ALCOHOL AND AGGRESSION: THE ROLE OF BEHAVIOR CONTINGENCIES AND INSTIGATOR INTENT

by

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ABSTRACT

The purpose of the present investigation was to determine the mediating effect of alcohol, behavior contingencies and instigator-intent on aggression in male social drinkers. Aggression was assessed by the intensity and duration of shocks administered to a bogus partner in a modified Buss aggression procedure. The investigation was carried out in two experiments employing randomized 3 x 2 factorial designs. In the first experiment, groups of inebriated, placebo and sober subjects were exposed to aversive contingencies which were either correlated or noncorrelated with their aggressive responses. In the second experiment, similar groups were exposed to aversive stimulation given with either neutral or malicious instigator-intent. In both experiments, the inebriated subjects were significantly more aggressive than either the placebo or sober subjects. Moreover, the intoxicated subjects displayed equally aggressive response patterns under both contingency conditions as well as under the two instigator-intent types. The nonintoxicated subjects displayed differential response patterns affected by the contingency and intent manipulations. The unique response pattern of the intoxicated subjects is attributed to the disrupting effect of alcohol on information processing.

SOMMAIRE

Cette recherche a pour but de déterminer les effets de l'alcool, des contingences du comportement, et des intentions d'un provocateur, sur les manifestations agressives de buveurs d'occasion. Les manifestations agressives sont évaluées à l'aide de l'intensité et de la durée de chocs électriques administrés à un sujet fictif selon une modification de l'épreuve d'agression de Buss. Cette recherche est constituée de deux expériences utilisant des modèles factoriels aléatoires 3 x 2. Dans la première expérience, trois groupes de sujets (sobres, ayant bu une boisson alcoolisée fictive ou une boisson alcoolisée réelle) fusent soummis de façon directe à des situations désagréables qui étaient soient corrélées soit non-corrélées avec les manifestation agressives de leurs réponses. Dans la seconde expérience, les mêmes trois types de groupes furent exposés à des stimulations désagréables présentées de façon soit malveillante soit neutre. Dans les deux expériences, les buveurs d'occasion se révélèrent significativement plus agressifs que les sujets sobres et ceux qui burent de l'alcool fictif. De plus, les sujets intoxiqués se montrèrent aussi agressifs dans leurs réponses lorsqu'ils furent soummis aux conditions de contingence du comportement que lorsque les deux types de stimulation leur furent adressées. Les réponses des sujets nonintoxiqués varièrent suivant les conditions de contingence

et le type de stimulation. Le mode particulier de réponse des sujets intoxiqués est expliqué par l'effet dévastateur de l'alcool sur le traitement de l'information.

PREFACE

The conception of the present investigation was influenced by the changing trends of drug abuse in North America. As increasing numbers of youths turn toward alcohol in their search for euphoria, and with the climbing crime rate in the cities and rural areas, the notorious relationship between alcohol and aggression becomes of particular concern.

The contribution of the present investigation to original knowledge is threefold. First, the effect of alcohol on two implicated aggression-controlling factors has been examined for the first time. The intoxicated subject's ability to process information concerning antecedents and consequences of his aggressive behavior was assessed. Second, new modifications of the laboratory-aggression paradigm were instituted. A pure tone was used as an instigating stimulus, and in one study as an aversive behavior contingency. Both auditory and visual contingency-information were given to the subjects and a video-tape deception was employed. Third, the use of subjects from a diverse background, instead of the commonly used student population, is unique in this area of research. It is hoped that the above contributions to knowledge will aid in the understanding of the alcohol-aggression relationship and in furthering more effective measures of aggression-control.

Due to the manuscript format of the thesis, and due to the fact that similar procedures and instruments were used in the two experiments, textual repetitions were necessary.

Finally, in compliance with the requirements of the Faculty of Graduate Studies and Research of McGill University, the full text of regulation (7) is given below.

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TABLE OF CONTENTS

LIST OF TABLES

Page

EFFECTS OF ALCOHOL AND BEHAVIOR CONTINGENCIES ON HUMAN AGGRESSION:

EFFECTS OF ALCOHOL AND INSTIGATOR-INTENT ON HUMAN AGGRESSION:

GENERAL INTRODUCTION

The Problem

The legality and availability of alcohol to the human consumer has caused the substance to become the most used and abused drug in western society. Canadian surveys of the extent and patterns of alcohol use report an increase in the incidence of alcohol consumption by high school students (Annis, Klug, & Blackwell, 1971; Fejen & Smart, 1972; Halpern & Mori, 1970; Hayashi, 1968; Narcotic Addiction Foundation of British Columbia, 1971; Rootman, Clark, & Oakley, 1972; Smart, Fejen & Alexander, 1972; Whitehead, 1970), as well as among college and university students (Bilodeau, 1971; Ledain, Campbell, Lehman, Stein, & Bertrand, 1973; Lubin, Blumberger, Diez d'Aux, Garfinkle, Goldhamer, Groulx, Kahn, & Weiner, 1971). The high frequency of alcohol consumption in the adult population has also been widely reported (Addiction Research Foundation, 1971; deLint, Schmidt, & Pernanen, 1970).

Apart from being the most popular drug, alcohol has also been found to have a consistent relationship to crime and other aggressive behaviors. Ingested alcohol has been identified in individuals perpetrating acts of violent assault (Mayfield, 1976) and violence in response to stress (Nicol, Gunn, Gristwood, Foggitt, & Watson, 1973). Virkkunen (1974), in a survey of homicides committed in Sweden, reported that alcohol was often present in the blood of both

the aggressor and the victim. A high frequency of violent crimes has been linked to alcoholics (Haberman & Baden, 1974; Lindelius & Salum, 1975) as well as to individuals who were under the acute influence of alcohol (Fitzpatrick, 1974). Alcohol intoxication has also been linked with aggression directed toward the self. Suicidal behavior has been observed in intoxicated alcoholics (Hagnell, Nyman, & Tunvig, 1973) as well as in nonalcoholic inebriated individuals (Buglass & McCulloch, 1970). Finally, some reports have associated alcohol intoxication with increases in risk-taking while driving (Cohen, Dearnalay, & Hansel, 1958; Teger, Katkin, & Pruitt, 1969) and with aggression, paranoid ideation and chronic anger leading to automobile accidents (Selzier, Payne, Westervelt, & Quinn, 1967).

In the attempt to understand the alcohol-aggression relationship traditional concepts seem to be of little promise. Proponents of the personality-trait approach have attempted to identify the excessive drinker and the aggressor in terms of their personality characteristics. A thorough review of the alcoholism assessment literature by Miller (1976), cites numerous studies reporting elevated Minnesota Multiphasic Personality Inventory (MMPI) scales and unique scores on other personality tests (e.g. TAT, Jung Scale, Rorschach) associated with alcoholism. These findings indicate that a wide range of traits, but no single profile, has

been implicated in the search for the alcoholic personality. Thus, Miller stresses the importance of using a multimodal assessment of alcoholics.

Whereas some studies have not been able to differentiate between groups of alcoholics and nonalcoholics by means of personality measures (Donovan & O'Leary, 1975), other studies were more successful in that endeavor (Loper, Kammeier, & Hoffman, 1973; Orford, 1976; Skinner, Jackson, & Hoffman, 1974). However, Rosen (1960) suggested that personality differences found between alcoholics and nonalcoholics are most likely due to sampling biases.

Nevertheless, in a recent and comprehensive work, Pihl & Spiers (1978) have critically examined the construct of the elusive "addictive personality". After reviewing 650 pertinent studies these authors conclude that though persistent, the addictive personality has not, to date, been unequivocally defined.

Studies attempting to identify the aggressive personality present similarly inconclusive findings. Cochrane (1975a, 1975b) proposes to define the aggressive personality based on nonsystematic psychodiagnostics and psychiatric ratings. The commonly accepted description of aggressors as overcontrolled individuals (Megargee, 1966) has been disputed by a study of imprisoned murderers who were not found to demonstrate this trait (Lester, Perdue, & Brookhart, 1974). In reviewing the descriptors of aggressive children,

Feshbach (1970) concludes that there seems to be no single cluster of traits comprising their personalities.

The commonly held hypothesis that men are almost always more aggressive than women (Buss, 1966b, 1971) has recently been challenged. In a review of the experimental literature, Frodi, Macaulay & Thome (1977) found no support for such a claim. It was stressed that when aggression is perceived as justified, women may act as aggressively as men. These authors suggested however, that men and women may react differently to external cues and provocation. It has been suggested, in this respect, that the interaction between constitutional and situational variables may prove as an important determinant of aggressive behavior (Wilkins, Scharff, & Schlottmann, 1974). Finally, the attempt to formally link aggressive characteristics and excessive drinkers has so far been unsuccessful (Kristianson, 1974; Ruff, Ayers, & Templer, 1975).

The plethora of studies on the behavior of excessive drinkers and perpetrators of aggressive acts to date forward little support for the existence of specific personality profiles. There is increasing evidence, however, that both phenomena are functions of situational determinants. The present investigation focuses on the extent to which alcohol mediates the effect of situational factors in the control of aggressive responsiveness. The existence of such a mediating

influence becomes evident through the study of aggression and both the pharmacological and extrapharmacological effects of alcohol.

The research into the alcohol-aggression relationship is comprised of studies proposing numerous formulations of aggression. These studies employ a variety of experimental paradigms in which diverse applications of alcohol are made. To facilitate the understanding of the effect of alcohol on aggression it is first necessary to review theoretical positions and empirical studies of aggression as well as data on some non-aggressive effects of alcohol.

Theories of Aggression

Over the years, several theories have been forwarded in an attempted to explain aggressive behavior. The instinct theory, popular during the earlier phases of this field's development, proposed that organisms are innately endowed with aggressive drives that must be discharged periodically. Freud and Lorenz were this theory's main proponents. In his psychoanalytic approach to the study of aggression, Freud (1920) used the term thanatos, or death force, for what he believed to be man's second major instinct aimed at the destruction of life. Freud argued that through the mechanism of displacement man can direct this destructive force toward others. Hence, the origin of aggression lays in the re-direction of the self-destruction instinct. Freud stressed

the inevitability of aggression in humans and suggested that the performance of some aggressive acts may reduce the accumulated instinctual energy thus preventing subsequent more dangerous acts. This concept of catharsis has since been criticized by some of Freud's followers (Hartmann, Kris, & Loewenstein, 1949) and disputed by others (Doob & Wood, 1972; Geen, Stonner, & Shope, 1975).

The ethological approach of Lorenz (1966) suggests the existence of a fighting instinct in lower animals and man. This product of evolution is presumed to generate aggressive energy. This energy is believed by ethologists to be released as a function of environmental stimuli and the amount of accumulated aggressive energy. Passage of time since the performance of the last aggressive act is thought to play an important role in lowering the aggression-release threshold. Lorenz's claim that all organisms have innate inhibitors of intra-species aggression is weakened in view of the historical and contemporary perpetration of aggressive acts by humans. Lorenz claimed, therefore, that man's cognitive processing of intent as well as his possession of weapons obviate many controlling mechanisms of aggression.

A more contemporary theory of aggression is drive theory. Forwarding the frustration-aggression model, Dollard, Doob, Miller, Mowrer, & Sears (1939) suggested that frustration gives rise to an aggressive drive which unleashes aggressive behavior. Frustration in this theory replaced

the instinct as activating source. However, as Bandura (1977a) suggests, the concept of frustration lost its specific meaning by subsuming a diverse set of conditions such as physical assault, deprivation, insult, thwarting, harassment and defeat. Furthermore, the instigation of aggression does not always require frustration (Berkowitz, 1969, 1973).

Another source of confusion regarding drive theory is its use of the concept of arousal. Frustration-induced arousal is assumed to be labeled anger by the frustrated individual who subsequently attacks his tormentor (Berkowitz, Lipinski, & Angulo, 1969). However, numerous studies have reported that similar physiological arousal can lead to labels of a variety of emotions (Mandler, 1975; Schachter & Singer, 1962). In addition, individuals who are prone to aggress may do so under emotional arousal from different sources (Rule & Neasdale, 1976a). Finally, general arousal has been found to serve as an aggression-eliciting cue. As a result, one reformulation of drive theory (Zillmann, 1978) conceptualized frustration or anger arousal as a facilitating rather than a necessary condition for aggression.

The analysis of aggression within a social learning model is currently gaining wide support (Buss, 1971; Zillmann, 1978). The theory's major proponent (Bandura, 1973, 1977a, 1977b) identifies three classes of components which are crucial for the understanding of the phenomenon: (a) The

origins of aggression, (b) the instigators of aggression, and (c) the regulators of aggression. Bandura sees aggressive behavior as an acquired skill. The skill's acquisition can take place either through observational learning, for example, by observing aggressive models (Bandura, Ross, & Ross, 1963a), or through direct experience such as aggressive shaping (Patterson, Littman, & Bricker, 1967). Structural factors such as physical build, canine teeth, jaws and muscle are aggression determining variables primarily in animals. Humans' dependence on biological structure for aggressive behavior is believed reduced due to man's ability to develop destructive weapons, to organize socially for collective action and to process symbolic language.

