (non-aggressive) solutions if provided. As only the two latter elements of social information processing appear to have been altered by alcohol, this study does not provide particularly compelling support for the social information-processing hypothesis.

Alcohol-Aggression Relationship as a Function of Diminished Self-Awareness

Hull (1981) has posed a hypothesis not unlike the one detailed above. Instead of a problem interpreting the actions of others, Hulls model suggests that alcohol leads to aggression through a reduction in self-awareness. Alcohol interferes with self-relevant social and environmental information in such a way that the individual is made detached from the self-referential consequences of behaviour. This model was predicated on the fact that individuals who are low in self-awareness

have been shown to be more likely to be aggressive (Scheier, Fenigstein, & Buss, 1974), as well as be more likely to cheat (Diener & Wallbom, 1976) and steal (Beamon, Klentz, Diener & Svanum, 1979). One study (Bailey, Leonard, Cranston, & Taylor, 1983) did provide some support for this theory; participants were tested on an aggression paradigm either in the presence of a mirror or a video camera (i.e. high self-awareness) or alone (i.e. low self-awareness). Individuals in the high self-awareness condition were less aggressive than those in the low self-awareness condition. However, it may well be that the presence of the mirror or camera did not so much raise self-awareness as it did a suspicion that someone was watching, a factor repeatedly demonstrated to modify aggressive responses (Dengerink, 1971; Dorsky & Taylor, 1972; Taylor & Sears, 1988; Taylor & Gammon, 1976).

Alcohol-Aggression Relationship as a Function of Faulty Attentional Allocation

Related to perhaps all of the above is the attention-allocation model of Steele and colleagues (Steele & Josephs, 1990; Steele & Southwick, 1985). This theory suggests that alcohol heightens aggression through an interruption of information processing, and a reduction of attentional resources to only the most salient of environmental cues. As such, alcohol reduces the probability of incorporating all relevant aspects of a situation into the production of a response option. That is, the

intoxicated individual is more likely to act aggressively as a response to salient cues of provocation, without appropriate recognition of important yet less conspicuous cues indicating the benefits of behavioural inhibition.

This model is similar to that of Taylor and Leonard (1983), which presents behaviour as the consequence of an interaction between alcohol and the relative strength of environmental cues. For example, in the absence of instigative cues, a sober individual always acts pro-socially. In the same sober individual, when instigative cues exist in the absence of inhibitory cues, aggressive results. On the other hand, in situations where instigative and inhibitory cues co-exist, alcohol becomes an important factor, reducing the likelihood of attending to, understanding, and/or utilising relevant cues for inhibition.

Alcohol & Aggression – Pharmacokinetics and Other Related Issues

Another relevant issue is the complexity of the stimulant-sedative effects of alcohol, and more specifically their relationship to the blood-alcohol concentration (BAC) curve. The majority of laboratory studies of the effects of alcohol administer a dose of alcohol and then at some later point compel the participant to perform in some fashion. The important issue here is that alcohol has categorically different pharmacological and behavioural effects depending on the temporal proximity to consumption. On the ascending limb of the BAC curve, alcohol has stimulating, activating, and euphoric effects, followed by sedative, depressing and

dysphoric effects on the descending limb (Jones & Jones, 1976; Martin, Earlywine, Musty, Perrine, & Swift, 1993). This pattern is typically referred to as the biphasic effects of alcohol. Beyond factors such as arousal and mood, such capacities as memory (Jones, 1973), attention (Hurst & Bagley, 1972), psychomotor performance (Savoie, Emory & Thomas, 1988), and reaction time have been demonstrated to be more affected on the ascending limb than a corresponding BAC on the descending limb. A previously mentioned study (Giancola & Zeichner, 1997) demonstrated that aggression is much more pronounced at 0.08% on the ascending limb of the BAC curve than it is at 0.08% on the descending limb.

Furthermore, there are issues of dose that should be alluded to; the effect of manipulating dose of alcohol on aggressive response has been examined in several studies. Unfortunately, there is little agreement among studies that vary dose about what constitutes a "low", a "medium", or a "high" dose. For instance, while one study reports administering "0.25, 1.00 and 1.75 oz per 40 lbs. of body weight" (Taylor, Gammon & Capasso, 1976), another reports administering "0.2, 0.67 and 1.32 ml per kg of body weight" (Peterson, et al., 1990). Generally, doses that produce blood-alcohol concentrations in the 0.08 to 0.10 % range are considered high doses. Doses that produce blood-alcohol concentrations in the 0.04 to 0.06 % range are moderate doses; doses beneath this are low doses.

Typically, studies manipulating dose tend to show that participants in high dose conditions are more aggressive than those in a low-dose group. In fact, a series of studies by Taylor and colleagues concluded that aggression manifested on the competitive reaction-time paradigm (Taylor, 1967) was "a positive linear function of the dosage of alcohol consumed" (Taylor & Chermack, 1993; Taylor & Gammon, 1975; Taylor, et al., 1976). Not all studies have replicated this effect, but those which have not are susceptible to methodological critique due to usage of the Buss aggression paradigm (critiqued previously; Bennett, Buss, & Carpenter, 1969; Gustafson, 1984), or for low statistical power (Cherek, Steinberg, & Vines, 1984; Cherek, Steinberg, & Manno, 1985). (Methodologically, there are obvious problems with this type of research, beyond the fact that different researchers administer different doses as "low" versus "high". Foremost, of course, is the issue of tolerance; for the established drinker a 1.0 ml/kg body weight "high" dose may not be particularly intoxicating; the resulting 0.04% blood-alcohol concentration from a "moderate" dose may not produce even perceptible subjective effect or behavioural consequence. Conversely, for the naïve drinker, a 0.67 ml/kg dose may produce impairment to the extent that any sort of testing is not possible; Hiltunen, 1997; Fillmore & Vogel-Sprott, 1997; Zack & Vogel-Sprott, 1995.)

Beyond the issues of pharmacokinetics, there is also evidence to suggest that the beverage type has an influence on propensity for

aggression. Several studies have demonstrated that the consumption of distilled spirits produces greater increases in verbal (Boyatzis, 1974; Takala, Pihkanen, Markkanen, 1957) and physical (Murdoch & Pihl, 1988; Murdoch, Pihl, & Ross, 1988; Pihl, Smith & Farrall, 1984) aggression than does consumption of beer, even when volume is controlled for. Other studies have suggested that vodka elicits greater aggression than bourbon (Taylor & Gammon, 1975; Taylor et al., 1976), and that wine is the beverage least likely to produce aggression, although this result may have been due to low blood alcohol concentrations (Gustafson, 1990).

The reasons for these differences are not well understood, but theories based on both expectancy and pharmacological factors have been presented. In terms of the former theory, it has been suggested that those who drink distilled beverages expect that these beverages will make them more aggressive; conversely, those who drink beverages such as wine do not expect any aggression eliciting properties (Lindeman & Lang, 1986; Pihl et al., 1984). In terms of the latter theory, some researchers (Greenberg, 1970; Katkin, Hayes, Teger, & Prutt, 1970) have alluded to congener content; "congeners" are chemical compounds other than ethanol which are commonly found in alcoholic beverages. The theory posits that congener concentration influences alcohol absorption rates (Taylor & Leonard, 1983), such that lower congener beverages such as distilled spirits (Greizerstein, 1981) will be absorbed more quickly, leading to

higher blood-alcohol concentrations at testing time, and therefore greater aggression. This theory, however, is problematic, because although it potentially explains the difference between distilled beverages and beer, it fails to do so for the differences observed between wine and beer; that is, although wine has fewer congeners than beer (Greizerstein, 1981), it is beer which has been shown to produce greater aggression (Gustafson, 1988a, 1988b; Murdoch & Pihl, 1988).

The Alcohol-Aggression Relationship – A Meta-Cognitive Phenomenon?

It can be argued that the majority of the aforementioned hypotheses that purport to explain the alcohol-aggression relationship are inherently cognitive in nature. In each of them there is an interference with the appraisal of something; what differs among them is what it is that is being appraised, be it the self, behaviour of others, cues of fear, pain, reward, or something other. As such, it appears reasonable to suggest that the aggression eliciting effects of alcohol are putatively meta-cognitive; i.e. involving the interference of perhaps many elements of cognition (Giancola, 2000a).

Until recently, the extent to which alcohol interfered with various aspects of cognitive functioning was surprisingly under-investigated; in fact, the majority of the studies conducted on the relationship between alcohol and cognition have been so in the last fifteen years. In general, the accumulated literature suggests that acute alcohol intoxication impairs a

variety of aspects of cognition, including episodic memory (Tiplady et al., 1999), verbal and spatial learning (Mungas, Ehlers, & Wall, 1994), and visuospatial attention (Post, Lott, Maddock, & Beede, 1996). However, the literature also suggests that alcohol's most pronounced effects appear to be on cognitive abilities associated with prefrontal cortex. In an early study, alcohol significantly impaired tests associated with prefrontal cortex, but had a less pronounced effect on tests associated with temporal cortex, and did not appear to impair performance on standard intelligence tests (Peterson, Rothfleisch, Zelazo, & Pihl, 1990). Subsequently, a variety of studies have been conducted which demonstrate alcohol's interference with cognitive capacities thought to be mediated by prefrontal cortex, including attention, abstract reasoning, abstraction, and working memory (Lyvers & Maltzman, 1991; Arbuckle, Chaikelson, & Gold, 1994; Sayette, 1994). Several of these studies have repeated the suggestion of Peterson and colleagues (199o) that alcohol preferentially affects cognitive abilities thought to be pre-frontally mediated. This suggestion appears to be congruous with recent neuroimaging studies which suggest acute alcohol intoxication reduces glucose metabolism predominantly in the prefrontal cortex (de Wit, Metz, Wagner, & Cooper, 1990; Volkow et al., 1990; Volkow, Wang & Doria, 1995).

There is considerable 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

(either idiopathic or alcohol-induced) can produce increased likelihood of aggressive behaviour.

Neuropsychology and Aggression

The notion that aggression can be related to certain brain structures is certainly not a new one. Nor is the notion that the structures in question may well be specific to, or at least related to, the frontal lobes. Many researchers have suggested a role for frontal dysfunction in aggressive, violent or antisocial behaviour (e.g., Gorenstein, 1982; Yeudall, Fedora & Fromm, 1987; Lueger& Gill, 1990; Raine, 1993; Moffitt, Lynam & Silva, 1994; Kandell & Freed, 1989). Most research has attempted to associate elements of maladaptive social behaviour to specific brain dysfunction. These sorts of unsuitable social behaviours include the failure to inhibit inappropriate responses, low frustration tolerance, poor attention span, deficits in planning, irritability, and deficits in behavioural organisation (Beaumont, 1983; Cummings, 1995; Mega & Cummings, 1994; Stuss & Benson, 1984).

Patients with frontal lobe lesions often manifest a broad and diverse pattern of behavioural and social deficits like that mentioned above. This is perhaps not surprising; the frontal cortex can been seen as being particularly important in the control of social behaviour from both an evolutionary and anatomical perspective. Phylogenetically speaking, the prefrontal cortex is the most proximally developed area of cortex (Kolb & Whishaw, 1990; MacLean, 1990), and in the human comprises a

relatively huge 33% of the neocortex (Fuster, 1989), as opposed to only 17% in chimpanzees. As a percentage of cortex, the frontal lobes decrease down through lower primates and into other mammals, reaching such low percentages as 3.5% of the neocortex in cats (Raine, 1993). Clearly this proliferation of frontal cortex corresponds to the proliferation of complex social rules, norms and mores which have become a part of day-to-day human existence (Luria, 1976, 1980).

From an anatomic point of view, the frontal cortex can be seen as significant as it connects in either an afferent or efferent fashion to virtually all brain structures (Cummings, 1995). Notable of these connections are those with the limbic system, a group of interconnected subcortical brain structures consisting of the hippocampus, amygdala, fornix, septum, cingulate gyrus, and mammillary bodies. The frontal cortex also connects directly with the hypothalamus and with several thalamic nucleii (Cummings, 1995). The connections to the limbic system are thought to be particularly important as this system is widely believed to be involved in the mediation of emotional behaviour, as well as learning and memory.

Evidence associating frontal lobe abnormalities with the control of human social behaviour comes from three sources: clinical reports of behaviour in patients with frontal lobe lesions; neuroimaging studies of aggressive individuals and violent criminals; and neuropsychological test studies of individuals who likewise manifest aberrant social behaviour.

Clinical Reports of Behaviour of Patients with Frontal Lobe Lesions

Although the most famous sufferer of frontal lobe damage died some one hundred and forty years ago, active discussions of his injury and the consequences thereof continue (Damasio, 1994). In 1848, Phineas Gage was a young, friendly, formal, conscientious construction foreman. He was in charge of a crew of railway construction workers; specifically they are a detonation team, blasting holes in rock for the construction of a new railway. One summer afternoon, Gage, in a moment of inattentiveness, set off an explosion without achieving proper distance. A three and one-half foot long iron tamping rod, used to compress the charge, was blown directly and violently into his face. The pointed end of the rod entered Gage's left cheek under the eye, and exited out the top of his head. Gage survived this injury, and perhaps as astonishingly, the inevitable series of ensuing infections. He demonstrated an exceptional recovery, physically, but his friends and family soon began to note definite changes in him and his nature.

Although seemingly not impaired in an intellectual sense, Gage's friends agreed that "Gage was no longer Gage" (Damasio, 1994). Where he was once staid and sombre, Gage is now intemperate, irreverent, profane, and capricious. His physician wrote that "the equilibrium... between intellectual faculty and animal propensities" (Harlow, 1868) had been destroyed. Soon let go by the railroad company, Gage's downward

spiral continued. Wandering afar, prone to "drinking and brawling", Gage was reduced to working as an attraction at Barnum's museum in New York. By 1860, twelve years after the accident, the unemployable Gage was living with his mother in San Francisco. There he died in 1861, after a prolonged series of seizures, at thirty-eight years of age (Damasio, 1994).

Recently, neuroimaging techniques were used to demonstrate that the lesion involved areas of the ventromedial region of both frontal lobes (Damasio, Grabowski, Frank, Galaburda & Damasio, 1994). While perhaps the most famous and notorious case of personality change subsequent to frontal lobe damage, Gage is by no means the only case. In fact, after decades of studies that have examined damage to the frontal cortex, a pattern of changes has been alluded to, which includes argumentativeness, irritability, impulsivity, loss of social grace, and a fundamental disregard for behavioural consequences (Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Miller, 1999; Miller, Darby, Benson, Cummings, et al., 1997). This pattern of deficits has become known as the "frontal lobe syndrome" (MacKinnon & Yudofsky, 1986; Mesulam, 1986). That is, in non-criminal populations, studies suggest a link between frontal lobe damage and reduced control of social behaviour.

Only quite recently have techniques been developed to examine the structure and function of the brain. These techniques, which I will generally refer to as neuroimaging techniques, have been utilised to

examine criminal offenders and other violent offenders in order to further examine the hypothesis that frontal lobe dysfunction is related to aggression and violent crime.

Neuroimaging Studies and Aggression

There has been in the last decade dramatic advancements in brain imagine techniques, and as a result these new techniques have allowed researchers another means of gaining insight into the neuropsychology of aggression. However, due to constraints of time, money, and availability, imaging studies of aggression are still relatively scarce, and moreover, the methodologies have been inconsistent. However, that is not to say that these studies offer us no relevant information.

The first neuroimaging studies used computerised axial tomography (CT), usually in combination with some other assessment technique, such as electroencephalography (EEG) or neuropsychological testing. This is the least instructive of the literatures detailing imaging studies of aggressive populations. First of all, although clearly a breakthrough technique when it was developed, CT compares poorly with current imaging techniques; CT slice thickness is approximately 10 mm. This relatively poor spatial resolution does not lend itself to the accurate assessment of substantive cortical abnormalities. Second, most of the studies using CT examined sexual offenders rather than non-sexual violent

offenders (see Raine 1993 for a review). As a result, the conclusions we can make about brain structure in non-sexual violent offenders are limited.

The studies using magnetic resonance imaging (MRI) have been only slightly more common. The first was conducted by Tonkonogy (1991), who used both CT and MRI to examine a sample of 87 psychiatric patients. Of those 87 patients, 14 had histories of frequent aggressive behaviour. The results suggested that these latter 14 were more likely to have lesions of the anterior-inferior areas of the temporal lobes than those with no history of aggression. However, this study has been criticised due to the fact that every member of the original sample had significant brain pathology; as such, the comparison between those with and without violent histories is likely of questionable utility (Raine & Buchsbaum, 1996). Other uses of MRI have been quite few. One case report detailed use of MRI to demonstrate brain structure impingement secondary to an arachnoid cyst in a 65-year old man with no violent history who had spontaneously manifested extreme impulse aggression. The cyst effaced ventral frontal, anterior temporal and insular cortical gyri (Relkin, Plum, Mattis, Eidelberg & Tranel, 1996). Another study, of perhaps greater applicability, used structural magnetic resonance imaging to compare 21 participants with antisocial personality disorder (APD) with 3 different control groups (34 healthy participants, 26 participants with substance dependence, and 21 psychiatric controls). The APD group showed an

11.0% reduction in prefrontal grey matter volume in the absence of ostensible brain lesions (Raine, Lencz, Bihrle, LaCasse & Colletti, 2000).

Whereas MRI and CT are structural imaging techniques, positron emission tomography (PET) and regional cerebral blood flow (RCBF) are functional imaging techniques. Moreover, these latter techniques have been used much more excessively in the assessment of violent offenders. The first of these sorts of attempts were not very successful; Graber and colleagues, for example, used RCBF to compare paedophiles with rapists, as opposed to sexually violent compared with non-sexually violent, or even violent versus control (Graber, Hartmann, Ciffman, Huey & Golden, 1982). This study demonstrated reduced RCBF in the pedophiles but not the rapists. Several members of this group conducted another study, comparing 16 child molesters with 16 normal controls, again using RCBF, and this time demonstrated reduced RCBF specific to the molesters that was particulary pronounced in the frontal cortex (Hendricks, Fitzpatrick, Hartmann, Quaife, Stratbucker & Graber, 1988).

Since these early efforts, several studies have focussed specifically on non-sexual violent crime. Volkow & Tancredi (1987), for example, assessed 4 violent patients and 4 normal controls using PET, and demonstrated compromised function of the frontal cortex in those with histories of violence. These researchers and their colleagues followed this study with another, larger study (Volkow, Tancredi, Grant, Gillespie, Valentine, Mullani, Wang, & Hollister, 1995). In this latter study, PET

was used to evaluate regional brain glucose metabolism in eight normal subjects and eight psychiatric patients with a history of repetitive violent behaviour. Seven of the patients showed widespread regions of low brain metabolism relative to the normal comparison subjects which, although varying somewhat, tended to be specific to medial temporal and prefrontal cortices.

Raine and colleagues used PET to study 22 murderers versus 22 controls (Raine, Buchsbaum, Stanley, Lottenberg, Abel, & Stoddard, 1994). The results, while not demonstrating lesions, per se, showed more frontal abnormalities in the murderers. A subsequent study, with 41 murderers and 41 age- and sex-match controls, again used PET imaging techniques to examine brain function (Raine, Buchsbaum, & LaCasse, 1997). The murderers were characterised by reduced glucose metabolism in the prefrontal cortex, superior parietal gyrus, left angular gyrus, and the corpus collosum. This group has also suggested a distinction between predatory and affective murderers, wherein the former stalk their victims in a calm fashion, and the latter kill in spontaneous impulsive acts. In a study comparing these two kinds of offenders (Raine, Meloy, Bihrle, Stoddard, LaCasse & Buchsbaum, 1998), PET was used to assess left and right hemisphere prefrontal and subcortical regions in 15 predatory murderers, nine affective murderers and 41 controls. Affective murderers relative to comparisons had lower left and right prefrontal functioning. In

contrast, predatory murderers had prefrontal functioning that was more equivalent to comparisons.

Another imaging technique, single photon emission computerised tomography (SPECT) has also been used to study the neurology of violent offenders. In one study (Amen, Stubblefield, Carmichael & Thisted, 1996), forty adolescents and adults who exhibited aggressive behaviour within the six months prior to evaluation (by physically attacking another person or destroying property) were evaluated with brain SPECT imaging. A control group of 40 psychiatric patients who had never been reported to exhibit aggressive behaviour were also studied. The brain SPECT patterns of the group with aggressive behaviour showed significant differences from the control group in several areas of brain. These findings were most often manifested in decreased activity in the prefrontal cortex and anteromedial portions of the frontal lobes, as well as left-sided increased activity in the temporal lobe and limbic system. In the most recent study (Soderstrom, Tullberg, Wikkelso, Ekholm & Forsman, 2000), the researchers retrospectively examined MRI, RCBF and SPECT images from pre-trial forensic psychiatric investigations of 21 subjects convicted of impulsive violent crimes, controlling for major mental disorder, substance abuse, and current medication. In 16 of 21 subjects, visual assessment of SPECT scans showed some hypoperfusion in the temporal and/or frontal lobes.

The evidence suggesting a relationship between frontal lobe dysfunction to violence or increased aggression is not insignificant, but is in no way unequivocal. Although these imaging technologies continue to advance, the cost of these studies is prohibitive, and the resulting relative scarcity of these studies makes drawing conclusions difficult. Although many 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. Moreover, imaging studies tend to contribute little to our understanding of what specific pattern of cognitive deficits characterise, for example, predatory versus affective murderers.

Another research technique has been to use neuropsychological tests so as to observe cognitive functions putatively associated with frontal dysfunction, and to associate those with antisocial or criminal behaviour.

Neuropsychological Studies and Aggression

Systematic attempts to relate frontal dysfunction to aggression and violent crime, assessing criminal offenders and other violent individuals with established neurological batteries, are at least two decades old. One study (Yeudall & Fromm-Auch, 1979), for example, 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, which was composed of 86 violent offenders, demonstrated significantly more anterior neuropsychological dysfunction than the 79 normal controls. Another study using the Luria-Nebraska Neuropsychological Battery (LNNB; Bryant, Scott, Golden, & Tori, 1984) found histories of violence in 73% of participants classified as brain damaged compared to only 28% of those classified as normal. However, Yeudall, Fromm-Auch and Davies (1982) failed to replicate the HRTNB-aggression relationship in violent delinquents, and Brickman, McManus, Grapentine & Alessi (1984) did not demonstrate the same relationship between LNNB indication of frontal deficit and aggression.

It appears from these studies as if frontal abnormalities are involved in antisocial behaviour, in some fashion, but there are inconsistencies that make conclusions tenuous. A related hypothesis is that the frontal deficits related to heightened aggression are localised predominantly in the left hemisphere (Flor-Henry, 1973). This hypothesis suggests that neuropsychological tests that are not sensitive to laterality will not be as likely to demonstrate the relationship. Yeudall and his colleagues (Yeudall, 1977, 1980; Yeudall, Fedora, & Fromm-Auch, 1987) have suggested that aggression may be specific to left frontal and anterior-temporal cortex. In fact, in one study, 76% of offenders had dysfunction specific to the frontal and temporal cortex (Yeudall & Flor-Henry, unpublished; reported in Raine & Buchbaum, 1996).

Although there has not continued to be an emphasis on laterality, more recent studies of neuropsychological function in forensic patients have continued to add support to the theory of prefrontal abnormality (Barratt, Stanford, Kent & Felthaus, 1997; Foster, Hillbrand & Silverstein, 1993). This relationship has also been demonstrated in younger boys; one study assessed aggression in boys relying on teacher- and parent ratings, and tested the boys on a large battery of neuropsychological tests. The results demonstrated a strong relationship between physical aggression in boys and their scores on tests of prefrontal function. However, the relationship between aggression and scores on other tests was not significant (Seguin, et al., 1995).

There is also a large literature detailing the neuropsychology of aggression in patients with psychiatric diagnoses. Diagnoses characterised by aggression, including antisocial personality disorder (Gorenstein, 1987; Malloy, Noel, Longabaugh & Beatty, 1990), psychopathy (Lapierre, Braun & Hodgins, 1995), conduct disorder (Lueger & Gill) and attention-deficit hyperactivity disorder (Barkley, 1997) have all been subject to neuropsychological studies. Integrative reviews of these studies concluded generally that these individuals perform more poorly on tests generally thought to be mediated by prefrontal cortex rather than tests thought to be mediated by posterior brain regions (Giancola, 2000a; Golden, Jackson, Peterson-Rohne & Gontkovsky, 1996). Furthermore, studies have demonstrated frontal dysfunction in aggressive psychiatric

populations not ordinarily characterised by aggression, such as patients suffering schizophrenia (Rasmussen, Levanden & Sletvold, 1995).

It should be pointed out that the results of some of the above studies should be considered with some caution. Many of the studies have been criticised for failing to control for possibly confounding variables. For example, some researchers have pointed out that along with deficits in tests thought to measure function of the prefrontal cortex, many of the population referred to above (both forensic and psychiatric) have also been shown to have verbal deficits (Giancola, 2000a). However, studies which have methodologically or statistically accounted for these verbal deficits continue to find a significant relationship between frontal measures and aggression (Giancola & Mezzich, 2000; Mofitt, 1993; Seguin, et al., 1995). Studies have also been criticised for failing to consider substance abuse, for inadequate diagnostic techniques, and for combining small samples with large test batteries (Hart, Forth, & Hare, 1990).

That caveat being issued, studies continue to advance in their sophistication, in terms of eliminating third-variable explanations. For example, the relationship between aggression and frontal-lobe tests demonstrated by Seguin and colleagues (1995) was drawn into question by suggestions of other mediating variables (Pennington & Ozonoff, 1996). However, subsequent work by the former authors demonstrated that the relationship was still in evidence even after accounting for attention-

deficit hyperactivity, memory, and intelligence (Seguin, Boulerice, Harden, Tremblay, & Pihl, 1999).

Although the literature relating aggressive behaviour to neuropsychological tests is relatively consistent, it is based on a great number of tests, the specificity of many of which remains in doubt. Besides the tests alluded to above, the Wisconsin Card Sort Test (Heaton, 1981), the Porteus Maze Test (Porteus, 1965), the Motor Restraint Test (Parsons, Tarter, & Edelberg, 1972), the Tower of Hanoi Test (Welsh, Pennington, Ozonoff, Rouse, & McCabe, 1990), the Continuous Performance Test (Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956) and the Stroop Test (MacLeod, 1991) have all been used to investigate aggression (Giancola, 2000a). Although the results of these studies are helpful, better developed and validated tests of prefrontal function are desirable so as to lend further credence to the notion that prefrontal abnormality is related to propensity for aggressive behaviour.

Tests of Dorsolateral Prefrontal Function and Aggression

There are two recently developed neuropsychological tests that have begun to garner significant research interest. These new tests, developed by researchers at the Montreal Neurological Institute, are essentially adaptations of tests classically used with monkeys for human use (Roberts, 1998). These include the self-ordered pointing task (which was itself derived from the internal and external generated sequencing

tasks; Brody & Pribram, 1978) and the conditional associative-learning task. The self-ordered pointing task (SOP; Petrides & Milner, 1982) and the conditional associative-learning task (CALT; Petrides, 1985a, 1985b) both appear to assess some element of function of the dorsolateral prefrontal cortex.

Positron emission tomography, conducted with normal volunteers, while executing a modified version of the CALT, demonstrated activation of cytoarchitectonic area 8 of the dorsolateral frontal cortex. Conversely, 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). Further validation of the relationship between these tests and prefrontal cortex comes from studies demonstrating that patients with unilateral lesions of the frontal lobe perform poorly on these tasks (Petrides & Milner, 1982; Petrides, 1985a). Corresponding to the subtle differences in the neuroanatomical area associated with each test, impairment on each appears related to a slightly different cognitive problem. Impairment on the CALT appears due to problems selecting a correct response from a set of responses (Petrides, 1985a) whereas problems on the SOP appear due to poor monitoring of responses (Petrides & Milner, 1982). Generally speaking, the CALT can be conceptualised as a test of strategy formulation and implementation, whereas the SOP can be considered more a classic test of working memory.

These tests were first used to assess likelihood of aggression in normal male volunteers, in two independent labs, one in Montreal (Lau, Peterson & Pihl, 1995) and one in Athens, Georgia (Giancola & Zeichner, 1994). In both these studies, the authors tested their participants on these tests, and then tested their subjects on a laboratory measure of aggression (in both cases versions of the aforementioned Taylor competitive reaction-time task). Whereas Giancola & Zeichner correlated the neuropsychological test scores with the dependant measures from the laboratory measure of aggression, Lau and colleagues instead divided their subjects into high- and low-function quartiles and utilised a between-groups ANOVA design. Furthermore, whereas Giancola and Zeichner did not involve alcohol, Lau and colleagues administered half their participants alcohol, making their ANOVA a factorial design. Regardless of these methodological differences, both research teams demonstrated a strong relationship between scores on these neuropsychological tests, and propensity for acting aggressively when provoked.

In a subsequent study, Lau and Pihl (1996) again tested normal male participants on these tests, and again split their participants into high and low quartile groups. Furthermore, they again tested using the Taylor aggression task. However, in this study they offered their participants contingent monetary reward in order to decrease their aggression. That is, all participants faced the same provocation, but those who inhibited their own retaliatory aggression were rewarded for doing so. Lau and Pihl found that

while the participants in the quartile of high neuropsychological performance inhibited their aggression to earn reward, the men in the low-performance did not; the responses of this latter group were just as if no contingent money was available. This result led these researchers to suggest that increased aggression in these men was a function of the inability to inhibit impulsivity.

These two new neuropsychological tests, which appeared to be sensitive to the integrity of the prefrontal cortex, appear to be able to differentiate aggressive from non-aggressive men. These tests have also been used to show an association between poor scores and increased propensity for aggression in young boys (Seguin, Pihl, Harden, Tremblay, et al, 1995) and conduct disordered adolescent girls (Giancola, Martin, Tarter, Pelham, et al., 1996).

Prefrontal Function versus Executive Cognitive Function

In the studies that follow, the reader will note that although the neuroanatomical issues addressed above are alluded to, there is greater emphasis on the term "executive function" than there is specific referral to neuroanatomy. The reason for this distinction is simple; it is an abstraction to suggest that the dorsolateral prefrontal cortex, the prefrontal cortex, the frontal lobes, or indeed any brain structure functions independently to produce aggressive behaviour (Roberts, Robbins, & Weiskrantz, 1998). Alluding to neuroanatomy in a causal fashion presupposes a level of functional neuroanatomical understanding which

we simply do not have (Moffitt, 1990; Cummings, 1995). Equivalence between executive functioning and prefrontal cortex cannot be assumed, especially when one takes into account the reality of patients with the "dysexecutive syndrome" secondary to lesions to brain areas other than the frontal lobes (Roberts, 1998; Baddeley & Della Sala, 1998). To allude to the cognitive capacities that are likely subserved, at least in part, by these neuroanatomical areas is far more readily justified.

"Executive cognitive function" is a higher-order cognitive construct that has been conceptualised and defined in multitudinous ways (Robbins, 1998). Generally, the term refers to mechanisms by which performance is optimised in situations demanding operation of various cognitive processes, including abstraction, sequencing, strategy formulation, set shifting, and planning (Baddeley, 1986). Generally, one can think of executive function as the ability to plan, initiate, and maintain or alter some form of goal-directed behaviour. It is clearly a complex cognitive construct, components of which may include: utilisation of information held temporarily or "on-line" (i.e. working memory; Goldman-Rakic, 1987, 1992); utilisation of various attentional processes (Shallice, 1982); inhibition of unsuitable inclinations (Shallice & Burgess, 1993); and/or monitoring of behaviour from an emotional, motivational, or affective point of view (Damasio, 1994; Petrides 1996). Attempts have been made to fractionate executive functioning into a number of distinct cognitive processes (e.g. Baddeley & Della Salla, 1998; Shallice &

Burgess, 1998), as well as to partition the prefrontal function in terms of which of these cognitive capacities certain areas mediate (Robbins, 1998). However, there remains no consistent agreement as to the definition of executive function, there is no consistent agreement of the specifics of neuroanatomy, and perhaps most importantly, there exists no consistent agreement on what neuropsychological tests actually assess this construct. Pennington and Ozonoff (1996), in a review of the association of executive function to childhood psychopathology, point out that dozens of tests of the construct currently exist.

The tests used in this dissertation, the Conditional Associative Learning Test and the Self-Ordered Pointing Test, are but two of these tests. However, these tests were selected from the many due to their theory-based construction, and their established validity via neuroimaging techniques. Moreover, as mentioned, these tests clearly assess cognitive capacities (strategy formulation, working memory, response monitoring, inhibition) consistent with those typically associated with executive function.

Introduction to Study One

At the time this dissertation was commenced, there were three studies relevant to my general topic that had already been conducted, and were either published or under review. These studies suggested that men with poor executive functioning were more aggressive than peers

(Giancola & Zeichner, 1994; Lau, Pihl & Peterson, 1995), and that when provoked, these men were also unable to inhibit aggressive responses in order to gain monetary reward (Lau & Pihl, 1996).

From this point, there were clearly several different directions in which continued studies could proceed. The first study contained in this dissertation was intended to examine whether alcohol intoxication had the capacity to render cognitively intact men impulsively aggressive. More specifically, it was of interest whether men with above average ECF scores would be impaired by alcohol to the extent that they would fail to inhibit aggressive behaviour in the face of monetary reward, as did the low-ECF (non-intoxicated) men in the Lau & Pihl (1996) study.

From a theoretical point of view, the importance of the study was not only in that it was intended to examine the disinhibitory properties of alcohol, but also in that it was intended to offer insight into the issue of control. That is, although the literature clearly demonstrates that alcohol significantly increases aggression in between-group designs, the fact remains that the majority of men do not become aggressive when alcohol-intoxicated, even when significantly provoked. Moreover, it had been demonstrated experimentally that certain manipulations reduce alcohol-related aggression (for example, Taylor & Jeavons, 1985), but no mechanism had been proposed for this effect. Thus, whereas the previous studies had investigated poor executive function vis a vis its relation to alcohol and disinhibition of behaviour, this study was interested in

executive function, alcohol, and cue-appropriate inhibition of asocial, aggressive behaviour.

STUDY ONE

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Cognitive Functioning and the Inhibition of Alcohol-Induced Aggression*

PETER N. S. HOAKEN, B.A., JEAN-MARC ASSAAD, B.A., &

ROBERT O. PIHL, PH.D.

Department of Psychology, McGill University, 1205 Dr. Penfield Ave,

Montreal,

Quebec, H3A 1B1, Canada

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ABSTRACT. *Objective*: A highly replicable research finding is that alcohol intoxication tends to induce aggressive responding. Recent research investigating the role of cognitive function in this relationship has shown that individuals who perform poorly on certain cognitive tasks have difficulty responding to contingencies to inhibit aggression, while high performers do not. High performers, however, show increased aggression while intoxicated. This study investigated whether subjects with above average cognitive functioning would inhibit aggression in order to attain monetary reward. *Method*: Men (N = 43), aged 18-30, selected on the basis of high performance on a neuropsychological test putatively assessing function of dorsolateral prefrontal cortex, the conditional associative learning task, participated in a modified version of the Taylor Aggression Task. Half the subjects were acutely alcohol intoxicated, the other half were sober. Furthermore, half the subjects in each of these groups received contingent monetary reward for choosing lower shocks. Aggression was defined as shock intensity delivered to a sham opponent. *Results*: Contrary to the hypothesis, intoxicated subjects, even though significantly impaired cognitively relative to their nonintoxicated peers ($F = 4.29$, 1/41 df, $p < .05$), appeared to have no difficulty inhibiting their aggression in order to gain monetary reward. That is, there was no difference between intoxicated and nonintoxicated subjects on the dependent variable, shock intensity, when contingent money was available ($F = .01$, 1/20 df, $p = .935$). *Conclusion*: This finding provides further evidence that alcohol-induced aggression is not

a uniform phenomenon, and it suggests a neuropsychological mechanism that may mediate the relationship. It may be that individuals with above average cognitive abilities retain sufficient residual functioning to inhibit aggressive responding, even when acutely alcohol intoxicated. (J. Stud. Alcohol, 59: 599-607, 1998)

VIOLENCE is a serious problem in North America, with one of the most important situational determinants of aggression being alcohol intoxication (Baron and Richardson, 1994). Research has shown that more than half of all violent crimes are immediately preceded by the consumption of alcohol by the offender (Murdoch et al., 1990), but this correlational research leaves the role of alcohol in some question. In order to better study the phenomenon, aggressive reactions have been elicited in laboratory experimentation. Various forms of provocation have all been shown to elicit strong counter-aggression in research subjects (Donnerstein et al., 1975; Geen, 1968; Wilson and Rogers, 1975; Taylor, 1967). Bushman and Cooper's (1990) meta-analytic study of 30 relevant experimental studies concluded that individuals were significantly more verbally and physically aggressive while alcohol-intoxicated.

Research has demonstrated relationships between many characteristics and aggressive response to provocation, including personality traits, genetic factors and sex differences (Baron and Richardson, 1994; Wood et al., 1991). One factor that may be particularly important is cognitive function, specifically those capabilities thought to be mediated by prefrontal cortex and related structures. The dorsolateral prefrontal-subcortical circuit, specifically, is a neuroanatomical pathway of considerable interest. This pathway consists of dorsolateral prefrontal cortex and projections to and from several subcortical structures, including the caudate nucleus, globus pallidus, substantia nigra and several thalamic nuclei

(Cummings, 1995). Tests thought to assess the functioning of this circuit require such capabilities as active monitoring, integration of information from both the internal and exterior worlds and calculation of appropriate behavioral responses (Seguin et al., 1995; Fuster, 1989). There is considerable evidence that associates frontal lobe deficits with decreased regulation of human social behavior. Individuals with frontal lobe damage often manifest a "disinhibition syndrome" (Hecaen and Albert, 1978), and even in nonlesioned individuals there is evidence of this relationship (Seguin et al., 1995; Raine et al., 1994). Not surprisingly, then, the role of the frontal lobes in aggression has begun to garner significant research interest.

Studies have shown that alcohol is pharmacologically capable of impairing performance on neuropsychological tests that measure the ability to plan, organize behavior, and think abstractly (Peterson et al., 1990). Research has also implicated the functioning of the frontal lobes in aggressive responding in alcohol-intoxicated subjects. Lau et al. (1995) and Giancola and Zeichner (1994) both used tests of dorsolateral frontal cortex (Petrides et al., 1993) to divide subjects into high and low quartiles. Both studies found heightened aggression on the Taylor Aggression Paradigm associated with impaired performance on these tests. Lau and Pihl (1996) again split subjects into high and low quartiles and offered these nonintoxicated subjects contingent monetary reward in order to decrease their aggression. They found that only subjects with high frontal functioning suppressed aggression in order to gain money, leading them to suggest that

increased aggression in a sample of men with decreased frontal lobe functioning was a function of the inability to inhibit impulsivity.

What is not known is whether subjects with baseline high frontal functioning would, if acutely alcohol-intoxicated, act like those subjects with frontal impairments; i.e, when intoxicated, will they become impulsive and be unable to identify and utilize more adaptive behavioral responses. If this is the case, then this would provide additional evidence implicating alcohol-induced cognitive dysfunction in the manifestation of aggression. This is the rationale of the current study.

This study used monetary reward as a contingency to attempt to inhibit aggressive responding in intoxicated and nonintoxicated subjects, all of whom had demonstrated high levels of performance on a test of prefrontal function and who showed overall high cognitive function prior to the alcohol condition. Neuropsychological tests were administered to assess prefrontal and temporal/hippocampal functioning, both pre drug and postdrug administration. It was specifically hypothesized that (1) only the no-alcohol group would be able to reduce their aggression in response to monetary reward; (2) subjects in the alcohol condition would demonstrate diminished frontal lobe function, as assessed by the neuropsychological tests, relative to the subjects in the no-alcohol condition; and (3) the no-alcohol group and the alcohol group would not differ on tests of temporal lobe/hippocampal functioning. The third hypothesis is important because it pertains to a methodological issue not addressed in previous studies, that of

double-dissociation. Tests that assess function of prefrontal cortex and associated structures should be interpreted in the context of other nonfrontal tests (Anderson et al., 1991; Luria and Homskaya, 1964), in order to conclude that the prefrontal cortex is involved specifically.

Method

Participants

Nonalcoholic male social drinkers, as assessed by a score of less than 5 on a brief form of the Michigan Alcoholism Screening Test (Pokorny et al., 1972) and a brief interview, aged 18-30, in good self-reported physical and mental health, were recruited through newspaper and campus advertisements. Those receiving medical treatment that contraindicated alcohol consumption, who had sustained a serious injury to the head, who had a diagnosed learning disability, or who were familiar with psychological experimentation were excluded from participation. Subjects who met all of the inclusion criteria (N = 98) were then administered the spatial conditioned associative-learning task (SCALT; Petrides, 1985a), and a short form of the Weschler Adult Intelligence Scale (WAIS-R; Wechsler, 1981). If they were able to complete the CALT in under 100 trials and achieved full-scale IQ estimates of over 100, they were included in the experiment (N = 52). Ability to complete the CALT in under 100 trials was intended to provide subjects in an upper quartile of function; WAIS-R estimates over 100 were intended to ensure sound overall cognitive ability. Subjects were randomly

assigned to one of four groups: either the alcohol-intoxicated or nonintoxicated conditions and either the inhibition or no-inhibition groups.

Apparatus

Aggression was elicited and assessed with a modified version of 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 lit up to indicate shock level chosen by the opponent. An IBM-compatible personal computer was used to run the aggression task and to 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 administrations of shocks to his fictitious opponent by viewing a DC ammeter provided for that purpose. A prerecorded videotape of the sham opponent receiving instructions regarding performance of the aggression task was played to the subject to reinforce the subject's belief in the existence of the opponent.

For those subjects in the inhibition condition, a cardboard display of eight monetary values, decreasing from 40 to five cents in five-cent increments, was placed immediately above the lights indicating the different shock levels (i.e., reward increased as shock level decreased). In this condition, during each trial, the subject could see how much money corresponded to each choice of shock and, upon the completion of each

trial, could monitor the total amount of money gained to that point by viewing a counter situated next to the aggression machine.

The spatial conditioned associative-learning task was used to screen subjects. Each one of six randomly placed lamps was 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 tester would respond with the word "wrong," until the subject pointed to the correct card, at which point the tester responded, "right," and the next trial began. The subject's task was to learn these associations so that when a given light was presented, the correct card would be chosen. Completion criterion was 18 consecutive trials without an error. Individuals with dorsolateral prefrontal lesions have been shown to perform poorly on this task; furthermore, 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 et al., 1993). 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, 1985a).

The Block Design, Information, and Vocabulary subtests of the WAIS-R were administered to provide estimates of full-scale IQ (Brooker and Cyr, 1986) and overall cognitive ability. Also administered prior to

drink administration was the Logical Memory subtest of the Weschler Memory Scale (Wechsler, 1945).

Subsequent to the aggression task paradigm, the nonspatial conditioned associative-learning task was also administered to all subjects. In this task, the subject was shown six hand postures by the experimenter and told that each hand posture was associated with a certain colored ball. The subject was not informed of the pairings. The tester would respond with the word "wrong," until the subject pointed to the correct card, at which point the tester responded, "right," and the next trial began. The task was that the subject had to learn to associate each hand posture with the appropriately colored ball. Completion criterion is 18 consecutive trials without an error. This task is essentially analogous to the spatial conditioned associative-learning task; it, too, is sensitive to frontal lobe dysfunction (Petrides, 1985b).

Also following administration of the Taylor aggression task, subjects were administered the Word Fluency task, which is also associated with the dorsolateral prefrontal circuit (Milner, 1964; Cummings, 1995). Specifically, the Word Fluency test appears to measure spontaneous verbal fluency, a capacity much impaired in patients with frontal lobe lesions. Subjects were also administered the Paired Associates (abstract) task, a task of short-term verbal memory, which is thought to assess capabilities associated with hippocampal and other temporal lobe structure functions (Milner, 1975).

Procedure

Subjects who met inclusion criteria were asked not to consume drugs or alcohol for at least 24 hours prior to testing. Upon arriving at the laboratory on the first day, subjects signed an informed consent form and provided demographic data. The subjects then completed the initial battery of neuropsychological and intelligence tests. Those meeting the performance criteria were scheduled for a second session.

Within seven days of the first session, subjects participated in the drug challenge, additional neuropsychological tests and the Taylor task. After measurement of blood alcohol concentration (BAC) to ensure sobriety, subjects were randomly assigned to the alcohol or no alcohol conditions. BAC was determined using an Alco-sensor III (Thomas Ltd.). In the alcohol condition, subjects were administered 1 ml per kg of body weight 95% alcohol USP units in three drinks of a one part alcohol to seven parts orange juice solution. In the sober condition, the men were administered three drinks of orange juice of equivalent volume. In each condition, participants were told explicitly what they were drinking. Drinks were consumed over a 20-minute period. A 20-minute waiting period followed to allow the men in the alcohol condition time to reach near-peak BAC. BACs were then taken and recorded.

Subjects were also assigned randomly to the inhibited or noninhibited conditions. In the inhibited condition, subjects received 40 cents for choosing Shock Level 1, the lowest, with the amounts decreasing

by 5 cents for each level to a value of 5 cents for selecting Shock Level 8, the highest. In the control (noninhibited) condition, there was no monetary reward associated with the subject's shock choice.

Each subject's pain threshold for electric shock was determined by delivering a series of shocks from 0-255 units (0-5.63 ma). The shocks increased stepwise by 5 units (5 units equals approximately 0.11 ma) at a constant rate. Each subject was 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. The next shock therefore was one step lower than the shock that induced pressing the button. Pressing the button upon three consecutive presentations of the same shock intensity stopped shock delivery. This shock intensity was defined as the subject's pain threshold.

The aggression task was then introduced as a competitive reaction-time task. Each subject 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 subject would be informed of the opponent's shock choice. If the subject "lost" that trial, he received that shock. Shock Levels 1-8 increased from 28 units for Level 1 to 100% of the subject's given pain threshold for Level 8, with the 6 intermediate shock levels being equal to 28 units plus 23%, 31%, 39%, 76%, 84%, and 92% of the difference between the subject's given pain threshold and the initial 28-unit level. The nature of the increases of the shock intensity was

decided upon in order to clearly define those shocks thought to be minimally provoking (Levels 1-4) and those thought to be maximally provoking (Levels 5-8). If the subject "won" the reaction time trial, he would then administer the previously chosen shock to his opponent.

Subjects in the inhibited condition were also 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 all of the trials.

Following these instructions, 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. In fact, the subject was watching a prerecorded videotape of a fictitious opponent receiving instructions.

The task itself consisted of 26 consecutive trials: a block of 12 trials followed by a transition trial, a second block of 12 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 either 1 second or 2 second duration. All subjects received three shocks at each level alternately winning one trial, losing two trials and winning two trials, losing one trial.

If the subject was to receive two shocks at a certain level, he would receive one of 1 second and one of 2 second duration. All subjects lost the transition trial and won the final trial. In both cases the opponent's shock choice was a Level 5.

Following the aggression task, subjects were interviewed to verify the success of the deception, debriefed and the necessity for deception was fully explained. No subject was adversely affected by the deception, according to self-report. The experimenter questioned the subjects rigorously regarding the completeness of the deception. Deception was rated by two experimenters subsequent to debriefing. Only subjects who were deemed to have been completely deceived, who reported no suspicions or doubts, were included in the analysis. Seven subjects were eliminated because they were deemed to not have been deceived. These seven subjects were basically evenly distributed among the four groups. One subject was eliminated because he failed to provide meaningful data, and one subject was excluded due to an adverse reaction to the alcohol. In all, meaningful and valid data was acquired from 43 subjects. All subjects were paid $5 an hour to compensate for lost time.

The objective measures of aggression were: 1) the intensity of shock the subject selected for the first trial, and 2) the mean shock selected for both the high and low provocation conditions. 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 both low and high provocation.

Results

Subject Measures

The following analysis is based on 43 subjects (sober/no inhibition, N = 11, Group 1; sober/inhibited, N = 11, Group 2; intoxicated/no inhibition, N = 10, Group 3; intoxicated/inhibited, N = 11, Group 4). Mean age, years of education, beverages per week, beverages per occasion, neighborhood code (an approximate of socioeconomic status) and IQ are presented in Table 1. Univariate analyses of variance (ANOVAs) demonstrated no significant differences between the four groups on any of the demographic variables.