Among the instigators of aggressive responses, Bandura (1977b) mentions biologically-based motivators (i.e., internal and external aversive stimulation) and cognitively-based motivators (i.e., the anticipation of consequences through cognitive representation). Whereas these instigators are aversive motivators of aggression, the social learning approach also accommodates motivators such as anticipated positive consequences and modeling that justify aggressive behavior. Finally, the process of socialization is conceptualized as an instigator in as much as it teaches the individual to obey orders to aggress (Milgram, 1963, 1974) or to perceive aggression as having an altruistic value (Buss, 1961).

The regulators of aggressive behavior seem to be the most encompassing component of the theory. These modifiers are external reinforcers such as tangible rewards (Buss, 1971), social rewards (Geen & Stonner, 1971), expression of harm (Buss, 1966a), reduction of aversive treatment (Patterson et al., 1967), punishment (Bandura, 1969), vicarious reinforcement (Bandura, Ross, & Ross, 1963b) and self-reinforcement such as self-reward and self-punishment for aggression (Bandura & Walters, 1959).

The last model of aggression to be reviewed in this section stems from attribution theory. Forwarded by Heider (1958) and further developed by others (Jones & Davis, 1965; Kelley, 1967), this theory deals with the ways individuals ascribe causality of events and dispositional characteristics to other persons' behavior. Some aggression-related issues are examined within an attributional framework. These issues are the labeling of emotional states, the labeling of behavior as aggressive and the perception of intentionality. Perhaps the most famous study related to attributional processes is the one by Schachter and Singer (1962) in which drug-induced arousal states were interpreted by subjects as emotional states resulting from situational variables. The importance of the attributions made by subjects concerning the source of their arousal has been demonstrated by Berkowitz et al. (1969) as well as in the studies of Baron and Bell (1975, 1976). The first study reported a reduction

of aggression in subjects who perceived their arousal to be inappropriately high. The other two studies indicated that the attribution of extremely aversive environmental conditions to the victim seemed inappropriate and resulted in a reduction of aggression toward the victim. Moderate discomfort, however, was attributed to the victim and the arousal, relabeled anger. Previously established emotional states have been found to intensify due to misattributions made concerning different sources of arousal (Zillmann, 1971; Zillmann & Bryant, 1974). Tedeschi, Smith & Brown (1974) suggested that the label aggression may be applied by an observer perceiving intentional coercion used by an instigator. Aggression was reported to increase toward individuals seen as aggressive instigators (Borden, Bowen, & Taylor, 1971). When the aggressive intentions of the instigator were communicated to the responder, the latter's aggression was found to be strongly affected by that information (Taylor & Pisano, 1971). In summary, according to the attributional model, when arousal labeled anger is attributed to an instigator who is perceived as having aggressive intentions, the necessary conditions for an aggressive response are met (Greenwell & Dengerink, 1973).

Systematic Studies of Human Aggression

The bulk of the studies on human aggression examine this phenomenon in a laboratory-controlled situation. The

experimental paradigms most widely used are modifications of those forwarded by Buss (1961) and Berkowitz (1965). Physical aggression in these paradigms is operationally defined as the subject's preparedness to administer aversive electrical shocks to a confederate in a bogus learning experiment. The shocks are presented as facilitating the learning process. In actuality, no electric shock is delivered or received. The index of aggression is the shock intensity, duration or latency of shock the subject believes he administers.

Several criticisms have been leveled at this paradigm. Schuck and Pisor (1974) criticized the Buss paradigm in terms of its artificiality, its being unethical, and, as others (Baron & Eggleston, 1972) have shown, the aggressive behavior under study could be construed by the subject as acts of altruism. The latter problem was effectively solved by replacing the learning task with a competition task (Taylor & Gammon, 1976). In this reaction-time competition, the slower performer receives an electric shock preselected by the winner. The ethical concern regarding the use of the Buss paradigm has been attenuated in as much as the thorough debriefing discussions which are undertaken are successful in preventing guilt feelings and self-esteem doubts from occurring in the subject following the experiment. However, no definite data concerning this issue are available.

The extensive and almost unique use of this paradigm in the study of aggression has raised questions concerning the artificiality of this paradigm's internal validity. This extensive use of the Buss paradigm may have caused researchers to obtain paradigm-specific results (Stonner, 1976). This problem is attenuated by finding consistent response patterns across laboratories, researchers and procedures (Goldstein, Davis, & Herman, 1975). The Buss paradigm has received support concerning its external validity. Children who were aggressive in the laboratory were also rated as such by their peers in natural settings (Williams, Meyerson, Eron, & Semler, 1967). Furthermore, increased aggression as a result of exposure to aggressive stimuli (e.g., guns, see Berkowitz & LePage, 1967) was found related to aggressive behavior of drivers in a field experiment (Turner, Layton, & Simons, 1975). Also, in a study relating aggression observed in the laboratory to aggression of adults in naturalistic social situations, prisoners were found to be more aggressive in the laboratory than were student controls (Wolfe & Baron, 1971).

The major contribution of the Buss paradigm lies in its widespread use. It encouraged detailed examination of situational variables playing a role in the instigation and regulation of human aggression. Among the instigating variables examined by studies are the different types of frustration used (Buss, 1963; Rule & Percival, 1971), a variety of

aggressive cues, such as weapons, film violence (Bandura, 1973; Berkowitz, 1973, 1974; Berkowitz & Alioto, 1973; Berkowitz & Geen, 1966; Berkowitz & LePage, 1967) and arousal from different sources (Berkowitz et al., 1969; Geen & O'Neal, 1969; Rule & Hewitt, 1971; Rule & Nesdale, 1974).

Among aggression-regulating variables examined by studies focusing on the aggressor are the justification for behaving aggressively (Brock & Buss, 1964), the need to restore psychological equity (Berscheid, Boye, & Walster, 1968), whether the aggressor acts alone, by an intermediary (Baker & Schaie, 1969), or in the presence of a group (Diener, 1976), whether the aggressor feels responsible for his acts (Diener, Dineen, Enderson, Beaman, & Frazer, 1975), his degree of self awareness (Rule, Nesdale, & Dyck, 1975) and the identification of the aggressor with the winner in a previewed violence film (Leyens & Pilus, 1973). Variables implicated by studies assessing the victim's role in the regulation of aggression are familiarity with the victim (Larsen, Lancaster, Lesh, Redding, White, & Larsen, 1976), the victim's sex (Buss, 1971; Taylor, & Epstein, 1967), his identity (Berkowitz & Geen, 1967) and his ability to retaliate (Baron, 1971, 1973).

A careful examination of the multivariate aggressive behavior investigated in these studies reveals the necessity to differentiate between two types of aggression. Buss (1971) suggested the differentiation between angry aggression and

instrumental aggression. The former type includes responses which are elicited by anger inducers such as insult, attack, annoyance, etc. These responses are usually accompanied by a state of arousal labeled anger and aimed at inflicting discomfort such as pain, embarrassment, etc. Instrumental aggression is initiated through competition or a sought reinforcer which is in another person's possession. The accompanying state of arousal is not labeled anger and the aggressive responses aim to acquire the reinforcer. Subsumed under instrumental aggression is the response aimed to terminate aversive stimulation. As will be seen below, this aggression-type differentiation is useful in facilitating the accounting for conflicting findings concerning the mediating effects of alcohol on aggression.

General Considerations from Alcohol Research

This section will attempt to enumerate alcohol-related effects on human behavior which are pertinent to the study of alcohol mediated aggression. Commonly, the user of drugs can tell the difference between the subjective effects of a marihuana cigarette, an amphetamine pill, a morphine injection and a drink of alcohol. The differences, however, are often related to gustatory and olfactory cues, effects of the route of administration, and the environmental circumstances surfounding the drug's use. Without these cues differentiation between drugs becomes quite difficult (Jones & Stone, 1970).

In general, ingestion of alcohol has been associated with a reduction in sensory acuity, a decrease in the drinker's attention span to environmental stimuli, disinhibition of emotional expression, progressive drowsiness, sleep and coma. Yet, the sequence of these effects varies with the drinker's past experience with alcohol, the setting in which the alcohol is drunk, the dose and the rate of ingestion (Kalant, 1970). Thus, it seems that pharmacological and nonpharmacological variables interact in the determination of the substance's effects on human behavior. The variety of factors and effects necessitates a closer look at the substance's characteristics.

Alcohol, or ethanol, is produced through fermentation of a variety of products such as grain and potato mashes, fruit juices, beet and cane sugar molasses, and waste liquor from sulfite factories. The preparation of pure ethanol involves a distillation process regardless of the alcohol's origins. The main components of alcoholic beverages are water and ethanol. Beers, table wines, dessert or cocktail wines, liqueurs or cordials, and distilled spirits, differ from each other in the mode of preparation of the fermented product, chemicals added and ethanol content (for detailed description, see Wallgreen & Barry, 1970, chap.2). In addition to ethanol and water, alcoholic beverages often contain small quantities of substances referred to as congeners. Typically, these are methanol, fusel oil, acids, esters, aldehydes, and other organic and inorganic compounds. The congeners have been

reported to have a variety of effects on behavior (Leake & Silverman, 1966, pp.160), a fact which makes comparison between studies using beverages of different congener content more difficult. Distilled spirits of different congener content are used in laboratory research due to their relatively high ethanol content and widespread use by the human consumer. There are many experimenters, however, who administer pure ethanol to subjects to obtain better dose control and "clean" ethanol effects.

One of the indices used for assessing the level of intoxication reached by an alcohol-ingesting organism is the blood alcohol concentration (BAC) level. During the absorption and distribution of alcohol in the body, arterial and capillary blood reflects rather closely the alcohol concentration in the brain (Wallgreen & Barry, 1970, p.34). Thus, the BAC level is often assessed in blood samples. This index is commonly expressed as a percentage; grams of alcohol per 100 ml of blood.

Another, and perhaps more popular, method of BAC assessment is through the analysis of breath samples. It has been shown, that as the lung capillaries are very permeable to ethanol, expired air provides a good estimate of arterial plasma alcohol concentrations (Anthonisen & Crone, 1956). Due to the convenience this method offers for human behavioral experimentation, it is used in spite of its lesser degree of accuracy.

Several routes of alcohol administration are available: Oral administration, intraperitoneal administration, intravenous injection, subcutaneous injection, inhalation and through the skin. The more popular method used with human subjects is oral administration. This method presents several problems. It was found (Schwagmeyer, 1937) that food will delay the absorption of alcohol producing a slower rise and a lower peak of the BAC curve. Different alcoholic beverages were also found to produce a variety of absorption rates as they interact with other substances present in the intestines (Pihkanen, 1957).

The time it takes to eliminate ingested alcohol from the body determines the duration of a given dose's action. As most of the ethanol is oxidized and eventually disposed of as carbon dioxide and water, increased respiration will shorten the elimination period. More important however, elimination rate has been linked to dose and shown to decrease as dose increases (Ekman, Frankenhaeuser, Goldberg, Hagdahl, & Myrsten, 1964). In addition, speed of drinking has been shown to affect elimination in that fast drinkers took longer to eliminate a given dose of alcohol than slow drinkers (Jones & Vega, 1973). The time it takes to reach the elimination phase (i.e., the descending limb of the BAC curve) varies. There is a consensus regarding the passage of approximately 90 minutes following oral administration of

80 proof and 100 proof vodka until that phase is reached (Fregly, Bergstedt, & Graybiel, 1967). In summation, factors such as time passage following ingestion, drinking speed, dose, and body weight of drinker, determine the characteristics of the effects of alcohol in terms of the ascending and descending limbs of the BAC curve. This curve is important for the understanding of the drug's effects on physiological, intellectual and emotional functions (Grenell, 1972; Jones & Vega, 1972; Russell & Menrabian, 1975). As will be seen hereafter, the effect of alcohol on these functions is an important determinant of the alcohol-mediated aggressive response.

Like the role of arousal in aggression, the labeling of an emotional state leading to the expression of aggression is influenced by external as well as by internal cues (Schachter & Singer, 1962). The role of internal sensations in the evaluation of their state of intoxication has been demonstrated by experienced drinkers (Bois & Vogel-Sprott, 1974). Thus, physiological effects of alcohol such as changes in heart rate, skin temperature, and the Galvanic Skin Response, may lend themselves to the drinker's attributional interpretations.