Pain threshold measures

As can be observed by the means in Table 1, rather large differences were observed for pain threshold between the groups. An ANOVA conducted on pain threshold revealed no significant differences between the four groups on this variable; however, because of this large variance, all analyses of shock selection were subsequently analyzed using this variable as a covariate.

Neuropsychological test measures

Mean scores on the spatial conditioned associative-learning task (CALT), nonspatial conditioned associative learning task (NSCALT) and Word Fluency, Paired Associates and Logical Memory tests are presented

in Table 2. Univariate ANOVAs conducted on the cognitive tests administered before beverage consumption demonstrated no difference between the intoxicated and nonintoxicated groups (collapsed over inhibition condition) on any of total trials to completion for the CALT, errors on the CALT or score on the Logical Memory task.

Analyses of the cognitive tests administered after beverage consumption, however, demonstrated diminished performance on frontal lobe tasks in the intoxicated group. When the NSCALT is administered subsequent to the CALT there is usually a significant learning effect. This was demonstrated in the sober groups, who were able to complete the NSCALT in 20.0 fewer trials and with 17.1 fewer errors, on average. The intoxicated group, conversely, were able to complete the NSCALT in only 1.5 fewer trials and in fact made 5.6 more errors. An ANCOVA, using the scores on the spatial conditional associative-learning task as the covariate, demonstrated a significant difference between the intoxicated and nonintoxicated groups on trials to completion for the nonspatial conditioned associative-learning task ($\underline{F} = 4.29$, 1/41 df, $\underline{p} < .05$). The total errors on the NSCALT was also significantly greater in the intoxicated group ($\underline{F} = 6.84$, 1/41 df, $\underline{p} < .05$). These results are presented in Figure 1.

The intoxicated subjects fared significantly worse than their sober peers on the other test thought to assess frontal lobe function, the Word Fluency task ($\underline{F} = 6.09$, 1/41, $\underline{p} < .05$). These findings all confirm our second hypothesis.

An ANOVA conducted on the Paired Associates scores demonstrated no difference between the intoxicated and non-intoxicated groups ($F = .68$, 1/41 df, $p = .42$). this finding suggess no significant temporal/hippocampal impairment and supports our third hypothesis.

Blood alcohol concentrations

A series of ANOVAs revealed no significant differences between the two intoxicated groups in terms of blood alcohol concentrations at the time of administration of any of the experimental measures. Means are presented in Table 3.

Initial shock setting

A 2 (Drug) X 2 (Inhibition) ANOVA, with pain threshold as a covariate, conducted on the initial shock choice, revealed no significant interaction between drug and inhibition. Significant main effects for both drug ($F = 5.01$, 1/40 df, $p < .05$) and inhibition ($F = 13.07$, 1/40 df, $p < .001$) on initial shock choice were found, with higher initial shock selections in the intoxicated and in the noninhibited groups.

Shock Intensity Measures

A 2 (Inhibition/No Inhibition) X 2 (Drug) X 2 (Provocation) mixed-design ANOVA, with provocation as a repeated measure, and again with pain threshold as a covariate, was conducted on shock intensity. No significant three-way interaction was found. Of the two-way interactions, only a significant interaction between drug and inhibition ($F = 4.54$, 1/39

df, $p < .05$) was found. A significant main effect of provocation was also attained ($F = 52.19$, 1/39 df, $p < .001$).

An analysis of simple main effects indicated a significant effect of drug in the non-inhibited condition ($F = 6.59$, 1/19 df, $p < .05$), with intoxicated subjects choosing higher shocks than their sober peers. There was no simple main effect for drug in the inhibited condition ($F = .01$, 1/20 df, $p = .935$). These two results together suggest that the contingent money served to reduce aggressive responding in the intoxicated subjects.

A significant simple main effect of inhibition in the intoxicated condition ($F = 14.05$, 1/19 df, $p < .001$) was found, with inhibited subjects choosing lower shocks than noninhibited subjects. This result is contrary to the first hypothesis. The simple main effect of inhibition in the nonintoxicated group did not reach significance , however ($F = 2.29$, 1/20 df, $p = .146$). The failure of this effect to reach significance was possibly due to a floor effect.

These results are all presented in Figure 2. As can be seen, when no contingent money was available, intoxicated subjects reacted more aggressively than nonintoxicated subjects. This was not a surprising result. Also not surprising was the finding that the presentation of contingent money made sober subjects even less aggressive than the sober subjects for whom no such incentive was available. However, what was not anticipated was that the presentation of contingent money would

profoundly reduce aggression in response to provocation in the intoxicated group.

Discussion

Inhibition of inappropriate or impulsive behaviours, with adaptive shifting to alternative behaviors, are functions attributed to the dorsolateral prefrontal-subcortical circuit (Moffitt and Henry, 1989; Cummings, 1995). As such, individuals with prefrontal dysfunction would be expected to both respond more aggressively when presented with provocation or punishment and fail to inhibit their aggressive behavior when presented with contingent monetary reward. Both of these phenomena have been demonstrated by experimental work (Lau et al., 1995; Lau and Pihl, 1996). Furthermore, alcohol has been demonstrated to interfere with these cognitive abilities (Peterson et al., 1990).

Therefore, it was not unreasonable to hypothesize that subjects with relatively high prefrontal function would, if their functioning was diminished by alcohol, act not unlike subjects with preexisting prefrontal deficits. This hypothesis was, however, not confirmed by the experiment. Specifically, although the group who consumed alcohol became significantly more aggressive than the group who remained sober when no inhibitory influence was exerted, they acted almost exactly like their sober peers when the contingent money was available.

That this population of subjects with above average cognitive abilities were able to inhibit their aggression is an important finding,

because it provides evidence for another variable that may mediate control of alcohol-induced aggression. Although heightened aggression in intoxicated subjects is a highly replicable finding, researchers have demonstrated that it is not a uniform phenomenon. Giancola and Zeichner (1995) have shown gender differences in alcohol-related aggression, with men becoming more aggressive towards men but not towards women. Bailey et al. (1983) demonstrated that intoxicated subjects made self-aware (by having them participate in the presence of a mirror and a video camera) are less aggressive than intoxicated subjects not made self-aware. Furthermore, two studies (Jeavons and Taylor, 1985; Taylor et al., 1976) demonstrated that nonimpaired college students were able to regulate their aggression while intoxicated. This result in the current study suggests the likely neuropsychological mechanism that underlies the findings in these two studies.

Further elucidation of the factors that control the alcohol-aggression relationship would seem to be an important endeavor. If subjects who appear to have low prefrontal function are unable to inhibit aggression to gain reward, then of interest is why those who demonstrate high frontal function do seem able. A possible interpretation of the results of this study is that the population chosen for the experiment, subjects with above average cognitive abilities, retain sufficient residual functioning, even when acutely intoxicated, to inhibit aggressive responding when in the presence of appropriate cues (i.e., although they

are impaired, they retain the ability to monitor the environment and to find and utilize alternative, more adaptive, behavioral responses).

There is some evidence to support the notion of residual function: Although the subjects in the intoxicated group were significantly impaired relative to their peers, their performance on the nonspatial conditioned associative-learning task appears to approximate (certainly within a standard deviation) the population mean. This is a tentative claim, because normative data for the NSCALT are only in the process of being collected. The mean referred to above is based on fewer than 200 subjects, all tested while nonintoxicated.

The results of this study suggest a neuropsychological or cognitive variable that may mediate the alcohol-aggression relationship. However, it should be pointed out that there are some methodological issues that should be addressed in future experiments. For instance, in order to avoid an enormous sample size, this study did not manipulate expectancy through use of a balanced-placebo design. It is possible that an expectancy manipulation might have significantly altered the results. However, this seems unlikely, as four meta-analyses have all concluded that alcohol expectancies play a negligible role in the production of aggression (Bushman, 1993; Bushman and Cooper, 1990; Hull and Bond, 1986; Steele and Southwick, 1985). Along the same lines, a study comparing the same manipulation with both high and low frontal

functioners would have been preferable for interpretation of results, but that again would have necessitated an unwieldy sample size.

Furthermore, this study used what might be considered a somewhat nonrepresentative sample. Of the 43 subjects used in the analysis, 28 were undergraduate university students and the remainder were from the general community. The sample is, therefore, less than ideal, and the extent to which this result will generalize to the population as a whole might be limited. That so many of the subjects were college students, however, does help to support that contention that they had "above average" cognitive abilities. It would have been necessary to screen a great many more subjects in order to recruit out sample, if the sample had been taken exclusively from the general community. This does, however, suggest another possible bias: One might suggest that the results of this study are due to demographic or personality variables, in that the studied population might have preexisting tendencies to be less aggressive than would a population of subjects with lower cognitive abilities. However, this is likely not the case. In the group of subjects who consumed alcohol, and for whom contingent money was not available, responding was quite aggressive. Their responses to provocation were in no way less aggressive than the responses of other populations, in other studies. Therefore, it is unlikely that this population has any pacifistic bias.

Another possible criticism is that certain demand characteristics caused the results; specifically, the intoxicated subjects for whom money

was available inhibited their aggression because of the demand characteristic. However, this seems unlikely in the context of the results of Lau and Pihl (1996). Exactly the same paradigm was used in this study, and the subjects in Lau and Pihl's study did not inhibit their aggression at all.

In conclusion, the present study demonstrates that the failure to inhibit aggressive responding while acutely alcohol intoxicated is not a uniform phenomenon. Furthermore, it suggests a mechanism that may mediate the alcohol-aggression relationship. The results suggest that alcohol seems to impair prefrontal function in a group of subjects with above average cognitive abilities, but that these subjects likely retain sufficient residual functioning to be able to inhibit their aggression. This study demonstrates that simple neurocognitive measures may provide important information regarding the possibility of controlling aggression in alcohol-intoxicated subjects.

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Table 1. Mean (+/- SD) demographics by group.

Demographic Data	Group 1 (Sober /NI) (n = 11)	Group 2 (Sober/I) (n = 11)	Group 3 (Intox/NI) (n = 12)	Group 4 (Intox/I) (n = 11)
Age	20.6 (2.9)	18.8 (0.6)	19.3 (0.7)	19 (1.0)
Years of Education	13.5 (1.0)	13.0 (1.1)	13.6 (0.7)	13.5 (0.7)
Salary Code (SES)	1.6 (1.3)	1.0 (1.0)	1.4 (1.0)	1.3 (0.5)
Beverages Per Week	13.2 (3.1)	7.9 (7.8)	11.4 (6.6)	10.0 (6.9)
Beverages Per Occasion	7.7 (3.1)	5.4 (3.1)	7.5 (2.7)	6.6 (1.5)
Neighbourhood Code	3.3 (0.8)	3.7 (0.8)	3.7 (0.9)	3.4 (0.7)
IQ	116.5 (9.6)	124.9 (12.8)	116.9 (8.3)	122.2 (10.9)
Pain Threshold	102.3 (65.1)	70.5 (62.4)	116.3 (93.1)	92.9 (72.2)

Table 2. Mean (+/- SD) test scores by group.

	Group 1 (Sober/ NI)	Group 2 (Sober/I)	Group 3 (Intox/NI)	Group 4 (Intox/I)
CALT – Total Number of Trials	68.4 (25.9)	69.3 (19.3)	58.4 (22.1)	61.8 (18.0)
CALT – Total Number of Errors	44.1 (28.5)	37.2 (20.5)	26.8 (17.1)	37.9 (22.5)
NSCALT – Total Number of Trials	42.4 (30.5)	55.2 (21.3)	55.8 (16.0)	59.2 (23.0)
NSCALT – Total Number of Errors	18.5 (12.9)	29.5 (16.2)	34.1 (18.0)	35.4 (13.7)
Logical Memory	10.8 (3.4)	10.3 (2.8)	10.2 (3.1)	9.2 (2.5)
Word Fluency	47.2 (8.2)	46.5 (7.9)	41.3 (10.6)	37.5 (6.1)
Paired Associates	7.5 (3.2)	4.6 (2.5)	5.1 (3.4)	5.5 (2.3)

Figures represent means and, in brackets, standard deviations

Table 3. Mean (+/- SD) blood alcohol concentrations per task by group.

	Group1 (Sober/NI)	Group 2 (Sober/I)	Group 3 (Intox/NI)	Group 4 (Intox/I)
BAC NSCALT	0.0 (0.0)	0.0 (0.0)	0.083 (0.011)	0.081 (0.009)
BAC Paired Associates	0.0 (0.0)	0.0 (0.0)	0.084 (0.011)	0.080 (0.008)
BAC Word Fluency	0.0 (0.0)	0.0 (0.0)	0.081 (0.014)	0.080 (0.008)
BAC Taylor Task	0.0 (0.0)	0.0 (0.0)	0.083 (0.012)	0.082 (0.011)

Figures represent means and, in brackets, standard deviations

Figure Caption

Figure 1: Mean difference of number of trials to completion of task, and number of errors made, between the spatial and the non-spatial conditional associative learning tasks, for sober and intoxicated groups. See Results for complete interpretation.

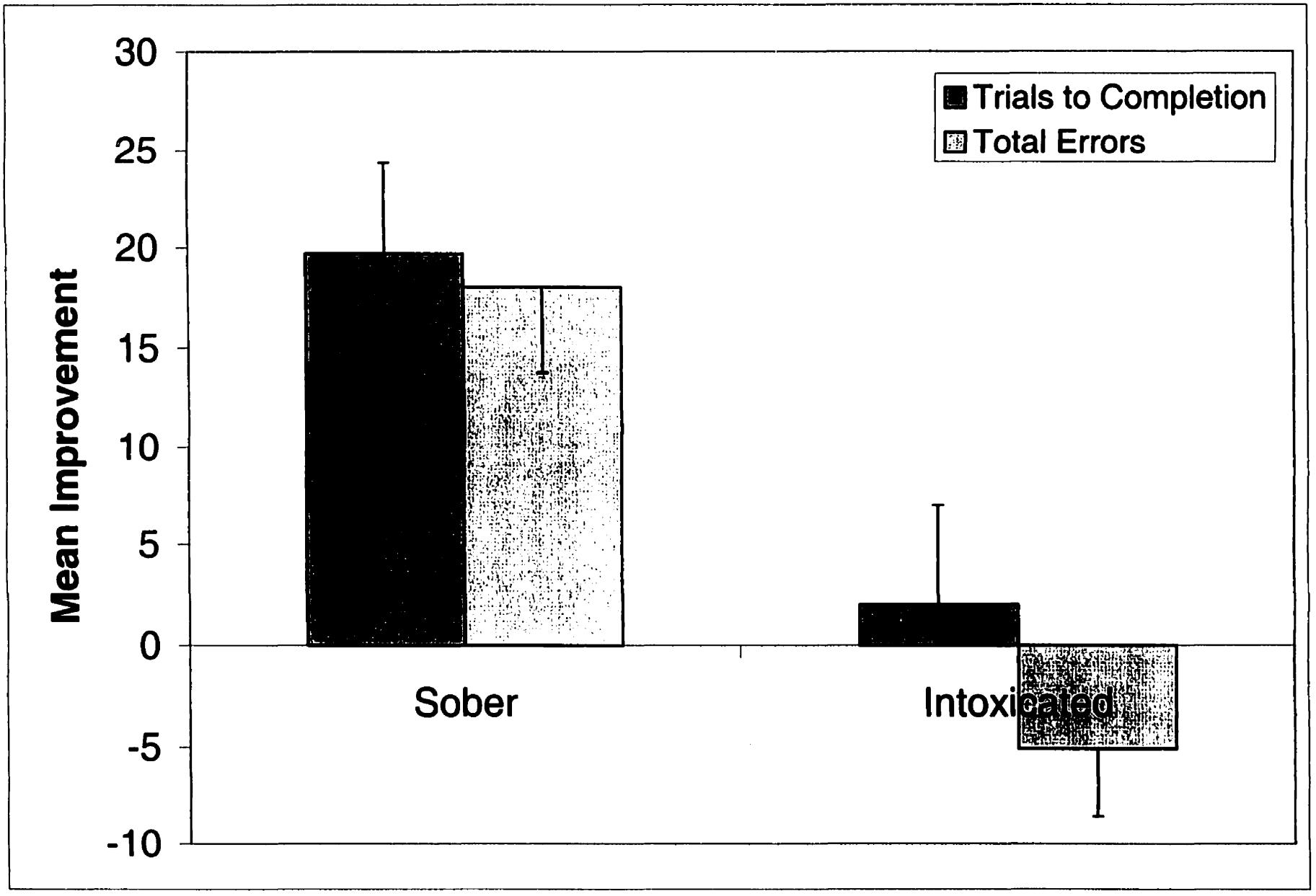
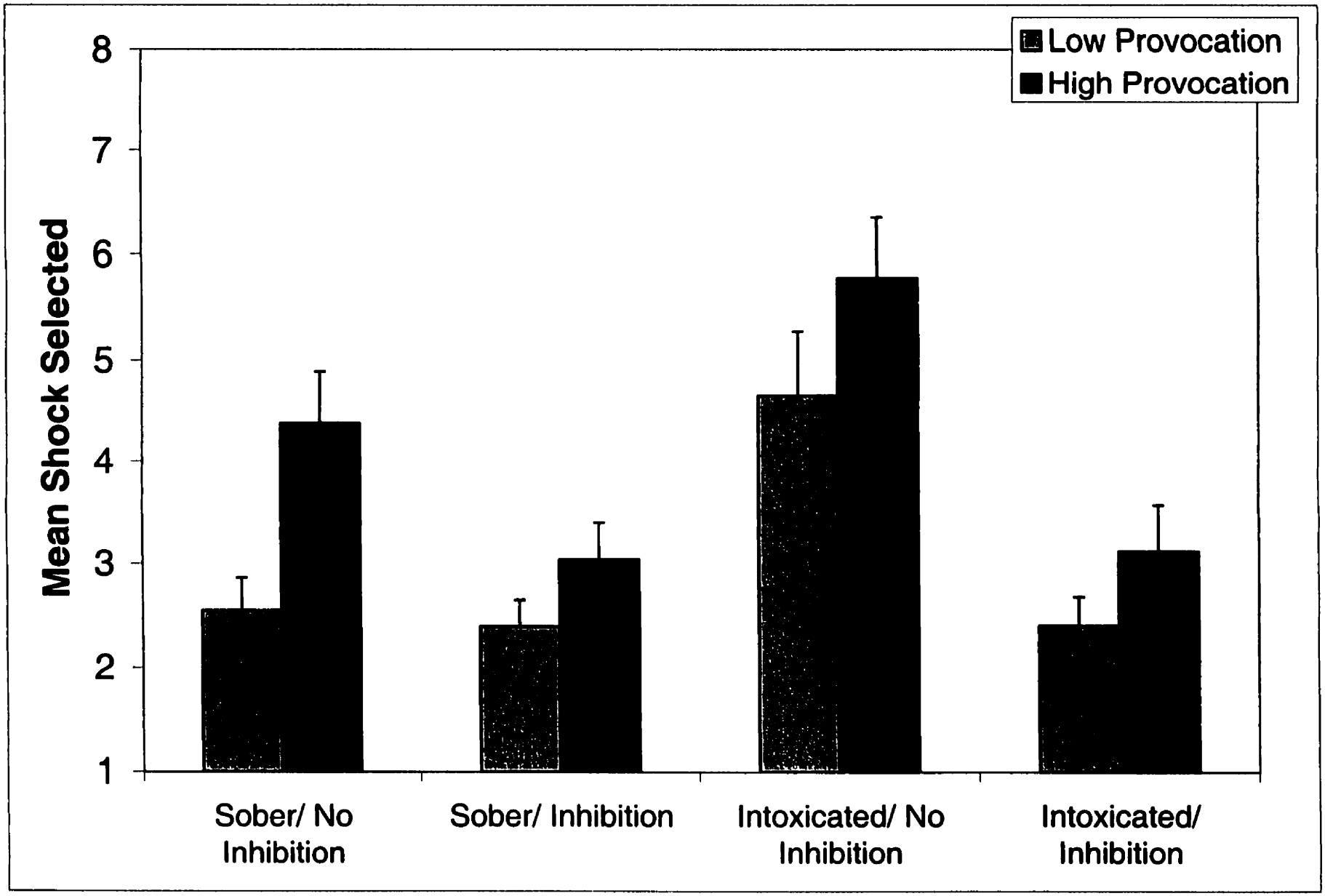

30
25
20
15
10
5
0
-5
-10
Mean Improvement
Trials to Completion
Total Errors
Sober
Intoxicated

Figure Caption

Figure 2: Mean shock intensity chosen, by provocation level, for each of the four groups. See Results for complete interpretation.


Low Provocation
High Provocation
Mean Shock Selected
8
7
6
5
4
3
2
1
Sober/ No Inhibition
Sober/ Inhibition
Intoxicated/ No Inhibition
Intoxicated/ Inhibition

Bridge to Study Two

The first study includes several interesting findings. First of all, the study replicates several previous findings, notably that aggression increases as a function of increased provocation, and that intoxicated males are more aggressive than non-intoxicated peers. Moreover, this study provides a rationale for why it is that although the large number of experimental between-group studies demonstrate unequivocally that alcohol induces heightened aggression, not all (in fact the minority of) individuals act aggressively when intoxicated. This study replicates the necessary condition of provocation, as mentioned, but also suggests the importance of cues which signal inhibition, and more importantly, the ability to heed them.

That is, this paper demonstrates that individuals with above average executive function can overcome the normally aggression-eliciting effects of alcohol, and act as passively as their sober peers, when appropriately motivated. This finding is in direct contrast to the finding of Lau and Pihl (1996) who demonstrated that low-ECF individuals were likely to act aggressively, and were likely to ignore cues which indicated benefits of inhibition, even when non-intoxicated. These two results suggest a continuum of interactive effects of pre-existing cognitive capacity and alcohol intoxication. Very low cognitive performers are likeliest to be aggressive, even when sober; many individuals in the middle of the distribution may become aggressive only when alcohol

sufficiently impairs their executive function; and those at the high end of the distribution are likely to make use of residual cognitive function to inhibit aggressive responses even when acute intoxicated. This study (and pre-existing others) stimulated the hypothesis that interference with executive cognitive functioning might, at least in some individuals, underlie the alcohol-aggression relationship. (Please refer to Appendix C for an integrative review of the literature supporting this theory.)

Study One also initiated an interest in the issue of who does and does not react aggressively in response to alcohol. Consideration of this literature soon led to one of the most consistent differences in the aggression literature – that women are less aggressive than men, even when intoxicated. However, much of this literature was somewhat dated, and interestingly, the more recent the study, the less pronounced the gender difference effect (Hyde, 1984). Moreover, although women commit a significant percentage of violent crimes (U.S. Bureau of Justice Statistics), and although there is recent convincing evidence that they may be as likely as men to act aggressively in certain contexts (Archer, 2000), there is relatively little research on aggression in women. Moreover, whether the aggression-eliciting effects of alcohol demonstrated in men held consistent in women was a greatly under-investigated issue. Thus, Study Two became a two-factorial study, examining men and women, both sober and intoxicated, on the Taylor aggression paradigm.

STUDY TWO

Hoaken, P.N.S. & Pihl, R.O. (2000) The effects of alcohol intoxication on aggressive responses in men and women.

Alcohol and Alcoholism, 35(5), 471-477.

Running Head: GENDER DIFFERENCES IN ALCOHOL-INDUCED AGGRESSION

The Effects of Alcohol-Intoxication on Aggressive Responses in Men and Women

Peter N.S. Hoaken & R.O. Pihl

Department of Psychology

McGill University

Montreal, Quebec, Canada

Keywords: Alcohol, aggression, sex differences.

Correspondence regarding this manuscript should be sent to: Peter N. S. Hoaken, Department of Psychology, 1205 Dr. Penfield Ave., McGill University, Montreal, PQ, Canada, H3A 1B1. Telephone: 514-398-6117. E-mail: hoaken@hebb.psych.mcgill.ca FAX: 514-398-4896

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Abstract

A considerable literature, clinical and experimental, has demonstrated the aggression-eliciting effects of alcohol intoxication. However, the focus of the experimental literature has been primarily on men and the studies of women have been inconclusive. This study was conducted to test for possible gender differences in the manifestation of alcohol-induced aggression. Participants were 54 males and 60 females, aged 18-30, who competed in a competitive aggression paradigm either sober or intoxicated. As expected, intoxicated men were more aggressive than their sober peers. However, under high provocation, both sober and intoxicated, women manifested aggression comparable to the intoxicated men. This study suggests women can be as aggressive as men and that alcohol intoxication does not seem to be as important a determining factor.

The Effects of Alcohol-Intoxication on Aggressive Responses in Men and Women

The antecedents of aggression and violent crime are clearly multifactorial, interactive, and often individualistic (Pihl, Peterson, and Lau, 1993; Giancola and Chermack, 1998). However, that being said, one of the most important situational determinants is alcohol intoxication (Graham, et al, 1998). There is a large, developed, and consistent experimental literature which demonstrates that alcohol intoxication increases likelihood of an aggressive response in men. In fact, several recent meta-analyses based on this literature have concluded unequivocally that alcohol leads to more verbal and physical aggression (Busman and Cooper, 1990; Bushman, 1993; Bushman 1996). There is, however, a relative paucity of research considering the aggression-eliciting effects of alcohol in women, and the existent studies are contradictory. Buss (1971) suggested that the enormous preponderance of studies of aggression on males was as it should be, as aggression was almost exclusively a male problem. One review concluded that "aggressive behavior is clearly sex differentiated by the age of 6" (Maccoby and Jacklin, 1980), and others have concluded that "males, as a group, are always more aggressive than females, as a group, regardless of how the aggression is expressed or measured" (Eron and Huesmann, 1989). These conclusions seem to implicitly suggest that women's aggression is inconsequential.

Recent crime statistics suggest that aggression in women is deserving of study: In the United States in 1996, women constituted 17.9% of all arrests for aggravated assault, 10.3% of all arrests for murder or non-negligent homicide, and 15.1% of all classes of violent crime (Unites States Bureau of Justice Statistics, 1998). Furthermore, some studies have suggested that woman may act aggressively towards their spouses as often as men do and that damage done in terms of level of medical care, days off work, and time spent bedridden may not be significantly different between male and female victims of domestic assault (Straus and Gelles, 1986; Langhinrichsen-Rohling, Neidig, and Thorn, 1995).

Despite the fact that the aggression-eliciting effects of alcohol is relatively clear in men, in women the little existent evidence is conflicting. For example, Giancola and Zeichner (1995a) found that neither blood alcohol concentration nor subjective impression of intoxication could predict aggressive responses in women. Non-published studies by Buss and colleagues (1970) and Ratliffe (1984) also failed to demonstrate alcohol-induced increases in aggression in women (see Gomberg, 1993, for details of these latter studies). Gustafson (1991) found that when offered a choice of an aggressive and non-aggressive response, women were much more inclined to use the non-aggressive response irrespective of alcohol dose. In contrast, Bond and Lader (1986) found that both a light and moderate dose of alcohol increased women's aggression when provoked, and

Rohsenow and Bachorowski (1984) found that a small amount of alcohol elicited an augmentation of women's verbally aggressive responses, but increasing alcohol dose had no further effect. Lastly, a recent study (Dougherty, Cherek, and Bennett, 1996) found that alcohol produced significant increases in women's aggression in response to alcohol on a point-subtraction laboratory measure of aggression.

What appears generally missing from this literature are direct comparisons of men and women, both sober and intoxicated, on measures of aggression. The one well-controlled study (Giancola and Zeichner, 1995b) which did investigate this subject found that intoxicated men are more aggressive than either intoxicated or non-intoxicated women, who did not differ from each other. This study also proposed a distinction between direct (shock intensity) and indirect (shock duration) forms of aggression. The authors found that men were likely to demonstrate both direct and indirect forms of aggression when intoxicated, and women the indirect form. This study utilised a complex design, in which beverage, gender, opponent gender, and provocation were all manipulated in a mixed design, which also lead to the conclusion that it is valid to always use same-sex "opponents" on the competitive aggression paradigm.

In the present study, we again examine both men and women, intoxicated and non-intoxicated, on a well validated laboratory measure of aggression, the competitive aggression task (Taylor, 1967). On the basis of the

accumulated literature, we hypothesize that: 1) both men and women, regardless of beverage type, would demonstrate heightened aggression to provocation; 2) that intoxicated men would be more aggressive than non-intoxicated men; 3) that intoxicated men would be more aggressive than either the intoxicated and non-intoxicated women's groups, which would in turn not differ from each other; 4) that sober men would be more aggressive than sober women; and 5) that women would be more likely to demonstrate aggression in an indirect rather than direct manner.

Method

Non-alcoholic male and female social drinkers, were recruited through local newspapers, and from the advertisement around the McGill University campus. Men and women, aged 18-30, all in good self-reported physical and mental health, served as subjects. Those receiving medical treatment that contraindicated alcohol consumption, who had sustained a serious injury to the head, who had a diagnosed learning disability, or who were familiar with psychological experimentation were excluded from participation. Participants were all administered a brief interview in order to assess current drinking patterns, in order to reasonably ascertain that all were capable of ingesting the experimental dose of alcohol with no adverse consequences. Conversely, all participants were required to score lower than 5 on a short form of the Michigan Alcohol Screening Test (Pokorny, Miller and Kaplan, 1972), in

order to assure none were alcohol-dependant. Subjects were also eliminated if they smoked more than one pack of cigarettes (25 cigarettes) or consumed more than 10 cups of coffee a day, in order to avoid the biases of short-term nicotine and/or caffeine withdrawal. All women were tested between day 5 and day 13 of their menstrual cycle. Testing directly subsequent to the termination of menses was intended not only to provide some control for hormonal fluctuations (Sutker, Goist, and King, 1987), but also to ascertain that the women were not pregnant. Furthermore, all women were required to sign a waiver certifying that they were not pregnant at the time of testing. All subjects were paid $5.00 an hour to compensate for lost time.

Apparatus

Aggression was elicited and assessed with a modified version of the competitive reaction-time task (Taylor, 1967). The goal of the aggression task is to see whether participants who consumed alcohol will respond with reciprocal aggression when provoked, compared to an active placebo or a control group (Lau, Pihl and Peterson, 1995; Gustafson, 1985). In this study the task board consisted of eight buttons, numbered from one to eight. Red lights situated above each button indicated the shock level chosen by the opponent when lit. An IBM compatible personal computer was used to run the aggression task and record data. Shocks were administered via a Mark I Behaviour Modifier (Farrall Instruments, Grand

Islands, NB), connected to an electrode attached to the inner forearm, below the elbow of the non-dominant hand. Each participant monitored administrations of shock to his/her fictitious opponent by viewing a DC ammeter provided for that purpose. A pre-recorded videotape of a same-sex sham opponent receiving instructions regarding performance of the aggression task was played for the participant to reinforce the subject's belief in the existence of the opponent.

Procedure

Testing was conducted over two days. On the first day, a battery of pencil-and-paper and experimenter delivered tests were administered. A short form of the Wechsler Adult Intelligence Scale- Revised (WAIS-R; Wechsler, 1981) including the Information, Block Design, and Vocabulary subtests was administered. Scores from these subtests were used to calculate estimates of full-scale IQ (Brooker & Cyr, 1986). Participants also filled in a questionnaire which asked them for their current income, current occupation, occupation of parents, and years of education. These variables were coded (Blishen, Carroll and Moore, 1987) and summed, and were intended to provide a measure of socioeconomic status. Subsequent to the first day of testing, participants were scheduled for the second day of testing.

Upon arriving at the lab for the second day, the participant's blood alcohol concentration (BAC) was measured to ensure sobriety. BAC was determined using an Alco-sensor III (Intoximeters, Inc., St. Louis, MO). The participant was then randomly assigned to the alcohol or sober condition. In the alcohol condition, the participant was administered 1 millilitre per kilogram of body weight 95% alcohol USP units in three drinks of a 1:7 alcohol:orange juice solution. In the sober condition, three drinks of orange juice of equivalent volume was administered. In each condition, participants were told explicitly what they were drinking. No placebo group was used, due to two considerations: First, expectancies do not appear to play a large role in the alcohol-aggression relationship (Giancola and Zeichner, 1997), and second, because a placebo design would have necessitated an unwieldy design demanding an unrealistic number of participants. Drinks were consumed over a twenty minute period. Another twenty minute waiting period followed to allow the subjects in the alcohol condition time to reach near peak BAC, which was again measured at this time.

Each participant's pain threshold for electric shock was determined by delivering a series of shocks from 0-255 units (0-5.63 ma) increased stepwise by 5 units at a constant rate. Each participant was instructed to press a button in response to any shock he/she regarded as painful (1) to stop the administration of the shock and (2) to reduce the level of the next

shock by one step. The next shock therefore was one step lower than the shock that induced pressing the button. Pressing the button upon three consecutive presentations of the same shock intensity stopped shock delivery. This shock intensity was defined as the participants pain threshold. The pain threshold evaluation procedure was explained in detail prior to beverage administration, but was conducted subsequent to consumption, simply because the experimenters were concerned about the effects of alcohol on pain sensitivity, especially the observation that pain sensitivity may actually increase on the ascending limb of the blood alcohol curve (Gustafson, 1985).

The aggression task was then introduced as a competitive reaction-time task. Each participant was instructed to select a shock level that he/she would deliver to his/her opponent after winning a reaction-time trial. Following each trial, the participant would be informed of the opponent's shock choice. If the participant "lost" that trial, he/she received that shock. Shock levels 1-8 increased from 28 units (sub-threshold but approaching threshold) for level 1 to 100% of the participants given pain threshold for level 8, with intermediate shock levels being equal to 28 units plus 23%, 31%, 39%, 76%, 84%, and 92% of the difference between the participants given pain threshold and the initial 28 unit level. The nature of the increases of the shock intensity was decided upon in order to clearly define those shocks thought to be minimally provoking (level 1-4)

and those thought to be maximally provoking (levels 5-8). If the participant had "won" the reaction time trial, he/she would then administer his/her previously chosen shock to the opponent.

Following these instructions the experimenter then left briefly, telling each participant that he was about to verify the readiness of the opponent. 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. In fact, the participant would watch a pre-recorded videotape of a fictitious opponent receiving instructions. Male subjects always competed against another male, and female subjects always competed against a female.

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 5-8 in the second block of trials. In the transition trial, the "opponent" always chose a shock level of 5, and this trial the subject always "lost"; this was intended to provide some assurance that the transition from low- to high-provocation was not too abrupt. The final trial also always had the "opponent" choosing a 5; this trial the subject always "won". The order of wins and losses as well as the opponent's shock choices were randomly assigned by the computer. However, all subjects "won" six trials at low

provocation and six trials at high provocation; in addition to the transition and final trials, all subjects "won" equally as many trials as they "lost". The opponent's shocks were all of either one-second or two second duration. The "opponent" chose each shock level three times, alternately winning one trial and losing two trials versus winning two trials and losing one trial. If the participant was to receive two shocks at a certain level, he/she would receive one each of one-second and two-second duration. The objective measures of aggression were the mean shock selected for both the high and low provocation conditions, and the shock duration for each of those conditions. It has been suggested that shock intensity is a measure of direct aggression, whereas shock duration is a measure of indirect aggression (Rogers, 1983; Zeichner, Giancola and Allen, 1994).

The Taylor Aggression Paradigm is one of the two most popularly utilised laboratory measures of aggression, and, having now been used for more than thirty years, is often referred to as the classic laboratory measure of aggression. That being said, recent criticism has been levelled at the validity of several aggression paradigms, the Taylor paradigm among them (Tedeschi & Quigley, 1996). However, a subsequent review (Giancola & Chermack), while agreeing with some criticisms of some paradigms, did not agree with the criticisms made of the Taylor paradigm, and argued that the Taylor paradigm has been shown several times to have good construct validity (Giancola & Zeichner, 1995c; Bernstein, Richardson, & Hammock, 1987), that it has demonstrated convergent

validity, in that it has been correlated positively with other measures of aggression, such as the Buss-Durkee Hostility Inventory, and that it has established discriminant validity, in that it has been shown to not correlate with other measures thought to be theoretically unrelated to aggression, such as competition, suspicion, or guilt. It has also been shown to have group discrimination ability; that is, individuals thought to be aggressive by nature appear so on this paradigm, while non-aggressive individuals do not (Giancola & Chermack, 1998).

Following the aggression task, BAC was taken and recorded, participants were interviewed to verify the success of the deception, and debriefed on the purpose of the study and the necessity for deception. All aspects of this study were approved by the McGill University Department of Psychology Ethics Committee, and all subjects provided informed consent prior to any involvement in the experiment.

Results

Subject Measures

A total of 114 participants were deemed admissible to participate in the complete study and were tested on the aggression paradigm. Participants were divided into four groups: Group 1 consisted of males in the sober condition (n=27); Group 2 consisted of males who consumed alcohol (n=27); Group 3 consisted of females in the sober condition

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Insert Table 1 about here

(n=30); and Group 4 consisted of females who consumed alcohol (n=30). Analyses of variance were conducted on all demographic variables to investigate potential differences between the groups; these tests revealed no differences between any of the groups on any of the variables presented in Table 1. The data intended to convey information on socioeconomic status is limited, because most subjects did not provide complete information. In fact, only one of the composite variables (years of education) was completed for even a majority of the participants. The data that was collected was summed and averaged; an analysis of variance indicated no significant differences between the groups.

Blood Alcohol Concentrations

An analysis of simple main effects determined that there were no differences between in blood alcohol concentrations between the two alcohol-consuming groups. Mean BAC's are represented in Table 1.

Shock Intensity Measure

A 2 (gender) X 2 (drug) X 2 (provocation) three-way mixed design analysis of variance was conducted on shock intensity, with provocation as a repeated measure. This analysis revealed a three-way interaction between gender, drug and provocation ($F(1, 110) = 4.06$, $p = .046$).

Further investigation of the nature of the relationship between these variables was possible through analysis of simple main effects. These analyses indicated that for men there was a significant simple main effect of alcohol, producing heightened aggression, in both low provocation ($F(1, 52) = 10.94$, $p = .0017$) and high provocation conditions ($F(1, 52) = 5.54$, $p = .022$). Furthermore, there was a significant simple main

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Insert Figure 1 about here

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effect for sober women to be more aggressive at high provocation than sober men ($F(1, 55) = 5.07$, $p = .028$). In addition, there was a significant simple main effect of provocation in all groups; sober men ($F(1, 26) = 21.81$, $p = .000$), intoxicated men ($F(1, 26) = 16.97$, $p = .000$), sober women ($F(1, 29) = 22.21$, $p = .000$), and intoxicated women ($F(1, 29) = 17.05$, $p = .000$) were all more aggressive in response to high provocation than they had been to low provocation. No other simple main effect was statistically significant; there were no differences between drunk men and drunk women, nor were there any differences between drunk and sober women, in low or high provocation conditions. These results are represented in Figure 1.

Shock Duration Measure

A 2 (gender) X 2 (drug) X 2 (provocation) three-way mixed design analysis of variance was conducted on shock duration, with provocation as a repeated measure. This analysis revealed a three-way interaction between gender, drug and provocation ($F(1, 110) = 5.59$, $p = .020$). Further investigation of the relationship of these variables was again conducted through analysis of simple main effects. These analyses demonstrated a significant simple main effect of alcohol (more aggression) on shock duration in men, for both the low provocation ($F(1, 52) = 4.74$, $p = .034$) and high provocation ($F(1, 52) = 8.75$, $p = .005$) conditions. There were a also simple main effect of gender in the intoxicated subjects, but only at high provocation ($F(1, 55) = 7.41$, $p = .009$). There was

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Insert Figure 2 about here

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a simple main effect for provocation for only one group, the intoxicated men ($F(1, 26) = 5.38$, $p = .029$). There were no other significant simple main effects. These results are represented in Figure 2.

Discussion

The first hypothesis, that aggression would increase as provocation increased, regardless of gender or beverage type, was supported, as was the second hypothesis, that intoxicated men would be more aggressive

than non-intoxicated men. The third hypothesis, that women would be less aggressive than intoxicated men, regardless of alcohol group, and that the sober and intoxicated women would not differ, was only partially supported. That is, although the intoxicated and non-intoxicated women did not differ, the magnitude of their responses were unexpected: Both groups acted with considerable aggression in response to provocation, and did not differ from intoxicated males. "The forth hypothesis, that sober men would be more aggressive than sober women, a hypothesis base primarily on crime statistics, was not supported; in fact, in the high provocation condition the opposite appeared to be the case, with sober women demonstrating significantly higher shock intensities than sober men. The fifth hypothesis, that women would be more likely to demonstrate indirect rather than direct aggression was also not supported; in fact the only group to demonstrate heightened shock duration were the intoxicated males.

This results of this study appear to dispute the conclusion that males are always more aggressive than females "regardless of how the aggression is expressed or measured" (Eron and Huesmann, 1989). The results show that these women manifested considerable direct aggression when highly provoked. What appears of considerable interest is why the women did not appear to react to the alcohol in the same fashion as did the men; that is, with facilitated or heightened aggression. This is an interesting issue

because although women commit fewer violent crimes than men, prevalence rates of alcohol use (not abuse) are not largely discrepant in the two genders, especially in late adolescence and early adulthood, a demographic group which manifests considerable rates of aggressive behaviour (White, Brick & Hansell, 1993).

There are a few studies which may help explain the present finding. More than two decades ago, a review of the then-accumulated literature on gender differences in aggression (Frodi, Macaulay, & Thome, 1977) concluded that women are likely to express considerable aggression when that aggression is perceived as justified. Bettencourt and Miller (1996) concluded some time later that the most important predictor of aggression in women is the form and the intensity of the provocation. Therefore, inconsistencies in the literature may be the result of the various experimental paradigms used, in that some may be more provocative than others. In the present study, for example, the fictitious opponent moves rather abruptly from a series of "low provocation" shocks to considerably more provocative attacks, regardless of the behaviour of the participant. As such, the paradigm is clearly physically provocative and retaliatory aggression may be considered "justified". An additional piece of supportive evidence is that one meta-analytic study of gender differences in aggression concluded that although there were fairly reliable gender differences in aggression, those differences were small, and there was a

trend for gender differences to be smaller the more proximal to the time the analysis was conducted, suggesting a trend of amelioration (Hyde, 1984). In addition, while it is known that women seem far less likely to commit planned acts of extreme aggression such as homicide, impulsive or reactive aggression does not appear to have a corresponding gender difference (Baron and Richardson, 1994). Thus, it may be that the form of the provocation in this study was sufficient to elicit considerable aggression in women, perhaps producing a ceiling effect, rendering the addition of alcohol intoxication as non-relevant.

We might also consider the extent to which alcohol effects are gender non-specific. There do appear to be metabolic differences between the genders which may alter the pharmacodynamics and pharmacokinetics of alcohol in women (Barros and Miczek, 1996). Several researchers have discussed the putative aggression-eliciting pharmacological effects of alcohol in men, including alteration of pain sensitivity, anxiolytic properties, increased psychomotor activation (Pihl and Peterson, 1995); perhaps the assumption that those same effects are experienced by women in the same way is erroneous. This data, along with the accumulated literature, seems to suggest that women's aggression is far more likely to be predicted by the nature of the provocation than by alcohol intoxication. Whereas in men intoxication seems to facilitate aggressive responses to provocation

that would normally not be responded to aggressively, in women this may not be the case. This is a hypothesis which merits further investigation.

This study also appears to draw into question the notion that women, if they are to aggress at all, will do so in an indirect rather than a direct manner. A long-standing assertion in the literature is that women will respond with different types of aggression than men. Lagerspetz, Björkqvist, and Peltonen (1988) showed that girls prefer more indirect means of aggression and also use a verbal variant of direct aggression. Bettencourt and Miller (1996) found that provocation had a greater effect on verbal aggression than on physical aggression in women. Björkqvist, Österman, and Lagerzpetz (1994) found that adolescent girls often use social manipulation, as opposed to direct confrontation, as a preferred method of aggression. The present results are not consistent with these studies, if in fact shock duration is a valid index of indirect aggression. "Shock duration" as it exists here, is essentially only a variant form of the presentation of a physical insult. Inasmuch as that is true, shock duration does vary from most definitions of indirect aggression, which emphasize social manipulation (Giancola and Zeichner, 1995b).

There are some concerns with the present study which should be addressed. First of all, it may be suggested that variants of the aggression task employed may not be valid with women. However, this appears not

to be the case: Gustafson (1986) has demonstrated that this paradigm is in fact valid in alcohol studies in women. It might be argued that the women appeared less familiar with alcohol, and attained slightly higher blood alcohol concentrations that did the men; as such, perhaps these women were too intoxicated to properly interpret the paradigm. This seems unlikely - the BAC's reached by the women in this study were almost exactly identical to those reached in other studies (eg. Giancola and Zeichner, 1995b; Gustafson, 1991), and as such seem wholly appropriate. Another concern might regard the drinking frequencies of the participants; they report rather heavy patterns of drinking, over ten drinks a week for the men. However, it may be that the participants we test provide us with slightly higher means in terms of drinks per week and drinks per occasion than what you would find in the normal population simply because we sometimes have to eliminate subjects who don't drink enough for our purposes. Because we give an intoxicating dose of alcohol, and ask participants to consume it relatively rapidly, we have to eliminate participants for whom this dose would potentially render them ill, or otherwise unable to continue participation. None of these participants. Finally, one might question the extent to which these results would generalise. It should be noted that although we do not claim to have a perfectly representative sample, participants were roughly equally sampled from university undergraduates and from an ad in a local paper, and as

such do not appear to be homogenous in terms of IQ, years of education, or socio-economic status.

In conclusion, this study provides new and provocative evidence that woman may act as aggressively as men on a laboratory measure of aggression, and furthermore, that this behaviour is not dependent upon, or influenced by, alcohol intoxication. This is a surprising result, based on the accumulated literature. However, considering recent crime data that suggests that women perpetrate a not insignificant percentage of violent crimes, it is a result that suggests further investigation.

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Table 1. Demographic data for all groups. Values are means +/- SD; IQ, intelligence quotient; BAC, blood-alcohol concentration.

	Men (Sober)	Men (Intoxicated)	Women (Sober)	Women (Intoxicated)
Age	19.55 (1.62)	21.66 (3.50)	21.60 (2.82)	21.70 (3.44)
IQ	100.72 (11.88)	108.63 (10.24)	109.64 (9.73)	107.07 (17.54)
BAC	.000 (.000)	.086 (.013)	.000 (.000)	.092 (.014)
Drinks per Occasion	6.16 (3.22)	6.19 (2.32)	4.57 (2.63)	5.70 (1.35)
Drinks per Week	10.54 (8.41)	10.25 (6.72)	8.07 (7.17)	7.64 (5.32)

Values are means +/- SD; IQ, intelligence quotient; BAC, blood-alcohol concentration.

Figure Caption

Figure 1. Mean shock intensity selected by group, in low and high provocation conditions.