Although ethanol is classified as a hypnotic or sedative drug, small or moderate doses have been shown to have a physiological stimulating effect. Heart rate and blood

pressure have registered consistent and transient small increases following ingestion of alcohol (Stein, Lieber, Leevy, Cherrick, & Abelmann, 1963). Inconsistent increases in heart rate coupled with decreases in blood pressure have been reported to occur following the ingestion of brandy but not following beer (Takala, Pihkanen, & Markkanen, 1957). An initial increase in heart rate with a subsequent return to normal as higher BAC levels are reached, has also been associated with an emotional response to the drinking experience (Alha, 1957).

Cutaneous vasodilatation, appearing as flushing, marks the effect of ethanol on regional blood flow. This blood flow, appearing to have a thermoregulatory function in the fingers, arms, feet and toes (Garlind, Goldberg, Graf, Perman, Strandell, & Ström, 1960), has been reported to increase following the ingestion of small and moderate doses of alcohol.

Increases of the Galvanic Skin Response (GSR) have also been associated with fluctuations in emotional states. Following a moderate dose of alcohol, GSR changes in response to a startling stimulus were reported to be signicantly lower than following a low dose of alcohol in subjects engaged in a card-sorting task (Carpenter, 1957). In contrast, another study (Coopersmith, 1964) reported elevated GSR responsiveness in inebriated subjects performing a word recognition task. Thus, in this study, it appeared that alcohol

increased the emotional response when subjects were engaged in difficult tasks. Whereas these data discrepancies may be due to situational factors and specific task requirements, they may also be due to the different doses of alcohol used (Idestrom & Cadenius, 1968). Thus, the inconsistent data associated with many alcohol-induced physiological changes prevent clear conclusions concerning the role of internal sensations in the drinker's attributional processes.

Alcohol-mediated cognitive functioning may be related to aggressive behavior in another way. In the case of the aroused inebriated individual, the effect of alcohol on risk-taking behavior may modify his response to instigation. Alcohol is believed to increase risk-taking behavior in humans. It appears that certain alcoholic beverages increase the level of risk-taking of drinkers (Cutter, Green, & Harford, 1973; Teger et al., 1969). However, it was observed that alcohol distorts the perception of how much risk is being taken and does not affect the level of subjective risk individuals are willing to take (Cohen et al., 1958; Katkin, Hayes, Teger, & Pruitt, 1970). Risk-taking behavior, often operationally defined as gambling decisions, has been shown to be dose related, in that low doses increased risk-taking while high doses produced a reduction in this behavior (Sjöberg, 1969).

The inebriated individual's ability to process the risk situation as defined by the above studies may be related

to the effects of alcohol on other intellectual functions such as problem-solving behavior, abstraction, learning and memory. Problem-solving behaviors are commonly assessed through tasks involving arithmetics, digit-symbol substitution, spatial relations, abstraction and calculus problems. Problem-solving behavior has been generally found to deteriorate under the effect of high doses of alcohol (Carpenter, 1962). Performance on digit-symbol tasks suffered following the ingestion of moderate doses as well (Muller, Tarpey, Giorgi, Mirone, & Rouke, 1964). Whereas low doses facilitated higher order problem solving (i.e., calculus problems), high doses had detrimental effect on this behavior (Carpenter, Moore, Snyder, & Lisansky, 1961).

Some studies related performance levels on the Raven Progressive Matrices test to the phase of alcohol elimination from the body. Deterioration in the performance on this test was observed as blood alcohol levels increased (Jones, 1974). The greater detrimental effect was noted during the elimination phase, that is, on the descending limb of the BAC curve (Jones & Vega, 1972; Jones & Bertera, 1974). These findings suggest that the arousing effect of low and moderate alcohol doses may facilitate some intellectual performance, and that this performance may deteriorate as the depressing effect of high doses sets in.

The notion that learning may continue under the influence of alcohol at low doses is supported by studies in

which social drinkers were successfully taught to discriminate between different low blood alcohol concentrations in themselves (Bois & Vogel-Sprott, 1974; Ogurzsoff & Vogel-Sprott, 1976). Furthermore, performance on a coding and vigilance task partially improved under low BAC levels (Vogel-Sprott, 1976). The effects of alcohol on short-term memory tested by Carpenter and Ross (1965) revealed linear performance deterioration of highly skilled subjects. Subjects with lesser degrees of proficiency demonstrated improved performance following low doses, as well as less absolute deterioration. Kalin (1964) tested long-term recall of Thematic Apperception Test (TAT) stories written the previous day while inebriated. His findings, consistent with numerous other studies, showed that story recall of these subjects was markedly poorer in comparison to controls. The phenomenon of "blackouts" was also demonstrated in the laboratory with alcoholics following moderate doses of alcohol (Diethelm & Barr, 1962).

In summary, it appears that the effects of alcohol on the reviewed intellectual functions are not only determined by the dose administered, but also by situational variables, task requirements and motivational factors. One remaining important variable, pertinent to the understanding of the alcohol-aggression relationship, is the substance's effect on emotions and their expression.

The extent to which alcohol affects human emotional states has been repeatedly linked to the drinker's ability to perceive his state of intoxication (Kastl, 1969). Inebriated subjects' ability to accurately estimate their level of intoxication has been established (Ekman et al., 1964), as well as the fact that this self-perception is made possible by attending to internal and external cues (Bois & Vogel-Sprott, 1974).

Numerous emotion-mediated behaviors have been reported to follow ingestion of alcohol: Increased talkativeness (Alha, 1951), argumentativeness (Loomis & West, 1958), exhilaration (Pihkanen, 1957), friendliness and happiness (Freed, 1970). Subjects reported increased "sexual power" (McGuire, Stein, & Mendelson, 1966), fantasies of personal power (McClelland, Davis, Kalin, & Wanner, 1972) and of physical aggression (Kalin, McClelland, & Kahn, 1965) following the ingestion of moderate and high doses of alcohol. However, feelings of detachment, depression, irritability and aggression have also been reported to occur in conjunction with alcohol intoxication (Fregly et al., 1967; Mendelson, LaDou, & Solomon, 1964).

These seemingly discrepant findings are believed to be closely related to the dose of ingested alcohol and the circumstances under which the alcohol is consumed (McClelland et al., 1972). In a three-emotional-dimensions

model (i.e., pleasure, arousal, dominance) Russell and Mehrabian (1975) suggested that the emotional state of the drinker is determined by his pre-drinking emotional state, the emotion-eliciting properties of the situation, the drinker's personality predispositions and the dose of alcohol ingested. According to these authors, an individual who experiences displeasure, moderate arousal and is neutral on the dominance dimension before drinking, and proceeds to consume a moderate dose of alcohol, may experience anger comprised of displeasure, high arousal and dominance resulting in hostility.

This interpretation of the effects of alcohol on emotion-mediated behaviors assumes that the drinker is aware of internal and external cues. However, with an increase in the ingested alcohol dose and a related modification of cognitive processes, these cues may play a different role in the control of behavior.

The hereto reviewed findings indicate that at low doses alcohol acts as a stimulant whereas at higher concentrations it acts as a depressant. This dual effect might be at first interpreted to result in a disinhibition of emotional expression which then leads to increased activity and approach of usually avoided situations. At higher points on the intoxication curve reduced activity, depression, and at times, avoidance behavior occur. Keeping in mind the

characteristics of alcohol and its effects on some physiological, intellectual and emotional functions in humans, one can proceed to review the studies examining alcohol-mediated aggression.

Effect of Alcohol on Aggression

In an attempt to understand the relationship between alcohol ingestion and the production of aggression, three basic questions have been raised (Carpenter & Armenti, 1972):
(a) Is aggression pharmacologically induced by alcohol?
(b) If aggression is caused by other central nervous system (CNS) arousal, does alcohol modify aggressive behavior?
(c) How does alcohol affect aggression in a social context?

The examination of animal behavior in a sterile environment is the best approach to determine whether aggression is pharmacologically induced by alcohol. Unfortunately, the evidence in this respect is scant. Chamove and Harlow (1970) noted increased self-aggression in individually housed monkeys who were administered alcohol in an ad-lib fashion. Testing the aggressive response in individually housed Siamese fighting fish, Raynes and Ryback (1970) found increases in aggression in those fish which were immersed in an ethanol solution. These findings were confounded by the introduction of a mirror into the fish tank. No increase in aggressive behavior was found in intoxicated rats (Cappell & Latané, 1969).

Aggression induced by hypothalamic stimulation of cats sheds some light on the mediating effects of alcohol. MacDonnell and Ehmer (1969) reported that alcohol intoxicated CNS-aroused cats took longer to become aroused and perform their attack on a target. This finding seemed more prominent when high alcohol doses were administered. However, alcohol seemed to increase the force of biting. In another study (MacDonnell & Fessock, 1972), alcohol intoxicated cats receiving no direct CNS stimulation displayed suppressed seizing and biting behavior. It appears, from this line of experiments, that alcohol in interaction with other arousal may increase the aggressive output in animals.

The effects of alcohol on human aggression behavior has been examined in a variety of contexts by several paradigms. Aggression had been assessed by indirect means such as check lists, projective and other tests, verbal interactions between subjects in simulated drinking-party situations and by the Buss paradigm assessing physical aggression.

Using the TAT projective test, Kalin et al. (1965) found no increases in aggression expressed by intoxicated subjects of a "discussion" group. However, the aggression score of subjects in a "fraternity party" group increased with the amount of alcohol consumed. The content of the stories obtained, mainly indicated an increase in thoughts of physical aggression and a decrease in aggression restraints.

These findings indicate that alcohol dose and social setting affected the stories' aggressive content. Another indirect observation of aggression was reported by Hetherington and Wray (1964). In this study, the subjects task was to rate aggressive or nonsense cartoons for their degree of humor. Inebriated subjects, high in need for aggression and social approval rated the aggressive cartoons as well as the nonsense cartoons as funny. Assuming a valid relationship between the personality dimension of need for aggression, the attachment of humor to aggressive cartoons and other aggressive behaviors, these findings suggest that the alcohol may cause the drinker to be less amenable to the controlling effect of social approval.

A more direct assessment of aggressive behavior was reported by Hartocollis (1962) observing the verbal interaction between subjects who have been intravenously injected with a moderate dose of alcohol. The subjects behavior was described as elated, aggressive, boisterous and hostile. In another study (Takala et al., 1957), verbal aggressive behavior was analyzed according to Bale's small-group interaction categories. Verbal aggression was found following ingestion of beer and brandy, whereas no aggressive responses were observed in the control group. In addition, the type of alcoholic beverage seemed to have a differential aggression-mediating effect as the brandy-group displayed more aggressive

responses than the beer consuming subjects. Similarly, increased verbal aggression due to the differential effect of two beverage types was observed in studies of simulated drinking parties (Boyatzis, 1974, 1975). In these studies, inebriated subjects experienced a "sense of power" and "bolstering for aggression", and displayed greater verbal aggression following the ingestion of distilled spirits than following the ingestion of beer.

The seemingly consistent relationship between alcohol and aggression is challenged by some studies of human physical aggression. In studies using the same experimental paradigm forwarded by Buss (1961), aggression is defined in terms of shock levels given to a "learner" by a subject assuming the teacher's role. In one study (Bennett, Buss, & Carpenter, 1969), ingestion of alcohol did not lead to significant increases in shock intensities administered, nor were the results related to the dose of alcohol ingested. The steady rise of the shocks administered during the experiment was attributed to apparatus-specific factors noted in previous research (Buss, 1963). In a more recent study (Lang, Goeckner, Adesso, & Marlatt, 1975), an increase in aggressive behavior was found in both inebriated subjects and in those who received a placebo beverage. The authors attributed the increase in aggression to the drinker's expectancies concerning drinking alcohol. As was suggested elsewhere (Sobell

& Sobell, 1975), the drinker may feel less responsible for his actions while intoxicated.

Different results were obtained by experimenters using a modification of the Buss paradigm. Replacing the "teacher-learner" task with a competitive reaction-time task, Shuntich and Taylor (1972) reported that subjects who ingested bourbon chose significantly higher shocks than placebo or no-drug controls. In similar experiments, where the aggressor had knowledge of the shock levels chosen for him by his opponent, increased aggression was found to be related to dose and type of beverage consumed (Taylor & Gammon, 1975; Taylor, Vardaris, Rawitch, Gammon, Canston, & Lubetkin, 1976). In the Taylor and Gammon (1975) study, the effect of low and high doses of vodka were compared to that of low and high doses of bourbon. Both groups receiving high doses displayed more aggression than those receiving low doses. Whereas the high dose of alcohol seemed to facilitate expression of aggression, the low alcohol dose seemed to have an inhibitory effect. This effect was most pronounced in the vodka conditions. Similar findings were reported by Taylor et al. (1976).

Some data concerning the effects of situational variables on alcohol-mediated aggression are reported by Taylor and his colleagues. The presence of a third party during the experiment suppressed the overall aggressive output but had a smaller inhibiting effect on intoxicated subjects than

on controls (Taylor & Gammon, 1976). In another study (Taylor, Gammon, & Capasso, 1976) inebriated subjects displayed increased aggression only when exposed to a threat situation. Finally, in an attempt to examine the effect of frustration on alcohol-mediated aggression, Taylor, Schmutte and Leonard (1977) found that although the inebriated subjects were most aggressive, the frustration manipulation did not appear to influence their behavior. Rather, situational antecedents such as physical attack, social pressure or ingested alcohol, were implicated as strong determinants of physical aggression.

Several reasons can be suggested for the discrepancy between findings of studies on alcohol-mediated aggression. First, the different applications of the pharmacological variable. As shown earlier in this review, alcohol has been implicated as having an extensive effect on cognitive processes and emotion-mediated behaviors. Furthermore, types of alcoholic beverages, dose and time passage after ingestion were found to play a crucial role in determining the substance's effect. Hence, since different experimenters have administered a variety of alcoholic beverages at different doses, giving rise to a variety of BAC levels, discrepant findings can be expected. For example, in the Shuntich and Taylor (1972) study, a dose of 0.9 ml of 100 proof bourbon/kg body weight was administered. In the Bennett et al. (1969)

study, doses of .33, .67 and 1.0 ml of absolute alcohol/kg body weight in the form of vodka were given. Finally, the dose used in the Lang et al. (1975) study was 1.3 ml of absolute alcohol/kg body weight. Moreover, passage of time between commencement of drinking and the end of the experimental task is often not reported and the estimation of the subject's BAC levels during the experiment is based on a pre-task reading. These issues make the comparison between studies difficult as there is no certainty as to during which stage of the BAC curve the task was performed.

A second factor to consider is the role of subject arousal throughout the experiment. The bulk of studies on aggression associate arousal with the occurrence of aggression and see arousal as a facilitator of such a response (Donnerstein & Wilson, 1976; Epstein & Taylor, 1967; Geen & O'Neal, 1969). Yet some studies have found a reduction in aggression under certain levels of arousal (Baron & Bell, 1976; Bell & Baron, 1977). In most studies on alcohol-mediated aggression an adaptation of the inebriated subject to the arousing stimuli (e.g., noise, shock) is likely but is not controlled (Lang et al., 1975; Shuntich & Taylor, 1972; Taylor & Gammon, 1975).

A third possible reason for obtaining discrepant results in these studies are the task demands to which the subjects are exposed and the nature of the monitored response. Some experimenters have induced frustration in their

subjects by exposing them to loss in competitive tasks against retaliating opponents (Taylor & Gammon, 1975; Taylor et al., 1977) while monitoring angry-aggressive behavior. In contrast, other studies have monitored instrumental (or altruistic) aggression of subjects who were trying to facilitate their passive experimental partner's learning (Bennett et al., 1969; Lang et al., 1975).

Although scholars of alcohol-mediated aggression concur that alcohol has a disruptive effect on the drinker's cognitive processes, most do not address themselves specifically to the effects this disruption has on the production of aggression. The present investigation attempts to assess this effect in regard to the antecedents and consequences of the aggressive response.

The Present Investigation

The present investigation of alcohol-mediated aggression views this behavior within the social learning framework. It is believed that aggression is most commonly instigated by unpleasant arousal which the subject experiences. In the present two studies alcohol is conceptualized as a pharmacological agent that has a disinhibiting effect on human behavior. However, it is assumed that alcohol increases arousal and motivates approach behaviors essentially along the ascending limb of the BAC curve prior to the alcohol elimination phase. This effect is expected to facilitate

the expression of emotion-mediated behaviors of which aggression is one.

The present two studies attempt to elucidate two questions: (a) Does ingested alcohol modify a person's ability to use information concerning his behavior contingencies, thus affecting his aggressive behavior?, and (b) does ingested alcohol modify a person's ability to consider the instigator's intent in order to control his aggressive response?

It has been suggested (Bandura, 1969) that behavior is governed by anticipated outcomes based on previous consequences of a given behavior. The first study attempts to assess whether the inebriated individual maintains his ability to process information related to possible consequences of his aggressive behavior in a given situation. In this study, information concerning behavior contingencies is made available to the drinking individual. As the contingencies are aversive to the subject, it is of interest to establish whether he takes them into account in order to evaluate the level of risk he is taking by aggressing against another person. It is hypothesized that under the effect of alcohol the intoxicated person's response is strongly affected by the instigating aversive stimulation.

Among the variables implicated as comprising the agresive act is the perceived intent of the instigator (Buss,

1971). When extended one step further, this notion may suggest that the response of an individual who is aversively stimulated by another individual may be modified by the perceived intent with which the aversive stimulation is delivered. The second study presents two different types of instigator-intent to the inebriated subject. It is of interest to establish whether following the ingestion of alcohol a person maintains the ability to process the intent of the instigating agent, or whether, as in the first study, his response is controlled by the aversive stimulation per se.

The experimental paradigm used in the present two studies is similar to paradigms used in aggression research. A modification of the Buss (1961) aggression machine is used in an interaction between the subject and his partner (an operant conditioning apparatus) in a task presented as a reaction-time/pain-threshold procedure. The instigating stimulation used is a pure tone individually evaluated by each subject as aversive. This stimulation was chosen for the following reasons: (a) exposure to noise has been associated with aggressive behavior (Glass & Singer, 1972; Waybrew, 1967), (b) auditory sensitivity has been reported to deteriorate only slightly under the effect of moderate doses of alcohol (Schneider & Carpenter, 1969), (c) auditory stimulation has been shown to be aversive but harmless (Kryter, 1970), and (d) the tonal stimulation is readily

quantifiable and resistant to adaptation (Stockinger, Cooper, Meissner, & Jones, 1972), thus ensuring continued aversive arousal throughout the experiment.

In an attempt to avoid responses which are related to aversive stimulation delivered at increasing intensities throughout the task (Shuntich & Taylor, 1972), the aversive instigating stimulation in the present studies was comprised of different tone levels administered in a random order.

The alcohol administered to subjects in the present studies was 95% ethanol at a dose of 1.32 ml/kg body weight. This substance and dose were chosen in an attempt to avoid effects due to congener content and to give rise to BAC levels above the legal-driving level of .080%. A second Breathalyzer reading taken at the end of the experiment ensured an accurate estimate of the subjects' BAC level during the task. Placebo and sober groups were added in an attempt to control for expectancy effects (Lang et al., 1975).

In the present investigation, only one dose of alcohol was administered due to the complex experimental design. To examine the effect of an additional alcohol dose in a methodologically sound manner would have necessitated doubling the number of subjects. This would have been beyond this investigation's possibilities.

In some studies of alcohol-mediated aggression task instructions were given before drinking (Bennett et al., 1969) while in others the instructions were given after the subject

was inebriated (Shuntich & Taylor, 1972). In the present studies, regardless of when the instructions were given, a special comprehension test was instituted in an attempt to ascertain that the inebriated subjects understood and remembered the instructions before the task was commenced.

Finally, and perhaps most important, one ought to consider the nature of the population used in the present investigation. Volunteer subjects have often been shown to differ from nonvolunteer subjects (Rosnow & Rosenthal, 1966; Rosnow, Rosenthal, McConochie, & Arms, 1969), a point which is particularly crucial in aggression research. The use of volunteer college students in this area of research raises a question concerning the validity of the findings. As familiarity with this type of psychological experimentation on aggression spreads on campus, students become suspicious of the experimenter's explanation and may behave in a manner so as to counteract the manipulation's effects (Stonner, 1976). The samples used in the present investigation are comprised of subjects of diverse background. This fact may strengthen the validity of attempts to generalize from the present investigation's findings.

EFFECTS OF ALCOHOL AND BEHAVIOR CONTINGENCIES ON HUMAN AGGRESSION

ABSTRACT

The purpose of this study was to determine the mediating effects of alcohol and behavior contingencies on aggression in male social drinkers. Seventy-two subjects were randomly assigned to one of six groups in a 3 x 2 factorial design. To control for alcohol and expectation effects, one third of the subjects received alcoholic beverages, one third received placebo drinks and another third was not administered any beverages. Aggression was assessed by the intensity and duration of shocks administered to a bogus partner in a modification of the Buss aggression procedure. Half the subjects were exposed to aversive contingencies correlated with their aggressive responses, and half received random aversive contingencies. The inebriated subjects were significantly more aggressive than the noninebriated subjects ($p < .01$). Moreover, the former subjects displayed an equally aggressive pattern under both contingency conditions, whereas the nonintoxicated subjects displayed a differential response pattern affected by the contingency type. These findings are attributed to the disrupting effect of alcohol on information processing and the mediating effect of contingencies on the nonintoxicated individual's aggressive behavior.

INTRODUCTION

The frequency of use and abuse of alcohol and the well documented correlation between alcohol and aggression results in a topic of understandable concern. Alcohol is highly correlated with assaultive behavior (Mayfield, 1976; Nicol, Gunn, Gristwood, Foggit, & Watson, 1973), homicide (Virkkunen, 1974; Wolfgang & Strohm, 1957), suicide (Buglass & McCulloch, 1970; Hagnell, Nyman, & Tunvig, 1973) and violent and nonviolent crimes (Fitzpatrick, 1974; Lindelius & Salum, 1975; Sobell & Sobell, 1975).

To date, the attempt to identify the excessive drinker in terms of specific personality characteristics has failed (Donovan & O'Leary, 1975; Orford, 1976). Likewise, the existance of a definable aggressive personality continues to elude researchers (Lester, Perdue, Brooknart, 1974; Wilkins, Scharff, & Schlottmann, 1974). Even studies attempting to tie drinkers and aggressors in terms of personality traits have failed (Kristianson, 1974; Ruff, Ayers, & Templer, 1975). It appears that research into the relationship between alcohol and aggression would benefit from a laboratory interactionalistic perspective.

One theoretical framework which has been forwarded to explain the correlation between alcohol ingestion and aggressive behavior utilizes two related physiological models: In one, alcohol is assumed to have an "energizing" effect which may lead to aggressive behavior. Some infrahuman research, though inconclusive, has demonstrated increased attack behavior in

alcohol-intoxicated cats which simultaneously received electrical stimulation of the CNS (MacDonnell & Ehmer, 1969; MacDonnell & Flynn, 1966). Perhaps relatedly, human subjects who ingested alcohol report an increase in aggressive fantasies (Kalin, McLelland, & Kahn, 1965) and feelings of power and dominance (McClelland, Davis, Kalin, & Wanner, 1972; Russell & Mehrabian, 1975). The other model does not directly attribute induction of aggression to alcohol, but conceptualizes the substance as a disinhibitor of approach behaviors which may facilitate the occurrance of aggression. Whereas infrahuman studies indicate a reduction of avoidance behavior following the ingestion of alcohol (Wallgreen & Barry, 1970, for review), inebriated human subjects demonstrated increased verbal (Boyatzis, 1975) and physical aggressive behavior (Shuntich & Taylor, 1972). The increase of aggressive behavior in humans has been indirectly attributed to the anxiety reducing effect of alcohol (Marlatt, 1976) which may lead to an attenuation of the impact of physical or social consequence on the aggressing individual. However, the tension-reduction hypothesis concerning the effect of alcohol is a dubious proposition (Cappell & Herman, 1972).

To date, studies examining the effect of alcohol on human aggressive behavior in the laboratory have yielded equivocal results. Employing modifications of the basic experimental paradigm forwarded by Buss (1961), researchers

observing increased aggression in inebriated subjects (Shuntich & Taylor, 1972) attribute this increase to the type and dose of alcoholic beverage administered (Taylor & Gammon, 1975; Taylor, Vardaris, Rawitch, Gammon, Canston, & Lubetkin, 1976). In contrast, other studies have shown that alcohol did not lead to aggression (Bennett, Buss, & Carpenter, 1969) or that an increase in aggressive behavior occurred regardless of the alcohol content of the administered drink (Lang, Goeckner, Adesso, & Marlatt, 1975). These authors explained the latter result in terms of the expectancies drinkers have regarding the effects of alcohol.

One of the variables seen playing a role in the control of human aggression is the behavior contingency attached to the aggressive response; that is, the physical and social consequences of one's aggressive behavior. When retaliation from the victim of aggression was a threat (Baron, 1973), or when a potential aggressor stood to gain relatively little through his behavior (Baron, 1974), aggressive behavior was inhibited. Likewise, expectancy of retaliation caused a decrease in victim derogation by the aggressor (Berscheid, Boye, & Walster, 1968). Furthermore, the opponent's ability to match electric shock settings of the aggressor served as an effective inhibitor of aggression (Pisano & Taylor, 1971). Infliction of pain or harm on a victim, both of which may have led to social repercussions against the aggressor, have been shown to reduce subsequent physical aggression in the labora-

tory (Buss, 1966a; Swart & Berkowitz, 1976).