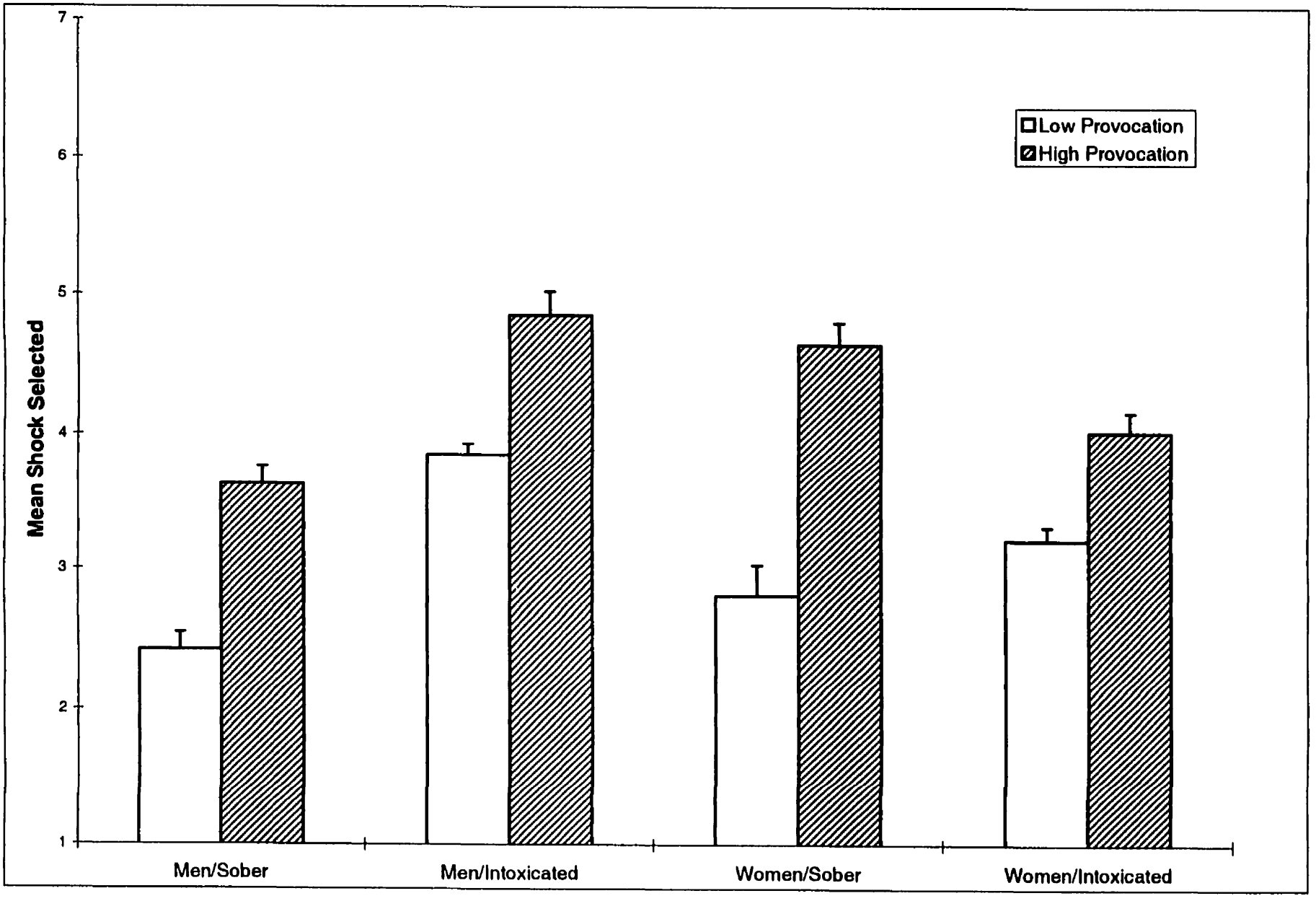
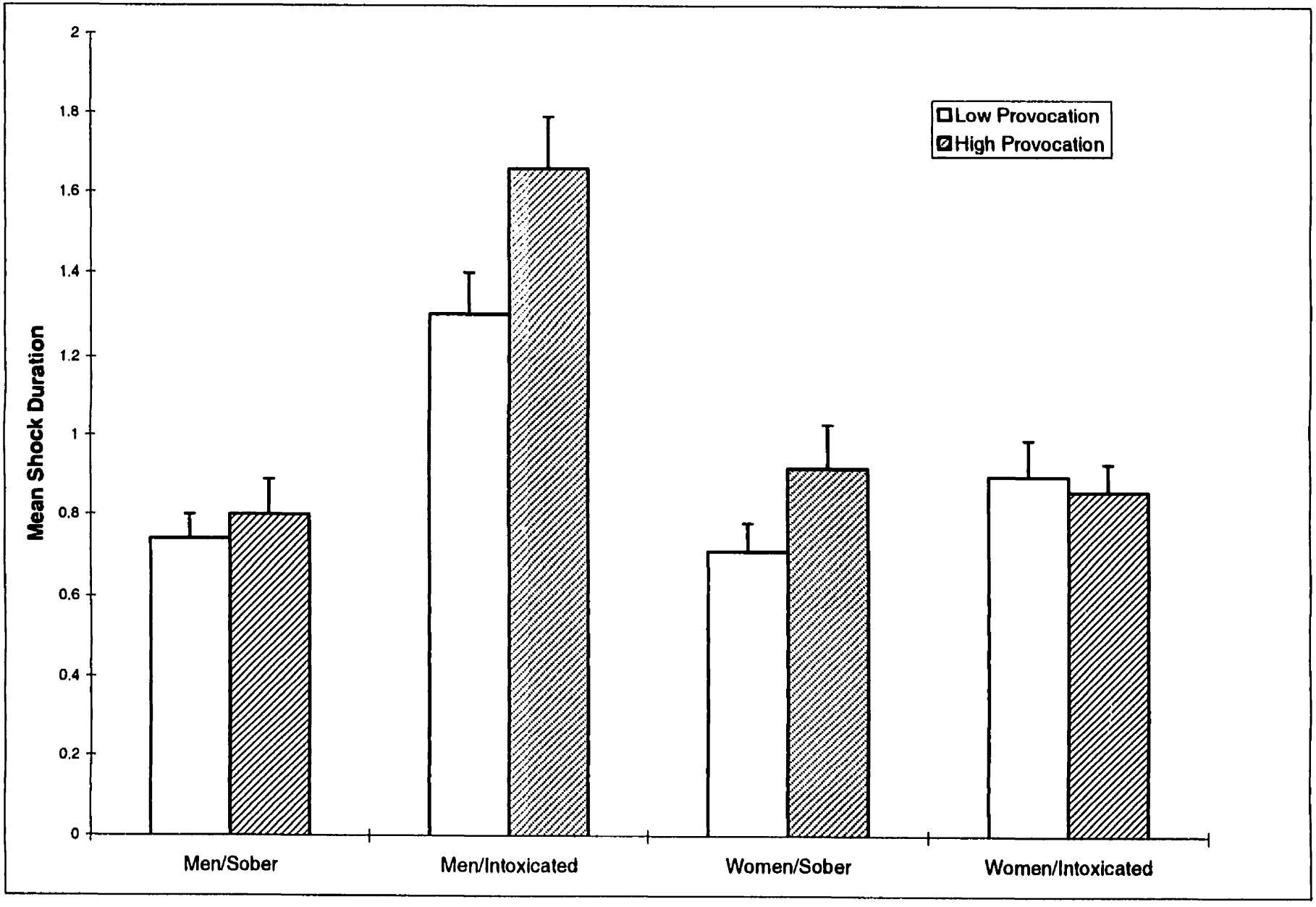

Mean Shock Selected
7
6
5
4
3
2
1
Low Provocation
High Provocation
Men/Sober
Men/Intoxicated
Women/Sober
Women/Intoxicated

Figure Caption

Figure 2. Mean shock duration selected by group, in low and high provocation conditions.


Mean Shock Duration
2
1.8
1.6
1.4
1.2
1
0.8
0.6
0.4
0.2
0
Low Provocation
High Provocation
Men/Sober
Men/Intoxicated
Women/Sober
Women/Intoxicated

Bridge to Study Three

The second study was interesting inasmuch as it did not correspond to many assumptions about the frequency and intensity of aggressive responses in women. Although, as noted, many researchers had suggested that women would always act less aggressively than men, in all contexts, this assertion did not prove to be the case. This study corresponds with a developing literature that suggests that with the right provocation, and in the right context, women are as likely as men to become aggressive, even physically aggressive (Archer, 2000).

What was also interesting was that the aggression-eliciting properties of alcohol did not appear to be consistent between men and women. That is, although there was a significant alcohol effect in men, with intoxicated men manifesting much more aggression than their non-intoxicated peers, this relationship is not in evidence in women. Women appear to manifest considerable aggression, when highly provoked, whether intoxicated or not. This is a curious finding, and we were at a loss as to explain it.

At the same time these aggression studies were being conducted, the lab was also collecting normative data on a computerised battery of executive functioning tasks. The majority of studies that had been conducted recently in the lab had all used men, and so there was a considerable database on norms for men. However, there was little normative data for women. As a result, we ran all women from this study

on the computerised battery, simply for the norms. This proved to be a fateful decision, in two ways. We realised later that we had executive function data for all the women, which would allow us to conduct post-hoc analyses to putatively explain the women's aggression. However, we also realised that we had no corresponding data for the men, which precluded a full and more comprehensive analysis.

Study three was intended to use the executive function data in a post-hoc analysis so as to understand the relationship between pre-existing levels of ECF, alcohol and aggression in these women. That is, we were interested in examining whether scores of executive function attained from the women in the Hoaken & Pihl (2000) study were related to magnitude of aggressive response, either in an interaction with alcohol, or by themselves.

STUDY THREE

Hoaken, P.N.S., Strickler, W. L. A., & Pihl, R.O. Does Executive Cognitive Function Mediate the Relationship Between Alcohol Intoxication and Aggression in Women? Manuscript under review, Aggressive Behavior.

Does Executive Cognitive Function Mediate the Relationship Between Alcohol Intoxication and Aggression in Women?

Peter N.S. Hoaken[1], Wendy L.A. Strickler[2], & R.O. Pihl[3]

[1]Department of Psychology, McGill University; [2]Department of Psychology, Department of School Psychology, University of Cincinnati. [3] Departments of Psychology and Psychiatry, McGill University.

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Corresponding Author: Peter N. S. Hoaken, Department of Psychology, 1205 Dr. Penfield Ave, McGill University, Montreal, PQ, Canada, H3A 1B1. Electronic mail may be sent via Internet to *hoaken@hebb.psych.mcgill.ca*

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Abstract

Objective: To examine both alcohol intoxication and executive function in as much as they might predict magnitude of aggressive response in women. Background: Two well-replicated findings are that alcohol intoxication and level of Executive Cognitive Function (ECF) are strongly related to aggressive responding in males. Studies conducted with women, however, are both inconsistent and rare. One recent study (Hoaken & Pihl, 2000) indicated that alcohol intoxication is less predictive of aggression in women than it is in men. The current study, a post-hoc analysis of that data set, is intended to investigate whether ECF independently or interactionally alters the intensity of women's aggressive response. Methods: Sixty women participants completed neurocognitive measures, intelligence and demographic measures, and were subsequently tested on the Taylor Aggression Paradigm, half sober, the other half acutely alcohol intoxicated. It was hypothesized that, like in men, intoxicated women would manifest greater aggression than their non-intoxicated peers. Results: The hypothesis that alcohol would increase magnitude of women's aggression was not supported. However, a series of post-hoc correlations revealed that women's aggression was significantly related to scores on tests of ECF. Discussion: This suggests that trait factors (ECF) are more important than state factors (intoxication) when predicting aggression in women. Furthermore, this helps to explain why the accumulated literature on the aggression-eliciting effects of alcohol in women is so contradictory.

Introduction

There is no question that interpersonal violence is a societal problem of considerable importance. In the United States alone, close to three million individuals are victims of violent crime each year (U.S. Bureau of Justice Statistics, 1997). Because men perpetrate the vast majority of violent crimes, it is not surprising that the research on aggression has been conducted almost exclusively with males. The theme of this male limited literature is that the mechanisms underlying aggression are multifactorial and likely interactional (Raine, 1993; Pihl & Peterson, 1995). Empirically, factors that have been implicated as predispositional include personality characteristics, cognitive abilities, and situational variables such as level of provocation, and drug or alcohol intoxication (Gustafson, 1991; Lau, Pihl, & Peterson, 1995).

Despite the empirical emphasis on men, and contrary to the widely held belief that female-perpetrated violent crimes are negligible, crime statistics suggest that aggression in women is not uncommon. In the United States in 1996, women constituted 17.9% of all arrests for aggravated assault, 10.3% of all arrests for murder or non-negligent homicide, and 15.1% of all classes of violent crime (U.S. Bureau of Justice Statistics, 1997). What is apparent is that aggression in women is not as rare as some may believe, and, for whatever reasons, some women are capable of extreme aggression. Therefore, to examine the

phenomenon of aggression in women appears consequential, with the costs of these offenses personally and societally significant.

The existent literature on aggression in women tends to focus on gender differences in aggression, showing that men are more aggressive as a group than women. The first extensive review of this literature (Maccoby & Jacklin, 1974) concluded that gender differences in aggression are considerable, that they exist in all cultures for which data is available, and that the difference is observable as early as age 2. Frodi, Macaulay, and Thome (1977), in a narrative review of studies of gender differences in adult aggression, concluded that although there were overall gender differences in aggression, the differences were far from consistent, with women as likely as men to aggress in certain situations. A meta-analytic investigation (Eagly & Steffen, 1986) concluded, similarly, that men were more aggressive on average, but that this difference was inconsistent. The authors also concluded that women were more likely to engage in psychological rather than physical aggression, were more likely to perceive that a behavior would lead to harm to a target, and were more likely to feel anxiety, guilt and fear as a result. They suggested that gender differences were aggression are likely a function of perceived consequences of aggression, which is learned as an aspect of gender roles.

Variables most commonly discussed as determinants of aggression in men are not well researched in women, or, if studied, remain somewhat equivocal. For example, a factor clearly implicated in acts of violent aggression perpetrated by men is alcohol intoxication (Pihl & Peterson,

1995). Several recent meta-analytic reviews of experimental studies conducted with men have demonstrated that alcohol intoxication is associated with increased aggression and that intoxicated individuals are both more verbally and physically aggressive than sober individuals (Bushman & Cooper, 1990; Bushman, 1996). A small number of studies exist with women but the results are highly contradictory. Giancola and Zeichner (1995a) found that neither blood alcohol concentration nor subjective impression of intoxication could predict aggressive responses in women. These authors, in a second study (Giancola & Zeichner, 1995b), found that intoxicated men were more aggressive than either sober and intoxicated women, who did not differ from each other. Gomberg (1993) reports two non-published studies, one by Buss and colleagues (1970), the other by Ratliff (1984) which also failed to demonstrate a heightening of aggression in alcohol-intoxicated women. In comparison, Bond and Lader (1986) found that both a light and moderate dose of alcohol increased women's aggression when provoked, and Rohsenow and Bachorowski (1984) found that a small amount of alcohol did elicit an increase in women's verbal aggressive responses, but increasing alcohol dose had no further effect. More recently, using a point-subtraction measure of aggression, Dougherty, Cherek and Bennett (1996) demonstrated significant increases in aggression in intoxicated women.

A more recent study, which was a direct comparison of men and women and the aggression-eliciting effects of alcohol, suggested that alcohol is a strong determinant of aggressive behavior in men, but not so

in women (Hoaken & Pihl, 2000). More specifically, testing aggression on a modified version of the Taylor (1967) aggression paradigm with all participants responding to a same-sex opponent, intoxicated men were much more aggressive than non-intoxicated men, but intoxicated women were not more aggressive than non-intoxicated women. However, that is not to say that the women in this study were all non-aggressive; in fact, both sober and intoxicated women demonstrated aggression comparable to the intoxicated men.

This result raises the important question of why alcohol had such a differential effect. There are theoretical mechanisms through which we believe alcohol induces aggressive behavior. Pihl, Peterson, and Lau (1993) have suggested four pharmacological effects that may influence the likelihood of aggressive responding; anxiolytic, stimulant, analgesic, and as a disrupter of so-called executive cognitive functioning. This last construct is typically conceptualized as a collection of cognitive abilities including strategy formulation, cognitive flexibility, and abstract reasoning, which have been collectively described as "the ability to organize a behavioral response to solve a complex problem" (Mega & Cummings, 1994).

Several studies have implicated the role of executive functioning in the regulation of aggressive behavior, in sober samples of both boys (Séguin, Pihl, Harden, Tremblay, & Boulerice, 1995) and men (Lau & Pihl, 1996; Hoaken, Assaad, & Pihl, 1998). Other studies (Peterson, Rothfleisch, Zelazo, & Pihl, 1990) have shown how intoxicating dosages

of alcohol impairs executive function, which may therefore be responsible for the increased aggression.

The current manuscript is intended to further investigate aggression in women, focusing on the contributing roles of alcohol intoxication and cognitive function. It intends to do this by subjecting the database from the aforementioned Hoaken and Pihl (2000) study to some post-hoc analyses. That is, we intend to examine whether scores of executive function attained from the women in that study are related to magnitude of aggressive response, either interactionally with alcohol or alone.

The comparison of intoxicated to non-intoxicated women reported below is repeated from the original reference. However, all subsequent analyses and interpretations are new. Consistent with the literature regarding alcohol, executive function and aggression developed with men, it was hypothesized that the executive cognitive function variables would be related to aggressive response in both intoxicated and non-intoxicated women.

Methods

Participants

Participants from the original study included 114 adult men and women. Participants included in the post-hoc analysis are 60 women ranging in age from 18 to 30. They were recruited from advertisements in local Montreal newspapers and the university undergraduate community.

Potential participants were excluded if they were thought to be alcohol or drug abusers, as assessed by a score of eight or greater on the brief version of the Michigan Alcoholism Screening Test (Pokorny, Miller, & Kaplan, 1972), and a brief interview about alcohol and drug use. Participants were also eliminated if they were not regular consumers of alcohol, had sustained a serious injury to the head, were familiar with psychological experimentation, were pregnant, and or deemed to not have been completely deceived by the Taylor Aggression Paradigm. All women were tested between days five and thirteen of their menstrual cycle in order to attempt to control for the effects of hormonal fluctuations (Sutker, Goist, & King, 1987).

Measures

A) Neurocognitive Measures: The Spatial Conditional Associative-Learning Task (SCALT; Petrides, 1985a) was performed on a computer which monitors and records the participant's matches and errors. The task consists of the presentation of six circular lights and six white rectangles presented on the screen. The lights are randomly lit one at a time, and the participant must point, using a mouse, to each rectangle until she correctly clicks on the specific card paired with that light. The participant's task is to learn and then remember the association between the circles and the rectangles such that when a light is lit, the participant is able to choose the correct rectangle. The task is repeated until the participant matches the lights and rectangles correctly on 18 consecutive trials.

The Non-Spatial Associative-Learning Task (NSCALT; Petrides, 1985b) consists of the same set-up as the SCALT; however, the six lights and rectangles are replaced by six colored squares and six semi-concrete pictures (e.g. looks like an abstract flower).

In the Self-Ordered Pointing Task (SOP) concrete version (Petrides and Milner, 1982), participants are presented with 12 familiar pictures (e.g. an umbrella, eyeglasses) arranged in a 3x4 matrix, again on a computer screen. The designs are the same on each of 12 consecutive screens; however, the position of the designs is randomly altered. The participant points to a picture, then the screen changes to the next one. The participant's task is to point to a different picture on each screen, without pointing to the same picture twice. Furthermore, the participant is not allowed to point to the same relative position more than twice in a row. Therefore, the participant must choose one of the twelve pictures for each of the twelve screens. The SOP abstract version is the same set-up as the concrete version; however, the twelve images are abstract black and white prints, which cannot be verbally identified (i.e. they cannot be named). Again, these tasks were administered, recorded and scored by computer.

Positron emission tomography scans have shown in the non-computerized version that these four tasks selectively activate dorsolateral prefrontal cortex (Petrides, Alivisatos, Evans, & Meyer, 1993a; Petrides, Alivisatos, Meyer & Evans, 1993b), an area distinctly associated with executive functioning (Cummings, 1994).

Executive function measures were, at this time, being collected from large numbers of participants in order to create normative data of the tests in question. Thus, it is simply a case of good fortune that this data exists for the 60 women who were included in the Hoaken & Pihl (2000) aggression study. However, this data was not collected from the men; sufficient normative data was deemed to have already been collected from men, and so in the absence of foresight, only the women from the Hoaken & Pihl (2000) study can be included in the post-hoc analysis to examine influence of executive function.

B) Intelligence Measures: A short form of the Weschler Adult Intelligence Scale-Revised (WAIS-R), including the Information, Block Design, and Vocabulary subtests was administered to all participants. Scores from these subtests were used to calculate estimates of full-scale IQ (Brooker and Cyr, 1986).

C) Aggression Measure: The Taylor (1967) Aggression Paradigm was used to elicit and assess aggression. The paradigm was presented to the participant as a competitive reaction time task in which she would compete against an opponent in an adjoining room. The opponent was actually fictional and all shocks delivered to the participant were predetermined by the experimenters. The participant was seated in front of a console consisting of a panel with eight buttons (numbered 1-8) indicating the eight shock levels. The participant used these buttons to choose the shock intensity to administer to her (mock) opponent. The Taylor Aggression Paradigm is one of the two most popularly utilized

laboratory measures of aggression, and has established construct validity, convergent validity, and discriminant validity (Giancola & Chermack, 1998).

Procedure

Women who met inclusion criteria were asked not to consume drugs or alcohol for 24 hours prior to testing. Upon arrival, participants signed an informed consent form. During the first hours of the experiment, participants completed a battery of tests and questionnaires including the neurocognitive tasks, IQ tasks, and demographic information questionnaires. During the second half of the session, participants competed in the Taylor Aggression paradigm. The aggression paradigm was presented to participants as "An Investigation into the Effects of Alcohol Intoxication on Reaction Time" on the consent form. Participants were then randomly assigned to an alcohol or sober condition, thirty to the alcohol condition, thirty to the sober condition, and were given the appropriate drink. In the alcohol condition, participants were given 1.00ml alcohol/kg bodyweight 95% ethanol mixed with orange juice in a 7:1 orange juice:alcohol ratio. Participants in the control condition were given an equivalent amount of plain orange juice. In each condition, participants were told explicitly what they were drinking. In both groups, participants were instructed to consume the beverage at a consistent rate finishing at the end of a fifteen-minute consumption period. Anothdr twenty minute was allowed for absorption before continuation.

The Taylor Aggression Paradigm was then introduced. Following the trial, the participant was informed of the opponent's shock choice (as indicated by whichever light lit up above the buttons), and then whether she had won or lost the trial. If she won the trial, she administered her chosen shock to her opponent; if she lost, she received the shock ostensibly chosen by her opponent. Due to previous research (Giancola & Zeichner, 1995b) which suggests that women act equally aggressively towards men and women opponents on this paradigm, gender of the opponent was not manipulated.

The task consisted of 26 trials. The first 12 were low provocation trials in which the participant always received lower shock levels (1-4) from the "opponent". After a transition trial, the following 12 trials were high provocation trials in which the "opponent" always chose high shock levels (5-8) to administer to the participant.

The computer randomly generated which shocks were chosen (by the opponent) within each range as well as the order of wins and losses. However, total number of wins and losses was equal for each participant and the opponent's shocks were always of the same duration. The objective measures of aggression were the first intensity chosen which, because it is selected before the participant has any information about opponent intention, is considered unprovoked aggression, and also the mean intensity of shocks selected for each provocation level. All shocks administered were below a "pain threshold" previously established by the participant.

Following the task, participants were interviewed to verify the success of the deception. Participants who were deemed to have entertained any doubts about the stated purposes of the experimentation (i.e. the cover story) were eliminated. Participants rated their own and their opponent's performance, as well as rating how effective they felt the task was at measuring reaction time. Participants were also asked whether they found anything about the task odd or confusing.

Lastly, participants were given an extensive and sensitive debriefing in which we explained the study, and explained the necessity for that deception. Participants in the alcohol condition were retained in the laboratory until their blood alcohol level dropped below .02.

Results

Demographic and Sober Neurocognitive Data

A total of 86 women were deemed acceptable after phone screening and were brought into the lab to complete the full session of testing. Of these, 6 were eliminated after an interview regarding drinking practices (either because they drank too much, or not enough to be safely included in the protocol). One woman was eliminated due to familiarity with similar psychological experimentation. Twelve women were eliminated subsequent to an interview with a graduate student in clinical psychology due to suspicion of depression. A total of 67 women were tested on the aggression paradigm; of those 1 was eliminated due to a mechanical problem, and 6 were deemed not to have been completely

deceived. Of the 6 women who were not completely deceived, 4 were from the intoxicated group, and 2 were from the sober group.

There was no difference between the participants in the non-alcohol and alcohol conditions on any of the demographic variables, including age, IQ, and socioeconomic status. Participants also did not differ on pain threshold evaluation between the two groups (see Table I).

Insert Table I about here

In the alcohol condition, the mean blood alcohol concentration directly subsequent to the presentation of the Taylor Aggression Paradigm was 0.0784 (s.d.=0.010).

The four tests of executive functioning did not differ between the groups. Moreover, the scores were very similar to those attained in other studies in our lab, and as such appear to be representative of the abilities of the general population. Means and standard deviations of all tests are presented for the two groups appear in Table I.

Aggression Measures

A one-way analysis of variance (ANOVA) revealed no difference between the intoxicated and sober women on intensity of the first shock selected, a measure often thought to indicate unprovoked aggression ($F(1,58) = 1.37$, $p=.246$).

A two-way mixed design ANOVA was conducted to assess the effects of alcohol on aggression, the measure of aggression being mean shock intensity, using provocation block as a repeated measure. A significant provocation x alcohol interaction was found ($F(1,58) = 15.75$, $p<.001$). There was a significant main effect of provocation ($F(1,58) = 104.96$, $p<.001$), but no main effect of alcohol. Analysis of simple main effects indicates

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Insert Figure I about here

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an effect of provocation in both the intoxicated ($F(1,29) = 17.05$, $p<.001$) and the non-intoxicated women ($F(1,29) = 119.59$, $p<.001$). However, there appears to be no effect of alcohol in either the low provocation ($F(1,58) = 1.75$, ns) or the high provocation ($F(1,58) = 2.28$, ns) conditions. These results are represented in Figure I.

Relationship Between Aggression and Neurocognitive Variables

Table II presents correlations between tests of ECF and aggression. Only the concrete version of the SOP was significantly correlated with shock intensity at both low and high provocation. No other

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Insert Table II about here

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correlation was significant at the Bonferroni corrected significance level of p=.006. The abstract version of the SOP also appeared to be moderately related to aggression.

Given the apparent relationship between at least some of the measures of executive cognitive function and aggression, it was of interest to examine whether there was an interaction between baseline cognitive abilities, alcohol intoxication, and aggression. A three-way mixed design ANOVA was conducted, with alcohol as a between-participants variable, provocation as a repeated measure, and executive function also entered. The ECF factor was created by standardizing and combining the four putative tests of executive function, and then splitting participants into higher and lower halves based on this calculation. This standardization and combination was conducted due to the considerable inter-correlation between these four measures. Before this overall analysis was conducted, however, it was examined how randomly the participants had been distributed to the two alcohol conditions. An ANOVA revealed no difference between the groups on the combined z-score which represented the baseline measures of cognitive ability ($\underline{F}(1,58) = 1.88$, $\underline{ns}$.). Thus, it is clear that the results are not due to an accidental random assignment of more low-ECF women to the sober condition than to the alcohol condition.

A two-way ANOVA was conducted on initial shock choice, with alcohol and cognitive function split as factors. There was no significant interaction between alcohol and cognitive function ($\underline{F}(1,56) = 0.74$, $\underline{ns}$.),

nor was there a main effect for either alcohol ($F(1,56) = 1.37$, ns.).or cognitive function ($F(1,56) = 1.37$, ns.) alone.

A three-way ANOVA on shock intensity, with alcohol and cognitive function split as between-subjects factors and provocation as a repeated measure was conducted. This analysis revealed no significant three-way interactions. There was one significant two-way interaction, between provocation and alcohol ($F(1,56) = 5.99$, $p<.05$). No main effect of alcohol was indicated, but a main effect of cognitive function was found ($F(1,56) = 8.84$, $p<.01$), as was a significant main effect of provocation ($F(1,56) = 54.06$, $p<.001$).

Planned analysis of simple main effects of the three-way interaction was undertaken to better elucidate the nature of the relationship between the variables. The influence of the split in scores representing executive function proved to be quite interesting: There was a simple main effect of cognitive function in the intoxicated groups at high provocation, with participants with poor cognitive function selecting significantly higher shocks than their peers ($F(1,28) = 6.36$, $p<.05$). This simple main effect existed as a trend at low provocation, as well ($F(1,28) = 4.09$, $p<.10$) with participants with poorer cognitive function again selecting higher shock intensities. Furthermore, in sober groups at high provocation, there was again a simple main effect of cognitive function, again in the same direction ($F(1,28) = 6.61$, $p<.05$). At low

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Insert Figure II about here

provocation in the sober participants, this relationship approached but did not attain significance at the p=.10 level. There were no simple main effects of alcohol.

These results suggest that alcohol does not appear to interact with these pre-existing abilities to produce aggression beyond the effect of the cognitive factor. This is represented in Figure II.

Discussion

As was previously reported, the women in this study seemed to become notably aggressive, more so than was expected in light of the literature. Unexpectedly, they responded in an aggressive fashion when highly provoked, whether drunk or sober. That the non-intoxicated women reacted as aggressively as they did, at a level comparable to if not slightly higher than the intoxicated women, was noteworthy.

The post-hoc analyses provide considerable insight in terms of explaining why this was the case. Neurocognitive variables thought to represent executive cognitive functioning proved to be related to propensity for aggression in both the sober and drug conditions, and, to a lesser extent, in both high and low provocation conditions. Tests of these sorts of cognitive capacities have been found to be predictive of aggression in men (Lau, et al, 1995; Hoaken et al; 1998), and it certainly appears from these analyses that the same is true for women. However, where researchers surmise a possible interactive effect between executive

function and aggression in men, there is no evidence of such a relationship in these women.

What remains in question, however, is how these cognitive deficits lead, either directly or indirectly, to increased aggression. One possibility is that individuals who score poorly on these tests are more behaviorally impulsive. The dorsolateral prefrontal cortex, in collaboration with subcortical structures, appears to be responsible for organizing strategies, monitoring and/or sequencing of response, and understanding consequences of actions (Bechara, Damasio, Damasio, & Anderson, 1994). When confronted with a provocative situation, an individual must organize strategies on how to appropriately respond and then monitor responses and continue to strategize. Impairment of these abilities could easily result in a response of a more reflexive, impulsive manner, likely retaliatory, and of similar intensity to the provocation. As suggested by Eagly and Steffen (1986), it may be that women with poor executive functioning are unable to access the learned inhibitory gender-role behaviors when provoked, and as a result do not experience the anxiety, guilt and fear, the result of which is an impulsive aggressive response to the provocation. Another related possible explanation for the relationship between poor executive function and aggression is that in women with poor ECF there is a form of emotional/affective/physiological flattening such that the information-processing problem inherent in the aggression paradigm is non fear- or anxiety-eliciting. In a sense these women with

poor cognitive functions reflect "predispositional low arousability" (Newman & Wallace, 1993).

The finding that poor executive functioning appeared to also predict aggression in response to "low" provocation may suggest that for women with poor executive function provocation is perceived differently. It may be that these women are more likely to perceive a situation as provocative, and are more likely to act aggressively in response to minimally provocative acts. This suspicion is supported by the fact that high cognitive-functioning women reacted with shock intensities almost exactly like those chosen by the "opponent" in the low-provocation condition, while the women with poor functioning selected shock intensities which were higher than their opponent.

It is of course possible that the women in this study were as aggressive as they were because of some sampling bias. For example it could be that the results were due to a chance over-representation of women with poor cognitive functioning. However, this does not seem to be the case as when scores on the measures of executive functioning of the women in this study were compared against a larger database, the mean scores of both the intoxicated and sober groups were not significantly different from the larger overall sample mean.

This study suggests that alcohol intoxication may not have the same, clear aggression-eliciting effects in women that it does in men. But more importantly, it indicates that the trait factor of executive cognitive function is likely more predictive of propensity of responsive aggression

than the state factor of alcohol intoxication. This further analysis of Hoaken & Pihl (2000) is consequential inasmuch as it helps to explain why the accumulated literature on the aggression-eliciting effects of alcohol in women has long been so contradictory and enigmatic.

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Table I. Means and standard deviations for all demographic and other variables, for both the intoxicated and non-intoxicated groups.

Variable	No Alcohol	Alcohol
Age	21.8 (2.98)	21.7 (3.44
IQ	104.64 (9.73)	102.06 (17.50)
Socioeconomic Status	3.96 (0.83)	3.75 (1.06)
Drinks per Week	8.07 (4.17)	8.64 (5.32)
Drinks per Occasion	4.57 (2.63)	3.97 (1.35)
Cigarettes per Day	3.25 (2.53)	4.46 (6.03)
Brief MAST	2.85 (0.89)	2.59 (0.65)
BDI	5.56 (4.67)	5.40 (4.30)
Pain Threshold	61.00 (19.45)	65.56 (39.67)
Concrete Self-Ordered Pointing (errors)	2.60 (2.06)	3.33 (2.07)
Abstract Self-Ordered Pointing (errors)	4.43 (3.67)	5.46 (2.60)
Spatial CALT (trials to completion)	109.43 (59.44)	119.68 (54.92/
Non-Spatial CALT (trials to completion)	59.75 (43.77)	55.22 (47.38)

Table II. Correlations between neuropsychological test scores and aggression, at both low and high provocation.

Variable	Low Provocation	High Provocation
Concrete Self-Ordered Pointing	.39 (p=.002)	.43 (p=.001)
Abstract Self-Ordered Pointing	.27 (p=.038)	.29 (p=.023)
Spatial CALT	.14 p=.28	.14 (p=.28)
Non-Spatial CALT	.13 (p=.34)	.10 (p=.43)

Figure Caption

Figure I. Mean shock selected by alcohol group, for both low and high provocation conditions.

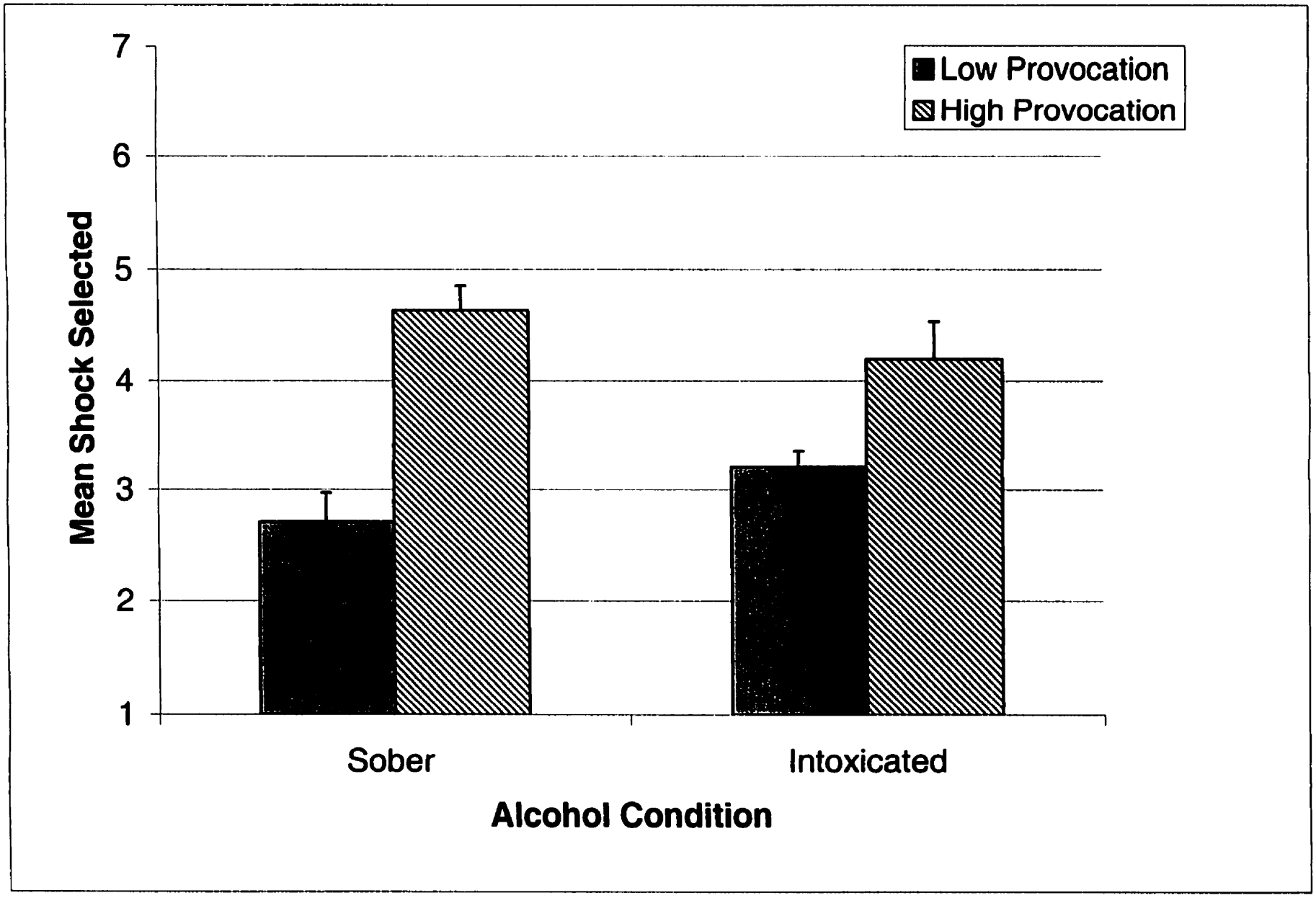
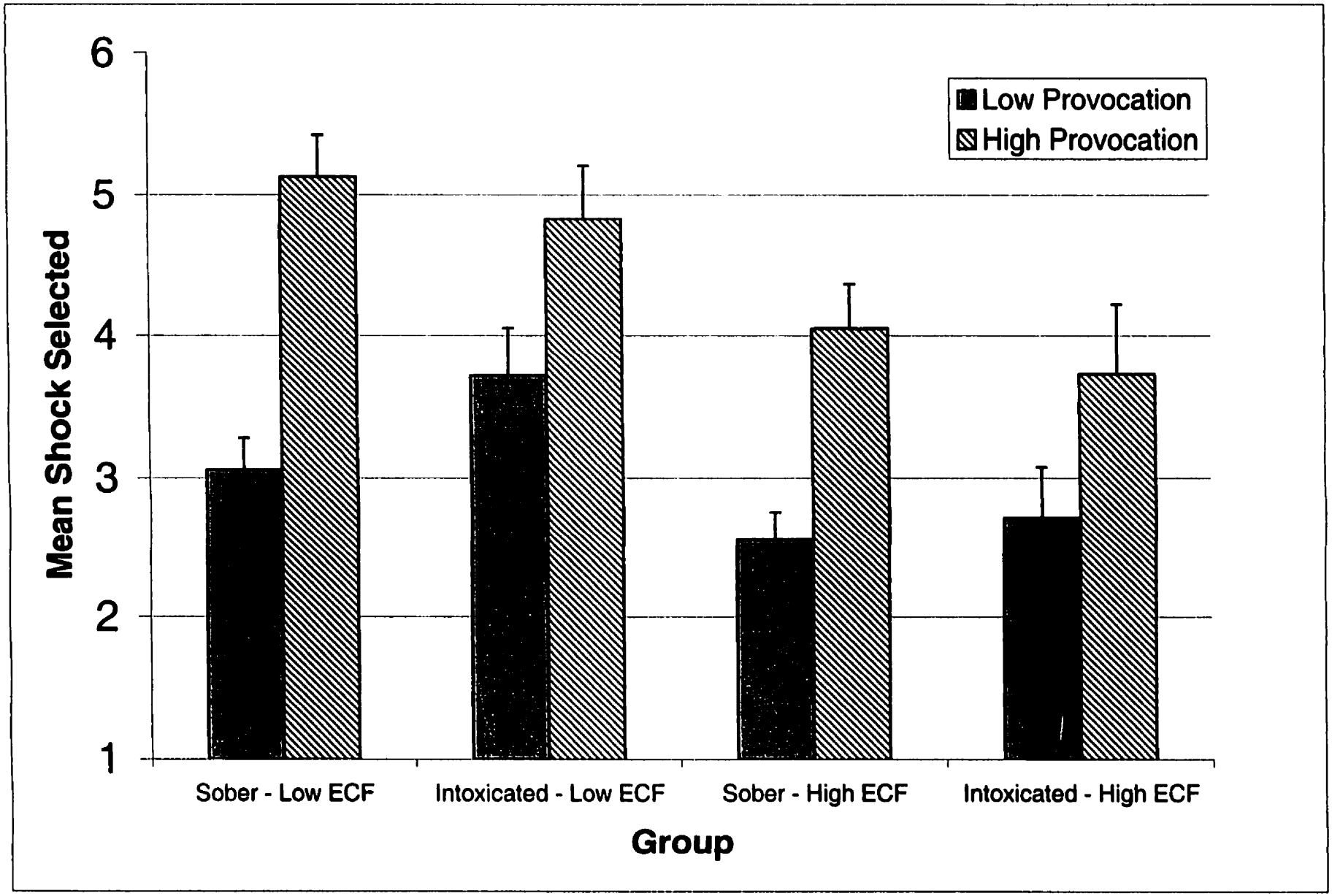

Low Provocation
High Provocation
Mean Shock Selected
7
6
5
4
3
2
1
Sober
Intoxicated
Alcohol Condition

Figure Caption

Figure II. Mean shock selected, for both high and low provocation conditions, for participants in both the high and low cognitive functioning group, by alcohol condition.


6
5
4
3
2
1
Mean Shock Selected
Low Provocation
High Provocation
Sober - Low ECF
Intoxicated - Low ECF
Sober - High ECF
Intoxicated - High ECF
Group

Bridge to Study Four

Study three demonstrates that below-average or aberrant executive cognitive function was a consequential condition for manifestation of aggressive behaviour in women, as it had previously been shown in men (Giancola, 1994; Lau, Pihl & Peterson, 1995; Hoaken, Assaad & Pihl, 1998). In women whose executive functioning is deficient, provocation is much more likely to lead to an aggressive response.

However, this result is idiosyncratic inasmuch as alcohol intoxication appears unimportant in terms of eliciting aggressive response, a finding not in keeping with the literature in men. Moreover, there was no ECF-alcohol interaction in these women. In fact, although poor baseline-ECF and alcohol intoxication (which is thought to interfere with ECF) both independently lead to heightened aggression, studies have not consistently demonstrated this interaction in men (Lau, Pihl & Peterson, 1995), which theory would suggest they should (Giancola, 2000). It could be that multi-factorial and mixed designs such as these are too complex to tease out the finer points of the ECF-alcohol-aggression relationship.

As such, the focus of these studies moved away from alcohol and focussed exclusively on the aggression-ECF relationship. That is, although we knew individuals with aberrant executive functioning were more likely to behave aggressively, we didn't know why this was the case. It is a question that still, to a certain extent, underlies the alcohol-aggression relationship; we do not know for a certainty whether the causes

are direct or indirect, and what are they, specifically. Many of the hypotheses which underlie the alcohol-aggression relationship appear worthy of investigation in the context of the ECF-aggression relationship, as well, including the notion that there is some interference in social-information processing (Dodge, 1986; Sayette, Wilson & Elias, 1993), some disruption in self-awareness (Hull, 1981), or some interference with threat-detection (Pihl, Peterson & Lau, 1993). The focus of the work became comparing low- versus high-ECF individuals on measures of aggression, but also attempting to examine why, specifically, low-ECF individuals were more likely to be aggressive.

Early research on the ECF-aggression link had suggested that low-ECF individuals act aggressively because of an inability to inhibit impulsive behaviours. That is, faced with provocation, these individuals react in kind in an impulsive, spontaneous, and rapid fashion, without proper appreciation of the possible consequences of the behaviour (Lau, et al., 1995; Lau & Pihl, 1996). Study four was intended to examine the validity of this explanation of the ECF-aggression relationship.

STUDY FOUR

Hoaken, P.N.S., Shaughnessy, V., & Pihl, R.O.

Executive Cognitive Functioning and Aggression: Is it an Issue of Impulsivity? Manuscript under review, Aggressive Behavior.

Executive Cognitive Functioning and Aggression:

Is it an Issue of Impulsivity?

Peter N. S. Hoaken[1], Valerie K. Shaughnessy[2], & R.O. Pihl[3]

[1]Department of Psychology, McGill University; [2]Department of Psychology, York University; [3]Departments of Psychology and Psychiatry, McGill University.

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Corresponding Author: Peter N. S. Hoaken, department of Psychology, 1205 Dr. Penfield Ave, McGill University, Montreal, PQ, Canada, H3A 1B1. Electronic mail may be sent via Internet to *hoaken@hebb.psych.mcgill.ca.*

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Abstract

Objective: To investigate the merits of the theory that individuals with poor executive function act aggressively due to impulsive, poorly considered, disinhibited behavioral responses to provocation.

Background: A large body of research has documented a relation between

the executive cognitive functions (ECF) and interpersonal aggressive behavior. A predominant theory proposes that individuals with poor ECF are more aggressive because they are unable to inhibit impulsive behaviors. However, evidence for this relationship is typically indirect. Methods: Forty-six healthy men and women completed neurocognitive measures of ECF, the Taylor Aggression Paradigm (TAP), and the Go/No-Go discrimination task, a behavioral measure of impulsivity. Also, impulsiveness of participant responses during the aggression task was directly assessed by measuring latency of responses to provocation ("set-time"). It was hypothesized that low-quartile scoring ECF men and women would perform more aggressively and more impulsively than high-quartile peers. Results: Consistent with expectations, results indicated that ECF was related to aggression and to impulsivity on the Go/No-Go task. However, low ECF men and women did not have shorter "set-times"; in fact, on this task low ECF participant's behavioral decisions appeared slightly slower than the high ECF participants. Discussion: In light of the above results, the authors speculate that a social information-processing problem may mediate the ECF aggression relationship, rather than altered impulsivity per se.

Introduction

There are several large and comprehensive bodies of research which demonstrate that impulsive, under-controlled, or dysregulated behavior predicts such problems as alcohol abuse (Colder & Chassin, 1997; Poulos, Le & Parker, 1995; Virkkunen & Linnoila, 1993), drug abuse (Luengo, Otero, Carrillo-de-la-Pena, & Miron, 1994; Allen, Moeller, Rhoades, & Cherek, 1998), gambling (Vitaro, Arseneault, & Tremblay, 1999; Langewisch & Frisch, 1998; Blaszczynski, Steel, & McConaghy, 1997), risky sexual behavior (Donohew, Zimmerman, Cupp, Novak, Colon, & Abell, 2000; Rawlings, Boldero, & Wiseman, 1995), and suicide (Oquendo & Mann, 2000; Suominen, Isometsa, Henriksson, Ostamo, & Loennqvist, 1997; Linnoila, DeJong, & Virkunnen, 1989). Also associated with impulsivity is interpersonal aggression and violent crime (Scarpa & Raine, 2000; Lane & Cherek, 2000; Wang & Diamond, 1999; Cherek, Moeller, Dougherty & Rhoades, 1997), complex social phenomena of which approximately three million people are victims each year (U.S. Bureau of Justice Statistics, 1997).

Although the causes of interpersonal aggression are clearly multifactorial, recent research on aggression has emphasized certain neurocognitive capacities as putative antecedents. Converging evidence has suggested that the so-called "executive cognitive functions" (ECF) may play an important role in mediating aggressive behavior (Moffitt, 1993; Hoaken, Giancola & Pihl, 1998b; Seguin, Pihl, Harden, Tremblay,

& Boulerice, 1995; Giancola, 1995; Giancola 2000). ECF has been conceptualized as the capacity to use certain "higher-order" cognitive abilities to adaptively regulate one's goal-directed behavior (Giancola, Martin, Tarter, Pelham, & Moss, 1996). These higher-order abilities include attention, planning, abstract reasoning, mental flexibility, temporal sequencing, hypothesis generation, self-monitoring, the organization of information in working memory, and the ability to use response feedback cues to regulate ongoing behavior (Foster, Eskes, & Stuss, 1994; Milner, Petrides & Smith, 1985; Stuss & Benson, 1984). From a neuroanatomical perspective, ECF has widely been thought to be subserved by neural systems involving prefrontal cortex and related subcortical structures (Roberts, Robbins & Weiskrantz, 1998; Cummings, 1995).

The study of clinical populations characterized by aggression has lent support to the importance of the executive functioning in the mediation of aggressive behavior. Conduct disordered adolescent males (Lueger & Gill, 1990) and females (Giancola & Mezzich, 2000) have performed poorly on neuropsychological tests of prefrontal and/or executive functioning. Tests of executive function have also been shown to be impaired in men with antisocial personality disorder (Malloy, Noel, Longabaugh, & Beattie, 1990). Studies utilizing experimental manipulations have also provided experimental evidence supporting the aggression-ECF relationship. Aggression has been linked to low scores on tests of ECF in samples of boys (Seguin et al., 1995; Giancola, Moss,

Martin, Levent, & Tarter, 1996), adolescent girls (Giancola, Mezzich & Tarter, 1998a, Giancola et al., 1998b), adult men (Giancola & Zeichner, 1994; Lau, Pihl, & Peterson, 1995; Hoaken, Assaad & Pihl, 1998a) and adult women (Hoaken, Strickler & Pihl, submitted manuscript).