To examine the controlling effect behavior contingencies have over aggressive behavior, a differentiation between instrumental and angry aggression is warranted (Buss, 1971; Feshbach, 1971). Instrumental aggression is seen as a response aimed at obtaining a reinforcer possessed by another or to avoid punishment by another. Anger is not necessarily associated with this response. An angry aggressive response is coupled with anger and aimed at inflicting discomfort or injury on a victim. This differentiation is necessary due to the possibility that behavior contingencies may have a stronger controlling effect on one type of aggressive behavior than on another.

If contingencies have significant import for aggressive behavior, the individual's ability to process information concerning contingencies becomes particularly crucial. Perhaps it is at this level that alcohol interacts with aggression determining factors.

When the effects of alcohol on risk-taking behavior is examined, it appears that certain alcoholic beverages increase the level of risk taken by individuals (Cutter, Green, & Harford, 1973; Teger, Katkin, & Pruitt, 1969), but that alcohol distorts the perception of how much risk is being taken and does not affect the level of the subjective risk individuals are willing to take (Cohen, 1960; Katkin, Hayes, Teger, & Pruitt, 1970). These studies, however, tap risk-taking

behavior on gambling and choice-dilemma tasks. Studies on laboratory aggression have demonstrated that the aggressiveness of an individual is a function of the probability of being retaliated against and of previous exposure to an aggressive model (Baron, 1971, 1973). One study reported increased aggression in inebriated individuals who were in a threat-situation (Taylor, Gammon, Capasso, 1976).

Considering the hereto mentioned variables, one may hypothesize a shift in behavior control under the influence of alcohol. Normally, when an individual is about to engage in aggressive behavior his responses are presumably controlled by anticipated consequences according to past experience. Under the effect of alcohol, however, experiential constraints may prove ineffective. In a state of arousal, after having ingested an adequate dose of alcohol, an individual may exhibit stimulus-bound behavior. In the case of the aggressing individual, his behavior may be more controlled by the arousing stimulus and less by the probable consequences of his behavior.

The present study attempts to elucidate the question of whether ingested alcohol interferes with a person's ability to process information concerning his behavior contingencies, an interference which may explain alcohol's relationship to the aggressive response. The basic paradigm forwarded by Buss (1961, p.47), utilizing an aggression machine, will be

employed. The subject's preparedness to administer painful electric shocks to another person, while shocks of negligible intensity are available, proves to be one of the laboratory controlled measures of human physical aggression to receive naturalistic validation (Wolfe & Baron, 1971). To control for the effects of expectancies associated with ingestion of alcohol (Lang et al., 1975) placebo and sober groups are added in the present study. As general arousal has been associated with the occurrance of aggression (Donnerstein & Wilson, 1976; Geen & O'Neal, 1969) an aversive tone is used in the present study both as an antecedent stimulus and as a consequence to the aggressive response. Finally, alcohol dose and drinking pace are controlled in order to achieve blood-alcohol concentrations conforming with those at which human subjects have exhibited an increased level of approach behaviors (Ekman, Frankenhaeser, Goldberg, Hagdahl, & Myrsten, 1964) and a disruption of cognitive processes (Jones & Vega, 1973).

METHOD

Subjects

Seventy-two male social drinkers, aged 18 to 35 (M age = 23.5 yr.), were recruited as paid subjects by means of local newspaper, radio and television advertisement. Initial telephone-screening followed by a personal interview and completion of a drinking-history and personal data questionnaire, ascertained that participating subjects met the following

requirements: (a) They did not have an alcoholism problem in the past or present, (b) were not treated or arrested in the past in connection with excessive drinking, (c) did not suffer any hearing impairment, (d) had no previous familiarity with psychological experimentation, and (e) drank regularly but not in excess of the equivalent alcohol content of eight beers per day.[1] These screening measures were instituted in order to increase sample homogeneity. The use of experienced drinkers ensured similar expectancies related to alcohol consumption. As a high alcohol tolerance could attenuate the effects of alcohol in the laboratory, heavy drinkers were avoided. Each of the participating subjects was asked to abstain from food and any drugs for a period of four hours prior to the experimental session.

Experimental Design

Two independent variables were manipulated: (a) Drug- subjects received either alcoholic beverages, placebo beverages or did not receive any beverage prior to the experimental session, and (b) behavior contingencies - subjects were retaliated against in either a pattern fully correlated with their responses or in a random pattern. A randomized, double-blind 3 x 2 factorial design with 12 subjects per cell was employed (see Table 1).

Insert Table 1 about here

Procedure

Upon arrival for the experimental session each subject was greeted by the experimenter and weighed. He was then asked whether he had refrained from eating and drinking any alcoholic beverages for at least four hours prior to his arrival. A breath sample was then analyzed by means of a Mate-A glass-tube analyzer to ascertain that the blood-alcohol concentration (BAC) of the subject was zero.

Assessment of aversive tone threshold. Each subject's aversive tone threshold was determined in the following manner: Segments of a continuous pure tone of 3000 Hz in discrete trials at different levels of loudness ranging from 60 dBA to 109 dBA sound pressure level were administered through headphones. Subjects were instructed to terminate the tone presentation as soon as it became aversive. Termination of the tone was effected by touching a response key of a Lafayette Multi-choice Reaction Timer. Criterion was set at five sec response latency following onset of tone. When the tone was terminated in less than five sec, the loudness level of the subsequent presentation was diminished by one dBA. When the tone was terminated later than five sec, the subsequent presentation's loudness level was augmented by one dBA. The lowest loudness level terminated in three successive trials under the latency criterion was taken as the subject's aversive tone threshold.

Following the tone procedure each subject completed a partial version of the Personality Research Form (Jackson, 1967) and the Locus of Control questionnaire (Rotter, 1966). During this stage an unobtrusive baseline heart-rate reading was taken by means of a Grass photo-electric transducer Model PTTI6 and recorded by a Grass Model 7P4 Tachograph.

Task and instruction. Following the completion of the questionnaires the task instructions were presented by the experimenter. The experimental paradigm employed used a modified version of the Buss (1961) aggression machine. The task was presented to the subject as a reaction-time/pain-perception task in which the subject was to be tested for reaction-time while his "partner" in the adjoining room was to be tested for perception of pain. The bogus partner was an operant conditioning apparatus. The subject was told that both behaviors would be examined under the influence of alcohol in the following manner. Shortly after the appearance of a light on the subject's console the partner would administer a tone through the subject's headphones.[2] After a five sec exposure to the tone, a second light would indicate to the subject to press, as fast as he could, one of the five buttons on his console. Pressing any one of these buttons would terminate the tone and at the same time deliver an electric shock to the partner. It was stressed that whereas button number 1 delivered a hardly noticeable shock, "just a tickle", buttons 2 through 5 activated

increasingly painful shock levels. The subject was led to believe that after receiving the shock the partner would communicate to him by means of a tone level of his choice (a contingency-tone) the degree of pain he felt. This self-terminating tone will last for five sec and will be accompanied by a light signal indicating to the subject which loudness level was chosen by the partner. This constituted one trial.

Each subject participated in 25 consecutive trials interspaced by 15 sec intervals to ensure clear differentiation between trials. The five tone levels used were meant to symbolically compare to the five shock intensities the subject could choose to administer.

Behavior contingencies. All subjects received the first tone's loudness levels in a random order, each level occurring five times. The subjects were divided into two groups, each group being exposed to a different contingency condition: (a) Correlated contingencies - the loudness levels of the contingency-tone delivered were in full correlation with the shock intensities chosen by the subject, and (b) random contingencies - the loudness levels of the contingency-tone were delivered in a random order, each level occurring five times.

The tone used in the experiment was pre-recorded on tape from a Tektronix FG502 Function Generator. This tone was selected to minimize habituation due to temporary auditory threshold shift (Stockinger, Cooper, Meisser, & Jones, 1972).

The light signal indicating the loudness level of the contingency-tone was instituted in order to ascertain visual as well as auditory contingency information.

Video deception. As the task instructions were given by the experimenter to the subject, the latter could observe himself and the experimenter on a 15 cm television monitor which was placed next to the shock console. This was done by means of a closed-circuit television system. Following the instructions the subject was advised that he would be able to observe on the monitor his partner being given his task instructions. As the experimenter left the subject's room a pre-recorded video tape was replayed on the monitor. This tape showed a male (amateur actor) waiting in a room as the experimenter entered. This actor was given a sample of the tone to be administered and examples of shock intensities 1 and 2. Finally, the experimenter turned to the camera and asked the subject to indicate if all was understood by pressing shock button number 1. This was intended to serve as a validation of the deception.

Drug conditions. At this point in the experiment subjects were assigned to one of three drug conditions: (a) Alcohol condition - subjects were administered 1.32 ml/kg body weight of 95% USP (United States Pharmacopoeia) alcohol in a 1:5 (alcohol:orange juice) solution divided into three drinks of equal quantity; (b) Placebo condition - subjects were administered three orange juice drinks of equal proportions

as in the alcohol condition, the alcohol part being replaced by juice and two cc of 95% USP alcohol layered on the drink's surface. Each glass was encased in a terry-cloth holder sprayed with 95% alcohol; (c) Sober condition - subjects were not administered any beverage prior to the experiment.

Drinks (or waiting period for subjects in the sober condition) were consumed in a carpeted, softly lit room, while the subject was seated in a cushioned chair listening to music of his choice. The drinks were prepared by a research assistant. In order to standardize drinking speed and duration, the subject was asked to finish his first drink in six minutes, his second drink in six minutes and his third in eight minutes. A waiting period of 20 minutes followed to allow the alcohol drinking subject to reach an elevated BAC level, but concurrently to provide sufficient time to complete the experiment before reaching BAC peak (Jones & Vega, 1973). At the end of this period, 40 minutes after commencement of drinking, a BAC reading was taken by means of a Smith & Wesson Breathalyzer Model 6000. Finally, the subject was asked to compare his present level of intoxication to the highest level of intoxication he had ever experienced on a seven-point Likert-type "how drunk" rating scale.

At this time the subject was asked to prepare for the task. In order to ascertain that the subject remembered, understood and believed the video deception, six questions pertaining to his task were asked. Once correctly answered

the task was commenced. Throughout the task period the subject was alone in the room. The sequence of the task was fully controlled by Foringer operant conditioning equipment. Four additional unobtrusive heart rate readings were taken throughout the task period. The subject's responses were monitored and recorded in an adjacent control room. After the task was completed the experimenter reentered the room. A second BAC reading was taken and the self-report "how drunk" scale was administered. The subject was then given a short questionnaire assessing his attitude toward the partner. Following this, a debriefing discussion was held and the subject was thanked and paid; those in the alcohol condition were driven home.

Measures of aggression. Two measures of physical aggression and one measure of verbal aggression were taken: (a) The average shock intensity the subject was "delivering" to his partner, (b) the duration of time the subject depressed each shock button, and (c) the subject's score on the post-task attitude questionnaire.

Ethical considerations. For ethical reasons due to the unusual demands put on the subject in this experiment the following steps were taken: (a) All subjects were "pure" volunteers and not coerced in any way to participate in the experiment, (b) the possibility of pressing only shock button number 1 (hardly noticeable shock) was clearly stated, (c) during the debriefing discussion the subject was assured that

he did not shock anyone and that his behavior was perfectly normal and comparable to other subjects' behavior in the same condition, and (d) several weeks following the experiment each subject received a letter explaining his role in the study, outlining the findings and reiterating the statement made in (c).

RESULTS

As subjects were randomly assigned to each of the six experimental groups analyses of variance were performed to assess variance differences between groups on the following variables: Age, years of formal education, socio-economic status (after Blishen, 1967), usual quantity of alcohol consumed, score on Locus of Control questionnaire and the personality scales of aggression, succorance, defendence, lying and social desirability. No significant differences between groups were found on any of these variables.

Alcohol Manipulation

BAC level. All subjects in the alcohol group achieved BAC levels well within the "social drinking" experience. BAC levels ranged between .085% and .150% ($\underline{M}_{BAC}$ = .089%) on the first reading and between .075% and .127% ($\underline{M}_{BAC}$ = .096%) on the second reading. No identifiable BAC levels were found in the subjects of the placebo condition.

"How drunk" scale. Subjects in the alcohol group indicated an average "drunk" score of 3.37 on the pre-task rating and 3.49 on the post-task rating. Placebo subjects averaged 3.08 on the first rating and 3.16 on the second. Two-tailed *t* tests performed to compare the means of the alcohol and placebo groups found the differences to be equally non-significant on the first, $t(46) = 0.72$, as well as on the second rating, $t(46) = 0.73$.

Analyses of variance for repeated measures were performed for comparing between drug groups and contingency conditions. Significant results were further analyzed by the Tukey Honestly Significant Difference test (Tukey, 1956).

Physical Aggression

Shock intensity. The shock intensity score for each subject was obtained by computing an average of all shock choices each multiplied by its respective level. Across contingency conditions alcohol-subjects administered significantly higher shocks than either the placebo-subjects, $Q = 27.43$, ($k = 3$, $df = 66$), $p < .01$, or the sober-subjects, $Q = 29.81$, ($k = 3$, $df = 66$), $p < .01$. No significant difference was found between the placebo and sober groups. When receiving correlated-contingencies, alcohol-subjects delivered higher shocks than either the placebo-subjects, $Q = 22.27$, ($k = 3$, $df = 66$), $p < .01$, or the sober-subjects, $Q = 24.77$, ($k = 3$, $df = 66$), $p < .01$. No significant difference was

found between the placebo and sober groups. Similar results were found under random-contingencies, $Q = 16.56$, ($k = 3$, $df = 66$), $p < .01$ and $Q = 17.45$, ($k = 3$, $df = 66$), $p < .01$, respectively. Behavior contingency effects within each drug condition were compared by two-tailed t tests. Whereas no significant difference was found between correlated and random contingencies in the alcohol group, both placebo and sober groups delivered significantly higher shocks under random contingencies than under correlated contingencies, $t(22) = 6.10$, $p < .005$ and $t(22) = 7.25$, $p < .005$, respectively. Means and standard deviation for this measure are shown in Table 2.

Insert Table 2 about here

Shock duration. No significant difference was found between correlated and random contingencies in the alcohol group. Moreover, the duration used for shock intensities 2 through 5 were significantly longer than the durations used for intensity 1, all $p < .01$. Subjects in the placebo group applied shocks of significantly longer duration under random contingencies than under correlated contingencies, $t(22) = 2.64$, $p < .01$. Subjects in the sober group responded similarly, $t(22) = 1.69$, $p < .05$. Subjects of both these groups used shorter durations for shock intensities 2 through 5 than for intensity 1, all $p < .05$. Under correlated contingencies,

alcohol-subjects used longer shock durations than either the placebo or sober group, $Q = 3.19$ and $Q = 4.16$ ($k = 3$, $df = 66$), $p < .01$, respectively. No significant difference was found between placebo and sober subjects in this condition. Under random contingencies no significant shock duration differences were found between any of the six groups. Mean shock durations for each shock intensity are shown in Figure 1.

Insert Figure 1 about here

Verbal Aggression

No main effects were found on the post-task measure of verbal aggression. Under correlated contingencies, no significant differences were found between alcohol-subjects and either the placebo or sober-subjects. Placebo-subjects, however, scored significantly higher on this measure than the subjects of the sober group in the same condition, $Q = 3.89$, ($k = 3$, $df = 66$), $p < .01$. Under random contingencies no significant differences were found. In both alcohol and placebo groups, subjects receiving random contingencies did not differ from those receiving correlated contingencies. However, the sober-subjects scored significantly higher under random than under correlated contingencies, $t(22) = 5.11$, $p < .005$.

Heart Rate

Analyses of variance for repeated measures revealed that alcohol-subjects' baseline heart rate in both contingency conditions was significantly lower than any of the subsequent readings, all $p < .01$. Only one in-task reading in the placebo group was significantly higher than baseline. No significant differences between heart rate readings were found in the sober group. Comparison between separate readings across drug group revealed no differences under correlated contingencies. Under random contingencies, however, the baseline reading was significantly lower than any subsequent reading, all $p < .01$. Contingency effects across drug groups are shown in Table 3.

Insert Table 3 about here

Correlations between Dependent Measures

Pearson product-moment coefficients were computed between shock intensity and shock duration. A strong correlation between these measures was found in the placebo/correlated contingencies group, $r = .79$, $p < .001$, but not in the placebo/random contingencies group. Strong correlations were also found in the sober/correlated contingencies group, $r = .63$, $p < .001$, and in the sober/random contingencies group, $r = .67$, $p < .001$. Post-task verbal aggression correlated with shock intensity, $r = .59$, $p < .001$, and with shock duration,

r = .75, p < .001, in the placebo/correlated contingencies group, and with shock intensity in the sober/correlated contingencies group, r = .60, p < .001.

Strong positive correlations between scores on the "how drunk" scale and dependent measures were found only in the placebo/correlated contingencies group. The pre-task rating correlated with shock intensity, r = .59, p < .001, and with shock duration, r = .63, p < .001. The post-task rating correlated with shock intensity, r = .70, p < .001, with shock duration, r = .69, p < .001, and with verbal aggression, r = .73, p < .001.

Correlations between Tone Stimulation and Dependent Measures

In order to assess subjects' response patterns to the loudness levels of the tone stimuli, shock intensity and shock duration were correlated with the first tone stimulus in each trial and with the contingency-tone of the preceding trial. As shown in Table 4, the strongest correlations between the first tone and shock intensity were found in the alcohol-group under both contingency conditions.

Insert Table 4 about here

As the effect of the stimulus-tone on the subjects' responses may have been combined with the effect of the contingency-tone, partial correlation coefficients were computed

controlling for the effect of the latter tone. As shown in Table 5, only the inebriated subjects' responses on the measure of shock intensity correlated strongly with the stimulus-tone.

Insert Table 5 about here

A multiple regression analysis was performed in order to determine the combined effect of both tone types on the subjects' responses, as well as the unique contribution of each to that effect. As shown in Table 6, on the measure of shock intensity, a strong stimulus-tone effect was found in the intoxicated groups. In contrast, a strong contingency-tone effect was found in the nonintoxicated groups.

On the measure of shock duration, only in the placebo/correlated contingencies group did the contingency-tone have a statistically significant effect, $F(1,297) = 18.79$, $p < .01$.

Insert Table 6 about here

Psychological Tests

Of all psychological measures administered, only Jackson's Aggression and Defendence scales correlated with

some of the dependent measures. In the alcohol/random contingencies group the Aggression scale score correlated with shock duration, $r = .64$, $p < .001$. In the placebo/correlated contingencies group the Aggression scale score correlated with shock intensity, $r = .65$, $p < .001$, with shock duration, $r = .56$, $p < .001$, and with verbal aggression, $r = .63$, $p < .001$. In the same group, Defendence scores correlated with shock intensity, $r = .67$, $p < .001$, and with verbal aggression, $r = .60$, $p < .01$.

Debriefing

Debriefing discussions revealed that no subject was aware of the deceptions employed in the present experiment. Subjects expressed their hope that they had not hurt their partner excessively. Whereas some subjects in the placebo group said their drinks were somewhat weak, others complimented the research assistant for his drink-mixing skills.

DISCUSSION

The findings of the present study demonstrate that both alcohol and behavior contingencies affect the production of aggressive behavior. The overall finding that subjects who were administered alcohol behaved more aggressively on measures of physical aggression than non-intoxicated subjects supports the conceptualization of alcohol as a facilitator of aggressive behavior. More important, however, is the finding that under

both contingency conditions the intoxicated subjects' behavior was equally aggressive. That is, regardless of the aversive consequences which their aggression held for them, alcohol-subjects overlooked the contingencies and responded forcefully to the arousing tone stimuli. Subjects in the placebo and sober groups seemed to take the contingencies into account and behaved aggressively only upon realizing that they cannot control the aversive consequences; both these groups were significantly more aggressive under random than under correlated contingencies. In fact, this finding is particularly evident on the shock duration measure. When receiving random contingencies the placebo and sober subjects may have been so incensed that differences between them and the inebriated subjects disappeared. Additional data demonstrating the inebriated subjects' unique response pattern are found upon examination of the shock durations used at separate shock intensities: Whereas alcohol-subjects generally applied longer durations to higher shock intensities, placebo and sober subjects displayed a reversed pattern.

These findings suggest that the controlling effect of behavior contingencies diminishes under the effect of alcohol. The question whether the inebriated individual's responses resemble stimulus-bound behavior receives a partial answer from the correlations between the tone stimuli and the shock intensity measure. Although the coefficients obtained were

not decisively high, it appears that alcohol-subjects' responses correlate strongly with the stimulus tone but not with the contingency tone. In contrast, the non-inebriated subjects displayed a strong relationship between their responses and the contingency tone. One could speculate that whereas the intoxicated subjects attended preferentially to the arousing stimulus and responded to it, the other subjects attended to the consequences of their behavior. This notion is supported by the results of the partial correlations and the multiple regression analysis. On the measure of shock intensity, when the effect of the contingency tone was partialled out, only the inebriated subjects' responses seemed strongly affected by the stimulus-tone loudness levels. On the same measure, the unique contributions of each of the tone types to the effect on the subjects' responses strongly suggest that whereas the intoxicated subjects responded primarily to the stimulus-tone, the nonintoxicated subjects' responses were strongly affected by their behavior contingencies.

The slightly different results regarding verbal aggression may have been due to the different nature of the measure. Here, unlike on the measures of physical aggression, the placebo subjects displayed a negative attitude toward their partner regardless of the contingencies to which they were exposed. It is probable that a slight expectancy effect

associated with the consumed beverage may have elevated the placebo-subject's verbal aggression under correlated contingencies. In fact, these subjects scored even higher on this measure than the sober subjects. It is important to note, however, that the expectancy effect in the present study did not appear to be as significant as in the study by Lang et al. (1975).

It appears that heart rate was affected to some extent by both the drug and contingencies manipulations. Conforming to what other studies have shown (Wallgreen & Barry, 1970, p.156), the dose of alcohol administered caused only a slight increase in heart rate. The fact that no difference was found between drug groups, and that a consistently higher in-task heart rate was monitored under random contingencies, may suggest that all subjects were more excited under this type of contingency. Admittedly, the use of heart rate as a measure of arousal is lacking. A multi-function measure (e.g. Galvanic Skin Response, blood-pressure and heart rate) would have been more adequate but beyond the present study's possibilities.

Three other results are deserving of some discussion. First, from the correlation computed between the dependent measures it seems that the subjects of the placebo and sober groups responded more consistently throughout the experiment. Under correlated contingencies, subjects who scored low on

the verbal aggression questionnaire, paired low shock intensities with short shock durations. This may indicate their attempt to control the consequences through the attenuation of their responses. Such a pattern could typify an instrumental response (Buss, 1971). This was not the case with the intoxicated subjects. Another finding of interest in this study are the positive correlations between the "how drunk" scale and the dependent measures found only in the placebo/correlated contingencies group. One way to account for this finding is to assert that simultaneously with experiencing a placebo response attributed to the drink they consumed, these subjects were aware of the control they had over the contingencies. Thus, the strong placebo responders who may have paid less attention to the contingencies, displayed increased aggressive behavior. The fact that no such correlations were found in the placebo/random contingencies group supports this explanation. The third finding of some interest is the fact that no consistent relationship was found between psychological tests and aggressive responses during and after the task. These results underscore the validity of conceptualizing aggressive behavior as a multivariate phenomenon which cannot be clearly linked to specific personality characteristics.

Several reasons can be given for the discrepancy between the findings of the present study and earlier research on

alcohol and aggression (Bennett et al., 1969; Lang et al., 1975). The nature of the arousing stimulus used in the present study was different from the provoking stimuli used in other studies. The present stimulus was not presented as a provocation but could have been construed as such by subjects who needed a justification for behaving aggressively. Moreover, an earlier study (Zeichner & Pihl, 1978) indicated that the aversiveness of the auditory arousing stimulus did not diminish or increase throughout the experiment. This may have been the case with some of the instigating stimuli used in other studies. Whereas this experiment used a relatively high dose of 95% alcohol, previous researchers administered alcoholic beverages of lower dose and higher congener content. This fact may have been responsible for some attenuation of the aggressive response examined in some of the earlier studies. The population used in the present experiment was decisively different from the populations used in other studies. As it is believed (Stonner, 1976) that student populations tend to display biased behaviors, subjects in earlier studies may have attempted to behave in a socially appropriate manner. The attempt to generalize from the present results may be more valid due to the present subjects' diverse backgrounds.

The findings of the present study suggest that the occurrance of aggressive behavior following the ingestion of

alcohol may be due to the individual's inability to process information pertinent to the consequences of his behavior. The ingested alcohol may decrease avoidance behavior usually motivated by fear of aversive stimuli, and may encourage the intoxicated person to take uncalculated risks. It appears that in a state of intoxication the inebriated person may respond aggressively to a stimulus to which he attributes his discomfort. The present experimental paradigm allowed subjects to use their aggressive responses instrumentally by controlling their behavior contingencies. In spite of this fact, it appeared that the inebriated subjects emitted angry aggressive responses whereas the non-intoxicated subjects often exhibited instrumental behavior where possible.