A predominant explanation for the relationship between executive cognitive functioning and aggression has been related to the concept of impulsivity. More specifically, it has been hypothesized that aggressive, low-ECF individuals are less able to inhibit impulsive behaviors (Lau et al., 1995). Lau and Pihl (1996) attempted to test this hypothesis by examining whether a monetary incentive could decrease aggressive responding in males. Individuals with poor measure of ECF (unlike those with high ECF; Hoaken et al., 1998a) were unable to inhibit aggressive responding in the presence of monetary reward; the authors suggested that this inability might be due to an inability to use inhibitory feedback cues to regulate behavior. The present study was intended to investigate the notion that individuals with poor ECF, individuals who are more likely to demonstrate aggression in response to provocation, are so because they are more behaviorally impulsive. However, investigating this putative relationship is not a simple endeavor because although "impulsivity" is an oft-discussed construct, it is one about which there is little definitional agreement in the clinical literature (Gerbing, Ahadi, & Patton, 1987; Parker, Bagby & Webster, 1993). There are multitudinous self-report measures of impulsivity (see Parker & Bagby, 1997, for a review) all of

which conceptualize impulsivity slightly differently. Not surprisingly, when these measures are intercorrelated there are at best inconsistent relationships between them (Luengo, Carrillo-de-la-Pena, & Otero, 1991). There are also several behavioral measures of impulsivity, which typically incorporate either some element of reaction time (e.g. the Matching Familiar Figures Test; Kagan, Rosman, Day, Albert & Phillips, 1964), or some perception of elapsed time (Barratt & Patton, 1983). The former of these are based on the notion that impulsive individuals have a tendency to make quick decisions and act without thinking; the latter on the notion that impulsive individuals tend to overestimate the amount of time that has passed. The intercorrelation of these behavioral measures also tends to be inconsistent. What is consistent, however, is that self-report and behavioral measures do not intercorrelate even minimally (Milich & Kramer, 1984; Gerbing et al, 1987; Malle & Neubauer, 1991; Helmers, Young & Phil, 1995).

While paper-and-pencil questionnaires are subject to biases such as self-awareness and demand characteristics, which may lead to inaccurate scores (Helmers, et al., 1995), behavioral measures of impulsivity tend to be more congruent with some theoretical definitions. One behavioral measure of impulsivity, the Go/No-Go task (Newman, Widom, & Nathan, 1985; Helmers et al., 1995), assesses the ability of a participant to withhold a response to a stimulus that has been previously paired with reward and/or punishment. It has been postulated that

impulsive individuals respond more frequently to negative stimuli due to an increased focus on reward and an inability to alter a dominant response set (Newman, 1987). An existing literature supports the Go/No-Go as a measure of behavioral impulsivity. Populations including incarcerated psychopaths, extraverts, juvenile delinquents (Newman, 1987; Newman, Patterson, Howland, & Nichols, 1990; Patterson, Kosson & Newman, 1987), and attention deficit hyperactivity disordered children (Iaboni, Douglas & Baker, 1995), have been found to make increased errors of commission, but similar errors of omission (failure to respond to a positive stimulus) on the reward-punishment version of the Go/No-Go task. In addition, psychopaths and extraverts were found to be less likely to slow down and respond more quickly after punishment (Newman, 1987). This tendency to speed up has been taken as further evidence for the failure of disinhibited individuals to learn from punishment, and has been incorporated into some working definitions of impulsivity.

Few studies investigate the aggression-impulsivity relationship directly. In one such study, LeMarquand and colleagues (1998) examined the relationship between tryptophan depletion, impulsivity and aggression in a sample of adolescent males selected for an extensive history of aggressive behavior. Aggressive individuals made more Go/No-Go "impulsive" errors than did non-aggressive individuals, and had lower scores on tests of executive function (LeMarquand, Pihl, Young, Tremblay, et al., 1998). The authors suggested that impulsivity and

executive function are correlated and underlie aggressive behavior. The study confirmed the impulsivity hypothesis of the low ECF-aggression relationship; however, only impulsivity and executive function, but not aggression, were measured directly. Cleare & Bond (1995) also endeavored to assess the relationship between impulsivity and aggression. Their study in healthy males included a tryptophan depletion manipulation, and measured behavioral aggression with a modified version of the Taylor (1967) aggression paradigm. These authors also assessed what they called "set-time". This variable represents the time taken by the participant to select a noise level to deliver to the opponent, and which they believed to represent the impulsive nature of the aggressive response. The results showed that individuals characterized by heightened aggression also displayed shorter "set-times", indicating to the authors an impulsive, disinhibited nature to the aggression.

Since men perpetuate the majority of violent crimes, it is not surprising that the bulk of the existing literature on aggression has been conducted on men. However, the commonly held view that women are not aggressive has recently been challenged. Aggression in women may be more common than it is perceived to be. In the United States, women committed 15% of all violent crimes (U.S. Department of Justice). Furthermore, evidence of female aggression toward spouses is as frequent or more so than male aggression toward another (Archer, 2000). Thus, as studies involving only men participants ignore the perpetrators of a

significant percentage of aggressive acts, this study included both men and women participants.

The present study, then, was conducted in order to use direct measures of aggression, executive function and impulsivity to examine the aforementioned theory of the ECF-aggression relationship. The study tested both men and women, who performed at the top- or bottom-quartile scores on measures of ECF, on the Go/No-Go paradigm, and on the Taylor aggression paradigm, with "set-time" measured on each trial. Consistent with the current literature, it was hypothesized that both men and women with low cognitive functioning would respond more aggressively to provocation. It was also expected that these individuals would display increased errors of commission on the Go/No-Go task, and would have shorter "set-times" on the Taylor paradigm (that is, they would be faster selecting shock intensities to deliver to their opponent). Consistent with the majority of studies (Eagly & Steffen, 1986) we hypothesized that men would be more aggressive than women.

Method

Participants

Male and female participants were recruited through both local newspaper advertisements, and campus recruiting. Two sources of participants was intended to preclude a homogenous sample, and to provide a representative sample of the general population. Only

participants aged 18-30 were recruited. Potential participants were eliminated if they had sustained a serious injury to the head, had a diagnosed reading or learning disability, or were familiar with psychological experimentation. Participants who met the criteria were invited to participate in the first phase of the experiment. On the first day, participants were tested on a battery of tests, including a general intelligence test, a test of memory, and several tests of executive cognitive functioning (ECF). Participants who fell into either the high- or low-quartile groups were invited to return for the second phase. On the second day, participants were administered behavioral tests of impulsivity and aggression. To control for the effect of hormone fluctuation on aggression, female participants were run between day five and thirteen of their menstrual cycles (Sutker, Goist & King, 1987). Females were eliminated from the study if they were pregnant.

Measures

Intelligence Measure

A short form of the Weschler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1981) was administered to all participants. This short form included the Information, Block Design, and Vocabulary subtests, scores from which were used to calculate estimates of full-scale IQ (Brooker and Cyr, 1986).

Memory Measure

A short form of the Weschler Memory Scale, Revised (WMS-R; Wechsler, 1987) was administered to all participants. This included the Figural Memory, Logical Memory, Visual Paired Associates, Verbal Paired Associates, and Figure Reproduction subtests, which were used to calculate the General Memory quotient (MQ).

Neurocognitive Measures.

Executive cognitive functioning (ECF) was assessed with the following neurocognitive tasks:

Spatial Conditional Association Learning Task (SCALT; Petrides, 1985). This task consists of six red lights in a circular array, and six black rectangles in a 2x3 matrix. Each light is associated with a rectangle. The aim of the task is to learn the six associations by trial-and-error and feedback from the experimenter. Each trial begins with the illumination of a random light. The participant is instructed to point to the rectangle they think is associated with the light. If the correct rectangle is indicated, the experimenter says "right", another light is lit and a new trial begins. If an incorrect rectangle is indicated, the experimenter responds "wrong", the light remains lit, and the participant continues selecting rectangles until the correct one is indicated. The task is terminated after 18 consecutive error-free trials are achieved, or after the completion of 180 trials. The dependent measures are the number of trials completed and the total

number of incorrect responses. This task is a measure of the ability to learn a series of conditional associations between unrelated stimuli. Studies have found that patients with frontal-lobe excisions, but not those with temporal lobe excisions, perform poorly on this task (Petrides, 1985). Furthermore, regional cerebral blood flow (rCBF) and positron emission tomography (PET) scans taken during this task evidenced the largest increase in blood flow in the posterior dorsolateral area of the prefrontal cortex (cytoarchitectonic area 8) in normal men (Petrides, Alivisatos, Evans, & Myers, 1993).

Self-Ordered Pointing Task (SOP; Milner Petrides, & Smith, 1985). This task assesses the ability to organize, plan and monitor a sequence of responses. Participants are presented with a 12-page booklet containing 12 pictures in a 3x4 matrix on each page. The same pictures, in a differing random arrangement, appear on each page. The participant is asked to point to a different picture on every page. A concrete and an abstract version of this task are administered. In the concrete version, the pictures are identifiable and familiar (e.g. sink, stove, tree). In the abstract version, the pictures are not identifiable and are difficult to encode verbally. Each version is repeated three times. The dependent measure is the number of errors committed. An error is defined as pointing to an already indicated picture within a set of 12 pages. Studies have reported that patients with frontal lobe, but not those with temporal lobe excisions perform poorly on this task (Petrides & Milner, 1982). Furthermore,

neuroimaging studies using PET and rCBF scans have shown the greatest increase in blood flow in the mid-dorsolateral region of the prefrontal cortex (cytoarchitectonic area 46 and 9) when this task was performed by normal males (Petrides et al., 1993).

Aggression Measure.

Aggression was elicited and assessed using a modified version of the Taylor Aggression Paradigm (TAP; Taylor, 1967). Participants competed against an "opponent" in a reaction-time trial. The opponent was actually fictional and all shocks delivered to the participant were predetermined by the experimenters. Participants were seated in front of a console consisting of a panel with eight lights (numbered 1-8) indicating the eight shock levels. Below each light was a corresponding button used to select shock levels to deliver to the "opponent". The lights represented shock intensities, one being the lowest and eight the highest. A ready-key, used as a reaction-time button and to administer shocks, sat in front of the participant. The shocks were delivered from a Mark I Behavior Modifier (Farall Instruments, Grand Islands, NB Canada) through an electrode placed on the inner forearm of the dominant hand, distal to the elbow. Participants used these buttons to choose the shock intensity to administer to the (mock) opponent. The dependent variable on the aggression paradigm was mean shock intensity selected.

The Taylor Aggression Paradigm is among the most popularly utilized laboratory measures of aggression, and is often referred to as the

classic laboratory measure of aggression. The Taylor paradigm has demonstrated construct validity, in that individuals thought to be aggressive by nature appear so on this paradigm, while non-aggressive individuals do not; has demonstrated convergent validity, in that it has been correlated positively with other measures of aggression; and has established discriminant validity, in that it has been shown to not correlate with other measures thought to be theoretically unrelated to aggression (Giancola & Zeichner, 1995; Bernstein, Richardson, & Hammock, 1987).

Impulsivity Measure.

The reward-punishment version of the Go/No-Go discrimination task was used to assess impulsivity. In this task, an IBM personal computer flashed a series of two-digit numerical stimuli. Participants learned by trial-and-error which stimuli were rewarding and which were punishing. Eight numerical stimuli were presented in a random order; four were rewarding, and four were punishing. Each stimulus was presented ten times, for a total of 80 trials. The stimuli were displayed on the screen until participants responded, or for up to three seconds. Lack of response lead to neither punishment nor reward. After each trial, the participants were given visual, auditory and monetary feedback. A correct response was followed by a high-pitched tone, the appearance of the word "correct" and the addition of money to the participant's tally of earnings. Similarly, an incorrect response was followed by a low-pitched tone, the appearance of the word "incorrect", and the subtraction of money from the

participant's tally. Participants began with one dollar, received 10 cents for every correct response, and lost 10 cents for every incorrect response. The dependent measures were the mean number of errors of commission (failure to inhibit a response to a punishing stimulus), as well as the mean number of errors of omission (failure to respond to a rewarding stimulus). The response time to stimulus response was also measured. Timing began when the stimulus appeared on the screen, and ended either when the participant responded, or at the end of the three second presentation. This variable was only collected for trials on which the participant responded (either an error of commission or a correct response).

Procedure

Participants who met the prescreening criteria were invited to participate in the first phase of the experiment, ECF screening. Upon arrival, participants completed a consent form, provided demographic data, then were tested on the WAIS-R and the WMS-R, followed by a series of neurocognitive tests – the SCALT, and the concrete and abstract versions of the SOP task. High- and low- quartile performers were invited to return for the second phase of the experiment. After the first phase, participants were paid $10 to compensate for lost time. Upon arrival for the second day of testing, participants completed a second consent form. Testing began with the Go/No-Go discrimination task. The instructions were presented, and the experimenter explained the nature of

the trial-and-error learning, as well as the monetary contingency. Practice trials, illustrating response options and their consequences, were demonstrated. The experimenter was not present during the task.

In preparation for the aggression paradigm, participants' individual shock ranges were determined. One-second shocks were administered at a sub-threshold level (28 ma) and increased in 0.10 ma increments every 5 seconds. By depressing the ready-key, participants indicated the first detectable shock, as well as a shock that has reached a painful level, and was the highest intensity they wished to receive. The range of intensities selected was divided into 7 equal increments. After the shock range had been determined, the examiner read scripted instructions. Next, the participant watched their opponent receive the instructions. In reality, it was a video recording of a confederate. This was done to ensure the believability of the deception, and to reinforce the instructions. Due to previous research (Giancola & Zeichner, 1995b) which suggests that women act equally aggressively towards men and women opponents on this paradigm, gender of the opponent was not manipulated. That is, men always "competed" against men and women against women. At this point, the experimenter answered any further questions and the paradigm began.

Each trial began with lights flashing from left to right, indicating that the participant should select a shock intensity to deliver to their "opponent", should they win the reaction-time trial. The dependent

measures were the mean shock intensity and set-time selected. Set-time was defined as the amount of time taken by the participant to select a shock intensity to deliver to their "opponent". Measurement began when the lights started flashing from left and right, and ended when the participant depressed the button below the light selecting a shock intensity. The reaction time competition ensued. Lights one and eight lit up indicating that the participant should depress the ready-key. After a variable length of time, all the lights illuminated, indicating that the participant should release the ready-key as quickly as possible. After the competition, a light illuminated corresponding to the shock intensity the "opponent" selected to administer to the participant. Next, the participant discovered whether they won or lost the trial, and either administered or received the shock. Participants delivered shocks by depressing the ready-key. Beside the participant was a DC ammeter indicating the level and duration of the shock administered. Audio prompts were given to indicate when to select a shock level, as well as whether the participants won or lost the trial.

There were 26 trials in total. Two sets of 12 trials were divided by a transition trial, and followed by a final trial. The first set of trials represented the low provocation condition during which the opponent selected shock levels between one and four. The second set of trials represented the high provocation condition. During this set of trials, the "opponent" selected shocks between level five and eight. In both the final

and transition trials, the "opponent" selected a shock level of five. All participants lost the transition trial and won the final trial. The "opponent's" shock selection, as well as the order of wins and losses, was randomized. Shock selection and data recording was done by an IBM personal computer.

Following the task, participants were given a questionnaire to assess their perception of the opponent, as well as their subjective rating of their aggression level to probe the completeness of the deception. The participants were then debriefed. Debriefing included an explanation of the deception, its necessity, and a request to refrain from revealing the deception to others. Participants were compensated $25 for their time, in addition to any money won on the Go/No-Go task.

Results

Participants

After telephone screening, 199 participants were invited into the lab and tested on the neurocognitive battery. Of these, 96 fell into either the low- or high- quartile groups and were deemed acceptable for the second phase of the study. Forty-two participants were eliminated because they did not want to participate, could not be conveniently scheduled, did not show up for the second session, or were lost over the course of the study. A total of 54 individuals were run on the aggression paradigm; of those, eight were eliminated because they were deemed not

to have been completely deceived. Of those, three men and three women from the high-executive function group and two women from the low-executive function group were not deceived. Thus, a total of 11 low- and 12 high- quartile women and 11 low- and 12 high- quartile men were run.

Demographic, Intelligence and Memory Measures

Analyses of variance were conducted on all demographic, intelligence and memory variables to investigate potential differences between the groups. These tests revealed no differences between any of the groups on any of the demographic variables including age, socio-economic status (based on Blishen, Carroll, & Moore, 1987) and alcohol and/or tobacco use. It should be noted that the data intended to convey information on socioeconomic status is limited, because most participants did not provide complete information; the data that was collected was summed and averaged. An

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Insert Tables I & II about here

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analysis of variance indicated no significant differences between the groups for any of these aforementioned variables.

The analysis of variance conducted on the intelligence measure (IQ estimate from the WAIS-R) did indicate a significant difference between groups [$F(3,42) = 3.93, p<.05$]. As a result, IQ was included as a

covariate for all further analyses. The analysis of variance for the memory measure (WMS-R general memory MQ) also did not indicate any significant differences between the groups. These results are represented in Table I. Performance of all groups on the measures of executive function are represented in Table II.

Aggression Measure

A 2 (quartile) x 2 (gender) x 2 (provocation) mixed design analysis of variance (ANOVA), with provocation as a repeated measure, and with IQ as a covariate, was conducted to assess the effect of gender and executive functioning on shock intensity selections on the TAP. The analysis revealed non-significant results for the

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Insert Figure I about here

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three-way and all three two-way interactions. Analysis of main effects revealed a significant effect of provocation [$F(1, 41) = 88.32, p<.001$], quartile [$F(1,41) = 6.50, p<.05$], and gender [$F(1,41) = 12.20, p<.01$]. These results supported the hypothesis that low-quartile (LQ) individuals would select higher shock intensities than their high-quartile (HQ) peers, and that high provocation would lead to an increase in aggressive responding. Men participants were more aggressive than women. Analysis of simple main effects indicated a significant effect of gender in

the LQ group under both low- [$F(1,20) = 5.84, p<.05$] and high- [$F(1,20) = 4.67, p<.05$] provocation conditions. Analysis of simple main effects of gender in the HQ group revealed a significant effect in the low provocation condition [$F(1,22) = 7.42, p<.05$] and a trend in the high provocation condition [$F(1,22) = 2.97, p<.10$]. Analysis of simple main effect of quartile in men revealed a significant effect in the low provocation [$F(1,21) = 6.76, p<.05$] condition and a trend in the high provocation [$F(1,21) = 3.21, p<.10$] condition. Analysis of simple main effect of quartile in women revealed a trend in the low provocation [$F(1,21) = 2.96, p<.10$] condition and an non-significant effect in the high provocation [$F(1,21) = 1.71, p=.21$] condition. These results are represented in Figure I.

Impulsivity Measures

Set-time. A 2 (quartile) x 2 (gender) x 2 (provocation) mixed design analysis of variance (ANOVA), with provocation as a repeated measure, and with IQ as a covariate, was conducted to assess the effect of gender, cognitive functioning quartile split and

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Insert Figure II about here

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provocation on the measure of mean set-time. There were no 3- or 2-way interactions. Analysis of main effects revealed a trend for quartile group

[$F(1,42) = 3.49, p<.10$]. Contrary to the hypothesis, the LQ participants were slower to select shock intensities. Analysis also revealed a trend for provocation [$F(1,42) = 3.25, p< .10$], with participants taking longer to select shock intensities at low provocation. The main effect of gender was non-significant [$F(1, 42) = 011, p=.74$]. These results are presented in Figure II.

Go/No-Go. A 2 (quartile) x 2 (gender) analysis of variance (ANOVA) was conducted to assess the effect of executive functioning and gender on the behavioral measure of impulsivity, errors of commission on the Go/No-Go task. The analysis of

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Insert Figure III about here

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gender x quartile interaction was non-significant [$F(1,39)=0.53, p=.47$]. Analysis of main effects revealed no significant gender effect [$F(1, 39) = 0.633, p=.43$], but a trend for executive function [$F(1,39) = 3.32, p<.10$], with LQ individuals making more errors of commission. These results are represented in Figure III.

Reaction Time to Stimulus Response. A 2 (quartile) x 2 (gender) analysis of variance (ANOVA) was conducted to assess the effect of executive functioning and gender on mean reaction time. The analysis revealed no significant gender x quartile

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Insert Figure IV about here

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interaction [$F(1,41) = 2.68$, $p=1.09$]. No significant main effect was found for quartile group [$F(1,41) = 0.294$, $p=.59$], nor was there a significant main effect for gender [$F(1,41) = 1.31$, $p=.39$]. The simple main effect of gender reaches a trend in the HQ group [$F(1,21) = 3.18$, $p<.10$], but is non-significant in the LQ group [$F(1,19) = 1.82$, $p>.19$]. There were no significant simple main effects. These results are represented in Figure IV.

Discussion

The results of this study support the existing literature which shows that individuals who perform poorly on test of executive cognitive function respond more aggressively to increasing provocation (Giancola & Zeichner, 1994; Lau et al., 1995). The consistency of this finding, across a range of experimental treatments and clinical literatures, provides strong support for the involvement of ECF in provoked aggressive behavior. That men were more aggressive than women also corresponds to the majority of manipulative studies (Eagly & Steffen, 1986), although not all (Hoaken & Pihl, 2000).

However, the two measures of impulsivity, "set-time" and the Go/No-Go task, seem to draw into some question the predominant disinhibition explanation of the ECF-aggression relationship. On the

aggression task LQ individuals were expected to make more rapid shock intensity selections, and thus, have shorter "set-times" than HQ individuals, indicating impulsive aggression. However, the opposite phenomenon was observed: Analysis revealed a trend for executive function, with the LQ men and women actually taking longer to select shock intensities than HQ individuals.

On the second measure of impulsivity, the Go/No-Go task, it was expected that ECF would be related to errors of commission, indicating an inability to inhibit responding. This hypothesis was partially supported. Errors of commission on the Go/No-Go task approached significance, with LQ men and women making more errors than HQ individuals. These results support the findings of LeMarquand and colleagues (1998), who reported an association between ECF, teacher-rated aggression, and errors of commission on the Go/No-Go task. Thus, the two putative measures of impulsivity seem to lend contradictory support for the aggression-impulsivity hypothesis.

It may be that this difference reflects definitional discrepancies on the construct of "impulsivity". Both male and female participants in low-ECF groups appear to fail to inhibit responses previously paired with punishment on the Go/No-Go. However, the "set-time" data for these participants is contrary to the expectancy that rapid action, and an absence of adequate forethought, characterizes impulsivity and impulsive aggression.

The apparent discrepancy between the two measures may be relative to the fact that aggression contains a social component and the Go/No-Go task does not. This suggests that individuals with poor executive function act aggressively not because they are impulsive, but rather because of some interruption of social-information processing. These individuals may not be able to use social cues to narrow response options, and hence may be overwhelmed by their response options and make inappropriate selections. There is evidence to suggest that prefrontal cortex is involved in the processing of both motivational and emotional stimuli, and moreover that prefrontal lesions may lead to disruptions in social behavior (Damasio, 1995; Anderson, Bechara, Damasio, Tranel, & Damasio, 1999). Adopting Robbins' (1998) description of executive function as the need to formulate new plans of action, to select appropriate responses, and monitor "behavior with respect to affective or emotional state" (Robbins, 1998) would suggest a model for the poor ECF-aggression relationship. It may be that participants in the our low-ECF group, like participants from multiple previous experiments, were not able to use social cues to narrow response options, consequently making inappropriate behavioral selections or responses. Overwhelmed by possibilities, social norms that disapprove of aggressive responses are searched for but not accessed; thus these individuals make bad social decisions but do so relatively slowly.

It may be, however, that the measure of "set-time" is confounded by matters of perception and motor performance. Barratt (1994) has pointed out that the relationship between impulsiveness and perceptual-motor tasks is a complex one, and that high-impulsiveness participants often perform less efficiently on tasks such as pursuit rotor, visual tracking, and some reaction-time tasks. In light of this, one might suggest that the "set-time" difference simply represents a difference in reaction time response to the "ready" signal at the beginning of each trial of the aggression paradigm. However, this assertion is drawn into question by the fact that low- and high-ECF participants in this study did not differ on the more representative measure of reaction time, the delay to stimulus response measure on the Go/No-Go task.

Although this study found evidence of a relationship between poor executive functioning and impulsivity, as have others, the relationship may be specific to non-social situations. Aggression is a primal social response option, a simple response option to an exceedingly rich and complex melange of contextual cues. It may be that individuals with poor ECF, demonstrating poor social information processing skills and an inability to cope with overwhelming response options, fail to access more socially-appropriate response options, and make default aggressive responses to provocative situations.

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Table I. Means (and Standard Deviations) for High-ECF and Low-ECF Men and Women on Demographic and Other Variables

	Men/High ECF	Men/Low ECF	Women/High ECF	Women/Low ECF
Age	20.25 (4.22)	23.54 (5.39)	22.41 (4.50)	23.45 (4.82)
Socioeconomic Status*	23.29 (5.16)	23.63 (5.39)	19.25 (6.26)	20.36 (6.69)
Drinks per Occasion	4.66 (4.37)	5.09 (3.53)	2.29 (.96)	3.99 (3.21)
Drinks per Week	9.75 (14.67)	9.27 (9.39)	5.54 (7.77)	7.27 (11.21)
Cigarettes per Day	2.16 (4.30)	5.72 (9.47)	3.91 (6.89)	4.18 (8.11)
WAIS-R (Estimate)	110.08 (9.78)	106.70 (10.11)	110.50 (10.31)	102.21 (15.43)
WMS-R	110.80 (11.79)	104.60 (10.02)	117.28 (11.83)	106.71 (11.24)

Table II. Means and Standard Deviations for High-ECF and Low-ECF Men and Women on Tests of Executive Function.

	Men/High ECF	Men/Low ECF	Women/High ECF	Women/Low ECF
Spatial CALT - # of Trials	64.33 (25.42)	167.91 (22.32)	69.08 (23.08)	145.36 (31.03)
Spatial CALT - # of Errors	30.00 (12.76)	161.54 (81.48)	33.41 (12.33)	115.54 (42.11)
Concrete SOP – Errors	1.75 (.86)	5.45 (2.38)	1.08 (.90)	4.81 (3.25)
Abstract SOP – Errors	1.33 (1.23)	5.90 (3.72)	1.91 (1.16)	6.27 (2.00)

Figure Caption

Figure 1. Mean shock intensity selected by gender and executive functioning split, for both low and high provocation conditions.

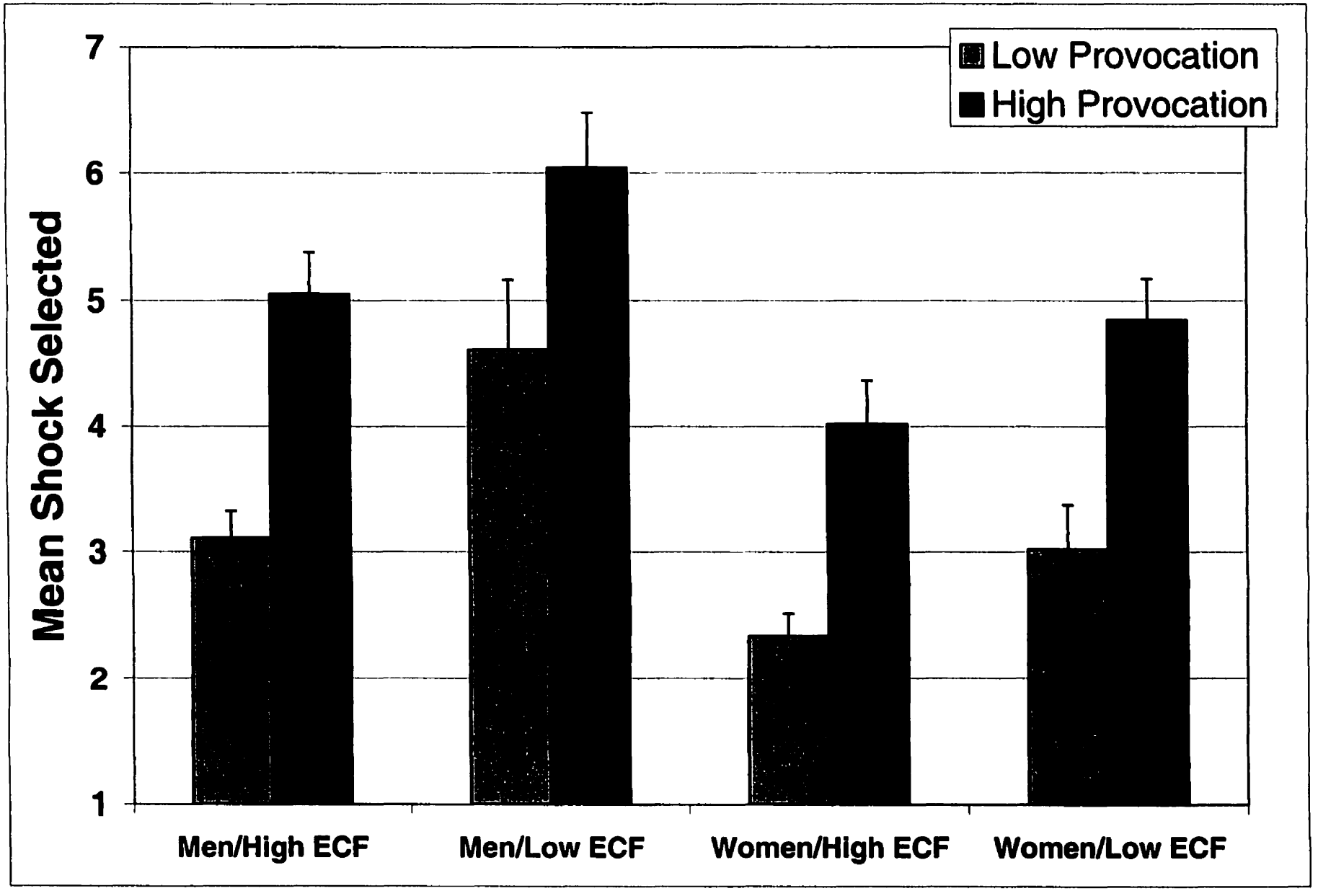

Low Provocation
High Provocation
Mean Shock Selected
7
6
5
4
3
2
1
Men/High ECF
Men/Low ECF
Women/High ECF
Women/Low ECF

Figure Caption

Figure II. Mean "set-time" (in seconds) by gender and executive functioning split, for both low and high provocation conditions.

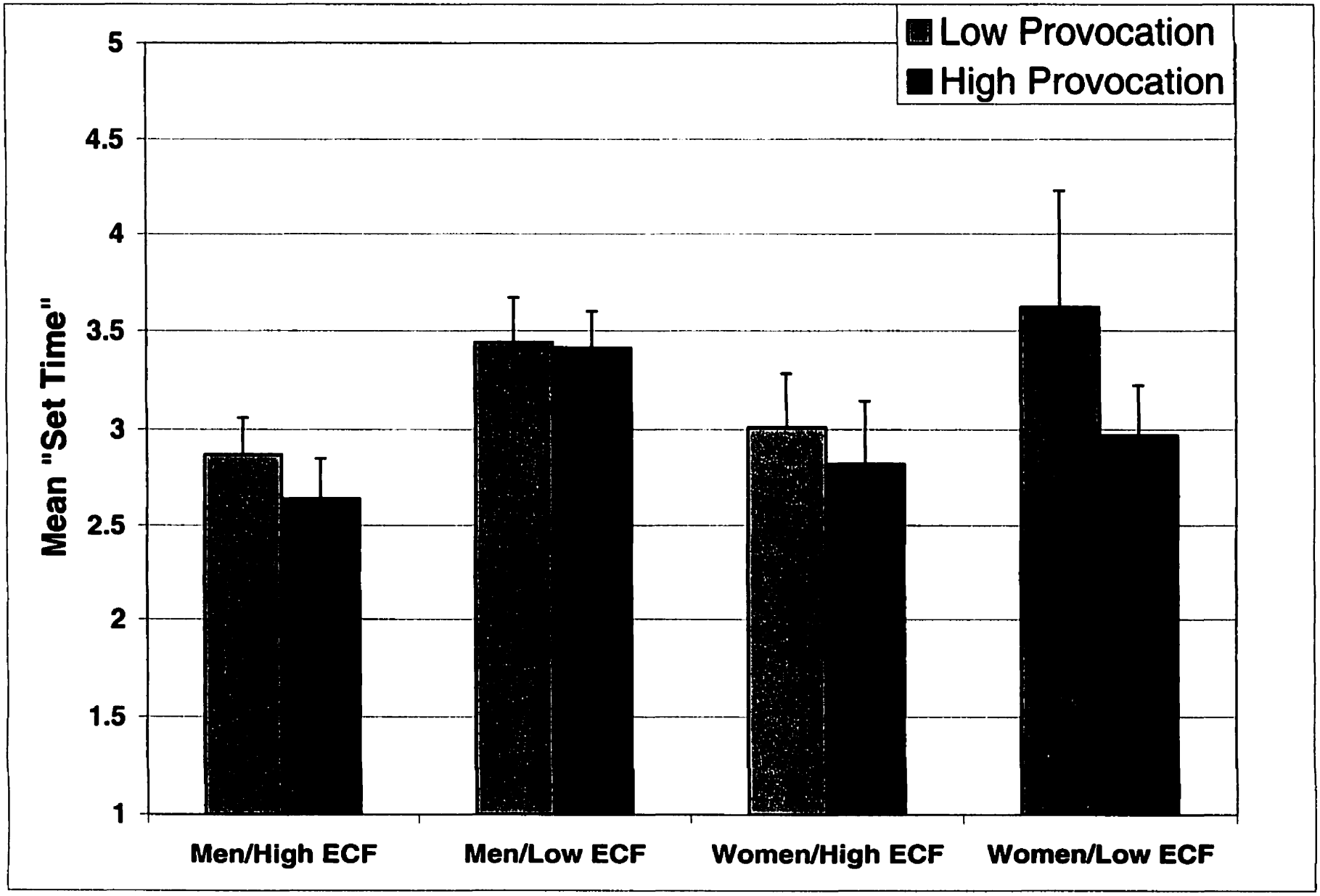

Low Provocation
High Provocation
Mean "Set Time"
5
4.5
4
3.5
3
2.5
2
1.5
1
Men/High ECF
Men/Low ECF
Women/High ECF
Women/Low ECF

Figure Caption

Figure III. Mean errors of commission on the Go/No-Go task by gender and executive functioning split.

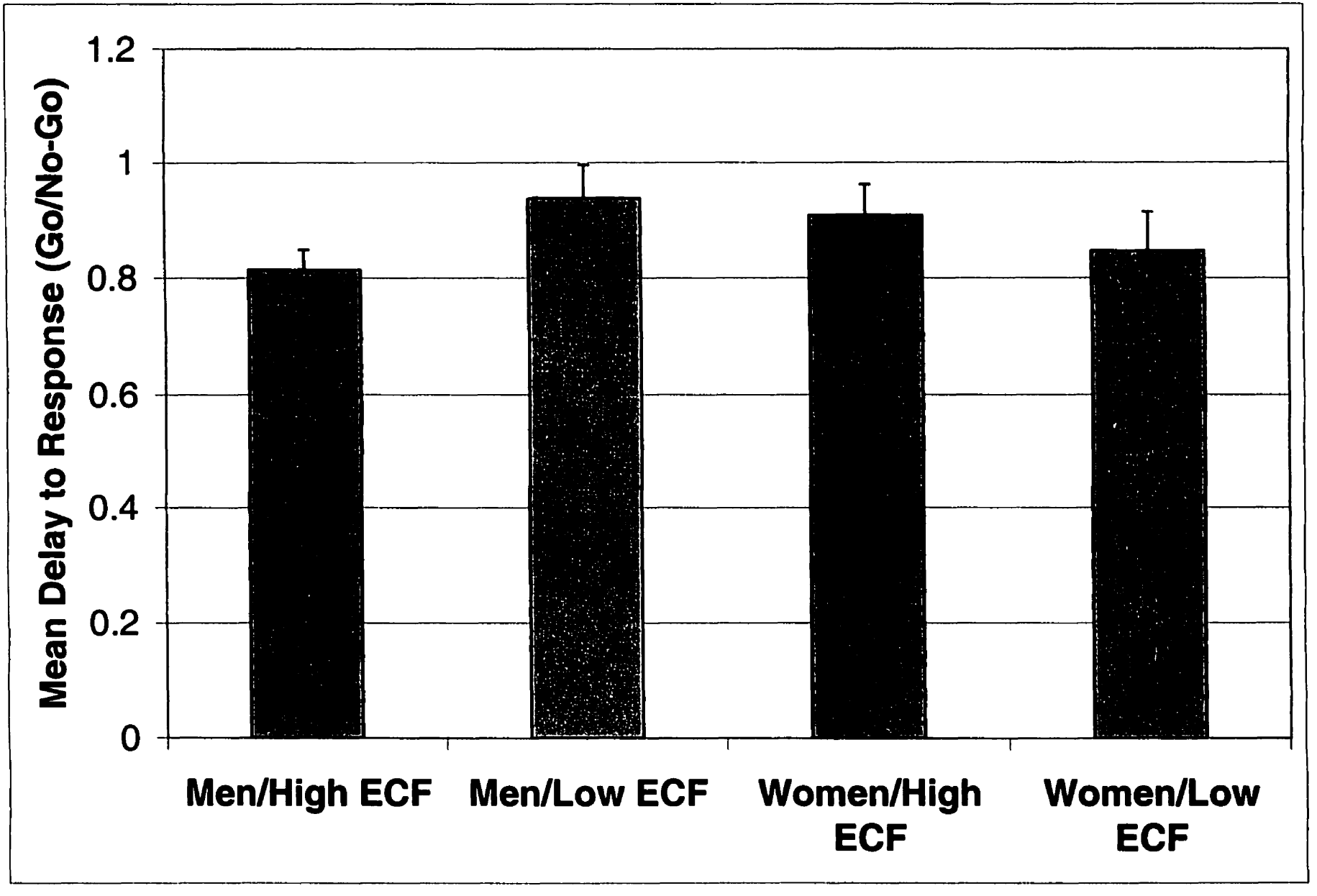
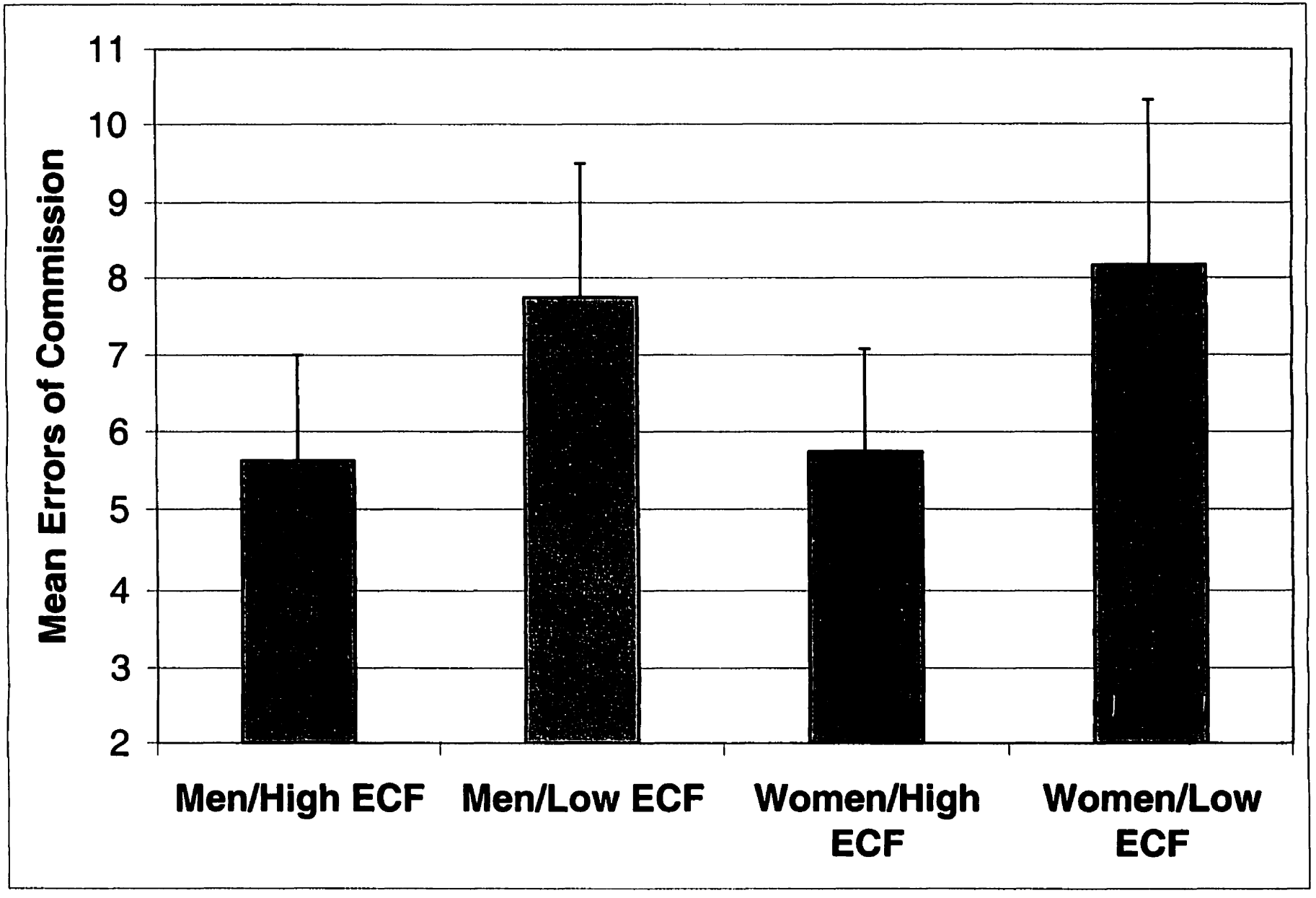

Mean Delay to Response (Go/No-Go)
1.2
1
0.8
0.6
0.4
0.2
0
Men/High ECF
Men/Low ECF
Women/High ECF
Women/Low ECF

Figure Caption

Figure IV. Mean reaction time to rewarding stimulus (in seconds) by gender and executive functioning split.


Mean Errors of Commission
11
10
9
8
7
6
5
4
3
2
Men/High ECF
Men/Low ECF
Women/High ECF
Women/Low ECF

General Discussion and Conclusions

The initial objective of this dissertation was to continue an already-initiated series of studies into the alcohol-aggression relationship in men, and the extent to which alcohol's interference with the so-called "executive cognitive functions" could explain this relationship. Although this rather narrow objective was pursued initially, the scope of the studies expanded considerably, making a concise review and synthesis of results problematic.

At the time this dissertation was being planned, as mentioned, there were relatively few studies specifically examined the relationships between poor executive function, alcohol intoxication, and aggressive behaviour (Giancola & Zeichner, 1994; Lau, et al., 1995; Lau & Pihl, 1996; Seguin et al, 1995). Thus, there were clearly several different directions in which continued studies could proceed. The first study contained in this dissertation was intended to examine whether alcohol intoxication had the capacity to render cognitively intact men impulsively aggressive. What was discovered was that alcohol does indeed impair ECF in these men – but apparently not enough to elicit impulsive aggression. The results of the second and third studies, intended to investigate gender-differences in aggression, and the possible role of executive function, suggest three important findings: women may not be so dissimilar from men in terms of their propensity to manifest aggression; alcohol appears to have less of an aggression-eliciting effect in women

than it does in men; and aggression in women appears highly related to level of executive function.

The results of the first three studies are consequential inasmuch as they help to elucidate the sometimes-inconsistent findings in two branches of the aggression literature; that of alcohol and aggression, and that of gender differences in aggression. Decades of studies have demonstrated that, in between-group designs, A) alcohol tends to make aggression more likely, and B) women are less aggressive than men. However, the fact remains that many people do not become aggressive in response to alcohol, and furthermore, many women are far more aggressive than the average man is. The studies appear to demonstrate that executive function is related to who would and would not become aggressive when provoked, irrespective of gender. Furthermore, it appeared important as a "protective" variable against alcohol-related aggression, again irrespective of gender. Previous investigations of alcohol and gender effects may have been corrupted by discrepant executive capacities represented in the groups to be compared.

The studies also appear to begin to elucidate how it is that poor versus superior executive functioning predisposes to, or protects from, aggressive and potentially injurious social interactions. Specifically, study four was intended to investigate one of the predominant hypotheses of the ECF-aggression relationship, that individuals with poor ECF act aggressively because they respond in kind to provocation in a rash, rapid,

impulsive nature, without consideration of consequences. This was a reasonable hypothesis, considering all the accumulated evidence, but was not one that was supported by the study. In fact, both men and women with low-ECF, despite behaving more aggressively, appeared to do so in a relatively slow, contemplative fashion. This may relate to the distinction between automatic and controlled cognitive processes; executive function is a complex cognitive process which involves the integration of several sources of sensory and affective input to reach a conclusion and guide behaviour (Robbins, 1998).

The impulsivity hypothesis was likely predicated on the notion that "poor ECF" or "low ECF" meant a bypassing of the executive process. Instead, it may be that the control aspects of executive function may be impassable, and what we observe in low-ECF participants is not a bypassing of the inhibitory function of ECF, per se, but instead the product of an underdeveloped or somehow faulty inhibitory process. According to a prominent theory, executive function should be conceptualised as an interaction of multiple information processing modules, all of which co-operate to result in an "integrated behavioural script" (Goldman-Rakic, 1987, 1998). The dysexecutive syndrome, manifested in these studies as heightened aggression, is therefore understood as a failure to adaptively retrieve information from various sources, including long-term memory, and process them "on-line". This process failure takes time, and thus behaviour associated with the dysexecutive syndrome should be perceived

as "making bad decisions slowly", rather than as an expression of impulsivity or disinhibition (Cockburn, 1995).

If the control aspects of executive function are not being bypassed (i.e. there is processing), but the response that is manifested is still maladaptive (i.e. aggression), then there must be other explanations for the ECF-aggression relationship. At this point it is instructive to reconsider the various explanations for the alcohol-aggression relationship. Several of those theories involved a utilisation of various sources of information, be they affective, cognitive, and/or physiological. Using this literature as a framework, it is possible to pose a speculative model the ECF-aggression relationship, and suggest directions for future research.

Directions for Future Research: A Speculative Model of the ECF-Aggression Relationship

The manner in which aberrant executive function (whatever its underlying reason) heightens likelihood of aggression is likely through an alteration of a variety of capacities and/or functions which interact to govern appropriate, pro-social behaviour. As the speculative literature on the alcohol-aggression relationship makes clear, these capacities are numerous and likely interactive. The rather deliberate responses of poor-ECF participants in study four suggest that aggression in not an automatic response. However, the processing that occurs prior to choice and utilisation of a specific action package is somehow altered, considering

that the selected response tends to be aggressive in nature. The relevant issue is *what* is altered.

The first element of this speculative model is social-cognitive in nature, and incorporates elements of Dodge's (1986) and Hull's (1981) models, both of which involve the perception of socially relevant behaviour and/or information. Dodge's model is based on the notion that alcohol interferes with social-information processing in such a way as to fail to interpret social interactions accurately, and generate appropriate behavioural responses. Hull's model, conversely, suggests that alcohol interferes with some element of self-awareness, such that social and environmental cues that would normally inhibit aggression are misperceived or simply missed.

As these are clearly complex cognitive processes, it seems wholly reasonable that individuals with poor executive functioning may have similar difficulties interpreting social behaviour - both their own and that of others. Dodge has previously extrapolated his theories beyond the alcohol-aggression relationship, demonstrating that adolescents with histories of aggression inaccurately interpret ambiguous interpersonal encounters (Dodge, Price, Bachorowski, & Newman, 1990). An important social aspect of Dodge's model, and in the inhibition of aggression, is empathy, or stated differently, the ability to "perspective take" (Davis, 1983). One research group has also postulated a relationship between cognitive processes and social processing, albeit in the opposite direction

presented here; Richardson and colleagues have suggested that it is a dispositional ability to take perspective which allows certain individuals to maintain sufficient cognitive function in threatening situations (Richardson, Hammock, Smith, Gardner, Signo, 1994).

In terms of faulty interpretation of behaviour of the self, there is some limited and preliminary data to implicate executive function. In a small exploratory study (Appendix A), a sample of high and low-ECF men and women were tested on the Taylor aggression paradigm, and also asked to self-rate their assertiveness. Assertion is typically defined as a pro-social and adaptive response to social interactions (Hollandsworth, 1977). Because we know low-ECF individuals to be more aggressive when provoked, and because provocation (in some form) is not an exceedingly low-frequency event, we would take self-reported assertion in the low-ECF participants to be support of a disrupted or inaccurate self-awareness. That is in fact what was demonstrated; low-ECF participants (irrespective of gender) were much more likely to self-rate themselves as assertive and pro-social than were their high-ECF counterparts (please see Appendix A for a fuller description of the study). Extant literature and this preliminary study conjoin to support the theory that some aspect of interrupted social-information processing is involved in the ECF-aggression relationship.

Another explanation of the alcohol-aggression relationship which may well be valid for the ECF-aggression relationship is that there is some interruption in the threat-detection system, such that cues that would

normally elicit psychophysiological arousal (i.e. fear responses) fail to do so. Another study peripheral to this dissertation lends credence to this claim (please see Appendix B). In this study, intoxicated participants both acted more aggressively than their non-intoxicated peers and had dramatically attenuated arousal responses to the presentation of threat (in terms of both heart rate increase and blood pressure increase).

This result may be relevant to the discussion at hand, if we relate this finding to the previously discussed literature that suggests alcohol-intoxication produces cognitive impairment specifically in capacities thought to be mediated by prefrontal cortex and associated structures. If that is in fact the case, then it may be that alcohol-intoxication produces a behavioural and a physiological response profile analogous to that of executive function abnormality. More plainly, it has been suggested that we may be able to model executive function deficits simply by alcohol-impairing normal individuals (Fillmore &Vogel-Sprott, 1999). Specifically, recent behavioural neuroimaging research has demonstrated that even moderate doses of alcohol can impair executive-type cognitive capacities, and that this effect is manifested as a result of decreased function of a frontostriatal network (Vogel-Sprott, Easdon, Fillmore, Finn, & Justus, 2001). In light of this, we might surmise that the pattern of diminished arousal and (concomitant increased aggression) in intoxicated participants may characterise low-ECF individuals as well.

Modelling the ECF-aggression relationship involves, then, not a rapid, rash, impulsive disinhibition of aggressive behaviour, but instead the likely failure of perhaps several parallel inhibitory mechanisms. The at-risk individual, with poor or somehow inadequate executive function, interacts socially in a variety of inept ways. Impaired by diminished self-awareness, and not inhibited by typical psychophysiological arousal, this individual fails to inhibit impulsive and likely provocative behaviour. Furthermore, the at-risk individual may fail to correctly interpret social situations in which another initiates a potentially provocative interaction, and by virtue of this may fail to initiate and utilise more adaptive behavioural response options (for example, assertion, submission, and/or flight).

What is at this time important to investigate is to what extent this currently speculative model can be validated. This could be accomplished in the following fashion; in terms of the social-information processing model, Dodge and Crick (1990) developed a laboratory protocol for assessing these sorts of capacities. As previously mentioned, one study (Sayette, Wilson & Elias, 1993) made use of this protocol to test the extent to which alcohol could interfere with these sorts of abilities, finding moderate confirmation. Examining the relationship between our measures of executive function and performance on this protocol would go a long way towards assessing this hypothesis.

In terms of the threat-detection hypothesis, testing would also be relatively simple. The relationship between executive function and psychophysiological measures of standard stress responses (heart rate, electrodermal response, perhaps respiration) in response to threatening situations could be easily examined. However, what would be important would be to vary the complexity of the threat. For example, while there may or not be aberrant response to a classic tone-shock paradigm (for example, see Conrod, Pihl & Ditto, 1995), it would be of greater interest to see what differential patterns of arousal would be produced by more complex threatening situations, specifically threatening situations in which there is some social component. This distinction is suggested due to the finding in Study Four that poor ECF-individuals react differently to simple tasks (Go/No-Go) than they do to complex ones in which there is some social component (Taylor aggression paradigm).

This speculative model is a useful tool for continued research into the ECF-aggression relationship, inasmuch as it is explanatory, yet is readily testable and imminently falsifiable. It may well be that the mechanisms alluded to here are not in any way related to the means by which inferior executive function potentiates aggressive behaviour. However, by exploring some of the putative mechanisms and proposing others, the construction of a model assists in advancing the understanding of who becomes aggressive and who does not, under what conditions, and moreover why. Continuing to test, modify and hopefully validate this

model will assist psychologists, clinicians, and perhaps even policy makers in ameliorating the societal malaise of interpersonal violence.

Conjectural Causes of Poor Executive Function

The participants described in this dissertation as having "poor", "sub-average" or even "aberrant" executive functioning have, in theory, some neurophysiological irregularity. That is, their brains do not appear as capable of the sorts of complex cognitive capacities which underlie the "executive cognitive" construct. But what is not widely speculated upon is why this is the case. Because participants in these studies were screened for head injury, psychiatric and physical illness, as well as current substance abuse, it is unlikely that the cognitive deficits observed were so for these reasons. More plainly, these cognitive abnormalities exist without any clear etiology, and likely without a lesion, per se. Discussion of what may cause ECF deficits is important inasmuch as primary interventions may become possible once the etiological factors are better understood.

There are several possible ways in which an individual may demonstrate poor executive function. Foremost of these is simply that there is considerable individual variation in brain development (Kolb & Wishaw, 1990). However, expression of complex cognitive capacities is necessarily the result of an interaction between heredity and environment. Prenatal exposure to alcohol has been recently demonstrated as a robust

predictor of executive function deficits (Mattson, Goodman, Caine, Delis, & Riley, 1999). Moreover, maternal malnutrition, exposure to infectious disease, and even psychosocial stress can have effects on neonate brain development, and, theoretically, future executive function (Mrzljak, 1990).

Post-natal influences are also likely important. Child malnutrition, exposure to neurotoxin, sensory and-or environmental deprivation may all lead to poor ECF (Moffitt, 1990). Considering that most neuroanatomists maintain that ECF is governed by a particularly rich connectivity between prefrontal cortex and subcortical structures (see Roberts, et al., 1998 for an extensive review), factors that facilitate synaptic branching in early life may be particularly important (Katz & Shatz, 1996). Executive functions are, like any other cognitive capacity, the end result of mechanisms fostering neural growth and diversity, triggered in response to complex experience (Quartz & Sejnowski, 1997).

Clearly, however, the manifestation of poor executive function is not exclusively related to structure. Function, in the form of neurotransmitter action, may also be highly important. Many researchers have suggested the importance of the serotonergic system in aggression (Virkkunin & Linnoila, 1993; Pihl & Lemarquand, 1998). Clinical populations characterised by increased aggression towards the self or others (including antisocial and borderline personality disorders) have been shown to have abnormalities in serotonergic function (Raine, 1993), and manipulations of the serotonergic system have been shown to be

capable of either increasing (Lemarquand et al., 1997) or decreasing aggression (Brizer, 1988). One of the main elements in most definitions of executive function is inhibition, especially inhibition of social an-or goal-directed behaviours. It has been postulated that serotonergic dysfunction may underlie inadequate function of the behavioural inhibition system (BIS; Gray, 1975; Fowles, 1988), which concomitantly underlies poorly governed, sometime antisocial behaviour (Scerbo & Raine, 1992). It may be that neuroanatomical insult and-or poverty of environmental stimulation has an effect on the functioning of the serotonergic system such that executive function is under-developed.

Potential Implications

As with any research project, the initial goal of these studies was, simply, a better understanding of the variables studied. However, it is insufficient to postulate a relationship between variables without further considering what this might practically mean. Although it is beyond the scope of the dissertation to comprehensively detail all practical applications, a few means by which the work may show future utility are briefly detailed below.

Early Interventions

If we are to accept the proposition that poor executive function is related to likelihood of aggressive response to provocative, disagreeable or otherwise hostile circumstances, then we should consider the extent to

which improvement of this capacity might ameliorate this relationship. Because interventions to reduce propensity for aggressive and/or antisocial acts are known to be more effective if instituted early in life (Kazdin, 1988), an emphasis should be put on early identification of problematic behaviours (i.e. behaviours which predict aggression or conduct disorder in children and antisociality or violent crime in adulthood). Once tools which reliably identify children as "at-risk" for aggressive or other unacceptable behaviours are established, then interventions should be aimed at strengthening the putative underlying cognitive deficit (Barkley, 1997). Various methods for accomplishing this have been suggested. For example, Giancola (2000b) has suggested that some of the same tools that can be used to measure certain aspects of executive function can also be used to strengthen these capacities. Specifically, tests of planning, sequencing, and strategy formulation could be administered repetitively, and, under appropriate tutelage, be used to strengthen the capacities they were intended to measure. There is evidence to suggest that this type of intervention is possible. Goldman and colleagues have demonstrated efficacy in neuropsychological remediating techniques with both young and older alcoholics (Forsberg & Goldman, 1987; Goldman, Klisz, & Williams, 1985). Perhaps more importantly, these neuropsychological benefits have been shown to generalise to other tasks involving similar cognitive capacities.

One might suggest that interventions of this type would be arduous for the adult, tedious for the child, and therefore difficult to implement. However, interventions need not be labour-intensive clinician or teacher, or boring for the child; evidence exists that interventions to enhance cognitive capacities can be implemented via computer, and in a fashion that is appealing to the child (Tallal et al., 1996). Subsequent to sufficient time mastering the computer tasks, at-risk children could be further taught to implement these newly developed skills in actual provocative dyads. Clinician feedback subsequent to the dyads serves to further shape and reinforce appropriate behavioural responses to previously problematic interpersonal situations.

Forensic Prediction

Aggression and violent crime carry enormous cost, both personally and socially. The clinical prediction of violent recidivism has a long but somewhat unsuccessful history. In fact, there is a voluminous literature which details the extent to which trained, experienced, and practising forensic clinicians are very poor at make accurate decisions regarding likelihood of aggression (see Quinsey, Harris, Rice, & Cormier, 1998, for an extensive review). It is readily apparent that clinicians tend to be susceptible to similar errors of judgement as are all humans (see Garb, 2000 for an extensive review). Perhaps the most profound error of

prediction is a failure to attend to all relevant, even if seemingly discrepant, sources of information.

Recent discussions on the prediction of aggression, dangerousness, and violent crime have suggested that mathematical (i.e. actuarial) models have much better predictive utility than do clinicians. Quinsey et al. (1998) review the literature on prediction of recidivism and demonstrate that actuarial approaches significantly out-perform "clinician judgement"; in the systematic consideration of archival data, the actuarial model made significantly fewer false positives (i.e. release of prisoners who then re-offend). Monahan (1981) details several issues that clinicians must attend to when making predictions about dangerousness. Among these are: history of violence, current stresses, base rate of violence among similar persons, demographic characteristics, and perhaps most importantly for the present discussion, cognitive and affective predispositions to violence.

As mentioned, research has begun to demonstrate the predictive utility of actuarial approaches to risk assessment. However, these approaches have been based almost exclusively on historical factors (history of crime, for example) and have not included, as Monahan suggested, other factors, cognitive ones specifically. It is possible that actuarial approaches including executive function as a predictor may out-predict models without it.

Limitations

Clearly, the studies in this dissertation suggest a feasible means of making predictions regarding the likelihood of aggressive response to certain provocative interpersonal situations. However, it must be acknowledged that the research herein was conducted with adult, non-clinical, non-forensic populations with no history of head injury, no substance abuse, or other form of psychopathology. In short, the model of the ECF-aggression relationship detailed herein is based on a not particularly representative sample, if in fact we expect this model to explain aggression and violent crime in vivo. Moreover, the participants were all between the ages of 18 and 30; this is the demographic group most likely to act aggressively (as indexed by rates of arrest for violent crime; US Bureau of Justice Statistics), but extrapolation to other age groups, especially adolescents, seems important.

The studies contained in this dissertation contribute much to the understanding of aggressive behaviour. As we continue to better understand the underlying causes of aggression, the better we are able to predict it, treat it, and most importantly, prevent it.

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APPENDIX A

(Intended for submission as a Brief Report, Aggressive Behavior)

Executive Cognitive Functioning, Aggression and Assertion: Is There a Problem of Social-Information Processing?

Peter N. S. Hoaken, Jane Simpson, Jennifer Crotogino,

& R.O. Pihl

Department of Psychology

McGill University

Recent research has demonstrated that poor performance on tests of executive cognitive function (ECF) predicts aggression in both men and women (Giancola & Zeichner, 1995; Hoaken, Strickler & Pihl, submitted manuscript). However, why individuals who appear deficient on these tests behave more aggressively than their peers remains poorly understood. One unexplored explanation for this relationship is that the cognitive capacities which underlie executive function are related to those which mediate social-information processing. This latter construct has been demonstrated previously to be related to aggressive behavior (Dodge and Crick, 1990; Sayette, Wilson, & Elias, 1993). This model suggests that aggressive individuals selectively attend to aggressive cues, are more likely to interpret ambiguous cues as aggressive, have a limited number of responses to choose from, and evaluate aggressive behavior more favorably in terms of expected outcomes and self-efficacy. A related possibility is that low-ECF, aggressive individuals also systematically misinterpret their own behavior, deeming it pro-social when it is not.

If that is the case, then these individuals are likely to confuse the concept of assertion. Assertion is a social behavior, like aggression, but is typically defined as a pro-social and adaptive response to social interactions (Hollandsworth, 1977), whereas aggression is usually defined as socially inappropriate, delivering threat and/or punishment (Baron & Richardson, 1994). Aggression may be manifested in low-ECF

individuals because they misinterpret other's actions as aggressive, or because they misperceive their own actions as assertive when they are not.

This study, using a state-measure of aggression and a trait measure of assertion, was designed to study this hypothesis. We hypothesize that low-ECF individuals will 1) be more aggressive on the aggression paradigm, and 2) self-report levels of assertion comparable to their high-ECF peers.

Method

Participants

Participants were recruited from McGill University and from a local Montreal newspaper. Participants were excluded if they had a previous head injury, a learning disability, or familiarity with psychological experimentation.

Measures

Neurocognitive Measures: Four tests of executive function were used. These included two versions of the conditional associative-learning task (CALT; Petrides, 1985) and two versions of the self-ordered pointing task (SOP; Petrides & Milner, 1982). Impairment on these task appears to reflect poor organizational skills, strategy formulation, and response monitoring (Petrides et al., 1993), the cognitive capacities underlying ECF (Mega & Cummings, 1994).

Assertion Measure: The Rathus Assertiveness Schedule (RAS; Rathus, 1973) is a 30-item self report measure. The RAS is a widely used, valid and reliable measure of how participants would behave in situations where assertive behavior could be used for profit.

Aggression Measure: The Taylor Aggression Paradigm (TAP; Taylor, 1967) is a behavioral measure of aggression, in which the participant is told he is competing with another participant (a sham "opponent") on a competitive reaction time task. This paradigm, detailed below, has demonstrable construct validity (Giancola & Chermack, 1998).

Procedure

On the first day of testing, participants gave informed consent, and completed the battery of neurocognitive tests. The four scores from the three neurocognitive tests were standardized and summed, and quartiles were calculated from the summed Z-scores. Participants within the highest or lowest 25% were booked for the second day of experimentation.

On the second day of testing, participants completed the Rathus Assertiveness Schedule, then "competed" on the Taylor paradigm. After a determination of participant pain threshold, the experimenter read the reaction-time instructions to the participant. The aggression paradigm consisted of 26 trials. At the beginning of each the trial the participant selected a shock level to administer to his or her "opponent", should he or

she win that given trial. The participant then "competed" on the reaction time trial, then was given feedback about success; wins allowing the participant to shock the "opponent", losses meaning the participant would be shocked. The first thirteen trials were "low provocation" trials where the participant only received low-level shocks, levels one (lowest) to four. The last thirteen trials were high provocation, where the "opponent" always chose a shock between levels five and eight (highest). The dependent measure was the mean level of shock chosen by the participant in each provocation level.

Lastly, the participant was debriefed; the experimenter explained the true nature of the study and the necessity for deception.

Results

A total of 88 men and women were brought into the lab after a phone screening to complete the battery of neurocognitive tests. Of these, 13 men and 18 women fell into high or low quartile groups, and completed the aggression paradigm. The sample was divided into four groups according to gender and ECF quartile (male/HQ, n=7; male/LQ n=6; female/HQ=9; female/LQ=9).

The aggression data was analyzed in a 2x2x2 mixed design analysis of variance (ANOVA) with provocation as a repeated measure. Independent variables were gender and cognitive functioning quartile. This analysis revealed no 3-way interaction. The three two-way

interactions were also non-significant. The three main effects were all significant.

Insert Figure 1 about here

Main effects of provocation ($F(1, 27) = 59.66$, $p = .000$), gender ($F(1,27) = 8.79$, $p = .006$), and quartile ($F(1,27) = 9.49$, $p = .005$) all replicate findings from past studies. These results are represented in Figure 1.

In order to examine the relationship between executive function and assertion, Rathus scores were correlated with standardized and summed ECF scores. The 2-tailed Pearson correlation coefficient, $r=.298$, $p=.104$. Although non-significant, this correlation is not

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Insert Figure 2 about here

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inconsiderable, and increases to $r=.369$, $p=.045$ with the elimination of one outlier. This relationship is represented in Figure 2.

Discussion

This study corroborates previous ones and supports our first hypothesis. However, the second hypothesis was not confirmed; the results indicate that although low-ECF individuals demonstrate more behavioral aggression than their peers, they almost without fail report themselves as behaviorally assertive and pro-social.

Dodge and Crick (1990) have argued that aggressive behavior is the result of a cognitive impairment in processing social cues. This model emphasizes the appraisal of external social cues, but the current study raises the possibility that aggressive individuals may also have difficulty appraising their own actions. Aggressive individuals may believe that harming or intending to harm another human being is a legitimate way to stand up for their rights or to get somebody to comply with a request. Low-ECF individuals, pseudo-psychopathically, may have difficulty differentiating pro- from anti-social behaviors.

Clearly, this study has major limitations. Only one assertion measure was used, a self-report measure, a trait measure, and a measure from which separate scores of aggression and assertion cannot be derived. Sample size was small. Behavioral measures of assertiveness were not utilized.

That being said, this study offers preliminary evidence that capacity for social-information processing, in terms of the analysis of self-action, merits further investigation and may help elucidate the ECF-aggression relationship.

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Figure Caption

Figure 1. Mean shock selected as a function of provocation and cognitive functioning quartile, for both men and women.

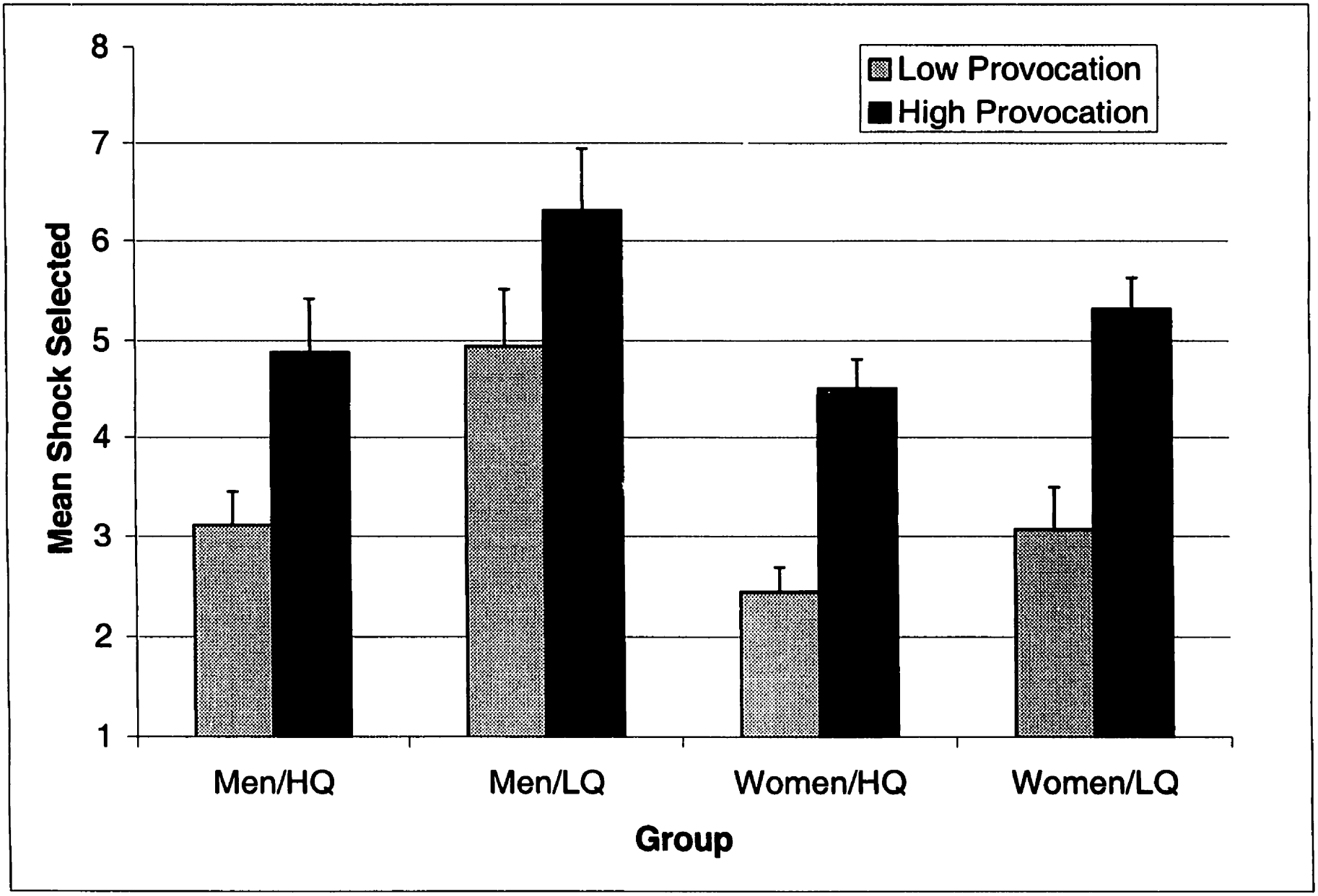
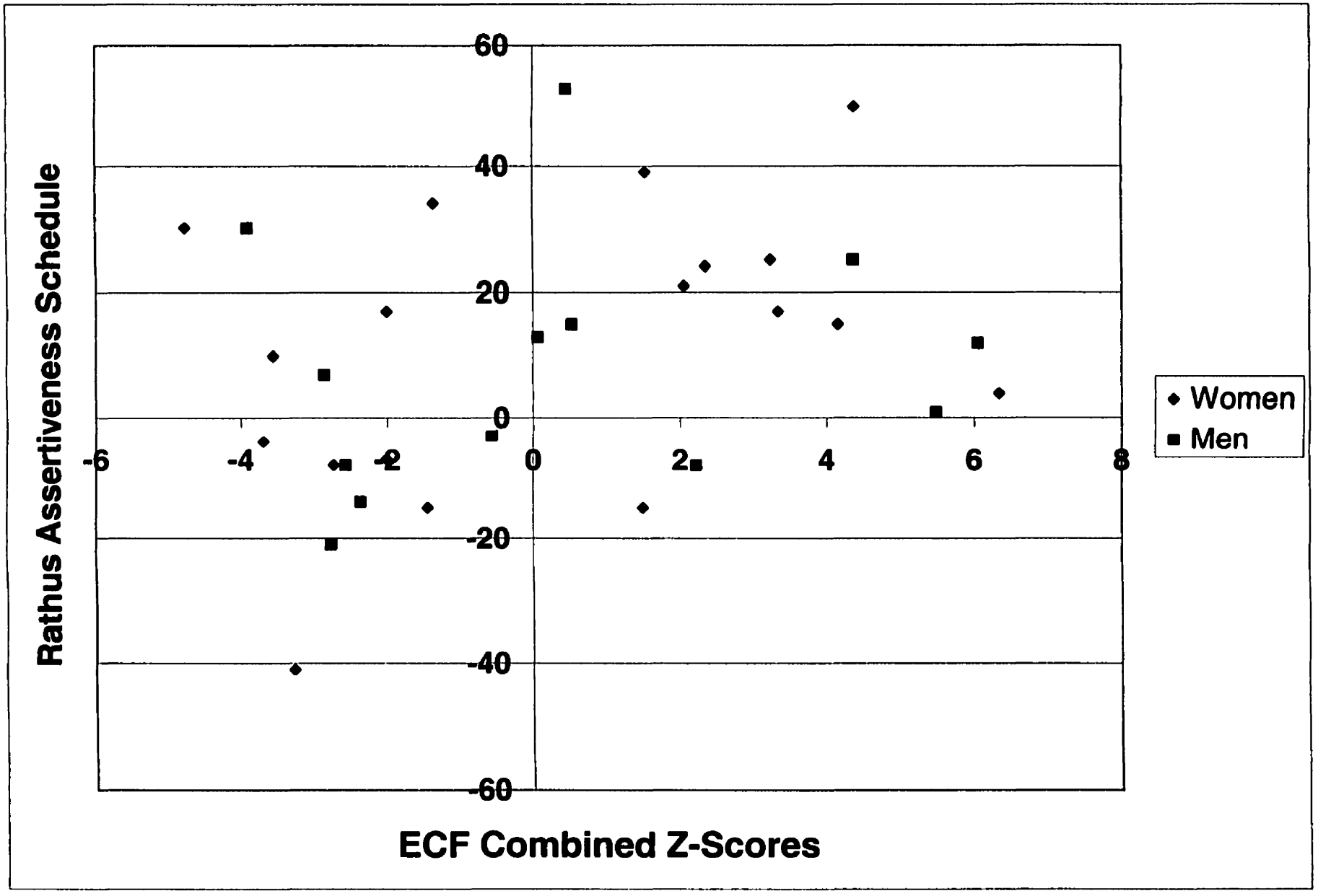

Low Provocation
High Provocation
Mean Shock Selected
8
7
6
5
4
3
2
1
Men/HQ
Men/LQ
Women/HQ
Women/LQ
Group

Figure Caption

Figure 2. Scatterplot representing Rathus Assertiveness schedule scores as a function of the standardized and combined executive cognitive function test scores.


Rathus Assertiveness Schedule
60
40
20
0
-20
-40
-60
-6
-4
-2
0
2
4
6
8
Women
Men
ECF Combined Z-Scores

APPENDIX B

(Manuscript in Progress. To be submitted to

Journal of Studies on Alcohol.)

Effects of Alcohol on Cardiovascular Reactivity and the Mediation of Aggressive Behaviour

Peter N.S. Hoaken, Tavis Campbell, & R.O. Pihl

Department of Psychology

McGill University

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Correspondance regarding this manuscript should be sent to: Peter N. S. Hoaken, Department of Psychology, 1205 Dr. Penfield Ave., McGill University, Montreal, PQ, Canada, H3A 1B1.

Abstract

Research has shown that the relationship between alcohol and aggression is multifactorial and complex. Recent models have proposed several pharmacological means by which alcohol may produce heightened aggression, among them that alcohol may both hyper-arouse the reward system and diminish the threat detection system. The current study examined these hypotheses employing heart rate and blood pressure as physiological indices of arousal, examining whether arousal differed by alcohol group, and if this related to level of aggression. Participants were 32 males and 32 females, aged 18-30, screened for physical and psychological disorder, who competed on the Taylor aggression paradigm. The gender groups were further split into half sober, half intoxicated. Arousal was measured at baseline, post beverage consumption, and post-aggression paradigm. Participants in the alcohol condition initially demonstrated slight heart rate elevations and blood pressure decreases. However, those participants showed little arousal in response to the aggression paradigm, while sober participants demonstrated considerable arousal on both indices. Intoxicated participants were more aggressive than sober controls; men and women did not differ significantly. Regression analyses demonstrated that change in systolic blood pressure from post-beverage consumption to post-aggression paradigm accounted for a significant percentage of the variance in terms of aggression

manifested. These results lend direct support to the stress-response dampening model of the alcohol-aggression relationship.

Effects of Alcohol on Cardiovascular Reactivity and the Mediation of Aggressive Behavior

One of the most important situational determinants of interpersonal aggression is alcohol intoxication. One study demonstrated that more than half of perpetrators and approximately 45% of victims of violent crime consumed alcohol prior to the offence in question (Murdoch, Pihl, and Ross, 1990). Furthermore, several meta-analyses of relevant experimental work have demonstrated unequivocally that intoxicated subjects are more verbally and physically aggressive than sober controls (Bushman & Cooper, 1990; Bushman, 1993). Thus, although the manifestation of aggression is clearly multifactorial and complex (Raine, 1993; Pihl & Peterson, 1995), alcohol intoxication appears to be of considerable importance. However, the mechanisms through which alcohol elicits heightened aggression are not completely understood.

Recent models (Pihl & Peterson, 1995; Pihl & Hoaken, 2001) have suggested several distinct but not necessarily mutually exclusive pharmacological effects of alcohol that may increase the likelihood of aggressive behavior. Included in these are the altering effects alcohol has on pain sensitivity (Gustafson 1985), and the altering effects alcohol has on certain cognitive abilities, specifically the so-called "executive cognitive functions" (Hoaken, Giancola & Pihl, 1998; Giancola, 2000). The models also suggest that alcohol may interfere with the system that mediates reward, as well as the system that governs threat detection. The evidence

for these latter hypotheses is currently, for the most part, indirect. As such, the present investigation was intended to assess, via cardiovascular indices of psychophysiological arousal, the extent to which evidence for these hypotheses could be demonstrated when eliciting alcohol-related aggression in a laboratory setting.

It is widely agreed that alcohol, particularly on the ascending limb of the blood-alcohol concentration curve, produces psychomotor stimulation. The psychomotor effects of alcohol, which are simplistically analogous to those of other stimulants, appear pharmacologically mediated by the dopaminerigic system (Gessa, Muntone, Collu, Vargiu, & Mereu, 1985). Alcohol appears, for example, to lead to increased firing of dopaminergic neurons in various areas of the mesocorticolimbic system (Harris, Brodie, & Dunwiddie, 1992). Whatever the mechanism, low and moderate doses of alcohol clearly lead to stimulation, in both animals and humans. In rats, for example, alcohol produces increased locomotion, rearing and exploration; in humans subjective increases in power, expansiveness, euphoria, and energy are reported. Alcohol has also been shown to lead to increased heart rate in humans (Rush, Higgins, Hughes, & Bickel, 1993; Conrod, Peterson, Pihl, & Mankowski, 1997). In certain populations, such as young males at high risk for alcoholism, this heart rate response is especially pronounced (Stewart et al. 1992), and sometimes manifests behaviorally as increased sensation seeking (Finn et al 1992), as well as approach behavior and dominance (Pihl & Peterson,

1995). Escalation of these sorts of behaviors leads to greater confrontation, greater provocation, and as a result, greater likelihood of an aggressive or violent encounter. As would be expected, these aforementioned 'at risk" young men have been shown to be much more likely to have histories of conduct disorder and antisocial personality (Pihl, Peterson, & Finn, 1990).

This is not to say that the stimulant effects of alcohol are apparent only in clinical populations. Indeed, there is much evidence that alcohol leads to heart rate increases in normal men (Higgins, Rush, Bickel, & Hughes, 1993; Bruce, Shestowsky, Meyerovitch, & Pihl, 1999) and women (Peterson, Pihl, Gianoulakis, Conrod, et al., 1996). If the cardiovascular stimulating effects of alcohol are demonstrable in normal participants, it may well be that the behavioral effects – i.e. approach, sensation seeking, dominance - are likely to be manifested as well.

Paradoxically, alcohol has also been associated with patterns of cardiovascular dampening. For example, although alcohol consumption alone has been shown to lead to increases in heart rate, it has at the same time occasioned decreases in blood pressure in both animals (Piano, Holm, Melchior, & Ferguson, 1991) and humans (Rush et al., 1993; Higgins et al., 1993). However, most of the research on the cardiovascular effects of alcohol consumption has developed out of what was first referred to as the tension-reduction hypothesis of alcohol consumption (Conger, 1951; Conger 1956), which was later modified to the drive-reduction theory

(Dollard & Miller, 1956), and eventually became known as alcohol stress-response dampening (SRD; Levenson, Sher, Grossman, Newman, & Newlin, 1980). What these theories all argued, essentially, is that alcohol is consumed because it is negatively reinforcing; people drink because of an observed amelioration of the adverse effects of various psychosocial stressors. Investigations of this hypothesis eventually revealed that when faced with stressful or provocative stimuli and/or situations, individuals who had consumed alcohol demonstrated a muting of typical psychophysiological stress responses. For example, alcohol's dampening effects on heart rate has been shown in response to non-social stimuli such as an aversive shock (Eisenhofer, Lambie, & Johnson, 1986; Levenson, Oyama, & Meek, 1987) and a loud noise (Lehrer & Taylor, 1974), as well as social interactions (Sher & Walitzer, 1986; Wilson, Abrams, & Lipscomb, 1980). Alcohol has also been shown to enact a stress-reduction effect on both systolic blood pressure (Niauria, Wilson, & Westrick, 1988; Zeichner, Edwards & Cohen, 1985) and diastolic blood pressure (Eisenhofer et al., 1986; Wilson, Brick, Adler, Cocco, & Breslin, 1989). It should be noted that while these studies found significant cardiovascular stress-dampening responses, this pattern of results is not always consistently demonstrated (see Sayette, 1993a for a review). Interpretability of this literature is made difficult due to inconsistency of the methodologies involved. It has been recommended that studies investigating the stress-response dampening effects of alcohol should

ensure pre- and post-measurement and incorporate a control group in order to distinguish the effects of alcohol consumption alone from the alcohol-stress interaction (Sayette, 1993a, 1993b).

The cardiovascular stress-dampening response is relevant to the alcohol-aggression relationship inasmuch as threat is typically seen as inhibiting aggressive responses; it has commonly been argued that there is a negative correlation between aggression and anxiety or fear. Specifically, those who have reason to fear that their aggression will be punished are less likely to engage in aggressive acts. Threat without the inhibiting effect of alcohol has been associated with increased blood pressure and heart rate (Sinha, Lovallo, & Parsons, 1992; Holmes, & Will, 1985; Farrington, 1997; Gerin, Pieper, Levy, and Pickering, 1992). These physiological responses can be seen as "reminders" of the socialization process; arousal means threat, and threat means punishment. Thus fear should adaptively inhibit the sorts of behaviors that might initiate an aggressive interaction. However, inhibition of the threat-detection system means that the inhibition is itself inhibited, and socially and interpersonally inappropriate behaviors are more likely to be manifested.

In summary, alcohol's psychomotor stimulant effects may lead to increased sensation seeking and approach behavior, clearly behaviors which carry an element of danger. However, at the same time alcohol's dampening effects on reactivity may attenuate the magnitude of the inhibitory effect anxiety or fear normally would exercise on the expression

of potentially dangerous behaviors. As anxiety cues are reduced, individuals may be more likely to engage in behavior that has been previously associated with punishment or threat. Both of these effects can be seen to theoretically predispose individuals to alcohol-elicited increases in aggressive behavior.

Although there is much indirect evidence to support these contentions, the direct evidence remains scarce. The current study is intended to examine the effects of alcohol on heart rate and both systolic and diastolic blood pressure, relative to non-intoxicated controls. Furthermore, it is intended to investigate whether the patterns of arousal manifested in response to alcohol change when there is an alcohol-provocation interaction; specifically, intoxicated and sober participants will compete on the Taylor (1967) aggression paradigm in order to assess the interactive effects of alcohol and this activity on cardiovascular indices of arousal.

While there is an abundance of evidence supporting a positive relationship between alcohol and aggression in men, relatively few studies have been performed with women, most of them recently. Although it has generally been argued that men are more aggressive than women (Bettencourt & Miller, 1996), there are studies which do not support this contention (Hoaken & Pihl, 2000; Archer, 2000), and moreover there has been considerable controversy about the conditions under which this relationship holds (Eagly & Stephen, 1996).

Some researchers have suggested that sex differences arise from biological causes (Maccoby & Jacklin, 1980), but other studies have failed to support assertions about biological sex differences (Tieger, 1980). Although there is a paucity of research comparing autonomic dampening responses to alcohol among women, there is some evidence that the genders respond similarly (Stewart & Pihl, 1994). Due to the disagreement regarding the aggression-eliciting effects of alcohol in women, and the paucity of data investigating biological mediating factors, this investigation tested both male and female participants.

Thus, the present study was designed to investigate the aggression-eliciting effects of alcohol in normal adult men and women. More specifically, it was designed to observe the differential patterns of cardiovascular arousal that are exhibited in response to only alcohol, then in response to a combination of alcohol and provocation. Consistent with previous studies, it was hypothesized that intoxicated participants would demonstrate slightly increased heart rate and slightly decreased blood pressure in response to the dose of alcohol (Bruce et al., 1999; Rush et al., 1993). In terms of response to provocation and aggression, it was hypothesized that the intoxicated participants would manifest greater aggression, but would demonstrate a less pronounced pattern of physiological arousal. Finally, although results of recent studies with similar procedures have demonstrated inconsistent results in terms of gender differences (Giancola & Zeichner, 1995; Hoaken & Pihl, 2000),

men were expected to be more aggressive than women in their responses, regardless of intoxication.

Method

Subjects

A total of 64 social drinkers (32 men, 32 women) between the ages of 18 and 30, in good physical and mental health, were recruited through a local newspaper and campus advertisements. The criteria for nonalcoholic status were a score of 5 or less on the Michigan Alcoholism Screening Test (MAST; Pokorny, Miller, & Kaplan, 1972) and a clinical interview regarding previous or present drug or alcohol related problems. Subjects who reported medical treatment that contraindicated alcohol consumption, serious head injury, a diagnosed learning disability, pregnancy, or familiarity with psychological experimentation were excluded from participation. In order to control for fluctuations in aggressive behavior and alcohol effects that may be associated with phase of the menstrual cycle, female subjects were tested during the follicular phase of their menstrual cycle (days 5-10 of cycle), as estimated by self-reports of length of their three previous menstrual periods (Sutger, Goist & King, 1987). All subjects were requested to fast for four hours prior to the alcohol session. Subjects were offered $5 an hour for their participation.

Measures and Apparatus

Aggression was elicited and assessed with a modified version of 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 lit up to indicate the shock level chosen by the opponent. An IBM-compatible personal computer was used to run the aggression task and to record data. Shocks were administered via the Mark I Behavior Modifier (Farrall Instruments, Grand Islands, NB, Canada), connected to an electrode attached to the inner forearm, below the elbow of the non-dominant hand. Each subject monitored administrations of shocks to their fictitious opponent by viewing a DC ammeter provided for that purpose. A pre-recorded videotape of the same-sex sham opponent receiving instructions regarding performance on the aggression task was played to the subject to reinforce the subject's belief in the existence of the opponent.

Dependent cardiovascular measures were systolic and diastolic blood pressure (SBP, DBP, in mmHG) and heart rate (HR: in beats per minute) and were recorded with a Sunbeam sphygmomanoter. Each determination requires approximately 30 seconds and provides a score for both blood pressure and HR. The blood pressure cuff was placed on the subject's non-dominant arm. Blood pressure and HR values were recorded as the average of three causal readings taken at each of the measurement periods. The first measurement was taken when the participant arrived at the laboratory, prior to the consumption of the

alcohol or orange juice pre-beverage (baseline). The second measurement was forty minutes following the first measurement, subsequent to the consumption of beverage, and before commencing the aggression paradigm (post-beverage). The third measurement was immediately following the aggression paradigm (post-provocation).

Procedure

Upon arriving at the lab, participant's blood alcohol concentration (BAC) was measured to ensure sobriety; this was conducted using the Alco-sensor III (Intoximeters, Inc., St. Louis MO). Participants were then randomly assigned to the alcohol or no alcohol conditions. In the alcohol condition, participants were administered 1 milliliter per kilogram of body weight 95% alcohol USP units in three drinks of a 1:7 alcohol:orange juice solution. In the sober condition, the participants were administered three drinks of orange juice of equivalent volume. In each condition, participants were told explicitly what they were drinking. Drinks were consumed over a twenty-minute period. A twenty-minute waiting period followed to allow the subjects in the alcohol condition time to reach near peak BAC. BACs were then taken and recorded.

Each participants pain threshold for electric shock was determined by delivering a series of shocks from 0-255 units (0-5.63 ma) which increased stepwise by 5 units at a constant rate. Each participant was instructed to press a button in response to any shock he/she regarded as

painful (1) to stop the administration of the shock and (2) to reduce the level of the next shock by one step. The next shock therefore was one step lower than the shock that induced pressing the button. Pressing the button upon three consecutive presentations of the same shock intensity stopped shock delivery. This shock intensity was defined as the participants pain threshold.

The aggression task was then introduced as a competitive reaction-time task. Each participant was instructed to select a shock level that he/she would deliver to her opponent after winning a reaction-time trial. Following the reaction time task, the participant would be informed of the opponent's shock choice. If the participant "lost" that trial, she received that shock. Shock levels 1-8 increased from 28 units for level 1 to 100% of the participants given pain threshold for level 8, with intermediate shock levels being equal to 28 units plus 23%, 31%, 39%, 76%, 84%, and 92% of the difference between the participants given pain threshold and the initial 28 unit level. The nature of the increases of the shock intensity was decided upon in order to clearly define those shocks thought to minimally provoking (level 1-4) and those thought to be maximally provoking (levels 5-8). If the participant had "won" the reaction time trial, he/she would then administer the previously chosen shock to his/her opponent.

Following the instructions the experimenter then left briefly, telling each participant 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. As noted earlier, the participant was actually watching a pre-recorded videotape of a fictitious opponent receiving instructions.

The task itself consisted of 26 consecutive trials including a block of 12 trials followed by a transition trial, a second block of 12 trials, and a final trial. The opponent's shock choices ranged from 1-4 in the first block and 5-8 in the second block of trials. The computer randomly assigned the order of wins and losses as well as the opponent's shock choices. The opponent's shocks were all of either one-second or two second 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. If the participant was to receive two shocks at a certain level, they would receive one each of one-second and two-second duration. In both the transition trial and the final trial the opponent's shock choice was a 5. The objective measure of aggression was the mean shock selected for both the high and low provocation conditions. This measure reflects the magnitude of an individual's aggressive response to both low and high provocation.

Following the task, participants were given a questionnaire to assess their perception of the opponent, as well as their subjective rating of their aggression level to probe the completeness of the deception. The participants were then debriefed. Debriefing included an explanation of

the deception, its necessity, and a request to refrain from revealing the deception to others. Participants were compensated for their time, and if in the sober condition, were allowed to leave. Participants in the alcohol condition were retained in the lab until their blood alcohol concentrations dropped to 0.02.

Results

Subject Measures

A total of 64 participants were deemed admissible to participate in the study and were tested on the Taylor paradigm. Participants were divided into four groups: Group 1 consisted of males who consumed alcohol (n=16); Group 2 consisted of sober males (n=16); Group 3 consisted of females who consumed alcohol (n=16); and Group 4 consisted of sober females (n=16). An analysis of variance was conducted on blood alcohol concentrations. There were no significant differences

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Insert Table 1 about here

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between post-beverage and post-provocation measures. There was also no gender difference, either at post-beverage or post-provocation. These results are represented in Table 1.

Aggression Measure

An 2 x2x2 mixed design ANOVA was conducted on mean shock selections, with provocation as a repeated measure and gender and alcohol condition as between subject factors. There was no significant 3-way

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Insert Figure 1 about here

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interaction, nor were any of the three two-way interactions significant. There was a significant main effect of provocation ($F(1,58)=62.24$, $p<.001$). There was also a significant main effect of alcohol condition ($F(1,58)=6.71$, $p<.05$). There was no main effect of gender ($F(1,58)=2.34$, $p=.13$). However, analysis of simple main effects allowed better understanding of the relationship between variables.

Effects of Alcohol Within Gender Factor: There was a simple main effect of alcohol in men in both low provocation ($F(1,28)=4.58$, $p<.05$) and high provocation conditions ($F(1,28)=4.65$, $p<.05$), with intoxicated men demonstrating greater aggression than sober men. Conversely, there was no simple main effect of alcohol in women, at either the low ($F(1,28)=1.59$, $p=.22$) or high provocation conditions ($F(1,28)=1.07$, $p=.31$).

Effects of Gender Within Alcohol Factor: In the sober condition, there was no significant simple main effect of gender in either the low or high provocation conditions; that is, men's and women's aggressive responses were no different when sober. In the alcohol condition, there

was also no significant main effects of gender at low provocation; however at high provocation there was a trend, demonstrating intoxicated men to be more aggressive than intoxicated women ($F(1,28)=3.10$, $p<.10$).

Physiological Measurements

Baseline Measures

A multivariate ANOVA was conducted on the four groups in terms of heart rate, systolic, and diastolic blood pressure at baseline measurement. As was expected, there was a significant effects of gender on both systolic ($F(1,60)=55.58$, $p<.001$) and diastolic ($F(1,60)=5.07$, $p<.05$) blood pressure (men as a group always have significantly greater blood pressures due to greater body mass). There was no significant difference between any of the groups in terms of heart rate. Means and standard deviations for all baseline physiological measures are revealed in Table 2. All subsequent analyses are conducted on mean change from

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Insert Table 2 about here

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baseline; due to the significant gender differences, graphical representations of the data will be split by gender, but all analyses will continue to include gender as a factor.

Heart Rate Changes to Alcohol and Provocation

As can be observed in Figure 2, differences were observed when comparing heart rate measured post-beverage with heart rate measured at baseline, and when comparing heart rate measured post-provocation with heart rate measured post-beverage. In order to test this, we

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Insert Figure 2 about here

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conducted a 2x2x2 mixed design ANOVA, with heart rate measurement (post-beverage and post-provocation) as a repeated measure, and gender and alcohol conditions as between-subjects factors. The three-way interaction was not significant. The interaction between gender and measurement time was not significant, nor was the interaction between gender and alcohol. However, there was a significant interaction between alcohol and measurement time ($F(1,60)=5.87$, $p<.05$). There was no significant effect of gender in terms of either change from baseline to post-beverage or post-beverage to post-provocation. There was a significant effect of alcohol from baseline to post-beverage ($F(1,62)=6.38$, $p<.05$) with the alcohol groups demonstrating significant increases in heart rate relative to the no alcohol groups. There was also a significant effect of alcohol from post-beverage to post-provocation ($F(1,62)=6.65$, $p<.05$)

with non-intoxicate groups demonstrating much greater heart rate increases to provocation than intoxicated groups.

Systolic Blood Pressure

As can be observed in Figure 3, little differences were observed when comparing systolic blood pressure measured at baseline to those measured at post-beverage; however, there was noticeable change in systolic blood pressure from post-beverage to post-provocation. In order to test this, we

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Insert Figure 3 about here

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conducted a 2x2x2 mixed design ANOVA, with systolic blood pressure measurement (post-beverage and post-provocation) as a repeated measure, and gender and alcohol conditions as between-subjects factors. The three-way interaction was not significant. The interaction between gender and measurement time was not significant, nor was the interaction between gender and alcohol. The interaction between alcohol and measurement time just failed to reach significance ($F(1,60)=3.89$, $p=.053$). There was, however, an overall main effect of alcohol ($F(1,60)=21.26$, $p<.001$).

There was no significant effect of gender in terms of either change from baseline to post-beverage or post-beverage to post-provocation. There was no significant effect of alcohol from baseline to post-beverage;

however, there was a significant effect of alcohol from post-beverage to post-provocation, with the no-alcohol groups demonstrating ($F(1,62)=19.07$, $p<.001$) significantly greater systolic blood pressure elevations than the alcohol groups during this period.

Diastolic Blood Pressure

As can be observed in Figure 4, little differences were observed when comparing diastolic blood pressure measured at baseline to those measured post-beverage; however, there was noticeable change in diastolic blood pressure from post-beverage to post-provocation. In order to test this,

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Insert Figure 4 about here

A 2x2x2 mixed design ANOVA was conducted, with diastolic blood pressure measurement (post-beverage and post-provocation) as a repeated measure, and gender and alcohol conditions as between-subjects factors. The three-way interaction was not significant. The interaction between alcohol and measurement time was not significant, nor was the interaction between gender and alcohol. The interaction between gender and measurement time approached significance ($F(1,60)=3.57$, $p=.063$). There was an overall main effect of alcohol ($F(1,60)=13.34$, $p<.005$) and measurement time ($F(1,60)=14.15$, $p<.001$).

There was a trend for gender in terms of change from baseline to post-beverage ($F(1,62)=3.65$, $p=.061$), with women demonstrating greater diastolic blood pressure decreases in response to alcohol than men. There was no gender effect in terms of post-beverage to post-provocation measurement of diastolic blood pressure. There was no significant effect of alcohol from baseline to post-beverage; however, there was a significant effect of alcohol from post-beverage to post-provocation, with the no-alcohol groups demonstrating ($F(1,62)=6.02$, $p<.05$) significantly greater diastolic blood pressure elevations than the alcohol groups during this period.