Clearly, questions concerning the inebriated person's ability to process crucial information remain. The interaction of alcohol with situational antecedents and their effect on emotional-mediated behaviors may carry import to the understanding of the alcohol-aggression relationship.

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Footnotes

1. Beer being the popular beverage during the summer in Canada, this limit was set to avoid heavier drinkers. In actuality, however, none of the participating subjects drank on the average in excess of 10 beers per week.

2. Five different loudness levels were used. The tones were set so that one level was six dBA below the subject's aversive threshold whereas the other four were louder in increments of six dBA. This produced one non-aversive and four aversive tone stimuli. An earlier study performed in the laboratory (Zeichner & Pihl, 1978) indicated that all levels were distinguishable from one another.

Table 1

Study Design and Placement of Subjects

	Correlated Contingencies	Random Contingencies
Alcohol	S_1	S_{37}
	.	.
	.	.
	S_{12}	S_{48}
Placebo	S_{13}	S_{49}
	.	.
	.	.
	S_{24}	S_{60}
Sober	S_{25}	S_{61}
	.	.
	.	.
	S_{36}	S_{72}

Table 2

Means and Standard Deviation for Shock Intensity

Drug Condition[a]	Correlated Contingencies	Random Contingencies
Alcohol		
M	4.13	4.34
SD	0.19	0.23
Placebo		
M	1.49	2.46
SD	0.37	0.47
Sober		
M	1.20	2.36
SD	0.16	0.66

[a] *N* = 72

Table 3

Mean Heart Rate for Behavior Contingencies across Drug Groups[a]

Heart Rate Reading	Correlated Contingencies	Random Contingencies	$\underline{t}$[b]
Baseline	77.32	76.40	1.10
T_3[c]	77.13	80.10	3.58*
T_9	78.20	82.2	4.82*
T_{15}	78.70	81.20	3.01*
T_{21}	78.70	82.00	3.98*

[a]Measure expressed in beats per minute.

[b]df = 22

[c]Serial number of trial in task at which reading was taken.

*$p < .005$

Table 4

Correlations between Tone Loudness Levels and Measures of Physical Aggression

Tone and Condition	Shock Intensity (r)	Shock Duration (r)
Stimulus-tone		
Alcohol/correlated contingencies	.44***	.00
Alcohol/random contingencies	.39***	.04
Placebo/correlated contingencies	.08	.04
Placebo/random contingencies	.05	.05
Sober/correlated contingencies	.05	.02
Sober/random contingencies	.14**	.01
Contingency-tone[a]		
Alcohol/correlated contingencies	.00	.08
Alcohol/random contingencies	.06	.04
Placebo/correlated contingencies	.27***	.24***
Placebo/random contingencies	.10*	.09
Sober/correlated contingencies	.13*	.08
Sober/random contingencies	.14*	.02

[a]For these coefficients each response was correlated with the contingency-tone of the preceding trial.

* $p < .05$

** $p < .01$

*** $p < .001$

Table 5

Partial Correlation between Tone Loudness Levels and Measures of Physical Aggression Controlling for Contingency-tone effects.[a]

Condition	Shock Intensity ($r_{si, s.c}$)[b]	Shock Duration ($r_{sd, s.c}$)[c]
Alcohol/Correlated Contingencies	.44*	.02
Alcohol/Random Contingencies	.40*	.04
Placebo/Correlated Contingencies	.08	.04
Placebo/Random Contingencies	.04	.04
Sober/Correlated Contingencies	.03	.01
Sober/Random Contingencies	.16	.01

[a]For these coefficients each response was correlated with the contingency-tone of the preceding trial.

[b]Partial correlation coefficient between shock intensity and stimulus-tone controlling for contingency-tone effect.

[c]Partial correlation coefficient between shock duration and stimulus-tone controlling for contingency-tone effect.

* $p < .001$

Table 6

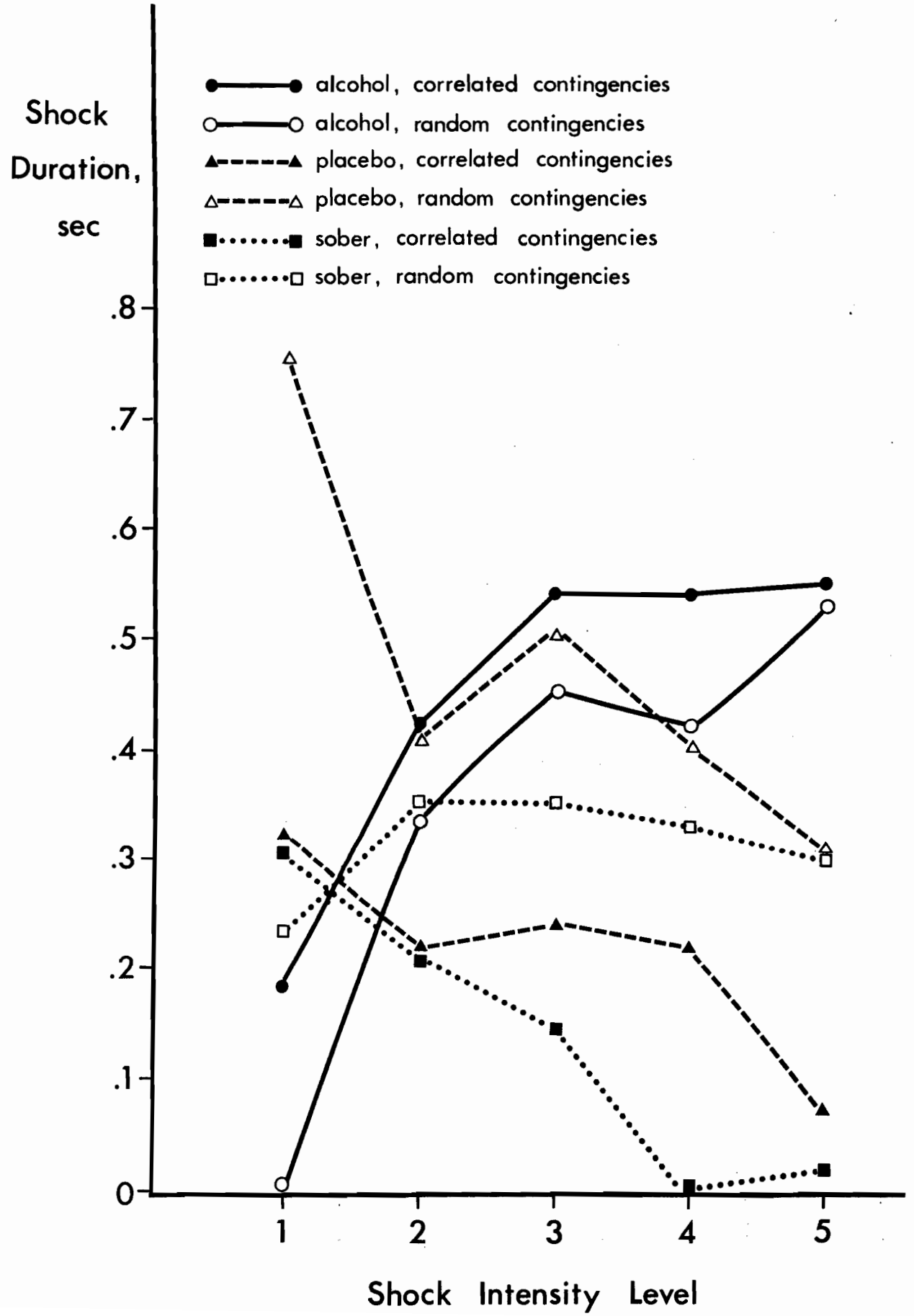
Multiple Regression Analysis for Tone Level Effect on Shock Intensity

Drug and Condition	Tone Type	β[a]	Std. Error β	F(1,297)	R	F(2,297)
Alcohol/Correlated Contingencies	s-t[b]	0.34	0.04	70.60**	.44	35.94**
	c-t[c]	0.01	0.05	0.09		
Alcohol/Random Contingencies	s-t	0.27	0.03	57.84**	.40	29.52**
	c-t	0.08	0.03	4.92*		
Placebo/Correlated Contingencies	s-t	0.04	0.03	1.80	.28	12.99**
	c-t	0.26	0.05	23.76**		
Placebo/Random Contingencies	s-t	0.03	0.05	0.48	.11	1.92
	c-t	0.84	0.05	2.92		
Sober/Correlated Contingencies	s-t	0.01	0.02	0.35	.14	2.83
	c-t	0.12	0.05	4.88*		
Sober/Random Contingencies	s-t	0.12	0.04	7.82**	.02	0.07
	c-t	0.11	0.04	7.43**		

a Regression coefficient
b Stimulus-tone
c Contingency-tone
* $p < .05$
** $p < .01$

Figure Caption

Figure 1. Mean shock duration for five shock intensity levels.


alcohol, correlated contingencies
alcohol, random contingencies
placebo, correlated contingencies
placebo, random contingencies
sober, correlated contingencies
sober, random contingencies
Shock Duration, sec
.8
.7
.6
.5
.4
.3
.2
.1
0
1
2
3
4
5
Shock Intensity Level

INTERMEDIUM

The previous study on the effects of alcohol and behavior contingencies on human aggression has yielded several interesting findings. Subjects who ingested alcohol displayed significantly higher aggressive behavior in comparison to subjects who received placebo beverages or to the sober subjects. This finding conforms to the positive relationship between alcohol and laboratory controlled aggression reported by several earlier studies.

More important, however, the findings of this study suggest that a disruption of information processing occurs following the ingestion of alcohol. The inebriated subjects in this study seemed not to process information concerning their behavior contingencies and aggressed toward their partner inspite of the resulting aversive consequences. Whereas these subjects displayed an angry-aggressive response pattern, the nonintoxicated subjects seemed to respond instrumentally. That is, they differentiated between the two types of contingency conditions and seemingly attempted to control the aversive consequences. Moreover, whereas the noninebriated subjects' behavior seemed to have been controlled by the contingencies, the inebriated subjects' responses resembled stimulus-bound behavior apparently being controlled by the aversive stimulation per se.

These findings raise some intriguing questions. As the inebriated subjects seemed to lose their ability to take into account the consequences of the aggressive interaction, will alcohol also modify their ability to process information concerning antecedants of a given situation? Are intoxicated individuals able to correctly perceive the intent with which they are stimulated by another person?

The following study attempts to examine the effects of alcohol and instigator-intent on the production of human aggression. Seventy-two different subjects were used. The basic aggression procedure used is the same as in the previous study with two important modifications. To simulate as closely as possible an aggressive interaction between drinkers, where malicious intent can be attributed to the instigator, the intent manipulation in this study was undertaken after the drinks have been administered. In addition, two intent-type conditions replaced the two contingency conditions.

This study also attempts to assess once more the angry versus instrumental response patterns of the subjects, as well as to examine whether the intoxicated subjects display response patterns resembling stimulus-bound behavior in the context of different instigator-intent types.

Due to the similar nature of the two studies several portions of the method section are redundant.

EFFECTS OF ALCOHOL AND INSTIGATOR-INTENT ON HUMAN AGGRESSION

ABSTRACT

The purpose of this study was to determine the mediating effects of alcohol and instigator-intent on aggression in male social drinkers. Seventy-two subjects were randomly assigned to one of six groups in a 3 x 2 factorial design. To control for alcohol and expectation effects, one third of the subjects received alcoholic beverages, one third received placebo drinks and another third was not administered any beverages. Aggression was assessed by the intensity and duration of shocks administered to a bogus partner in a modification of the Buss aggression procedure. Half the subjects were exposed to aversive stimulation administered with a neutral intent, and half were aversively stimulated with a malicious intent. The intoxicated subjects were significantly more aggressive than the nonintoxicated subjects ($p < .01$). Moreover, the inebriated subjects were significantly less affected by the intent manipulation than the noninebriated subjects. The latter subjects displayed a clear differential response pattern controlled by the intent of the instigator. These findings are attributed to the disrupting effect of alcohol on information processing and the mediating effect of instigator-intent on the noninebriated individual's aggressive behavior.

INTRODUCTION

The role of alcohol in the elicitation of human aggression has become an issue of increasing concern. Numerous instances of assaultive behavior (Mayfield, 1976), homicide (Virkkunen, 1974), suicide (Hagnell, Nyman, & Tunvig, 1973) and violent and nonviolent crimes (Haberman & Baden, 1974; Lindelius & Salum, 1976) have been reported to occur in conjunction with the ingestion of alcohol by the aggressor and often by the victim as well. Such reports have indirectly contributed to the conceptualization of alcohol as a chemical agent which increases approach behaviors. Some of these behaviors result in a variety of aggressive responses.