Relationship Between Physiological Response and Aggression

Regression analyses were conducted to determine the extent to which alcohol and situational factors interacted to affect the physiological indices, and the extent to which this physiological change accounted for variance in aggression manifested. Because there were no significant gender effects, groups were collapsed, and regression coefficients are based on all participants. Two stepwise regressions were conducted, the first on mean shock selected in the low provocation condition; the second on mean shock selected in the high provocation condition. Six predictive variables were included in the stepwise regression. They were: heart rate change from baseline to post-beverage; heart rate change from post-beverage to post-provocation; systolic blood pressure change for those same two periods; and diastolic blood pressure change for those same two periods. The first stepwise regression entered only one factor; change in

systolic blood pressure from post-beverage to post-provocation accounted for a significant percentage of the variance in mean shock selected at low provocation ($F(1,61) = 8.03$, $p = .006$, $R^2 = .116$). The second stepwise regression, for mean shock selected at high provocation, likewise entered only one factor, again change in systolic blood pressure from post-beverage to post-provocation ($F(1,61) = 9.81$, $p = .003$, $R^2 = .138$).

Discussion

This study is consequential inasmuch as not only does it replicate previous studies regarding the alcohol-aggression relationship, it provides direct support for a stress-response dampening model of that relationship. Moreover, the study contributes to a growing literature that suggests gender differences in alcohol-related aggression are not as great as once thought. These results will be discussed in turn.

The results of this study, first of all, replicate literally dozens of others that demonstrate that in between-group studies, an alcohol manipulation will make intoxicated participants act in a more aggressive fashion than their sober controls. This is no longer at issue; however, what is still largely not agreed upon are the mediating variables for this relationship, and a clear consensus on differential gender effects.

The demonstration that alcohol appears to attenuate cardiovascular reactivity in men and women is consistent with existing findings (Sayette, 1993a). However, that the stress-response dampening effects of alcohol

can be so directly related to likelihood of aggressive response to provocation appears a novel finding. This result is an important one because it sheds light on the issue of why some people react aggressively when alcohol-intoxicated and others do not. The results of this study suggest that it is not simply alcohol-administration, but instead the individually-specific susceptibility to alcohol's stress-dampening effects (Sher & Walitzer, 1986), that accounts for aggression. That is, the regressions appear to suggest that although intoxicated, some participants still manifested arousal, and acted relatively non-aggressively, whereas those who manifested more pronounced dampening were more likely to act aggressively. The fact that heart rate responses in response to alcohol and provocation do not predict aggression whereas blood pressure results do, might be considered an odd finding, as both as generally considered sympathetic nervous system responses (Mezzacappa, Kindlon, & Earls, 1996). However, dissociation of these two measures has been demonstrated in response to different types of stress and/or provocation, such as neuropsychological (Higgins et al., 1993) and intelligence test performance (Zeichner, Edwards, & Cohen, 1985).

In terms of gender differences, the intoxicated women in this study did appear to demonstrate slight increases in response to alcohol; these increases were not significant at either the low- or high-provocation conditions. The aggression of the men was not significantly greater at low provocation, but approached significance at high provocation. These

results, while not entirely consistent with previous studies, do correspond with recent studies that suggest that alcohol intoxication does not elicit aggression as pronounced in women as it does in men (Hoaken & Pihl, 2000), and that provoked sufficiently, women's aggression will not always be less than that of men (Eagly & Steffen, 1996). The fact that women, who experienced the same pattern of cardiovascular reactivity as men in both the alcohol and sober conditions but did not exhibit as pronounced a pattern of aggressive responding, may be related to socialization practices. Several observers (e.g. Buss, 1963; Bandura, 1973) have suggested that men and women may not differ in overall levels of aggression as much as they do in their preferred means of aggressing, with men showing a clearer tendency than women only in the case of physical violence. Women may be no less aggressive than men in non-physical ways, such as verbal expression. It may be that had women been offered an aggressive option more appealing to them, the pattern of their alcohol-related aggression would have more closely corresponded to their male counterparts.

The major limitation of this study was that cardiovascular responses were not measured during the aggression task. It is quite possible that measures taken following the aggression paradigm are not representative of cardiovascular reactivity during the task. Furthermore, it would have been beneficial to assess individual differences in anxiety and hostility levels, factors known to significantly influence cardiovascular responding to interpersonal stress (Jorgensen et al., 1996).

The current study presents findings that suggest that the stress dampening effects of alcohol have different implications on physical aggressing for men and women. While the cardiovascular responses between the sexes were equivalent, their pattern of aggressing following provocation was not. The results may best be understood by considering the different socialization histories of men and women and its result on their experience with different forms of aggression. Many approaches to the etiology of sex differences tend to consider such differences to be a product of learning, not heredity. In fact, it should be noted that most researchers do not propose that inherited biologically based sex differences account for all male-female differences in aggression. Instead, they generally propose that biological differences establish different backgrounds in the two sexes, against which environmental and situational forces operate.

In conclusion, irrespective of the gender differences, this study offers direct support for the arousal-dampening/anxiolytic models of the alcohol-aggression relationship. Intoxicated participants, as a whole, responded more aggressively than did their sober peers. Moreover, they demonstrated patterns of physiological arousal indicative of stimulation in response to alcohol alone, and patterns indicative of stress-response dampening when intoxicated and faced with a provocative situation.

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Table 1. Mean (and standard deviation) blood alcohol concentrations for all groups.

	Post-Beverage Measurement	Post-Provocation Measurement
Men/No Alcohol	.00 (.00)	.00 (.00)
Men/Alcohol	.073 (.023)	.076 (.026)
Women/No Alcohol	.00 (.00)	.00 (.00)
Women/Alcohol	.077 (.014)	.081 (.0096)

Table 2. Means (and standard deviations) of baseline measures of systolic blood pressure, diastolic blood pressure, and heart rate, for all groups.

	Systolic Blood Pressure	Diastolic Blood Pressure	Heart Rate (beats per minute)
Men/No Alcohol	119.75 (7.41)	69.31 (8.69)	67.81 (10.86)
Men/Alcohol	123.37 (9.66)	68.37 (7.57)	69.12 (8.32)
Women/No Alcohol	102.68 (9.97)	63.12 (7.65)	71.25 (12.30)
Women/Alcohol	105.68 (9.98)	65.5 (8.21)	69.93 (9.15)

Figure Caption

Figure 1. Mean shock selected (and S.E.M.) for both low and high provocation conditions, by gender group and alcohol condition.

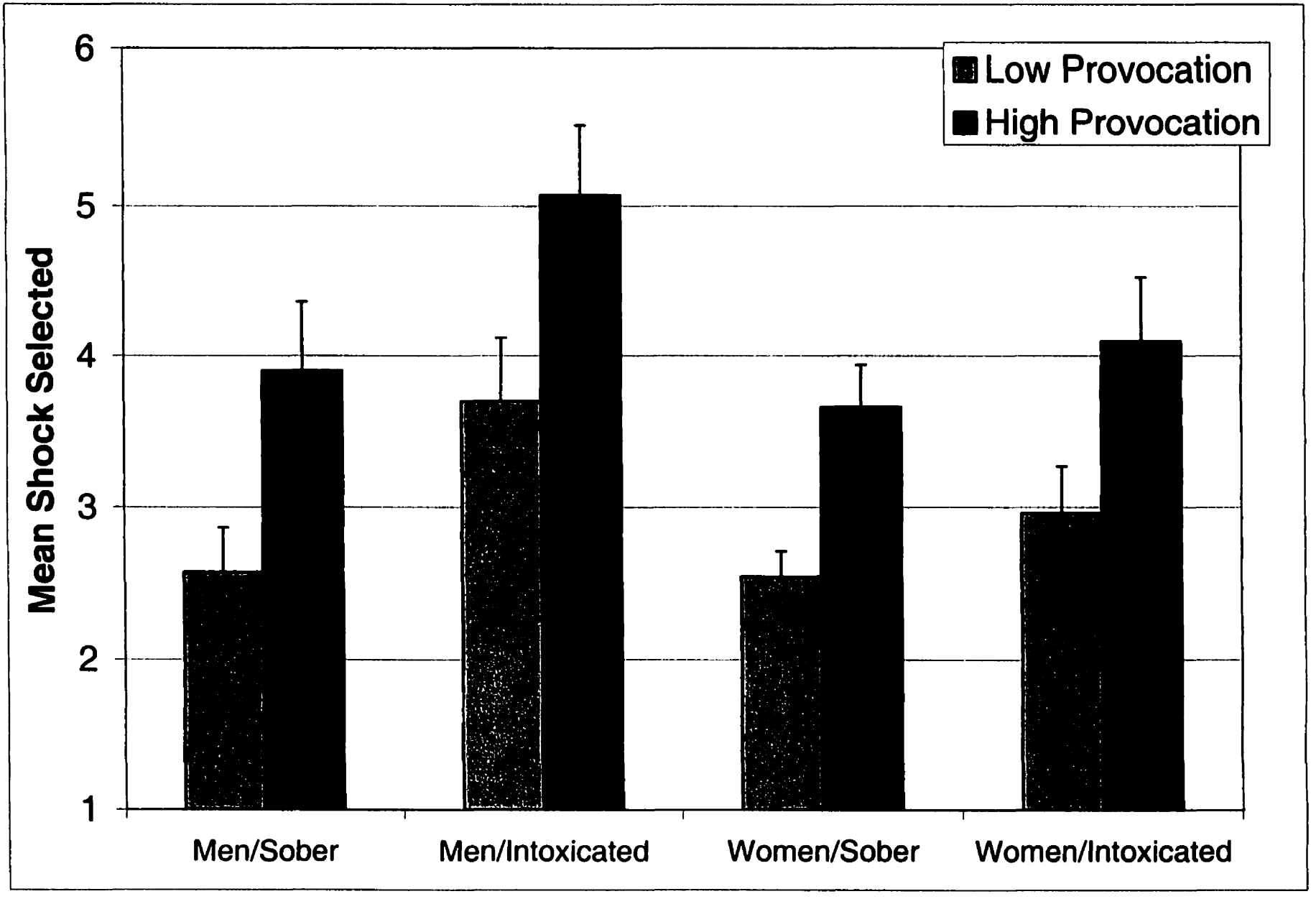
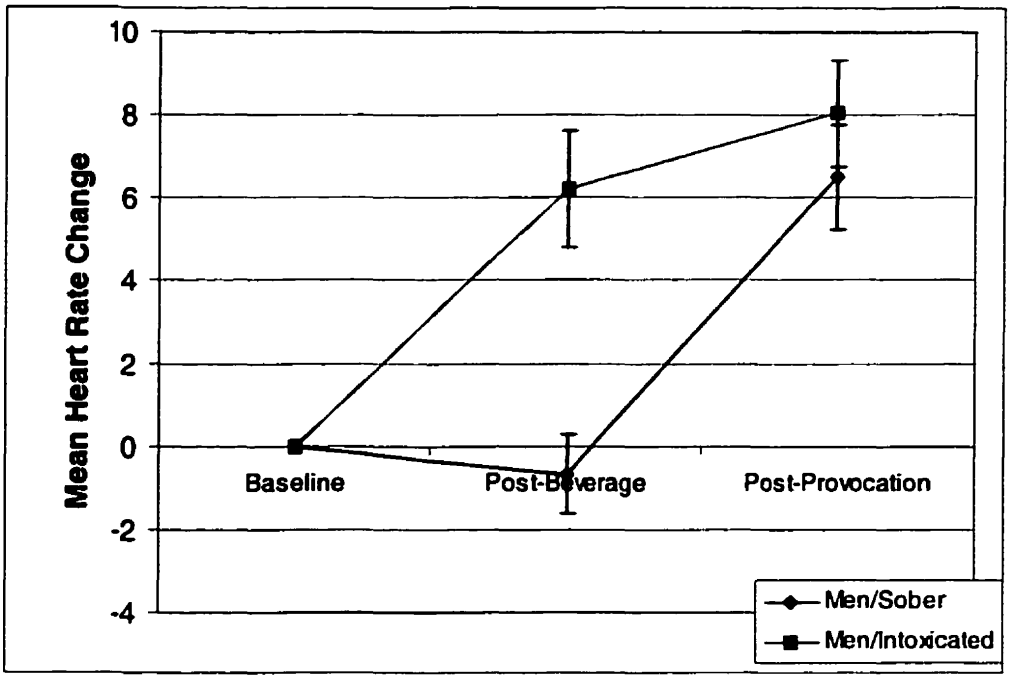

Low Provocation
High Provocation
Mean Shock Selected
6
5
4
3
2
1
Men/Sober
Men/Intoxicated
Women/Sober
Women/Intoxicated

Figure Caption

Figure 2. Mean change (and S.E.M.) from baseline in heart rate in response to beverage consumption and Taylor aggression paradigm, for each of the four groups.


Mean Heart Rate Change
10
8
6
4
2
0
-2
-4
Baseline
Post-Beverage
Post-Provocation
Men/Sober
Men/Intoxicated

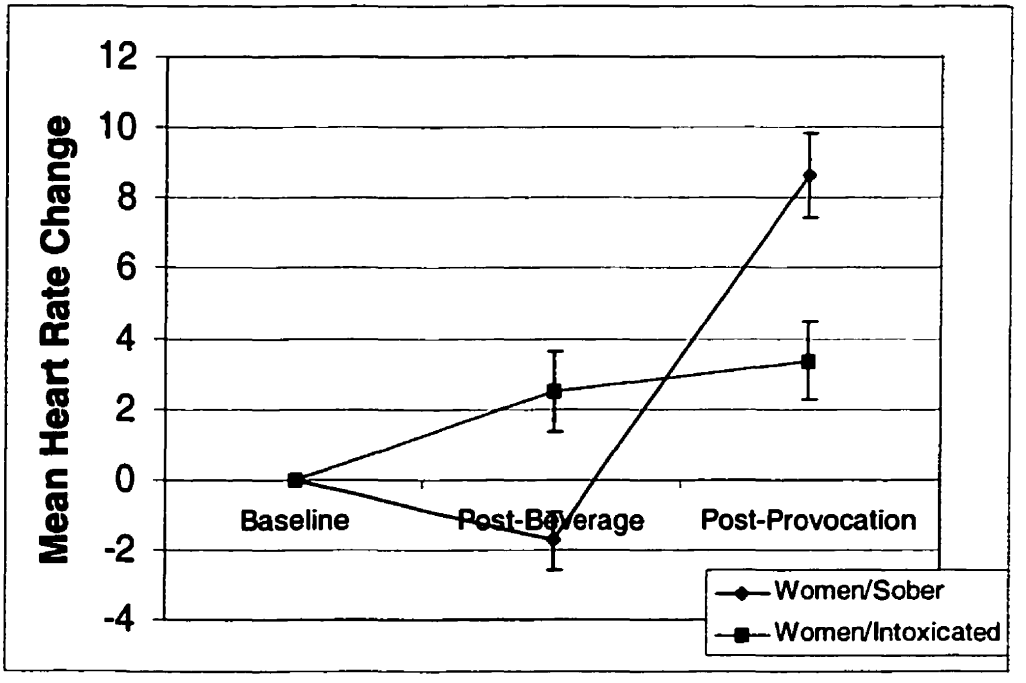
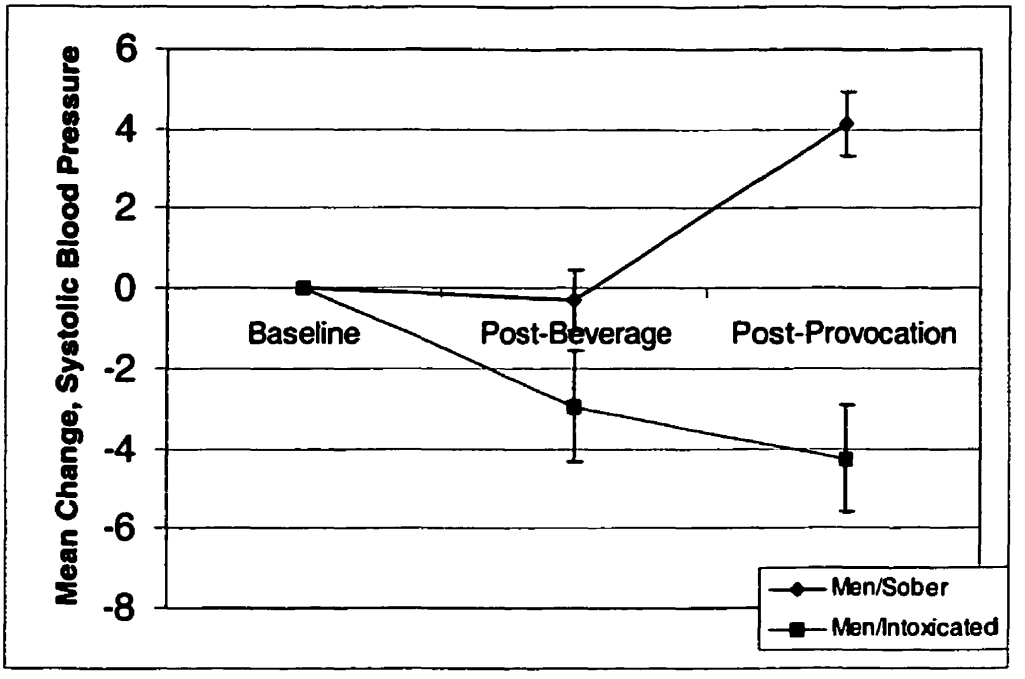

Mean Heart Rate Change
12
10
8
6
4
2
0
-2
-4
Baseline
Post-Beverage
Post-Provocation
Women/Sober
Women/Intoxicated

Figure Caption

Figure 3: Mean change (and S.E.M.) from baseline in systolic blood pressure in response to beverage consumption and Taylor aggression paradigm, for each of the four groups.


Mean Change, Systolic Blood Pressure
6
4
2
0
-2
-4
-6
-8
Baseline
Post-Beverage
Post-Provocation
Men/Sober
Men/Intoxicated

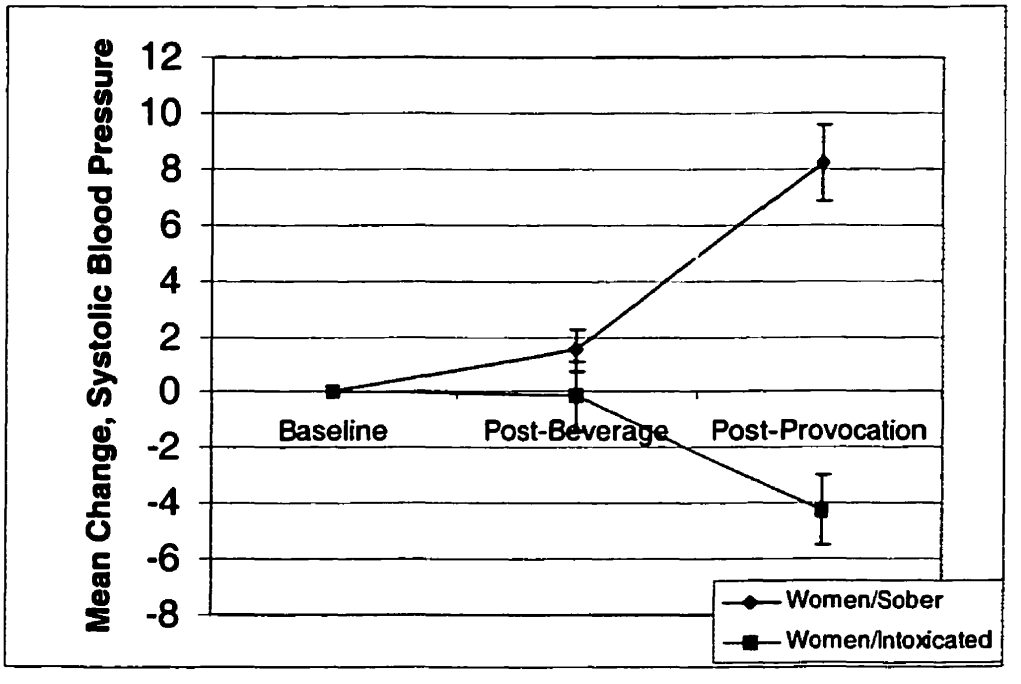
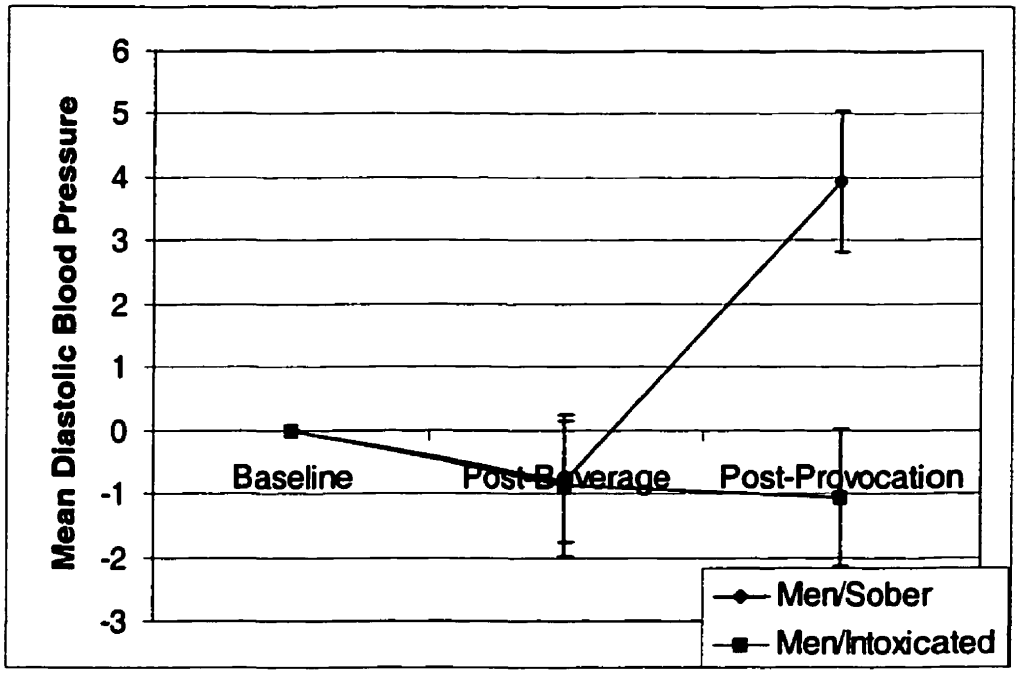
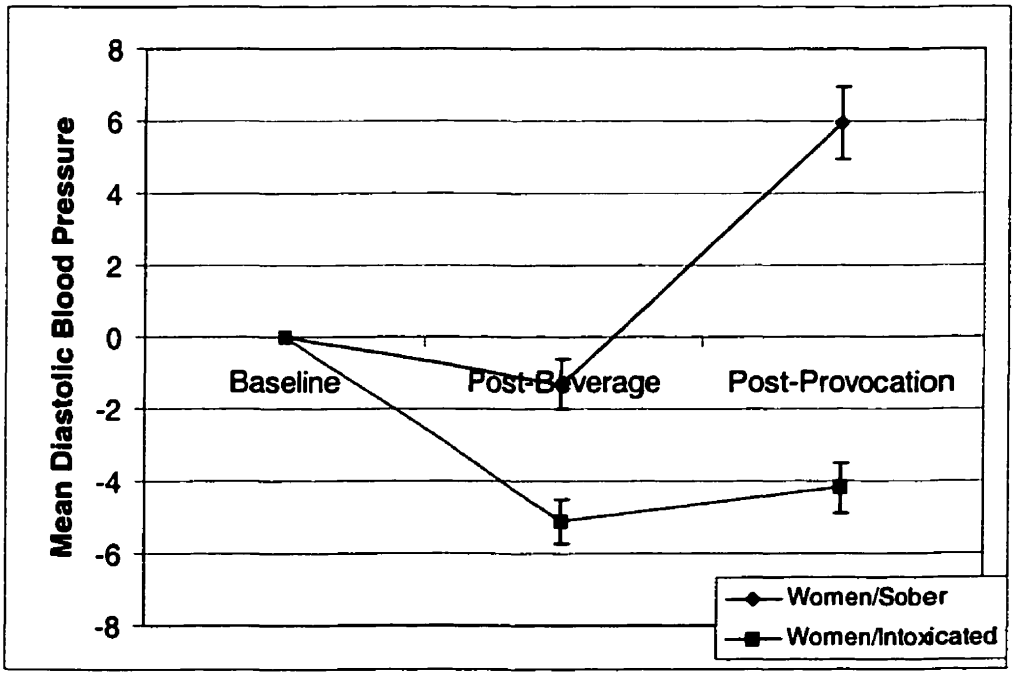

Mean Change, Systolic Blood Pressure
12
10
8
6
4
2
0
-2
-4
-6
-8
Baseline
Post-Beverage
Post-Provocation
Women/Sober
Women/Intoxicated

Figure Caption

Figure 4: Mean change (and S.E.M.) from baseline in diastolic blood pressure in response to beverage consumption and Taylor aggression paradigm, for each of the four groups.


Mean Diastolic Blood Pressure
6
5
4
3
2
1
0
-1
-2
-3
Baseline
Post-Beverage
Post-Provocation
Men/Sober
Men/Intoxicated


Mean Diastolic Blood Pressure
8
6
4
2
0
-2
-4
-6
-8
Baseline
Post-Beverage
Post-Provocation
Women/Sober
Women/Intoxicated

APPENDIX C

Hoaken, P.N.S., Giancola, P., & Pihl, R.O. (1998). Executive cognitive functions as mediators of alcohol-related aggression. Alcohol and Alcoholism, 33(1), 47-54.

EXECUTIVE COGNITIVE FUNCTIONS AS MEDIATORS OF ALCOHOL- RELATED AGGRESSION

PETER N. S. HOAKEN[1], PETER R. GIANCOLA[2], and ROBERT 0. PIHL[3]

Departments of [1]Psychology and [3]Psychiatry, McGill University, Canada and [2]Western Psychiatric Institute and Clinic, Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, USA

Abstract — A large body of literature has documented a relation between executive cognitive functioning (ECF) and aggression. ECF encompasses 'higher-order' mental abilities such as attention, planning, organization, abstract reasoning, and self-monitoring. ECF has been defined as the ability to utilize these functions to self-regulate goal-directed behaviour. The prefrontal cortex represents the primary neurological substrate that subserves ECF. Acute alcohol consumption has been shown to disrupt ECF/pretrontal cortical functioning. Literature is reviewed linking ECF/prefrontal cortical functioning, alcohol consumption, and aggressive behaviour. A hypothetical model, based on empirical data. is presented, suggesting that ECF/prefrontal cortical functioning is an underlying aetiological mechanism for the relation between acute alcohol consumption and aggressive behaviour.

INTRODUCTION

The relation between alcohol and aggression is both real and significant (Bushman and Cooper. 1990; Bushman, 1997). However, elucidating why this relation exists is exceedingly difficult and convoluted. Most researchers concur that the alcohol-aggression link is not simplistic: rather, an intricate multifactorial model, which includes pharmacological, cognitive, contextual, and psychological effects, should be invoked (Pihl et al., 1993; Chermack and Giancola, 1998). Specific to the pharmacological effect, it has been suggested that there are several ways that alcohol might facilitate aggression. It may act as an anxiolytic thus diminishing fear of retaliation, it may act as a psychomotor stimulant thus increasing novelty or sensation-seeking behaviour, or it may act as an analgesic thus reducing the salience of provocative painful stimuli (Pihl and Peterson, 1995). A depletion of this hypothetical model is illustrated in Fig. I.

Another possibility, which is the basis of this paper, is that alcohol disrupts executive cognitive functioning (ECF). ECF encompasses 'higher-order' mental abilities, such as attention, planning. organization, sequencing, abstract reasoning, selfmonitoring, and the ability to utilize external and internal feedback to adaptively modulate future behaviour (Stuss and Benson. 1984; Foster et ai., 1994). ECF has been defined as the ability toutilize these functions to self-regulate goal-directed behaviour (Giancola, 1995). There is substantial evidence to show that the

prefrontal cortex represents the primary neural substrate that subserves these functions (Stuss and Benson 1984; Giancola, 1995).

DOES ALCOHOL AFFECT COGNITION?

The notion that alcohol impairs various aspects of cognition is not new; indeed, studies on this issue date back almost 60 years. However, well-developed and standardized tests of cognitive functioning were either unavailable or unused in these studies. Examination of such literature led Peterson et al. (1990) to hypothesize that the cognitive effects of acute alcohol consumption are similar to the symptoms exhibited by patients with damage to the prefrontal cortex. This hypothesis was based on two considerations. First, ECF, which is particularly susceptible to the effects of alcohol, is governed by the prefrontal cortex. Second, individuals with prefrontal cortical lesions display behavioural characteristics reminiscent of alcohol-intoxicated individuals.

Peterson et al. (1990) administered low. moderate, and high doses of alcohol to subjects using a balanced-placebo design. These researchers were interested in the performance of subjects on tests measuring prefrontal cortical functioning, temporal functioning, and a variety of miscellaneous neuropsychological abilities. The results demonstrated that high doses of alcohol detrimentally affected prefrontal functioning which included planning, working memory and complex motor control. However, it had much less of an effect on 'non-frontal' tests. These findings showed that alcohol can disrupt ECF/prefrontal cortical

functioning in a fashion that produces a behavioural profile similar to patients with prefrontal cortical lesions. It is thus important to determine which prefrontal characteristics are associated with decreased regulation of human social behaviour, including a 'disinhibition syndrome', characterized by impulsivity, socially inappropriate behaviour, and aggression (Hecaen and Albert, 1978: Giancola, 1995).

IS ECF RELATED TO AGGRESSION?

A large research literature has demonstrated that ECF/prefrontal cortical functioning is related to aggressive behaviour (reviewed in Giancola, 1995). Individuals with psychiatric disorders characterized by disinhibited and aggressive behaviours such as antisocial personality disorder (Gorenstein, 1987), psychopathy (Smith et al.. 1992), substance use disorders (Tarter et al., 1989), conduct disorder (Moffitt, 1993), attention deficit hyperactivity disorder (Benson, 1991), and inattention/overactivity difficulties (McBurnett et al., 1993) have all been shown to perform poorly on tests of ECF.

Studies with lesioned populations also provide evidence supporting the ECF-aggression link. McAllister and Price (1987) found that 60% of psychiatric patients with prefrontal cortical pathology displayed disinhibited behaviour with affective lability, and 10% displayed violent outbursts. In addition, Heinrichs (1989) discovered that a prefrontal lesion was the best predictor of violent behaviour in a sample of 45

neuropsychiatric patients. Data from neurological case reports have also clarified the relation between ECF/prefrontal functioning and aggression. Clinical researchers have reported violent and aggressive behaviours in individuals with damage to the prefrontal cortex (Thompson, 1970; Cope and Donovan, 1979). Others have noted an inability to delay gratification, irresponsibility, sexual promiscuity, grand larceny, drug involvement, angry outbursts, arson, suspected rape, and physical violence in neurological patients who incurred damage to the prefrontal cortex during childhood (Price et al.. 1990). These patients developed normally until the damage was sustained. Another study noted sexual disinhibition, disobeying parental orders, and verbal and physical aggression in a 13-year-old girl suffering from partial complex seizures localized primarily in the prefrontal cortex (Boone et al., 1988).

Other studies with boys at high risk for substance abuse have also demonstrated this relation. Researchers at the Center for Education and Drug Abuse Research (CEDAR), in Pittsburgh, USA, have demonstrated that ECF was negatively related to both mother and teacher reports of aggression and delinquency in their children. Furthermore, ECF was also related to symptoms of conduct disorder and to aggressive responding on a point-subtraction laboratory measure of aggression (Giancola et al., 199611). In a subsequent study, it was found that ECF was effective in predicting reactive aggression at a 2-year follow up of the same boys (Giancola et al., 1996b). Finally, Giancola and colleagues

discovered that the ECF-aggression relation was also present in adolescent female substance abusers (Giancola et al., unpublished work) as well as normal college males (Giancola and Zeichner, 1994).

Neuroimaging studies with violent offenders have also implicated frontal cortex. Raine et al. (1994) conducted a positron emission tomographic study on 22 subjects accused of murder and 22 matched controls. The offenders as a group had significantly lower glucose metabolism in both medial and lateral prefrontal cortex relative to the controls; no other differences were found. Furthermore, these differences were not found to be a function of handedness, head injury, motivation, or mental illness. There are several other studies which suggest the same relationship - Mills and Raine (1994) reviewed 20 brain imaging studies which examined either brain structure or function in violent or sexual offenders. They concluded that frontal lobe dysfunction is associated with violent offending. As regards sexual offending, the situation is less clear, but these latter authors suggest that violent sexual offences are most likely to be associated with anterior brain dysfunction.

The association between ECF and aggression also extends to normal populations. Seguin et al. (1995) followed a cohort of boys for 12 years, since the boys were 6 years of age. The boys were tested on a series of cognitive tests, which included measures of ECF as well as tests of spatial learning, verbal learning and cerebral dominance. Using parent, teacher, and psychometric evaluations, the boys were divided into

one of three groups based on ratings from the preceding years: (1) stable aggressives, who demonstrated a stable and consistent pattern of physical aggression; (2) unstable aggressives, who had been identified as aggressive, but with less stability or consistency as the stable aggressives; (3) non-aggressives. As can be seen in Fig. 2, ECF was most strongly related to physically aggressive behaviour.

Other studies have assessed the interactive effects of ECF and alcohol on aggressive behaviour. Lau et al. (1995) separated subjects into 'high' or low' functioning groups based on their performance on two tests of ECF: the Self Ordered Pointing (SOP) test and the Spatial Conditional Associative Learning Test (SCALT) ;Petrides, 1985). Both tests assess the ability to process and manipulate large amounts of informa:ion in working memory. Half of the subjects in each group were administered alcohol, the other half remained sober. Aggression was then elicited and assessed using a modified version of the Taylor Aggression Paradigm (TAP; Taylor, 1967). The TAP places subjects in a situation whereby electric shocks are received from and administered to a fictitious 'opponent' during a competitive reaction time task. Physical aggression is operationalized as the shock intensities and durations selected by the subjects. Subjects are seated at a table in a quiet room. On the table facing the subject is situated the aggression console consisting of a black metal box equipped with an assortment of electrical push buttons and light emitting diodes. A reaction time lever is mounted on a small black box

placed just anterior to the console. Arranged on the console are eight shock lush buttons labelled '1' (low) to '8' (high). Subjects experience increasing levels of provocation (shocks) as the task proceeds.

As can be seen in Fig. 3, the results indicated hat provocation interacted with ECF to predict aggression. Low ECF subjects were more aggressive under conditions of high provocation than were high functioning subjects. Furthermore, when alcohol was involved, aggression increased as a function of heightened provocation for both groups. This led the researchers to conclude that alcohol intoxication and prefrontal cortical dysfunction are involved in the disinhibition of aggressive impulses (Lau et al., 1995).

In a subsequent study, Lau and Pihl (1996) tested the hypothesis that heightened aggression was due to an inability to inhibit impulsive behaviour by examining whether subjects with poor prefrontal functioning would be able to inhibit aggressive responses if offered contingent monetary reward. Again, the SCALT was used to classify subjects into high- and low-functioning groups. Aggression was measured using a modified version of the TAP. Monetary reward was introduced by providing subjects with higher amounts of money for administering lower intensity shocks (i.e. 40 cents for administering a '1' and only 5 cents for administering an '8'). Alcohol was not administered in this experiment. The results indicated that subjects with low ECF responded aggressively (did not inhibit aggression in order to gain reward), whereas

high functioning subjects were less aggressive (successfully inhibited aggression). The results were interpreted to suggest that men with dysfunctional ECF are more impulsive and less able to behave non-aggressively in provocative situations.

The results of these two studies suggested that compromised ECF/prefrontal functioning is associated with increased aggression in response to provocation. The next study by these researchers then involved the administration of alcohol to subjects with high baseline ECF/prefrontal functioning in order to determine whether they could inhibit their aggression for monetary reward (Hoaken et al., 1998). It was hypothesized that sober individuals would be able to inhibit their aggression in order to gain monetary reward, whereas intoxicated subjects would not be able to do so due to an alcohol-induced disruption in ECF.

This study involved screening subjects with the SCALT and a brief IQ test to ensure high ECF and sound overall cognitive ability. Subjects were randomly assigned to either an intoxicated or non-intoxicated condition, and then, either received or did not receive monetary reward. Aggression was measured using a modified version of the TAP identical to that used in the Lau et al. (1995) study. After completing the TAP, all subjects were administered a post-intoxication test of ECF/prefrontal cortical functioning: the non-spatial version of the CALT (NCALT). Results indicated .hat, compared to the sober group, the intoxicated group performed significantly worse. Given the high similarity between the

spatial and non-spatial 1A.LT, a learning effect is typically observed when the tests are administered in close temporal)roximity (as was done in this study). Hoaken el al. (1998) found that sober subjects completed the NCALT in 20 fewer trials than they had the CALT, and with 17 fewer errors. The intoxicated groups were unable to complete the NCALT in ewer trials, and as a result, made more errors. These data are presented in Fig. 4.

With respect to aggression, results indicated hat, when no contingent money was available, intoxicated subjects reacted more aggressively than did non-intoxicated subjects. In addition, sober subjects given monetary reward were less aggressive than their non-rewarded counterparts. However, an unexpected finding emerged: the presentation of contingent monetary reward profoundly reduced aggression in the intoxicated group (see Fig. 5). One plausible interpretation of this finding is that the sample chosen for the experiment (i.e. subjects with superior cognitive abilities), retained a sufficient amount of residual ECF, even when acutely intoxicated, to inhibit aggression given the appropriate inhibitory cues. These results are interesting, because, although there is a relation between alcohol and aggression, and although the majority of violent crimes occur when the perpetrator is intoxicated, the fact remains that the majority of individuals who become acutely intoxicated do not become aggressive. This study suggests that the retention of sufficient

residual ECF may afford the intoxicated individual the ability to inhibit an aggressive response given the presence of appropriate inhibitory cues.

ECF AND ALCOHOL-RELATED AGGRESSION

Evidence further implicating ECF in the expression of alcohol-related aggression, as well as the elucidation of a possible aetiological mechanism, comes from a recent study which assessed the biphasic effects of alcohol on human aggression (Giancola and Zeichner, unpublished work). Alcohol metabolism follows a biphasic trajectory characterized by an ascending followed by a descending limb representing respectively alcohol absorption and elimination from the body. This trajectory is referred to as the blood-alcohol concentration (BAG) curve. As has previously been mentioned, studies have shown that alcohol has pronounced effects on neuropsychological functioning, particularly ECF. However, these effects appear to differ reliably depending on which limb of the BAG curve they are measured. For example, alcohol has been shown to severely disrupt attention (Hurst and Bagley, 1972), abstract reasoning (Jones and Vega, 1972), short term memory (Jones, 1973), and arithmetic skills in the ascending limb of the BAC curve, whereas in the descending limb, these functions improve significantly as they return to their previous state) of equilibrium.

Interestingly, these data, in conjunction with the studies presented earlier documenting a relation) between ECF and aggression, can be used

to further bolster the hypothesis that ECF may serve is an aetiological mechanism for the alcohol-aggression relationship. As noted above, previous investigations have demonstrated a negative relation between ECF and aggressive behaviour. As such, if alcohol disrupts ECF on the ascending limb of the BAC curve to a greater extent than it does on the descending limb, it can be hypothesized that aggression should be greater on the ascending limb than on the descending limb. A recent study by Giancola and Zeicliner (unpublished) supported this hypothesis.

Aggression was measured using a modified version of the TAP (Giancola and Zeichner, 1995). Subjects were tested at a BAC of 0.08% either on the ascending or the descending limb of the BAC curve. Sober control groups were also used to take into account the effects of passage of time resulting from the greater amount of time the descending limb group was required to wait before testing compared to the ascending limb group. That is, it took approximately 45 min for subjects in the ascending limb group to reach a BAC of 0.08%, whereas it took subjects in the descending group an average of 3 hours. Therefore, subjects in the control groups were matched with subjects in the appropriate alcohol group for the amount of time spent waiting in the laboratory prior to testing. The control groups received no alcohol.

Although this study used the traditional measures of shock intensity and duration as indices of aggression, an additional metric was added. The particular version of the TAP used in this study had a range of

five shock buttons. The new measure consisted of the proportion of times the highest shock button (i.e. '5') was administered within each provocation condition. It has been suggested that this metric represents an extreme Form of aggression (Chermack and Taylor, 1995). Results indicated that subjects were significantly more aggressive on the ascending limb than on the descending limb of the BAC curve. Furthermore, control subjects did not differ from one another in aggression, suggesting that a passage of time effect did not confound the results. Finally, subjects who received alcohol and were tested 3n the descending limb were no more aggressive than either of the control groups indicating that alcohol does not appear to facilitate aggression on the descending limb (see Fig. 6). This finding is also interesting, because it speaks against the alcohol expectancy theory, which stipulates that intoxicated aggression results from the belief that alcohol increases aggression (McAndrew and Edgerton, 1969). If this were the case, it could be argued that aggression should have also been manifested on the descending limb.

CONCLUSIONS

In the context of the model presented earlier (see Fig. 1), the importance of ECF/prefrontal cortical functioning is underscored. Although alcohol-induced alterations in pain sensitivity, alterations in psychomotor stimulation, and alterations to the threat/anxiety system

all tend to increase the probability of aggression, it is likely that most of these symptoms are mediated by ECF. In fact, there are data suggesting that the prefrontal cortex is involved in the regulation of arousal and anxiety (Gray, 1982: Raine et al., 1991).

ECF has been shown to be related to aggressive behaviour in a growing number of studies. ECF also appears to predict which individuals will and will not become aggressive, either sober or under the influence of alcohol. As such, this area of research has important societal implications. Because of the exorbitant social costs that are associated with alcohol-related aggression, the ability to identify the operative mechanisms that mediate this relation is of great scientific import.

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Figure Caption

Figure 1. Theoretical model of the putative pharmacological mechanisms through which alcohol induces aggression. From Pihl & Hoaken, 1997.

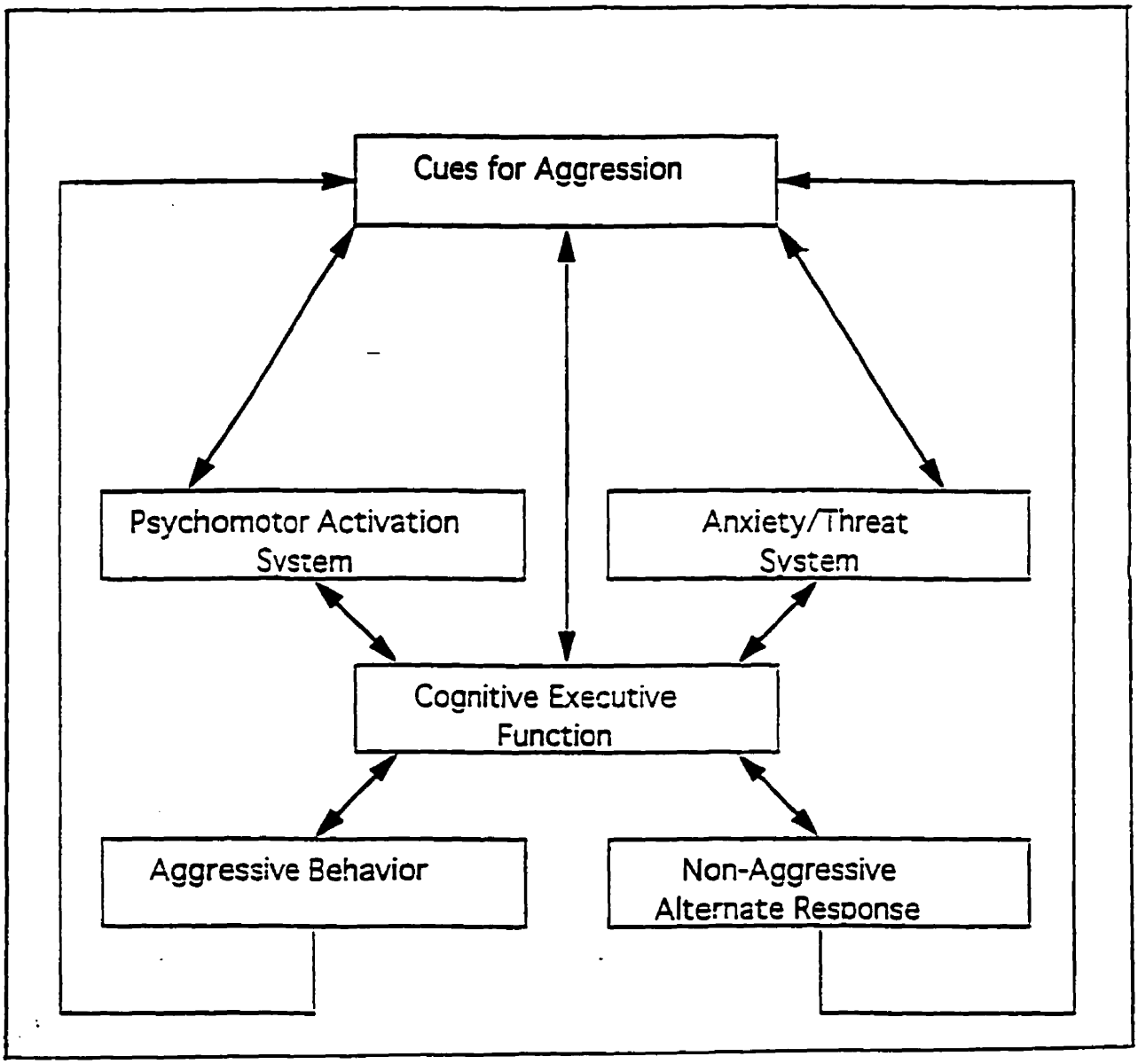

Cues for Aggression
Psychomotor Activation System
Anxiety/Threat System
Cognitive Executive Function
Aggressive Behavior
Non-Aggressive Alternate Response

Figure Caption

Figure 2. Adjusted mean regression factor scores by aggression group.

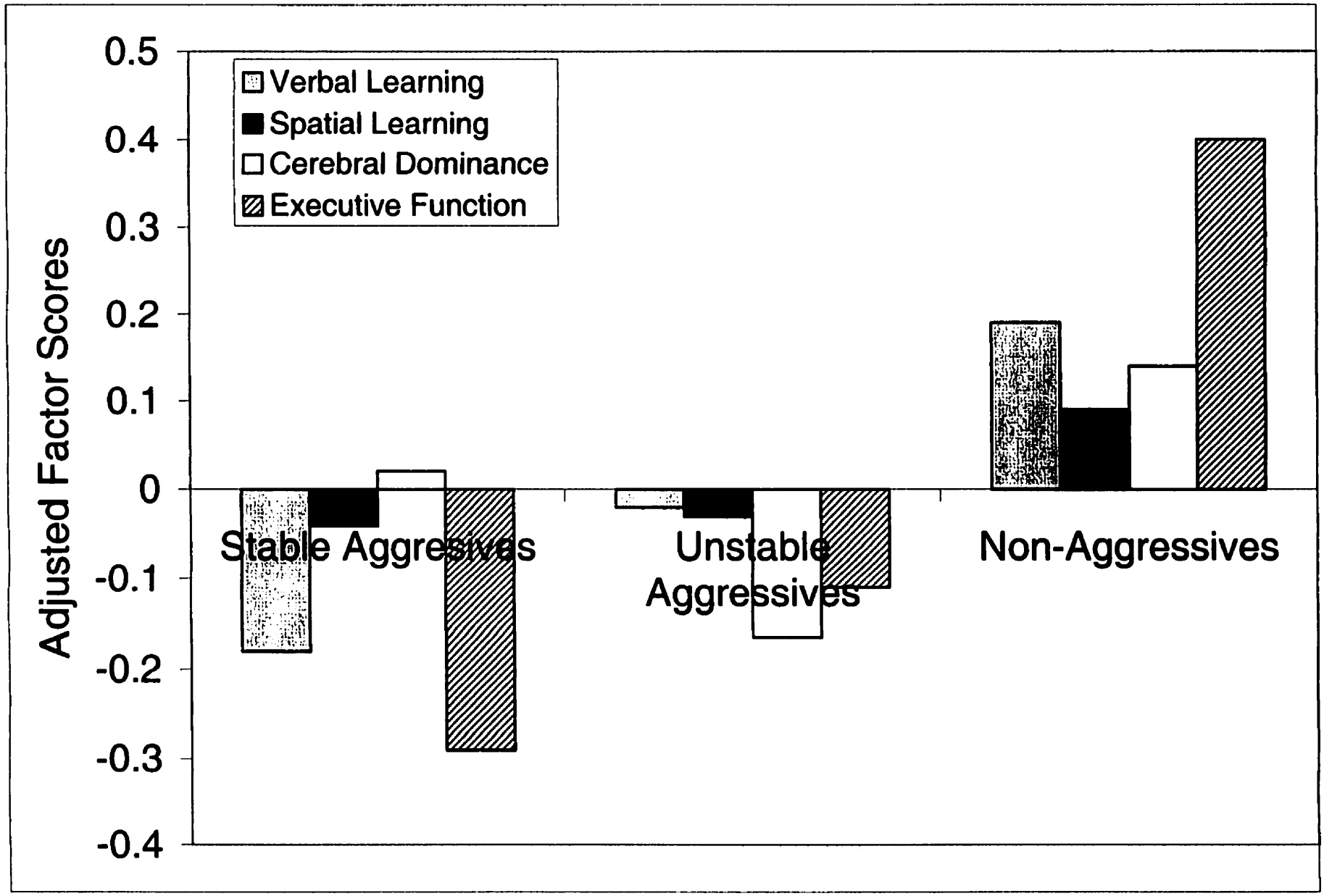

Verbal Learning
Spatial Learning
Cerebral Dominance
Executive Function
Adjusted Factor Scores
0.5
0.4
0.3
0.2
0.1
0
-0.1
-0.2
-0.3
-0.4
Stable Aggressives
Unstable Aggressives
Non-Aggressives

Figure Caption

Figure 3. Aggression in response to low and high provocation, in subjects whose performance on tests of executive functions fell into the lowest or highest performance quartiles.

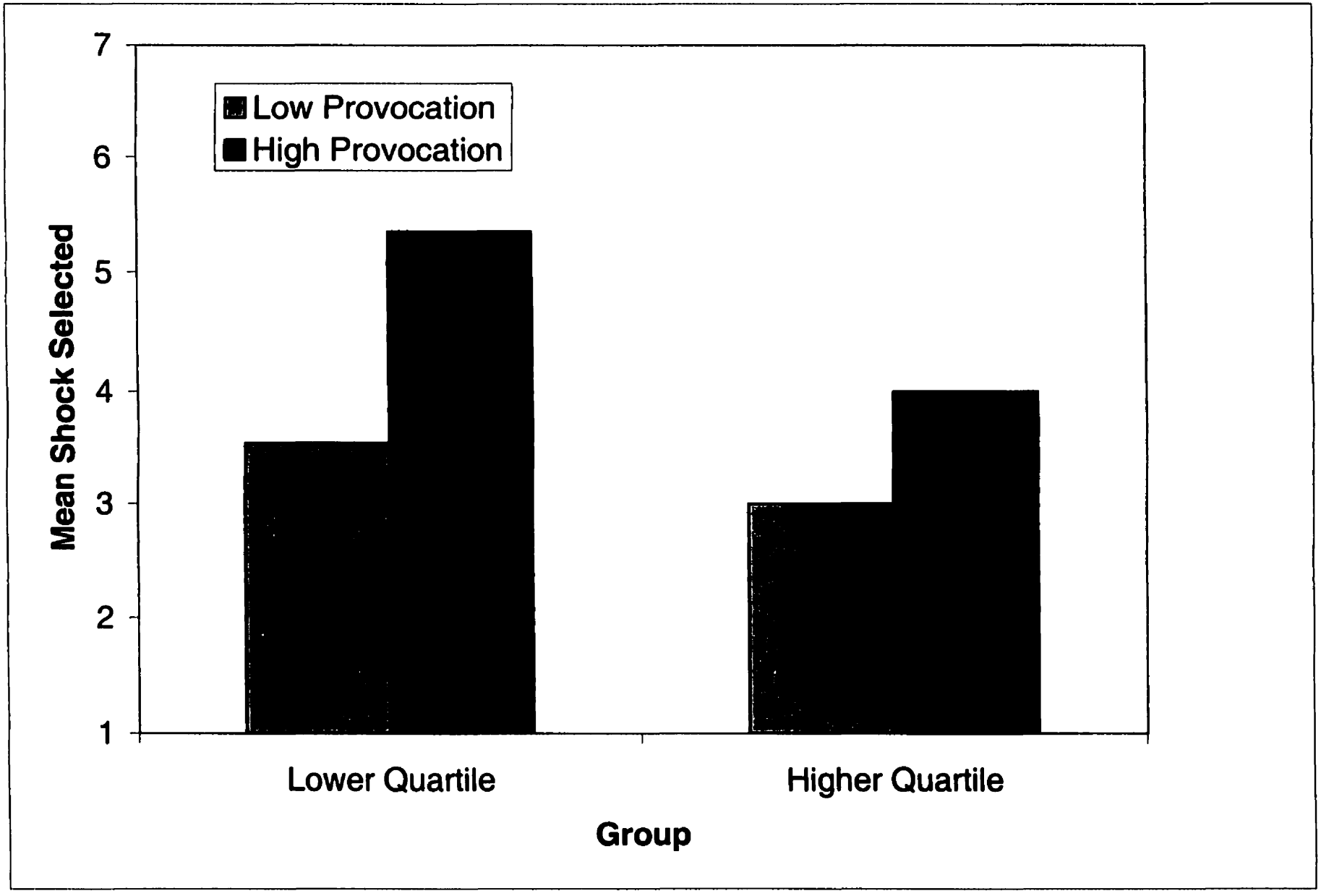

7
6
5
4
3
2
1
Low Provocation
High Provocation
Mean Shock Selected
Lower Quartile
Higher Quartile
Group

Figure Caption

Figure 4. Alcohol-induced changes in ability to perform a test of executive functions. From Hoaken et al., 1998.

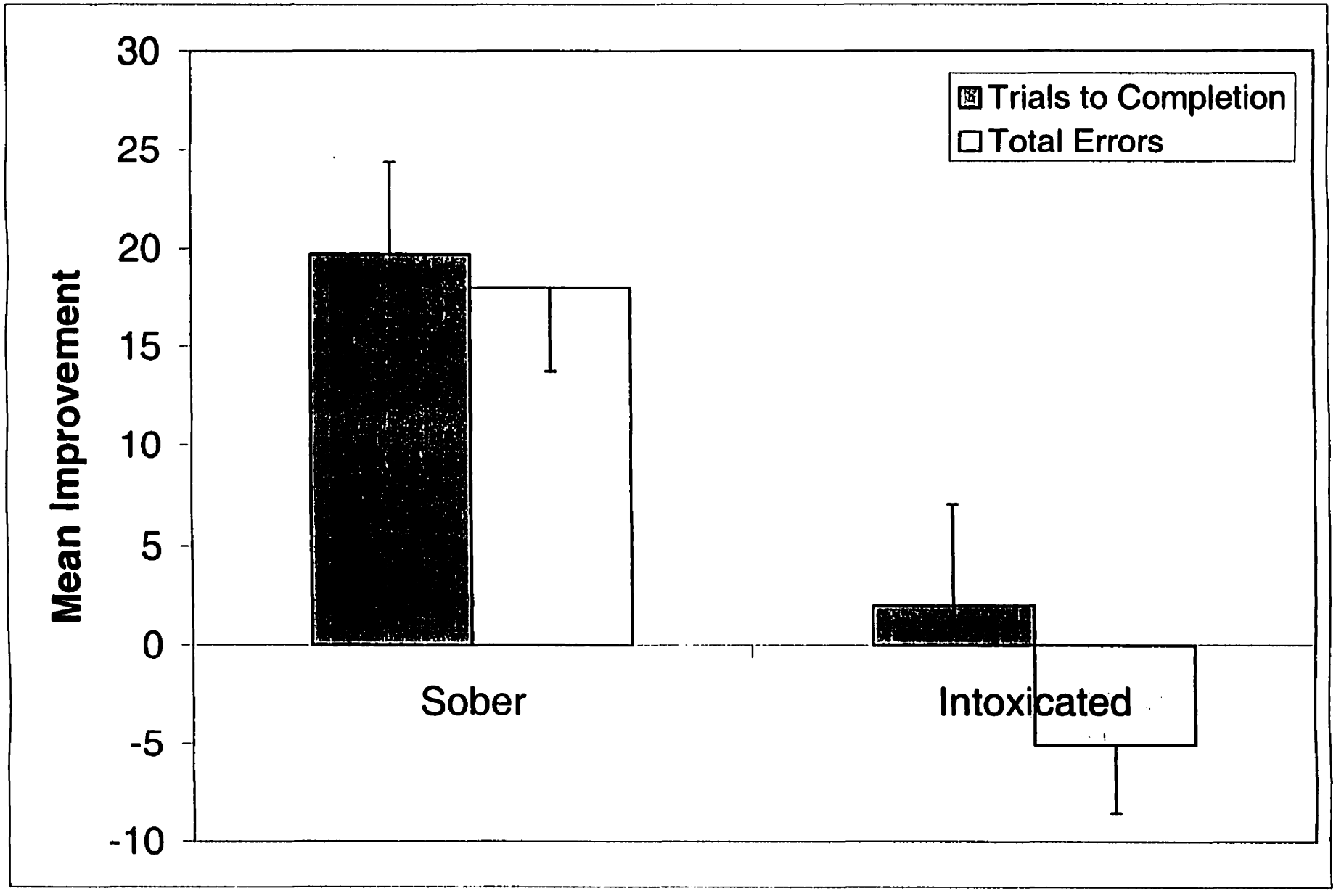

30
25
20
15
10
5
0
-5
-10
Mean Improvement
Trials to Completion
Total Errors
Sober
Intoxicated

Figure Caption

Figure 5. Effects of alcohol on ability to inhibit aggression in men with above-average ECF abilities. From Hoaken et al, 1998.

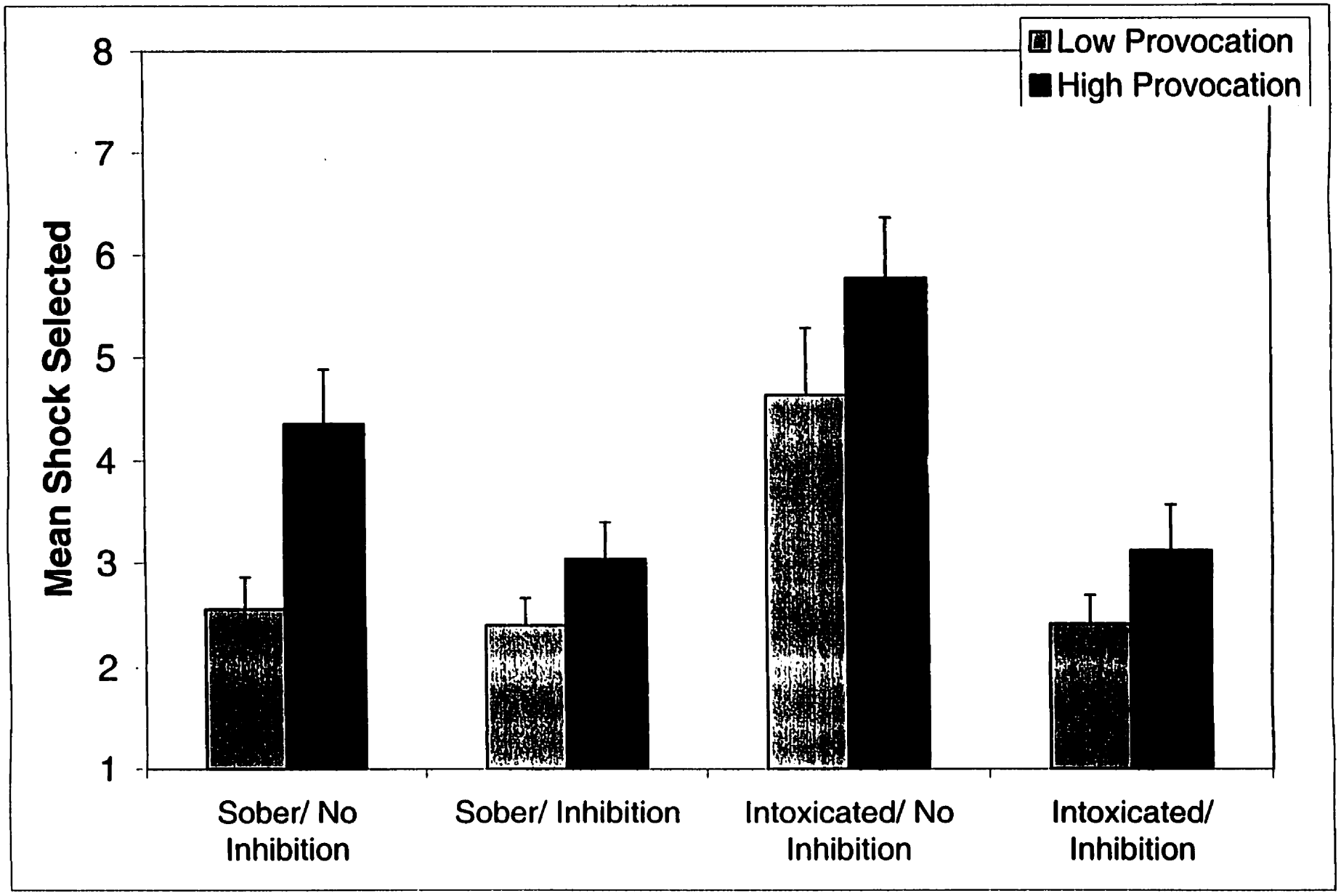
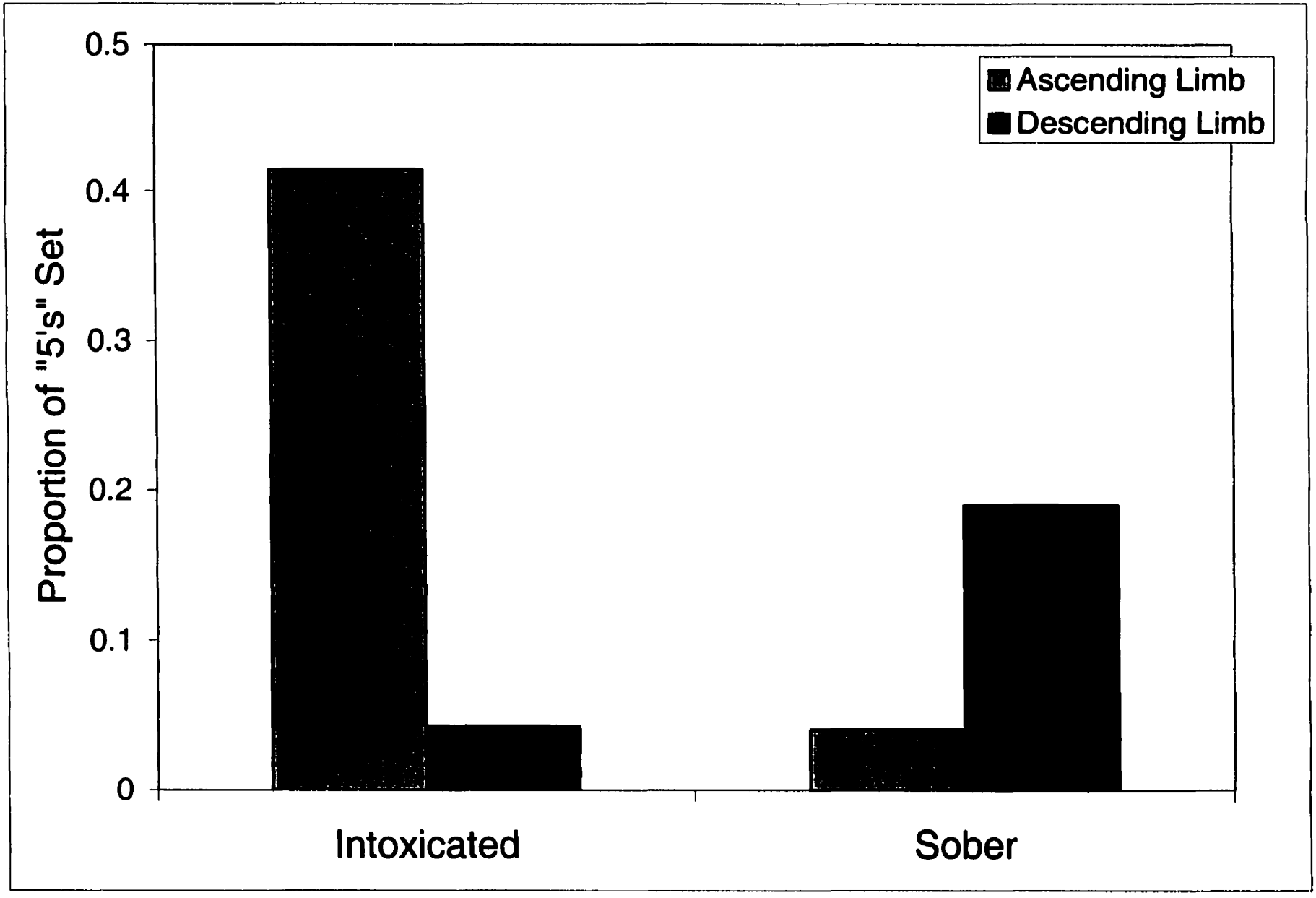

Low Provocation
High Provocation
Mean Shock Selected
8
7
6
5
4
3
2
1
Sober/ No Inhibition
Sober/ Inhibition
Intoxicated/ No Inhibition
Intoxicated/ Inhibition

Figure Caption

Figure 6. Proportion of '5s' set, by drug group and limb of the blood-alcohol concentration (BAC) curve.


0.5
0.4
0.3
0.2
0.1
0
Proportion of "5's" Set
Ascending Limb
Descending Limb
Intoxicated
Sober

APPENDIX D

Pihl, R.O., & Hoaken, P.N.S. (2001) Biological bases to addiction and aggression in close relationships. In: Wekerle, C. & Wall, A.M., (Eds.), The violence and addiction equation: Theoretical and clinical issues in substance abuse and relationship violence. Philadelphia, PA: Brunner/Mazel., In Press.

Biological Bases of Addiction and Aggression

In Close Relationships

R.O. Pihl & Peter N.S. Hoaken

Department of Psychology

McGill University

Running Head: BIOLOGICAL BASIS TO ADDICTION AND AGGRESSION

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Numerous current and substantive reviews of the biology of drug addiction exist (Pihl & Peterson, 1992). Less evident but still plentiful are those related to drug involved aggression, although often focus is specific to a single neurotransmitter (Pihl & Lemarquand 1998) or aspect of cognitive functioning (Hoaken, Giancola & Pihl, 1998). All of these reviews are consistent with the relatively recent trend, driven by rapid methodological innovation and daily discovery, of drawing upon biology to explain psychological and social phenomenon. For example, the current head of the National Institute of Drug Abuse illustrates this view in an article titled "Addiction is a Brain Disease, and it Matters", where he notes, "Addiction as a chronic, relapsing disease of the brain is a totally new concept for much of the general public, for many policy makers, and, sadly, for many health care professionals" (Leshner 1997, p. 46).

The present chapter will not be a collectively exhaustive review, covering already well worn ground; rather its goals are to present a structure which integrates information for both the biology of addictions and drug related aggression, and to link relevant information to the focus of this volume, "close relationships". The latter goal is particularly challenging as at face value what the biology of addiction and drug related aggression has to do with close relationships is not apparent. There is, of course, the obvious; aggression in humans and other primates is primarily a social behaviour. But beyond the blatant, just what is particular to close relationships which might interact with the biology of various drugs

requires equally apparent speculation. The two relevant literatures appear almost mutually exclusive: the former dealing with social psychological and sociological theories on addiction and aggression in close (read: family) relationships; the latter on the biology of addiction and violence. For example, there exists virtually no research on biological factors in marital aggression. Thus, writing a chapter on biological factors in close relationships poses a challenge.

This chapter thus begins by reviewing and synthesising the extant literature on the biology of addiction and aggression within the framework of putative motivation systems. This will be followed by a discussion of a new focus of attention in addiction and aggression research, the frontal cortex. The chapter concludes by examining how these biological factors impact "close" relationships and vice versa.

Biology and the Risk for Addictions and Aggression

There are many ways to organise an examination of the contribution of biology to addition and to the drug-aggression relationship. The perspective of level, i.e., genetic, biochemical-physiological and neurobiological, is a common approach. Each of these levels offer many facts which demonstrate how each in turn contributes to increased risk. Often lacking, however, is an explanatory model of how these facts interrelate and interact in what are highly complex causative equations. The approach of asking how these facts, genetic, biochemical,

physiological, et cetera impact biological systems demands integration and presents a more functional representation. General questions, such as what factors impact degree of risk for abuse, as well as specific, such as which alcohol-affected biological mechanisms alter provocation threshold in the alcohol-aggression relationship, can be addressed.

We have previously hypothesised the existence of at least four relevant biological systems, important to risk for drug abuse (Pihl & Peterson, 1995a) and drug-related aggression (Pihl & Peterson, 1995b), which are differentially responsive to various drugs and to which, for a myriad of reasons, individuals differ in their responsivity. Although these systems are theoretical, they synthesise and model current drug effect information from all levels, and further begin to allow some understanding to perplexing questions such as: why do only some individuals develop abuse/dependency problems; why do some individuals appear more susceptible to some drugs and not others; and why do individuals aggress when under the influence of a drug or drugs? Currently, the four systems are labelled: A) the cue for reward system, B) the cue for punishment system, C) the pain system, and D) the satiation systems. Each system encompasses the operative interaction of biological and experiential factors. This is essential as biological contributions are likely meaningful only in terms of what has happened, is happening, and will happen to the individual. In this regard, it needs to be said that the nature-nurture debate, in the extreme, which invariably arises when biological

explanations for a social phenomenon like drug abuse and drug-related aggression are considered, is a perversity of logic. Patently, we do not exist without a body nor in a vacuum. Thus the following discussion and speculation regarding the interaction between drugs and brain mechanisms which increase the likelihood of addiction and aggression should be viewed as only part of the story. The explanations are not reductionistic, rather they depend on contextual factors, past, present and future. On the other hand, it is equally incongruous but unfortunately common that some psycho-social explanations and adherents thereof seem to ignore the fact that the drugs of concern affect brain functioning. We shall now consider how drugs/alcohol affect these four systems, in "susceptible" individuals, and how the likelihood of addiction and aggression is therefore altered.

The Cue for Reward System

It is common to refer to drugs which are abused both legal and illegal as rewarding. Why else, the argument goes, would individuals steadfastly, and in the face of great negative consequences continue their seemingly single-minded drug taking, abusing behaviour? There exists a vast animal and human literature which has delineated the reinforcement properties of many drugs. Yet, to simply note that certain drugs are rewarding obscures complex relationships and even totally opposite reasons why different individuals abuse the same drug. The obvious conundrum regarding simple reward explanations is that primary

reinforcers like food, water, sex, to which some drug effects are often compared, actually result in satiation where these reinforcers are no longer temporarily effective. Satiation typically does not occur with for example drugs which increase stimulation by activating certain dopaminergic mechanisms. Thus, the importance of the hypothesised cue for reward system. A cue for reward is anything which previously has been contiguously associated with something basically rewarding, that is satiating, or with something that produces the cessation of punishment or threat or anything novel. Cues of reward (promise, if you will) in themselves result in psychological states of excitement, curiosity, pleasure and hope. This "system" is often referred to as the psychomotor system as its stimulation results in an activation of involvement, that is, movement toward biologically relevant stimuli. In addition, accompanying important subjective feelings of excitement, curiosity, euphoria, and of increased power and energy also occur. The seemingly biological purpose of these effects is to force us to approach that which may be primarily reinforcing. Animals will work to activate electrodes or chemicals that stimulate this system and such stimulation reinforces learning such as condition place learning (Fibiger & Phillips, 1988). It is also known that the degree of these effects depends on the density of dopamine receptors. Specifically implicated is the dopaminergic pathway of the ventral tegmental-nucleus accumbens area. (Koob 1992; Wise & Bozarth, 1987) Stimulant drugs such as cocaine and amphetamine in

particular activate this system but so does alcohol, THC, nicotine, PCP, and some prescribed drugs. Cocaine for example seems to slow dopamine re-uptake as does amphetamine which also releases dopamine. (Koob & Bloom, 1988). Use of these drugs in particular can result in sensitisation where other similar drugs become more effective (Wise 1988). For example, the high correlation, (r = .84) between cocaine and alcohol abuse is relevant (Helzer & Pryzbeck 1988). When cocaine and alcohol are combined, there is a substantial increase in cocaine related euphoria, improvement in alcohol psychomotor performance and an overall increase in heart rate (Farre et al. 1993). In fact, cocaine and alcohol interact to produce a metabolite, cocaethylene, which significantly increases dopaminergic effects on the cue for reward system which seems responsible for the enhanced euphoria-inducing effects when these drugs are combined (Jatlow et al. 1991, McCance-Katz et al. 1993).

Alcohol alone can and does effect this reward system. Alcohol produces effects on locomotor activity, which have been shown to be dopamine mediated (Dudek et al. 1984) and like other stimulant drugs can result in place preference learning particularly on the rising limb of the blood alcohol curve (Reviewed by Wise & Bozarth 1987). One notable outcome, also, like the other stimulants is that alcohol can cause an increase in resting heart rate (Sher et al. 1994, Finn & Pihl, 1987) along with concomitant positive subjective feelings (Martin et al. 1993, Conrod et al. 1998). In general these effects are time and dose limited (Jones et al.

1976; Stewart et al. 1992), and more importantly, they are also individually differential. In fact, genetic factors have been implicated in determining the degree of response to the stimulating and/or sedating effects of alcohol (Dudek et al. 1991). For example, particular alcohol preferring strains of rats when compared to other especially bred strains demonstrate heightened dopaminergic activity to alcohol, more sensitivity to the locomotor activity enhancing effects and more resistance to the sedating effects of alcohol (Gordon et al. 1993). These effects may be both direct or indirect were the activity of other biochemical systems which operate on the dopamine system are altered (Harris 1994). In humans, a substantive increase in heart rate to alcohol has been found in alcoholics (Peterson et al. 1996) and in some multigenerational sons of alcoholics who are at a 4 to 9 times increased risk for developing the disorder (Conrod et al. 1995; Conrod et al. 1998). Recently, Bruce et al. (1999) demonstrated additional positive effects of this response. Twenty four hours after intoxication individuals who showed this high heart rate response recalled more positive and fewer negative words than low heart rate responders from a list of words learned while intoxicated the previous day.

The above findings illustrate how variability in the functioning of the cue for reward system would effect susceptibility to addiction. However how this variability is relevant to a biological explanation of drug-related aggression is less clear. An answer comes from both data and theory. In a recent study, we challenged, with an intoxicating dose of

alcohol, young men who had been part of the longitudinal study which began when they were age 6 in kindergarten (Assaad et al. 1999). At that date, 12-13 years ago, these individuals were selected for the study by their teachers on the basis of their level of demonstrated aggressive school behaviour. Continuous testing throughout the years, in school, at home and in laboratory studies, confirmed the stability of behaviour for those who were highly and continuously aggressive from the others who were consistently non-aggressive. What the alcohol challenge data shows, is that those individuals who display a high heart rate response to alcohol are more likely to have a history of delinquent behaviour, engage in more fighting and in general display a gamut of antisocial acts when compared with other subjects. Further, these individuals are also more likely to display sensation seeking particularly disinhibited personality characteristics. These results might be interpreted to suggest that individuals with a heightened sensitivity of the cue for reward system are at risk for aggression and related behaviours. These results also replicate the well known fact that conduct disorder/ASPD and alcohol/drug abuse are frequent co-morbid disorders. The question of whether drugs/alcohol intoxication will increase or decrease aggression in these subjects is the focus of a current laboratory investigation. It is known that sons from a male limited tense family history of alcoholism (Type 2 Cloninger 1987) are prone to aggression when intoxicated as are women diagnosed with ASPD (Conrod et al. 1999). Finally, the literature, (detailed later in this

chapter) which relates frontal lobe functioning deficits, frequent in these subjects and alcohol produced frontal dysfunction in increased aggressivity is relevant. Substantial data exists detailing the inhibitory control that frontal structures play over the cues for reward system by effecting the release of dopamine and subsequent psychomotor behaviour.

Theoretically, how alcohol specifically effects the cues for reward system to potentiate the increased likelihood of aggression is illustrated in Figure 1. First, simply by promoting exploration, raising "excitement and curiosity" stimulation of this system increases the likelihood of confrontation. Additionally and importantly the effects of this system and the concomitant subjective responses are likely analgesic to that which is externally threatening and inhibitory to aggression. In that omnipresent valence between action/promise and inhibition/threat, by activating cues for reward, cues for threat are diminished. In a sense then the breaking of rules which by definition often leads to the unexpected becomes exciting and initiates activity. Further, in the extreme, and in particular it is the diminishment of sensitivity to threat and punishment by the activation of this approach behaviour which results in aggressive behaviours, dangerous to the self and others.

The Cue for Punishment (Threat) System

The majority of problem drinkers when asked why they drink will give an answer which fundamentally translates as, "to reduce stress".

Indeed, like benzodiazepines and barbiturates alcohol also has anxiolytic effects. Pharmacologically, alcohol and other anxiolytics operate on the major brain inhibitory neurotransmitter, GABA. It has been shown, for example, that alcohol effects the chloride ion channels at the GABA benzodiazepine receptor (Warnecke, 1991), increases the firing rate of GABA and thus increases inhibitory action on other neurons. Stress often represents threat which can be operationalized as a cue for punishment, as are pain, depriving situations, sensory over-stimulation, frustration, disappointments, social isolation and the occurrence of novelty. It appears a great deal of the mammalian brain is hard wired to deal with threat as the continued survival of the organism can rest on the speed of reactivity. It is a jungle out there, at least it used to be, and not reacting fast enough could quickly result in death. Thus the purpose of the threat system is to inhibit ongoing behaviour and stop us from getting hurt. This responsivity can take the form of specific fears or more generally anxiety. Fear protects from that which has previously produced harm and anxiety results in caution to cues of threat. For example, isolation from others is a threatening condition and it is anxiety that leads us to be careful and appropriate in the presence of others, alerting us to our possible, current, past and future faux pas.

As with cues of reward there appears to be differential individual sensitivity to cues of threat. Clearly, this reactivity can be taken to the pathological as witnessed in the anxiety disorders. This individual

sensitivity can well be genetic as, it has been shown that the high alcohol sensitive strain of rats and the long sleep strain of mice are much more responsive to the sedative effects of alcohol than their counterpart strains. (Crabbe 1989) This sensitivity to the sedative effect of alcohol is also exhibited by the fact that these animals will die from a lower dosage than controls. In humans the abuse of alcohol correlates with anxiety sensitivity in both clinical (Cox et al. 1993) and non clinical (Stewart et al. 1994) samples. Additionally, anxiety sensitive individuals have been shown to display a decrease in their stress response (called response dampening) when intoxicated (Stewart et al., 1994). It is logical to assume therefore that the heightened abuse pattern found in these individuals reflects a form of self-medication with alcohol activating the brain's inhibitory mechanisms and thus decreasing responsivity to threatening stimuli. Most notable in these threatening stimuli for anxiety sensitive individuals are cues attacking the integrity of the self. (Stewart et al, 1999). Some sons of alcoholics also show stress response dampening, but in the sober states are over-reactive to novel stimuli, likely related to a cognitive problem. For both these groups the modification of the threat response even though for different reasons explains the increased abuse potential these groups reflect. This dampening of the threat system also explains more generally the increased likelihood of aggression when intoxicated.

Figure 2 illustrates how threat inhibits aggression. Even given intense provocation, the possibility of retaliatory pain and injury (physical, psychological, social) controls the response. We have demonstrated this effect in numerous laboratory studies where knowledge of likely consequences, and the determined motivation of the attacker, inhibit retaliation when sober (Zeichner & Pihl, 1979, 1980). Indeed it is likely this inhibitory control is to a large degree what the socialisation process is all about. The presumed threat of retaliation and "conscience" even when provoked by the aggressor is what likely keeps most of us in check. This control, however, is somewhat soluable on alcohol. As Figure 2 illustrates, an intoxicating dose of alcohol is seen as inhibiting this inhibition. This specific conclusion is derived from numerous animal and human studies. Intoxicated rats typically venture from hiding even knowing a cat is present, which increases the likelihood of their demise (Blanchard et al. 1993). This diminishment of inhibition of control also explains the frequent role of drug intoxication in victims of violence. Intoxicated humans even when aware of the consequences will retaliate when provoked, i.e., knowing that a fight will likely erupt (Zeichner et al., 1982). Thus it is not the absence of knowledge, i.e. drug induced stupidity, which is the operative mechanism but likely the inhibition of threat control.

The Pain System

The pain or punishment system refers to where and how the negative consequences of behaviour which decrease the future likelihood of that behaviour operate. Neuroanatomically, there appears to be considerable overlap between structures which affect the affective aspects of pain and aggressive behaviour, including the prefrontal and orbital frontal cortex, the amygdala, various hypothalmic nuclei and the dorsal raphe nucleus. (Albert et al. 1993; Raine et al. 1998; Chapman 1996, Wills & Westlund (1997). Negative consequences which effect this system are many and divergent. They include not just the obvious, e.g. simple physical pain but also states of deprivation, sensory over-stimulation, frustration, disappointment and social isolation. The response to pain can be both inhibitory and anger inducing. Inhibition occurs in the sense of "once hurt, twice shy" and anger and aggression can often result into the putative purpose of the elimination of the presumed source of the pain. Thus fear and hate are coexisting emotional responses to a painful stimulus and those which decrease pain are negatively reinforcing. Analgesics such as the opiates operate on the periaqueductal gray brain area which is directly related to the mediation of pain (Panksepp, et al. 1985), although these drugs also effect the cue for reward area (Wise 1988). Further, their administration not only reduces physical pain but also social distress (Knowles et al. 1987), developed in maternally deprived stress induced animals. Interestingly, this effect can be reversed by the opiate

antagonist nalexone (Knowles et al. 1987). Alcohol, also impacts the pain system through that system's neurotransmitters, the endorphins. It has been shown that C57 BL/6J alcohol preferring mice have a particularly low pain threshold which is increased by opiate agonists which concomitantly decreases alcohol consumption (Gianoulakis & Gupta 1985). In our research, we have shown that sons from families with multigenerational male alcoholism reflect greater sober pain sensitivity (ratings to electric shock), than men with no family history of alcoholism (Stewart et al. 1995). We have also shown that these individuals display a substantive increase in plasma beta endorphin on the rising limb of the blood alcohol curve (Peterson et al. 1996). We obtained a correlation of .91 between blood alcohol and plasma endorphin levels in individuals who showed a high heart rate response versus one of .26 for negative family history controls. We have recently demonstrated that naltrexone blocks this response (Peterson et al. 1999).

The putative relationship between alcohol, the pain system and aggression, is highly complex. First, there is the situation where alcohol acts much like it does on the threat system decreasing/inhibiting the withdrawal response inherent in the pain response. Thus, a shift in the fear- anger valance occurs in favour of anger and retaliatory attack occurs. Certainly, in many species, non-avoidable pain is perhaps the most common and reliable procedure for eliciting aggression, both direct and indirect. To reiterate a point we have made a number of times in this

chapter, substantial provocation with negative stimuli is usually required to produce aggression in humans. Although, there are a myriad of different forms of aggression, retaliatory/defensive aggression predominates. In this sense drugs like alcohol also effect sensitivity to pain. While it is true, that alcohol is seen by many as a sedative and has in the past been used as a surgical anaesthetic (Mullen & Lockhart 1934) this effect is dose and individual dependent. At moderate dosages, and on the rising limb of the curve, alcohol has been shown to result in heightened ratings of pain sensitivity in a general population (Gustafson 1985), a response also seen in lowered pain thresholds in alcohol intoxicated rats (Gray 1982). Thus, alcohol and other drugs may contribute to aggression simply by increasing reactivity to pain, i.e. the significance of provocation.

The complexity of this situation however is demonstrated in some individuals who even on the rising limb of the blood alcohol curve seem to show less responsivity to pain. We have demonstrated (Pihl et al. 1990) on our laboratory aggression task that some men with a positive family history for alcoholism who tend to be more aggressive when sober and putatively more pain sensitive are less aggressive and reactive when intoxicated.

The Satiation System

The biological systems involved in unconditioned positive reinforcement comprise what we view as the satiation system. The

neurotransmitter serotonin (5HT) is uniquely involved in a wide range of basic functions including food and water intake, sexual behaviour and sleep. It appears that serotonin operates on these systems by generally providing a moderating influence on the activity of other neurotransmitters. For example, serotonin, has been shown to inhibit behaviour to threat (Sobrie, 1986) and to release dopamine and increase locomotion (Kriem et al. 1996) Indeed, the functioning of serotonin in the brain has been analogized as the maestro of an orchestra organising and controlling talented individualists and melding them into a harmonious unit (Pihl & Peterson 1995). At appropriate optimum levels of the functioning of the neurostransmitter, neuro-synchrony is thought to result, while at insufficient levels of functioning, dys-synchrony (Spoont, 1992).

Low or insufficient serotonergic functioning has been linked to aggression in both animals and humans (see Pihl & Lemarquand, 1998 for review). For example, mice bred to lack a specific serotonergic receptor are more aggressive when provoked than controls (Saudou et al. 1994), and when serotonergic activity is less, as measured by spinal fluid metabolites, monkeys are spontaneously more aggressive, have a higher mortality (Higley et al. 1996), and take more risks (Mehlman et al. 1994). Conversely, with higher serotonin levels monkeys display greater pro-social behaviour (Mehlman et al. 1995), and are more dominant (Higley et al. 1992). In a study where we manipulated the serotonergic precursor tryptophan, functionally depleting brain serotonin levels, we increased

level of aggression in vervet monkeys (Chamberlain et al. 1987) and humans (Pihl et al. 1995). The general disinhibiting effect of manipulating serotonin is also seen where drugs which produce low levels of release result in increased impulsivity whereas at higher levels and thus increased 5HT functioning there is a decrease in impulsivity. (Poulos et al. 1998). The literature with humans reflects a very similar story with low serotonergic functioning also measured in terms of cerebral spinal fluid metabolite levels being related to individual histories of aggressive impulsive behaviour (Brown et al. 1982) homicide (Lidberg et al. 1985) children with impulsive and aggressive behaviours (Kruesi et al. 1990) impulsive violent offenders (Virkunnen et al. 1994) individuals with serious suicide attempts (Virkunnen et al. 1989) impulsive individuals with alcoholic fathers (Linnoila et al. 1989), self-reported aggression in normals (Roy et al. 1988) and in alcoholic violent offenders (Virkunnen et al. 1994). That low levels or dysfunction of 5HT is involved in the expression of aggression/impulsivity is also supported by a large number of studies showing blunted hormonal responses to 5HT agonists. Demonstrated is lower receptor numbers or functioning in platelet 5HT re-uptake studies in impulsive/aggressive patient populations (see Pihl & Lemarquand 1997 for review). In experimental investigations involving acute tryptophan depletion similar conclusions are possible. What the data suggests is that 5HT is involved in controlling responsivity to stimuli for it is specifically

the response to provocation that is altered by reducing the serotonin levels. This is illustrated in figure 3 (Pihl et al. 1995).

Alcohol does affect serotonin levels although the relationship is complex (see Lemarquand et al. 1994a and 1994b for reviews). In numerous studies with various methodologies it appears that increased serotonergic function leads to decreased ethanol consumption. A low level of serotonergic functioning seems to be a risk factor for alcohol abuse in that while acute alcohol intake increases brain 5HT functioning, chronic intake may actually decrease it. We have speculated (Pihl & Lemarquand 1998) that such chronic consumption might lead to a general state of lowered 5HT functioning and thus a greater incidence of impulsivity and aggressive behaviour. Recent attention has focused on subtypes of individuals, particularly individuals with early onset of drinking problems and/or antisocial characteristics. The serotonin autoreceptor 5-HTIB has been implicated in mouse (Saudou et al. 1994) and human studies (Lappalainier et al. 1998) for both drinking and aggression. It is precisely this specificity upon which the system's approach presented in this review is based.

The Frontal Cortex: Executive Cognitive Function and Drug- Related Aggression

Figure 4 illustrates the relationship between the apparent two most important systems regarding drug related aggression, the cue for reward

and threat systems, and another biological variable which has recently begun to garner significant research interest in those interested in addiction and aggression - the function of the prefrontal cortex. The importance of this brain area for human behaviour is, simplistically, illustrated by its larger size relative to other cortical lobes, a fact which differentiates humans from other primates. In fact, research has demonstrated that disruption by drugs, or pre-existing dysfunction, of the cognitive capacities mediated by this brain area, capacities often labelled executive functions, impact directly on likelihood of aggression.

The executive cognitive functions comprise "higher-order" cognitive activities, including attention, planning, cognitive flexibility, abstract reasoning, self-monitoring, and the ability to integrate external and internal feedback in order to adaptively modulate further behaviour. Executive functioning is commonly conceptualised as the ability to use these functions to plan, initiate, and regulate goal-directed behaviour (Giancola et al., 1998). It appears that the prefrontal cortex represents the neural substrate that subserves these functions (Giancola, 1995; Stuss & Benson, 1984) with the dorsolateral prefrontal-subcortical circuit, specifically, the neuroanatomical pathway of considerable interest (Cummings, 1995). This pathway consists of dorsolateral prefrontal cortex and projections to and from several subcortical structures, including the caudate nucleus, globus pallidus, substantia nigra and several thalamic nuclei, thus interacting directly with the cue for reward and threat systems.

There is considerable evidence that associates frontal lobe deficits with decreased regulation of human social behaviour. Individuals with frontal lobe damage often manifest a "disinhibition syndrome" (Hecaen and Albert, 1978), and even in non-lesioned individuals there is evidence of this relationship: One study demonstrated a substantial relationship between aggressivity and scores on carefully selected tests of executive functioning in boys (Séguin et al., 1995). Further, from a neuroimaging standpoint, a study employing positron emmission tomography demonstrated that, while not lesioned per se, murderers demonstrated more prefrontal abnormalities than did matched controls (Raine et al., 1994). There have been a series of studies that show that psychiatric disorders characterised by antisocial behaviour such as conduct disorder (Moffitt, 1993), antisocial personality disorder (Gorenstein, 1987), and attention-deficit hyperactivity disorder (Barkley, 1997; Benson, 1991) are all also characterised by poorer performance on tests of executive function, relative to controls.

It is widely agreed that these cognitive capacities are related to heightened propensity for aggression, and there is substantial evidence that alcohol and other drugs interfere with these capacities. Peterson and colleagues (1990) were among the first to hypothesise that the prefrontal cortex may be particularly susceptible to alcohol intoxication. In order to test this hypothesis, the experimenters administered low, moderate, and high doses of alcohol to participants, in a balanced-placebo design

intended to eliminate the effects of expectancy, and then administered a battery of cognitive tests. The results, illustrated in figure 5, show that high doses of alcohol detrimentally affected a number of functions associated with the frontal lobes, including planning, memory, and complex motor control, but appeared to have a lesser effect on other "non-frontal" tests. In a subsequent study (Hoaken, Assaad, & Pihl, 1998), it was shown, through a repeated measures design, that alcohol significantly and specifically interfered with tests of executive function, even at a moderate dose. It is apparent, then, that alcohol produces cognitive deficits reminiscent of prefrontal damage. In a series of laboratory studies we have begun to examine the effects of both alcohol and executive functioning on behavioural measures of aggression. In the first study of this relationship (Lau, Pihl, & Peterson, 1995), two tests of executive functioning were used to screen male participants. Participants falling into either the highest or the lowest quartile of executive function were retained; half of each of these groups were administered alcohol, the other half remained sober. In this study, as provocation increased, the participants in the lower quartile became progressively and significantly more aggressive than those in the highest quartile. This led to the conclusion that alcohol intoxication and frontal lobe dysfunction were both involved in the disinhibition of aggressive impulses. Giancola and Zeichner (1994) also reached this conclusion, in a similar study.

In a subsequent study, Lau & Pihl (1996), in order to test their hypothesis that heightened aggression was due to an inability to inhibit impulsive behaviour, tested whether participants with poor executive functioning would be able to inihibit aggressive responses if offered contingent monetary reward for not responding aggressively. Again participants were classified into highest and lowest quartiles of executive function and tested sober for aggression. The results of this study showed that the men in the lower quartile, when highly provoked and paid to inhibit nonetheless responded aggressively, whereas the men in the upper quartile inhibited aggression. It appears as if men with these patterns of cognitive dysfunction are impulsive, and respond aggressively when provoked without being able to switch to other, more appropriate behaviours.

We know from decades of studies that drinking alcohol makes individuals more likely to manifest aggression, we also know that the vast majority of drinkers do not aggress when they become intoxicated. It may well be that sound executive functioning underlies this reality. It has been demonstrated that even when acutely alcohol-intoxicated, men with above-average executive functioning will inhibit aggression in order to gain reward. That is, even when legally drunk subjects seem to retain sufficient residual executive function to identify and utilize more appropriate behavioural responses to provocation (Hoaken et al., 1998). Although the aforementioned studies used men as participants, there is

accumulating evidence that executive functioning may also well be related to aggression in women. Hoaken, Strickler and Pihl (2000) conducted a study in which two groups were tested on the aggression paradigm, one sober, the other acutely alcohol intoxicated. What was surprising about this study was that alcohol intoxication did not appear to be as important as in previous studeis with men, as sober women were as aggressive as their intoxicated peers in the face of heightened provocation. What did appear to be consequential for predicting who would and who would not become aggressive was executive functioning. Measures of executive functioning taken before drink administration correlated very highly with aggression, irrespective of alcohol intoxication.

There are also studies that seem to suggest that poor executive functioning seems to predispose to alcohol and drug use and abuse (Pihl et al. 1990; Peterson & Pihl, 1990). What is important to consider regarding executive functioning is that this cortical area provides the context for integrating and interpreting stimuli. Expectancies and/or cognitive schemas blend past learning, including language and imitation, to colour event appraisal and determine behavioural outcome, which is a very social, culture specific process. One's general expectancy set then subsumes the regulation of anxiety/threat, psychomotor activity, as well as beliefs regarding drug effects and interpersonal aggression. Historically and individually determined behaviour, and its dysfunction, due to prior programming (or lack thereof), incapacity, or drug insult (albeit

temporary), can readily place the individual out of context and inappropriate.

Specific Mediating Factors of the "Close Relationship".

The reader may now wonder what is specific about a "close" relationship that may somehow alter the seemingly deterministic biological factors deliniated above. Three foci for consideration are assortative mating, genetic factors and the saliency of provocation.

Assortative Mating

Many studies, dating back decades, have observed that there appears to be a high incidence of shared morbidity of various forms of psychological distress within married couples (Galbaud du Fort, Kovess, & Boivin, 1994). What this observation means however is arguable. Possible explanations are: that a husband and wife living together apply profound influences on one another, and as such one partner may "infect" the other with abnormal behaviours or reactions; it may be the case that the breakdown of the (shared) social environment may lead to pathology in both partners; and/or it may simply be the case that when one partner enters treatment, treatment for the second becomes facilitated (Neilsen, 1964). Alternatively, there may be some tendency of couples to attract each other on the basis of some pre-existing similitude. This tendency for couples to be more similar for some phenotype or behaviour than would be expected if they were paired randomly has been referred to as spouse

similarity (assortative mating). This description appears to be particularly true for alcoholics where mutual partner diagnoses far exceed what would be expected by random pairing (Hall, Hesselbrock, & Stabenua, 1983a, 1983b; Jacob & Bremer, 1986). One study, for example, demonstrated that adult children of alcoholics marry other adult children of alcoholics at a rate more than twice that found in the general population (Black, Bucky, & Wilder-Padilla, 1986). This is important because if both members of a couple share a predisposition for alcoholism, they likely share underlying biological characteristics which are a basis for the increased risk of addiction and also possibly aggressive behaviour. Furthermore, if both members of a couple are intoxicated, provocation becomes that much more salient for both members, and as such, aggression becomes that much more likely. It has been demonstrated that more than half of all perpetrators and nearly half of all *victims* of violent crime are acutely intoxicated at the time of the crime, which certainly seems to support the contention that dual intoxication is a strong risk factor for aggressive interaction (Murdoch, Pihl, & Ross, 1990).

Evidence of assortative mating has also been found in heroin addiction (Anglin et al., 1987), schizophrenia (Alanen & Kinnumen, 1975), and antisocial personality (Guze et al., 1970; Cloninger et al., 1975; Kreuger et al., 1998). the latter associated with both poly-drug abuse and aggressive behaviour. Thus, spousal similarity may be an element of a

"close relationship" which leads to increased probability of both addiction and aggressive behaviour.

Genetic Considerations

Another aspect of close relations is the heritability of risk for addiction and aggression passed from parents to offspring. It is critical to view the family as an interacting unit and not explicitly focus on the couple/parents. Stated succinctly, "The child acts; the environment reacts; and the child reacts back in mutually interlocking-evocative interaction." (Caspi et al., 1987, p. 308).

Bi-directionality of causality has been demonstrated in a number of studies. In one illustrative study (Blackner et al. 1996), the researchers showed that parenting interacted with a child's difficult temperament to increase behaviour problems. Further, the added difficulties of a problematic offspring create and exacerbate existing parental relationship and individual problems. The literature on the heritability of risk for addiction and aggression is compelling. For example, regarding alcoholism, family, twin and adoption studies place the contribution of genes for both men and women at approximately 60%. (Cotton 1979; Heath et al. 1996; Cloninger et al. 1981). The search for candidate genes is currently exuberant with many and divergent findings reported. (See Goate & Edenberg 1999 for recent review). However, at this juncture only very preliminary speculation can be drawn and conclusions must wait considerable replication. Yet, what is well known is that phenotypic

expression presents a pattern of high risk for addiction and aggression. We and others have previously reviewed the behavioural characteristics of sons of alcoholics (Pihl et al. 1990; Sher 1991; Windle & Searles 1990), particularly the behavioural dysregulation, electrophysiological and biochemical idiosyncracies they display. Typically, found in these individuals are disciplinary problems with histories of impulsivity, rebellion, conduct disorder and antisociality. Very frequently aggressive behaviour both sober and intoxicated has been noted (Cloninger 1987). In addition, there is evidence that offspring of alcoholics have a greater rate of crime and violent crime than do controls and that violence in the father is related to addiction in the offspring. For example one Swedish adoption study showed a significant correlation between violent offences in the biological fathers and alcohol dependence in the male adoptees (Bohman et al., 1982).

The Saliency of Provocation

Mentioned previously is the increased saliency of provocation if both members of a close relationship are intoxicated. Numerous laboratory studies of the alcohol-aggression relationship clearly underscore the increased impact of provocation when intoxicated (Pihl et al. 1993). In fact, seemingly all putative variables that impact the relationship seem to interact with level of provocation. Specifically, drugs which enhance aggression appear to lower the reactivity/retaliation threshold of the individual. Even where particular neurotransmitters like

serotonin are involved the interaction with level of provocation has been demonstrated. (Smith et al, 1987; Pihl et al 1995). Both these studies used a tryptophan depletion paradigm (which dramatically decreases brain serotonin, with similar subjects, participating on the same aggression task. The earlier study used a noise as the aversive stimulus and found no effect whereas the latest research provoked with a brief electric shock and produced a strong tryptophan depletion effect when compared to two control conditions.

However, it might be that the saliency of the provocation in a potentially violent altercation between two individuals in a close relationship would serve to *decrease* the likelihood of aggression. One of the most important aspect of the executive functions is a behaviour-inhibition capacity. In light of this, we suggest that the inhibitory aspects of the frontal cortex may well mitigate aggression in close relationship more so than in other relationships. Plainly said, we are suggesting that in a close relationship, the salience of provocation makes situations less likely to become violent because of the increased involvement of the inhibitory aspects of the frontal cortex.

Conclusion

From a biological point of view, there are several factors which may place individuals "at risk" for either addiction or aggression, and often, both. As we have indicated in some detail, several biological

motivational systems, constantly interacting with each other, and mediated by the inhibitory aspects of the prefrontal cortex, mediate likelihood of these kinds of impulsive, maladaptive, disregulated behaviours. However, what we can say about the extent to which these factors are more or less in evidence in the context of a "close relationship" is limited. From a biological point of view, we hold the individual and his or her personal biological makeup to be most predictive addictive or aggressive propensity, and suggest that an individual "wired just right", if you will, will manifest the problematic behaviours irrespective of social milieu. However, that is not to say that the social milieu is unimportant. Future research would do well to attempt to elucidate the relative contributions of biology and social environment in the manifestation of addiction and aggression in close relationships.

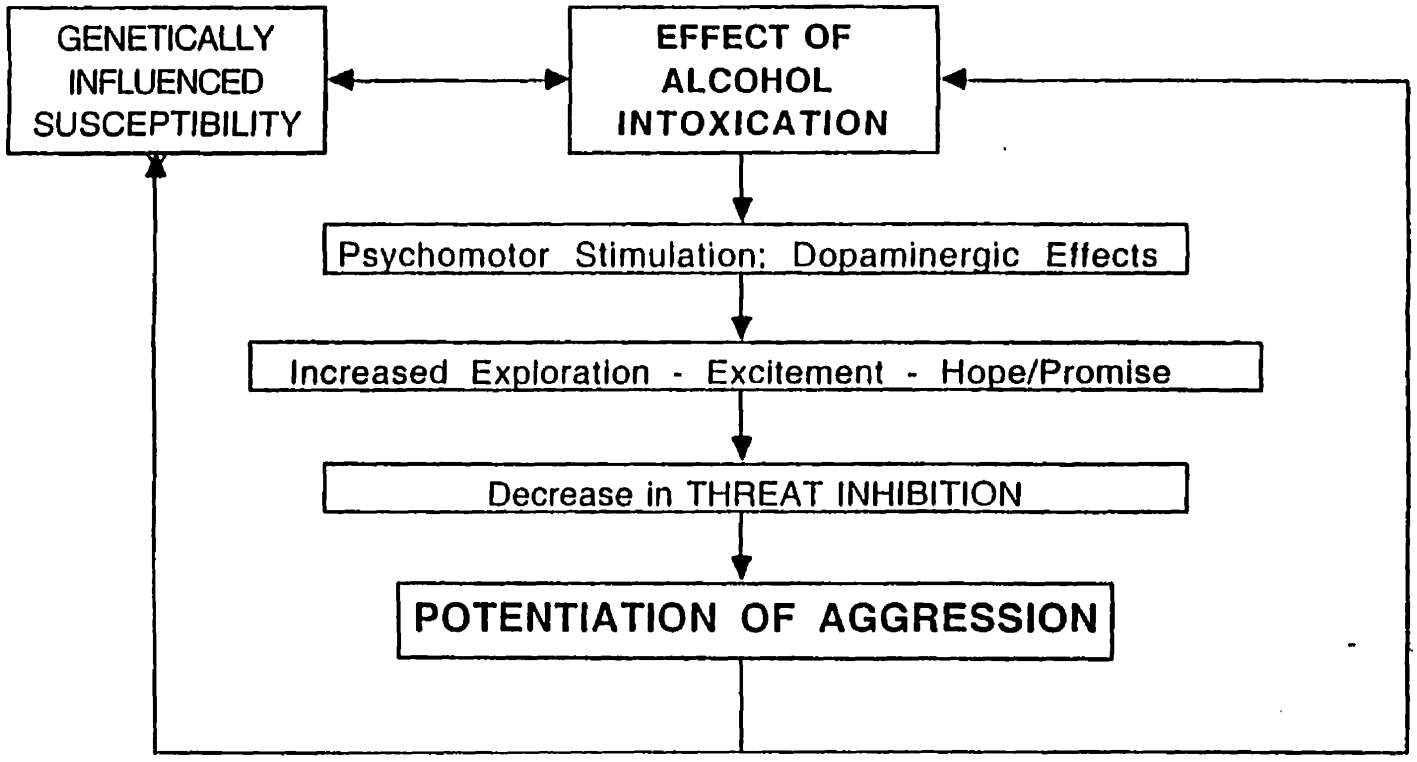

Figure 1. The effect of alcohol on the cue for reward system.

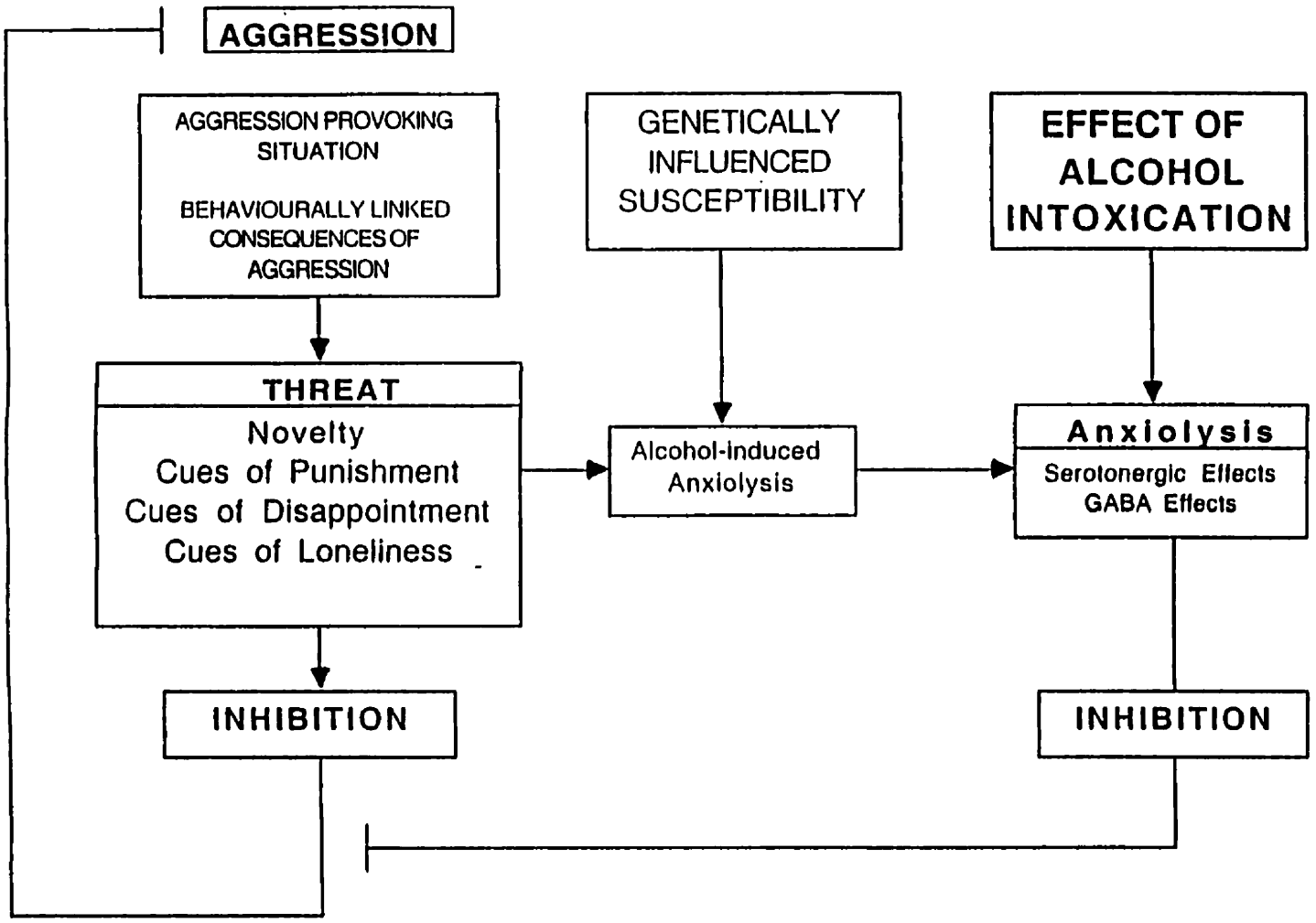

Figure 2. The effect of alcohol on the cue for punishment system.

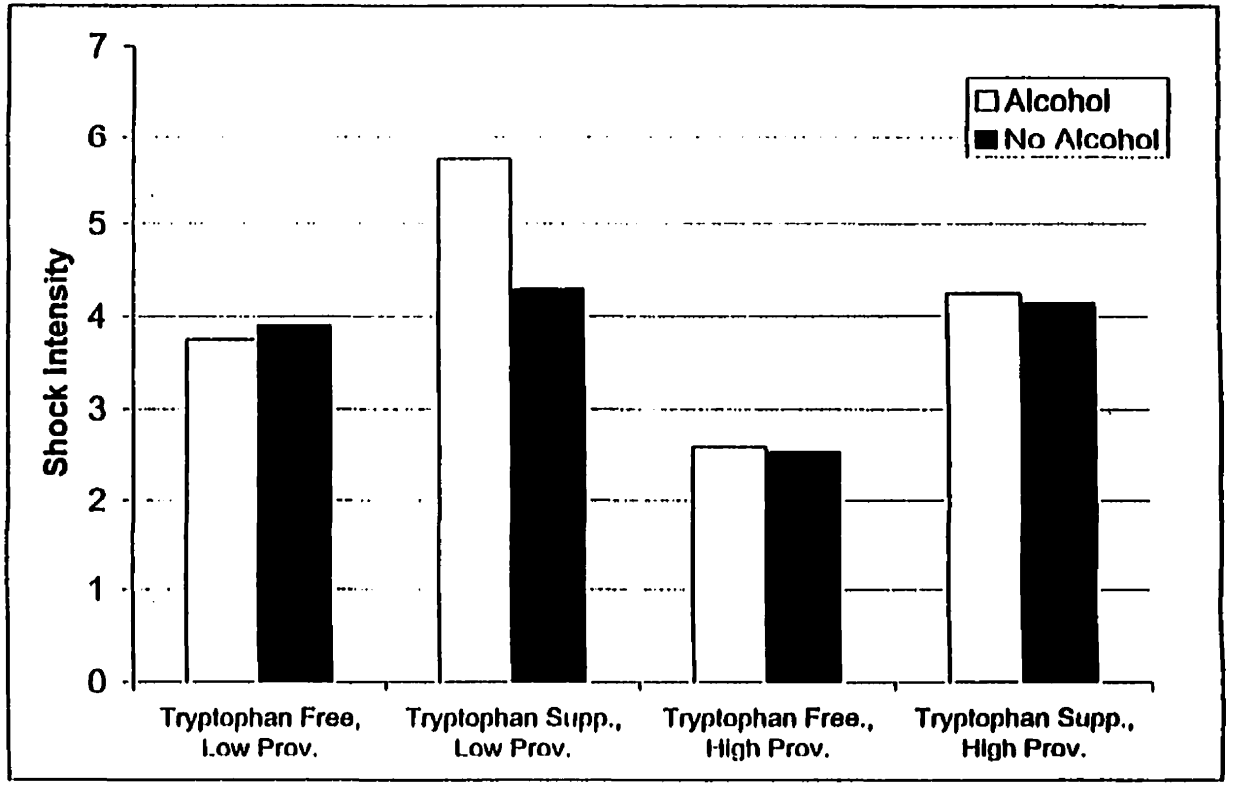

Figure 3. The effect of altered tryptophan levels and alcohol under low and high provocation on the intensity of shock administered to subjects.

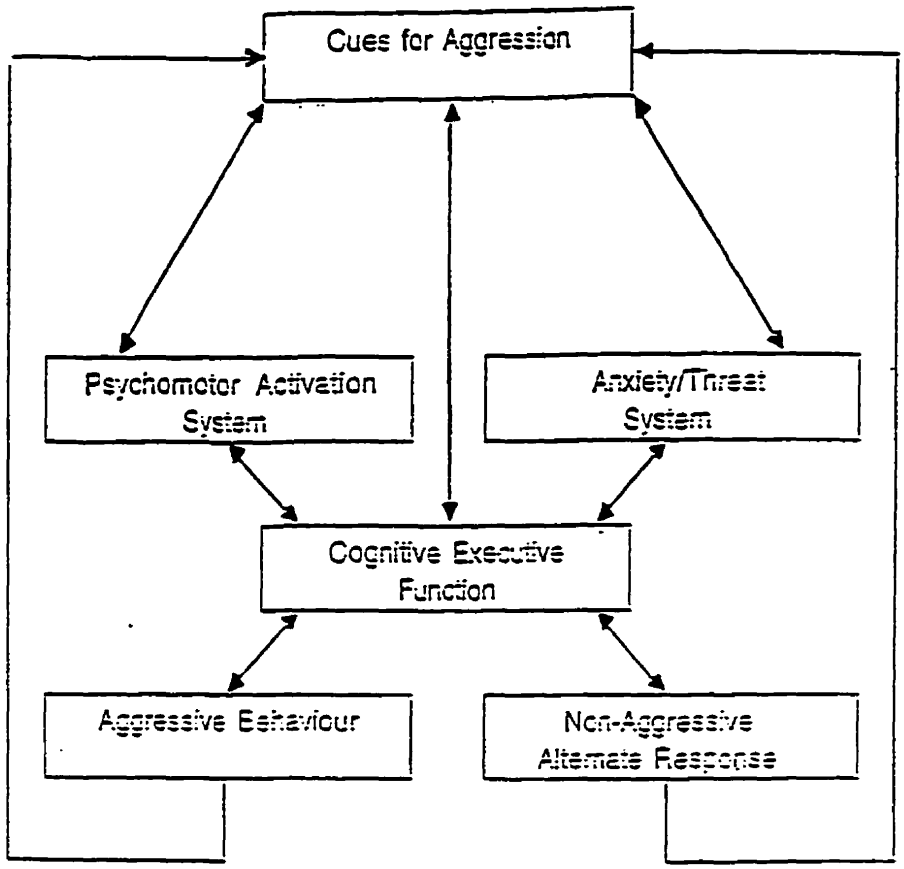

Figure 4. Schematic of the interrelationship between two systems and centrality of the executive function in the determination of aggressive and nonaggressive responding.

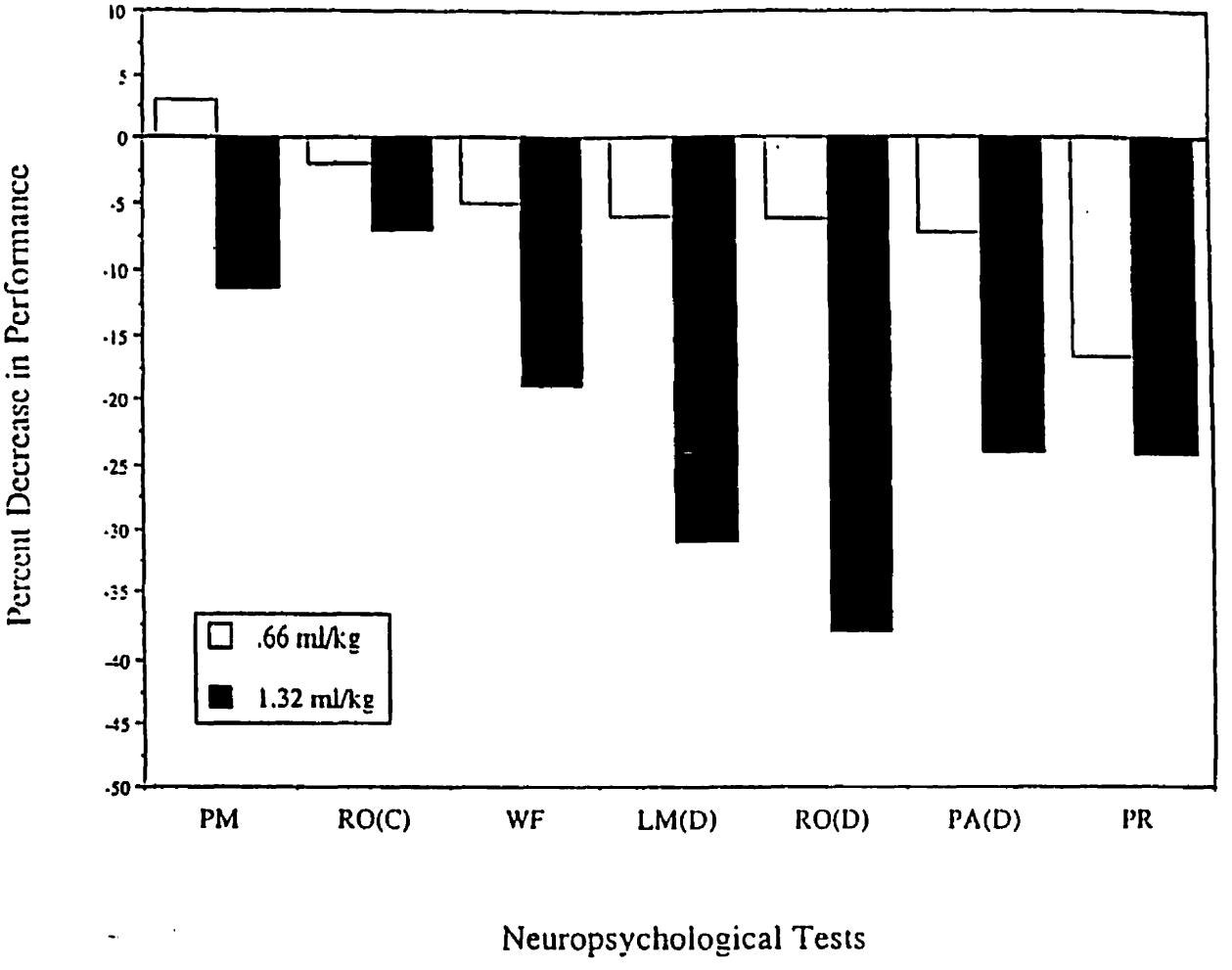

FIGURE 5. Dose-related effects of alcohol intoxication on cognitive performance: Tests associated with frontal function: PM (Porteus Maze), RO(C) (Rey-Osterreith, copy), WF (Thurstone Word Fluency); tests associated with hippocampal function: LM(D) (Logical Memory, Delayed), RO(D) (Rey-Osterreith, delay), PA(D) (Paired Associated, Delay); tests associated with motor function: PR (Pursuit Rotor) (Peterson et al., 1990)

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APPENDIX E

Pihl, R.O., & Hoaken, P.N.S. (1997). Clinical correlates and predictors of violence in patients with substance use disorders. Psychiatric Annals, 27(11), 735-740.

CLINICAL CORRELATES AND PREDICTORS OF VIOLENCE IN PATIENTS WITH SUBSTANCE USE DISORDERS

R.O. PIHL AND PETER N.S. HOAKEN

DEPARTMENT OF PSYCHOLOGY, MCGILL UNIVERSITY

Substance use/abuse and violence correlate and there are many aspects to this relationship. In general, at least four very differential but not necessarily mutually exclusive reasons for this relationship exist. These are: 1) Violent crimes can be committed to gain access to drugs or resources to purchase drugs, 2) violence appears to be a necessary means to resolve disputes among those involved in an illegal, unregulated, and ruleless business, 3) drug use and violent behavior can be the result of the same factors and exist coincidentally, and 4) certain drugs can increase the likelihood of violence because of their effect on the individual. Patients can exhibit substance abuse related violence for any one or combination of the above reasons. Thus, when confronted with violent behaviour, the first task of the astute clinician is to determine if substance abuse is involved, and to differentially diagnose which motivational condition or conditions is operative. This problem is not trivial as substance abuse disorders are common both alone and as a co-morbid disorder.

When ascertained by broad general surveys, substance abuse disorders are among the most prevalent psychiatric disorders, for one month, yearly, or lifetime diagnoses[1]. Although in recent years increasingly these prospective patients are being seen psychiatrically, for the most part they remain never treated, fall under the aegis of the legal system, or become involved in alternative approaches. However, given the importance of violent behavior as arguably the most important reason for psychiatric hospitalization[2], substance abuse might well underlie

behaviors frequently attributed to other disorders and explanations. Figure 1 illustrates the high co-morbidity that exists between substance abuse and other psychiatric disorders. These high levels of co-morbidity are not insignificant, especially when considered in collaboration with Figure 2, which represents the probability of violent behaviour by psychiatric diagnosis, and by gender. As can be seen, psychiatric patients with co-morbid substance abuse disorders constitute the greatest risk for violence.

We have primarily conceptualized four, of perhaps many, physiological effects of drugs on an individual that should increase the likelihood of violence[3]. The first of these is an alteration of the anxiety/threat system. In most cultures, individuals learn that heightened aggression is linked with increased likelihood of punishment. As such, cues which suggest the need to become aggressive elicit anxiety. When intoxicated by some drugs, however, such normal inhibiting mechanisms are themselves inhibited, leading to increased probability of the aggressive response. The second mechanism is an alteration of the psychomotor system, such that general excitation and reward are enhanced. Psychomotor stimulants potentiate motor behavior, including approach and attack behaviours, that would not ordinarily be manifested. Psychomotor stimulant properties, therefore, seem to potentiate aggression in humans through increased novelty- or sensation-seeking behavior. The third mechanism, which amounts to an alteration of the pain system, seems to operate in such a way that saliency of provocative stimuli are

increased. Defensive aggression is an unconditioned response; drugs which heighten pain sensitivity, therefore, increase likelihood of defensive aggression. The last mechanism is an alteration of the cognitive control system, which normally modulates the general motivational state. Certain drugs may interfere with those cognitive functions which underlie planning and the formulation of behavioral strategies, specifically through the initiation and maintenance of goal directed action in the face of external and internal regulatory cues.

These four systems, illustrated in Figure 3, apply differentially to the specific pharmacology of various drugs, and further encompass psychological effects, such as the expectation that a specific drug will lead to aggression, or that drug intoxication condones or excuses violence. It should also be noted that the four systems mentioned are in no way considered mutually exclusive, nor collectively exhaustive. Any drug can affect the functioning of one or more of these hypothesized systems, and what follows is a discussion of the prominent drugs involved in violence, and how each might affect these hypothesized systems.

ALCOHOL

Crime studies repeatedly demonstrate the high and significant involvement of alcohol in general violent behavior. In a review of 26 studies, involving 11 countries, it was determined that 62% of violent offenders had been drinking shortly before perpetrating a crime[4]. The rate

of intoxication for violent crimes was roughly double than that for non-violent crimes, and it was evident that in those studies where blood alcohol concentrations were measured, heavy drinking had been involved. Further, a chronic problem with alcohol increases risk. A recent study of homicidal offenders in Finland found that 39.2 % of male offenders and 32.1 % of female offenders met DSM-III-R criterion for alcoholism; the next highest rate for an Axis I disorder was approximately 6%, for schizophrenia[5]. Another recent study of a Danish birth cohort concluded that alcohol and drug use increased risk for criminal and violent behavior, among both mentally disordered and non-disordered subjects[6]. While crime study data are purely correlational, and thus open to many interpretations, manipulative controlled laboratory studies have confirmed alcohol's role in aggressive behavior. In the most recent meta-analysis[7], an effect size of .43 was calculated for intoxicated over non-intoxicated aggressive responding. The conclusion was drawn that alcohol does affect aggression, particularly in males, albeit through indirect means, likely the aforementioned mechanisms. Further, dose is important, as at high doses alcohol is both an analgesic and a psychomotor depressant, whereas at lower doses the opposite effects can occur.

There is no reason to believe that psychiatric patients would be any less susceptible than non-patients to the aggression-eliciting effects of moderate to heavy consumption of alcohol. On the contrary, two important factors may suggest a greater manifestation of aggression in this

population: First, certain groups of psychiatric patients are particularly vulnerable to alcohol abuse problems, and second, their condition may well interact with, and make them more susceptible to, the violence producing effects of alcohol. Thus, psychiatric patients must be considered a high risk for aggression if a co-morbid alcohol dependence exists.

BENZODIAZEPINES

Since their introduction in the early 1960's, there have been many case reports linking benzodiazepines with increased anger and aggression[8]. Given the designed effects of these drugs, specifically on the anxiety system, consistent with the theorizing represented in the model in Figure 3, this response should be expected. However, with the exception of case studies, experimental human and animal laboratory studies suggest the amount of violence produced by benzodiazepines pales when compared with that of alcohol. In fact, it is interesting to note that some clinicians have dismissed the clinical evidence[9], and in fact consider benzodiazepines as anti-aggression drugs[10]. The issue is extremely important, as benzodiazepines represent the most used psychotropic medication[11]. To properly answer the issue, two factors deserve further examination: Dose-related effects, and individual factors.

Dose appears to be an extremely important variable. The aforementioned discrepancy of clinician beliefs vis a vis the aggression-

eliciting effects of benzodiazepines may be related to a poor understanding of dose-related effects. Most case studies which report disinhibition of aggressive responses, and most manipulative controlled laboratory experiments, which do indeed seem to show benzodiazepine-related increases in aggression, involve relatively low doses[12,13,14]. On the other hand, discussion of benzodiazepines as anti-aggressive agents usually involve somewhat higher doses[15]. This is an important consideration, and is consistent with drugs such as alcohol which also effect the anxiety system producing reduced general functioning per ce at higher, approaching sedative, doses.

Differential susceptibility in certain individuals may also be predictive. Benzodiazepines have a heightening effect on aggression, mediated through the anxiety system, an effect that has been demonstrated repeatedly in both animal and human studies[13,16,17]. However, this effect, shown in research to be statistically significant, may not be clinically significant in most individuals. It is specific individual differences which can most likely explain who does and who does not become meaningfully more aggressive when under the influence of benzodiazepines. Specifically, reviews of the literature have suggested that increased aggression in response to typical doses of benzodiazepines is usually related to pre-existing brain damage[18], patient expectation of increased aggressivity[19], or pre-treatment level of hostility[19]. Furthermore,

benzodiazepine/alcohol combinations have been shown to produce heightened aggression greater than the expected sum of the two drugs[20].

Therefore, the perspicacious clinician needs to be certain that benzodiazepines are administered to an appropriate population, and explained sufficiently, in order to reduce possibility of disinhibited aggressive reactions. Patients with personality disorders or co-morbid alcohol-abuse disorders must be considered to be highly susceptible to the violence-inducing properties of the benzodiazepines.

PSYCHOSTIMULANTS

Although there are many case studies in the literature associating the various forms of amphetamine and cocaine use with heightened violence[21,22], the exact nature of the relationship remains in some question. There is an extensive animal literature suggesting behavioral effects of these drugs, including what appears to be defensive aggression. Attack responses, however, do not appear to be elicited[23,24].

In controlled laboratory studies with humans, amphetamine administration appears to produce increased competitiveness, excitability and volubility[24], but the literature is somewhat equivocal as to whether there is a significant increase in aggression[25,26,27]. Correspondingly, the limited literature has yet to unequivocally demonstrate a direct link between cocaine administration and increased aggression; only one

controlled study has thus far been conducted, and in this study only high dosages of cocaine were associated with increased aggression[28].

The issue, then, is how to account for the great number of case studies detailing violence associated with these drugs, and the general perception that this relationship exists. The explanations are likely indirect and multifactorial: First, individuals likely to abuse psychostimulants are, for the same reasons, more likely to manifest uninhibited acts of aggression. Antisocial personality and psychopathy have been associated with sensation-seeking, which, in turn, has been associated with a greater likelihood of initiation of drug use, particularly use of psychomotor stimulants[29]. It has been proposed that sensation seekers are chronically underaroused, and as such find the stimulating properties of these drugs preferentially reinforcing, and furthermore act out aggressively in order to attain alternative stimulation. A second possibility is that the intense psychological dependence associated with these drugs may make users in mild or severe withdrawal more aggressive. In one laboratory study[30], cocaine dependent subjects were significantly more aggressive than matched controls. A third and related possibility is that aggression of an instrumental nature may be used in order to attain more of the high cost desired drug. Furthermore, psychostimulants can increase delusions and hallucinations in patients with psychotic disorders. Because psychosis itself is associated with violence (most commonly mediated through paranoid ideation), patients with histories of both

paranoid delusions and psychostimulant use/abuse are potential violence risks.

PHENCYCLIDINE

Case reports suggest that violent behavior seems to at least occasionally follow self-administration of phencyclidine[24]. The animal literature also seems to suggest a relationship[31,32,33], although findings are somewhat ambiguous. In humans, the frequency and intensity of such a pharmacological effect, and in fact, whether such a relationship truly exists, is at this time in question, primarily because of a lack of controlled laboratory studies of phencyclidine and human aggression. Classification of Phencyclidine into a drug class is difficult, because it is not truly a hallucinogen, nor is it truly a psychomotor stimulant. Because its effects are so poorly understood, it is difficult to assess based on the model in Figure 3. It may be that phencyclidine-related aggression is manifested only in interaction with other variables. One study[34] found that personality characteristics and usage history (e.g. age of first administration) were predictive of aggressivity when phencyclidine-intoxicated. Importantly, it was also found that subjects with a past history of psychiatric hospitalization reported greater levels of assault when using phencyclidine than did subjects without psychiatric histories. Therefore, taking a careful history may facilitate prediction of aggression.

ANABOLIC STEROIDS

Numerous retrospective reports propose a link between violence and the use of these drugs[35,36,37]. This retrospective nature of the data is problematic as is the fact that the typical user represents a highly specific population possibly prone to violence in the first place. Athletes, male adolescents and young men are most frequently studied. Further, in these populations extensive use of alcohol in association with anabolic steroids has been reported[38,39]. A review of 13 studies of athletes found that 34% of the individuals reported behavioral side effects[40]. Heavy use seems particularly related to increased violence[41,42], yet negative findings have also been reported[43,44]. Clearly controlled investigations are needed, as well as increased understanding of the role of hormone levels in aggression.

CANNABIS

There is, and has been for a considerable period of time a debate over whether cannabis produces violence. This debate dates back at least to 1926, when a New Orleans newspaper exposed the "Menace of Marijuana", claiming that an association between the drug and crime existed, especially violent crime[45]. This was concluded despite the fact that at that time no studies on the effects of cannabis on aggression had ever been conducted. By the 1930's the Bureau of Narcotics had established the Marijuana Tax Act, which essentially banned the drug.

Currently available research, for the most part, demonstrates strongly that the relationship proposed several decades ago does not exist. Several studies have shown that human violent behavior is either decreased or unchanged with cannabis administration[24, 46,47,48]. The animal literature suggests the same relationship; cannabis tends to foster submissive behaviors, and suppress attack and threat behaviors[49,50]. Some animal studies, particularly with Wistar rats, have noted heightened aggression with cannabis administration. However, in these studies there is usually a third variable which might account for this result, for example REM sleep deprivation[51], social seclusion[52], or pretreatment with another drug[53]. It is possible that attention to these studies, or to case reports of violence in cannabis intoxicated individuals, might perpetuate prevalent misconceptions. It would seem that where violence and cannabis are linked one would be wise to investigate non-pharmacological factors.

OPIATES

According to a large body of animal literature, morphine and other opium derivatives temporarily reduce aggressive behavior[24,54,55]. The literature on humans seems to suggest the opposite relationship[56,57,58], but care must be taken in drawing conclusions from the few existent studies. What seems more certain is that while intermittent use of opiates seems to produce euphoria and feelings of well-being, chronic administration produces more complex changes in mood and behavior. Opiate

withdrawal is associated with feelings of confusion, hostility and suspicion in humans, and the animal research has demonstrated heightened aggression in animals during withdrawal, perhaps due to heightened pain sensitivity[24]. Drugs that block heroin withdrawal, then, may be helpful in management of aggressive impulses. Another consideration is the population who abuse opiates. There is some evidence that opiate abuses may have more pre-morbid feelings of rage[24]; it may also be that individuals who are abusers of opiates are more likely to be aggressive because of the reasons they abuse the drug, not because of the drug itself.

CONCLUSIONS

Is there a relationship between drugs and aggression? Clearly, the answer is a resounding yes. However, the nature of the relationship is interactional and multi-factorial, and, moreover, different for different classes of drug. Having said that, our recommendations for the clinical practice of violence prediction in patients with substance use disorders are as follows: 1) Differentiate the form of the drug-violence relationship. 2) Specific to the drug, determine whether direct pharmacological action of the drug, withdrawal from the drug, or craving for the drug is the likeliest producer of violence. 3) Determine how and if the drug or drugs of abuse interact with the basis of the clinical pathology. 4) Deal with the problem, seek or provide treatment for the substance use disorder, co-

morbid disorder, and lifestyle difficulties which greatly increase the risk for the individual.

Figures Headings

Figure 1: Co-Morbidity of drug and Alcohol Diagnoses with Prominent Other Diagnostic Categories.

Constructed from data presented in: Regier, D.A., Farmer, M.E., Rae, D.S., Locke, B.Z., Keith, S.J., Judd, L.L., & Goodwin, F.K. (1990) Comorbidity of mental disorders with alcohol and other drug abuse: results from the epidemiologic catchment area (ECA) study. Journal of the American Medical Association, 264(19), 2511-2518.

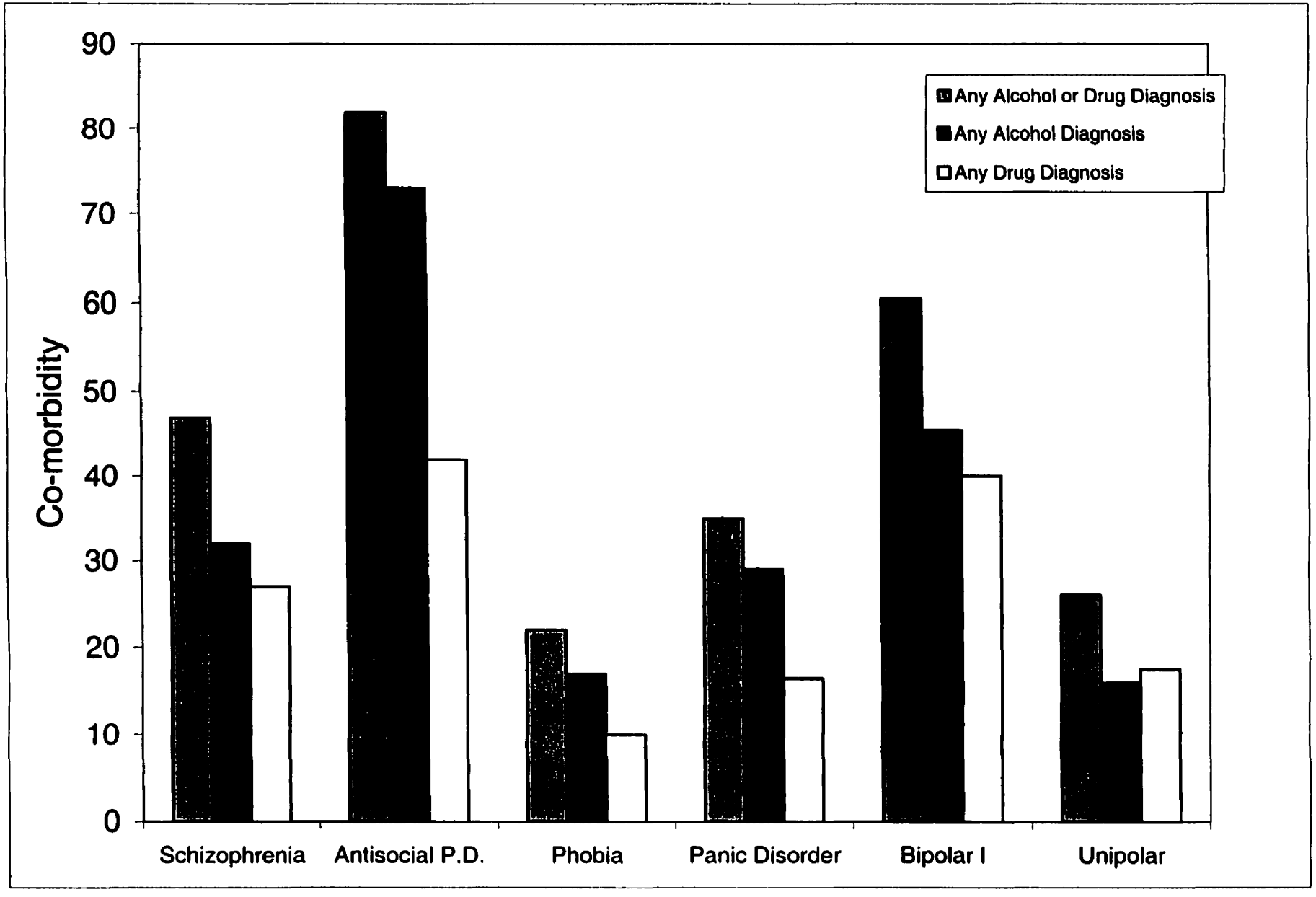

Co-morbidity
90
80
70
60
50
40
30
20
10
0
Any Alcohol or Drug Diagnosis
Any Alcohol Diagnosis
Any Drug Diagnosis
Schizophrenia
Antisocial P.D.
Phobia
Panic Disorder
Bipolar I
Unipolar

Figure 2: Probability of Violent Behavior in a One-Year Period by Diagnostic Class

From: Swanson, J.W. (1994). Mental disorder, substance abuse, and community violence: An epidemiological approach. In: Monahan, J., & Steadman, H.J. (Eds.) Violence and Mental Disorder: Developments in Risk Assessment. University of Chicago Press: Chicago. Used by permission.

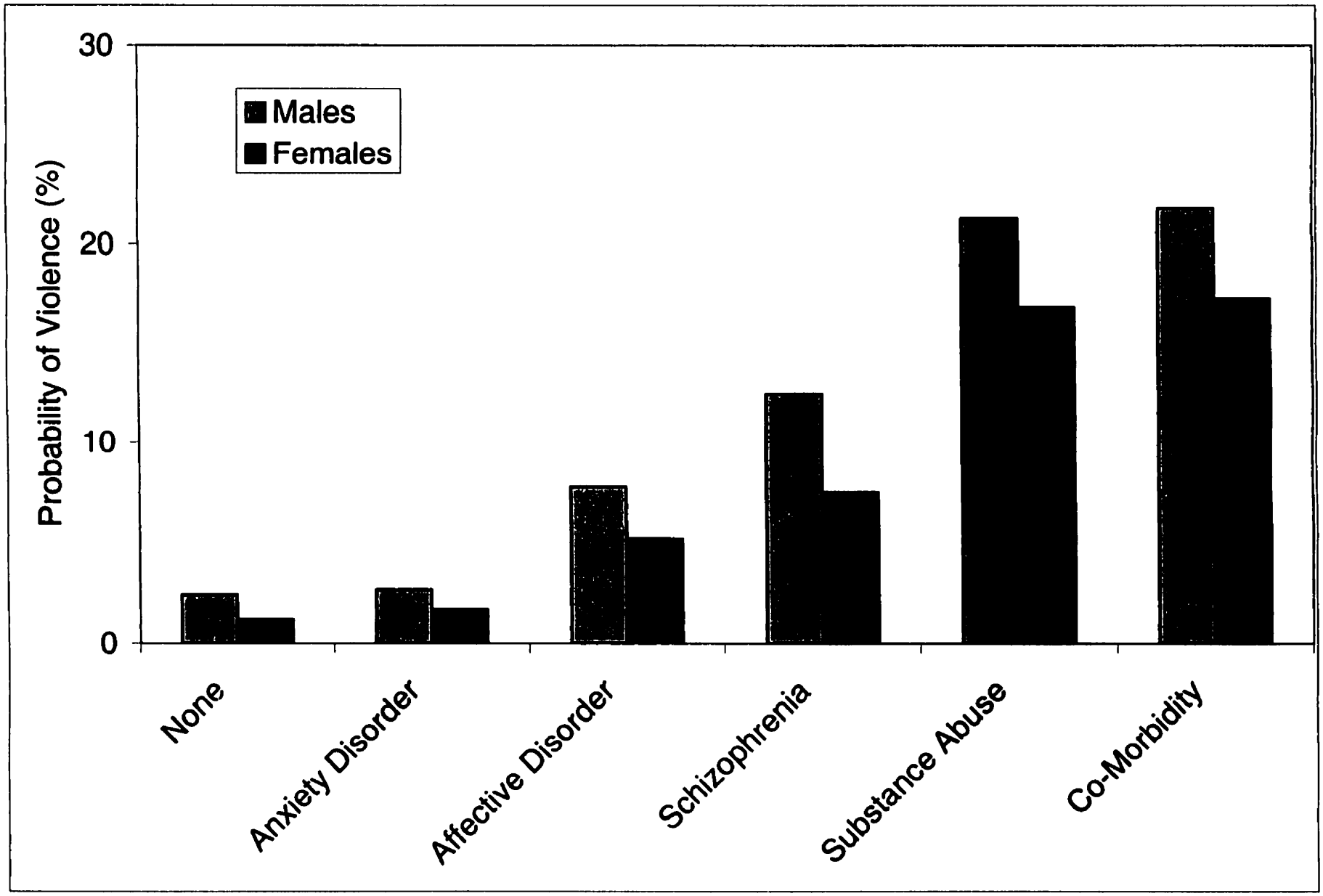
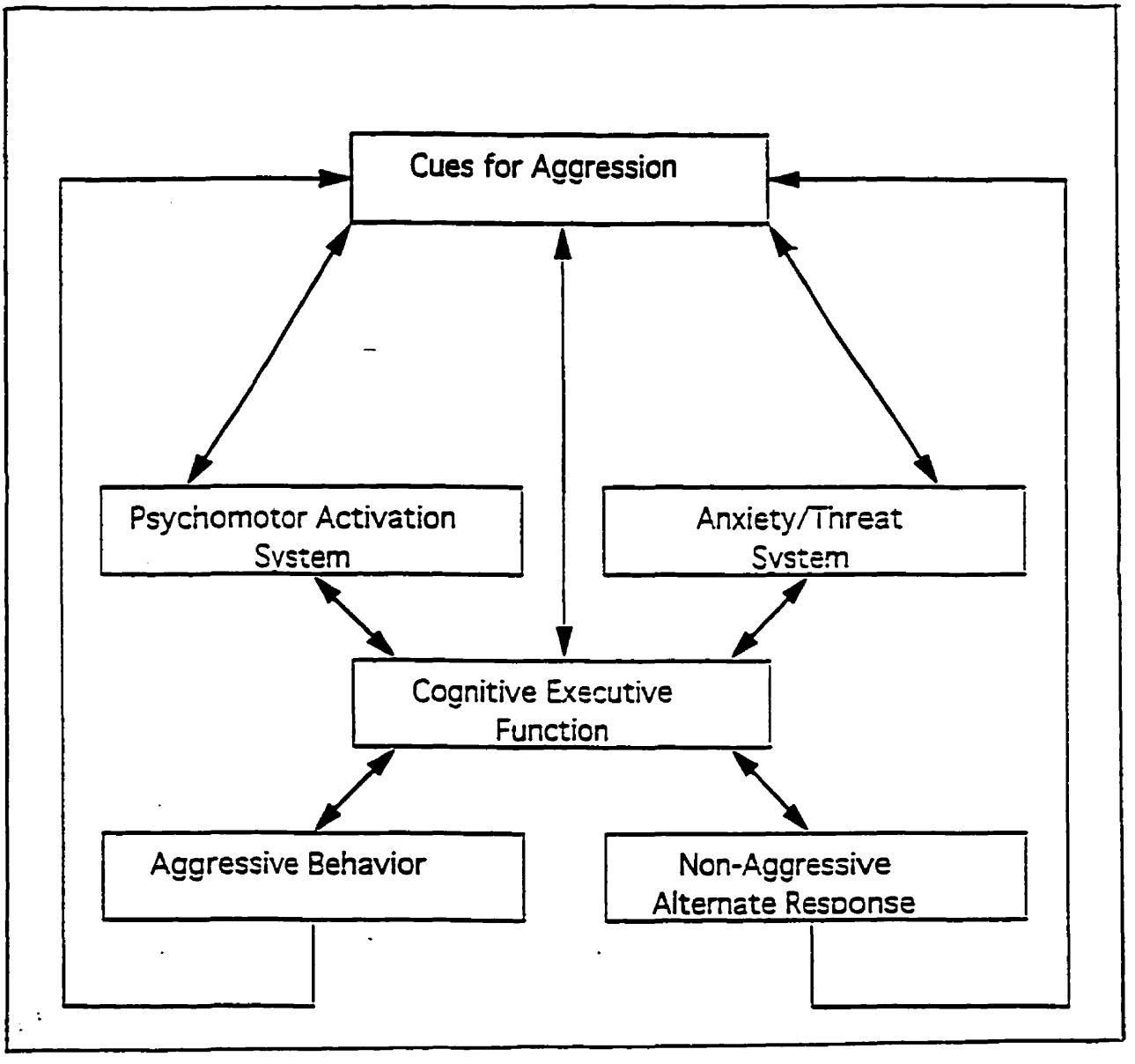

30
20
10
0
Probability of Violence (%)
Males
Females
None
Anxiety Disorder
Affective Disorder
Schizophrenia
Substance Abuse
Co-Morbidity

Figure 3: Model of Systems Which Mediate Drug-Violence Relationships


Cues for Aggression
Psychomotor Activation System
Anxiety/Threat System
Cognitive Executive Function
Aggressive Behavior
Non-Aggressive Alternate Response

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