Studies have generally failed to identify consistent personality characteristics of the excessive drinker (Kohn, 1974; Miller, 1976), as well as of the aggressive personality (Knott, Lasater, & Shuman, 1974; Larwood & LeGault, 1975). Moreover, researchers have been unsuccessful in linking drinkers and aggressors in terms of personality traits (Kristianson, 1974). It appears that a situational perspective may be able to provide greater understanding of the relationship between alcohol and aggression.

Most commonly, as demonstrated by studies on aggression in the laboratory, aggression against others by definition involves two parties: the instigator and the responder. The instigator may frustrate (Buss, 1963), threaten (Geen, Stonner, & Kelley, 1974), attack (Borden, Bowen, & Taylor, 1971)

or insult another person (Hartman, 1969). The latter may, in response, retaliate in kind (Pisano & Taylor, 1971) or resort to other measures (Berkowitz & Geen, 1967). Unless the responder chooses to remain passive or to negotiate in a nonaggressive manner, he will engage in aggressive behavior triggered by the instigator.

The aggressive exchange may be comprised of two kinds of responses (Buss, 1971) transmitted in either direction. The angry type of aggressive responses are triggered by anger inducers such as insult, attack, annoyance, etc., aimed at inflicting discomfort on another person. Instrumental aggression is seen as a response triggered by competition or a sought after reinforcer in the possession of another person (Baron, 1974; Buss, 1966b). This response is aimed to achieve acquisition of such a reinforcer. One of the factors determining whether or not the responder will engage in aggressive behavior is his perception of the instigator's intent.

The relationship between instigator-intent and aggressive behavior is best examined within the framework of attribution theory (Jones & Davis, 1965; Kelley, 1967). Within this model, the attributions a person makes about the source of his arousal may influence his emotional state (Schachter & Singer, 1962). Arousal from a variety of independent sources has been shown to be misattributed by subjects causing an intensification of previously established emotional states

(Zillmann, 1971; Zillman & Bryant, 1974). Also, attribution of discomfort to an instigator caused subjects to label the arousal they experienced as anger (Baron & Bell, 1976). Furthermore, using an attributional model, Tedeschi, Smith and Brown (1974) suggested that the label aggression may be applied by an observer perceiving intentional coercion used by an instigator. Thus, an individual who is being aversively aroused by an instigator and who labels his emotional state anger and the instigator's behavior aggression, may engage in aggressive behavior directed toward the instigator.

Studies examining how the responder's aggression is affected by his perception of the instigator found increased aggression directed toward individuals seen as aggressive instigators (Borden et al., 1971). Moreover, aggression was found to be an additive function of actual punishment and perception of the instigator's intended level of punishment (Taylor, 1967). When the aggressive intentions of the instigator were communicated to the responder, the latter's aggression was found to be strongly affected by that information (Taylor & Pisano, 1971). Interestingly, symbolic attack, i.e., the perceived intent of the attacker, appeared to be a more potent instigator of aggression than physical aggression per se (Greenwell & Dengerink, 1973).

To better understand what affects aggressive behavior in interaction with ingested alcohol, one should examine the

inebriated individual's ability to process information related to the instigator's behavior in a given situation. If information concerning the instigator's intent is made available to the drinking individual, does he take it into account when deciding which response to emit? Studies on alcohol-mediated physical aggression in the laboratory present inconsistent findings. Using modifications of the basic paradigm forwarded by Buss (1961), in which physical aggression is defined in terms of shock settings on an aggression machine, some studies have found increased aggression following the ingestion of alcohol (Shuntich & Taylor, 1972; Taylor & Gammon, 1975). In contrast, other studies have shown that alcohol did not lead to aggression (Bennett, Buss, & Carpenter, 1969) and that an increase in aggressive behavior occurred regardless of the alcohol content of the administered beverages (Lang, Goeckner, Adesso, & Marlatt, 1975).

Two recent studies examining the effects of alcohol on physical aggression seem to provide some clarifying data. In one (Taylor, Schmutte, & Leonard, 1977), frustration per se was found to be a weak determinant of aggression relative to situational antecedents like physical attack, social pressure or alcohol. In another study (Zeichner & Pihl, Note 1), inebriated subjects were found to be unable to process information concerning the consequences of their aggressive behavior. These authors suggested that alcohol

intoxication seems to effect a shift in the individual's behavior control. Situational constraints such as consequences were found to be ineffective determinants of behavior; these inebriated subjects displayed stimulus-bound responses.

The present study attempts to elucidate the question of whether ingested alcohol interferes with the intoxicated person's ability to use information concerning the instigator's intent in the modification of the former's aggressive behavior. The basic paradigm forwarded by Buss (1961, p.47) is employed. The subject's preparedness to administer painful electric shocks to another person, while shocks of negligible intensity are available, proves to be one of the laboratory controlled measures of human physical aggression to receive naturalistic validation (Wolfe & Baron, 1971). To control for effects of expectancies associated with ingestion of alcohol (Lang et al., 1975), placebo and sober conditions are added in the present study. As general arousal has been associated with the occurrence of aggression (Donnerstein & Wilson, 1976; Geen & O'Neal, 1969) an aversive tone is used in the present study as the instigating stimulus. Finally, alcohol dose and drinking pace are controlled in order to achieve blood-alcohol concentrations conforming with those at which human subjects were shown to exhibit increased approach behaviors (Ekman, Frankenhaeuser, Goldberg, Hagdahl,

& Myrsten, 1964) and disrupted cognitive performance (Jones & Vega, 1973).

METHOD

Subjects

Seventy-two male social drinkers, aged 18 to 35 (M age = 23.2 yr.), were recruited as paid subjects by means of local newspaper, radio and television advertisement. Initial telephone-screening followed by a personal interview and completion of a drinking-history and personal data questionnaire, ascertained that participating subjects met the following requirements: (a) They did not have an alcoholism problem in the past or present, (b) were not treated or arrested in the past in connection with excessive drinking (c) did not suffer any hearing impairment, (d) had no previous familiarity with psychological experimentation, and (e) drank regularly but not in excess of the equivalent alcohol content of eight beers per day.[1] These screening measures were instituted in order to increase sample homogeneity. The use of experienced drinkers ensured similar expectancies related to alcohol consumption. As a high alcohol tolerance could attenuate the effects of alcohol in the laboratory, heavy drinkers were avoided. Each of the participating subjects was asked to abstain from food and any drugs for a period of four hours prior to the experimental session.

Experimental Design

Two independent variables were manipulated in a randomized, double-blind 3 x 2 factorial design with 12 subjects per cell (Table 1): (a) Drug-subjects received either alcoholic beverages, placebo beverages or did not receive any beverage prior to the experimental session, and (b) instigator intent - subjects were aversively stimulated with either neutral or malicious intent.

Insert Table 1 about here

Procedure

Upon arrival for the experimental session each subject was greeted by the experimenter and weighed. He was then asked whether he had refrained from eating and drinking any alcoholic beverages for at least four hours prior to his arrival. A breath sample was then analyzed by means of a Mate-A glass-tube analyzer to ascertain that the blood-alcohol concentration (BAC) of the subject was zero.

Assessment of aversive tone threshold. Each subject's aversive tone threshold was determined in the following manner: Segments of a continuous pure tone of 3000 Hz in discrete trials at different levels of loudness ranging from 60 dBA to 109 dBA sound pressure level were administered through headphones. Subjects were instructed to terminate the tone presentation as soon as it became aversive. Termination of the tone was effected by touching a response key of a Lafayette Multi-choice Reaction Timer. Criterion was

set at five sec response latency following onset of tone. When the tone was terminated in less than five sec, the loudness level of the subsequent presentation was diminished by one dBA. When the tone was terminated later than five sec, the subsequent presentation's loudness level was augmented by one dBA. The lowest loudness level terminated in three successive trials under the latency criterion was taken as the subject's aversive tone threshold.

Following the tone procedure each subject completed a partial version of the Personality Research Form (Jackson, 1967) and the Locus of Control questionnaire (Rotter, 1966). During this stage an unobtrusive baseline heart rate reading was taken by means of a Grass photo-electric transducer Model PTTI6 and recorded by a Grass Model 7P4 Tachograph.

Drug conditions. After the completion of the questionnaires subjects were assigned to one of three drug conditions: (a) Alcohol condition - subjects were administered 1.32 ml/kg body weight of 95% USP (United States Pharmacopoeia) alcohol in a 1:5 (alcohol:orange juice) solution divided into three drinks of equal quantity; (b) Placebo condition - subjects were administered three orange juice drinks of equal proportions as in the alcohol condition, the alcohol part being replaced by juice and two cc of 95% USP alcohol layered on the drink's surface. Each glass was encased in a terry-cloth holder sprayed with 95% alcohol; (c) Sober condition -

subjects were not administered any beverage prior to the experiment.

Drinks (or a waiting period for subjects in the sober condition) were consumed in a carpeted, softly lit room, while the subject was seated in a cushioned chair listening to music of his choice. The drinks were prepared by a research assistant. In order to standardize drinking speed and duration, the subject was asked to finish his first drink in six minutes, his second drink in six minutes and his third in eight minutes. A waiting period of 20 minutes followed to allow the alcohol drinking subject to reach an elevated BAC level, but concurrently to provide sufficient time to complete the experiment before reaching BAC peak (Jones & Vega, 1973). At the end of this period, 40 minutes after commencement of drinking, a BAC reading was taken by means of a Smith & Wesson Breathalyzer Model 6000. Finally, the subject was asked to compare his present level of intoxication to the highest level of intoxication he had ever experienced on a seven-point Likert-type "how drunk" rating scale.

Task and instruction. Immediately following the drinking phase the task instructions were presented by the experimenter. The experimental paradigm employed used a modified version of the Buss (1961) aggression machine. The task was presented to the subject as a reaction-time/pain-

perception task in which the subject was to be tested for reaction-time while his "partner" in the adjoining room was to be tested for perception of pain. The bogus partner was an operant conditioning apparatus. The subject was told that both behaviors would be examined under the influence of alcohol in the following manner. Shortly after the appearance of a light on the subject's console the partner would administer a tone through the subject's headphones.[2] After a five sec exposure to the tone, a second light would indicate to the subject to press, as fast as he could, one of the five buttons on his console. Pressing any of these buttons would terminate the tone and at the same time deliver an electric shock to the partner. This constituted one trial. It was stressed that whereas button number 1 delivered a hardly noticeable shock, "just a tickle", buttons 2 through 5 activated increasingly painful shock levels. The subject was led to believe that after receiving the shock the partner would rate the level of pain he experienced on a chart.

Each subject participated in 25 consecutive trials interspaced by 15 sec intervals to ensure clear differentiation between trials. The five tone levels used were meant to symbolically compare to the five shock intensities the subject could choose to administer.

Intent conditions. All subjects received the tone's loudness levels in a random order, each level occurring five

times. The subjects were divided into two groups, each group receiving stimulation delivered with a different intent: (a) Neutral intent - The subject was told his partner was to administer the tones according to a predetermined list prepared by the experimenter. Thus, the partner was described as having no control over the tones he delivered; (b) Malicious intent - The subject was told that his partner was free to choose any tone level he wished. Thus, the partner in this condition was described as having full control over the tones he administered.

The tone used in the experiment was pre-recorded on tape from a Tektronix FG502 Function Generator. This tone was selected to minimize habituation due to temporary auditory threshold shift (Stockinger, Cooper, Meisser, & Jones, 1972).

Video deception. As the task instructions were given by the experimenter to the subject, the latter could observe himself and the experimenter on a 15 cm television monitor which was placed next to the shock console. This was done by means of a closed-circuit television system. Following the instructions the subject was advised that he would be able to observe on the monitor his partner being given his task instructions. As the experimenter left the subject's

room one of two pre-recorded video tapes was replayed on the monitor: (a) Neutral intent tape - this video tape showed a male (amateur actor) waiting in a room as the experimenter entered. This actor was given a sample of the tone to be administered, a list of predetermined tone levels, examples of shock intensities 1 and 2, and a chart to record his pain evaluation. The experimenter emphasized that no deviation from the prepared list is allowed. Finally, the experimenter turned to the camera and asked the subject to indicate that all was understood by pressing shock button number 1. This was intended to serve as a validation of the deception; (b) Malicious intent tape - this tape differed from the previous one in that no prepared tone list was used and the actor was explicitly given free choice in the tone levels he was to administer.

At this time the subject was asked to prepare for the task. In order to ascertain that the subject remembered, understood and believed the video deception, six questions pertaining to his task were asked. Once correctly answered the task was commenced. Throughout the task period the subject was alone in the room. The sequence of the task was fully controlled by Foringer operant conditioning equipment. Four additional unobtrusive heart rate readings were taken throughout the task period. The subject's responses were monitored and recorded in an adjacent control room. After the task was completed the experimenter reentered the room.

A second BAC reading was taken and the self-report "how drunk" scale was administered. The subject was then given a short questionnaire assessing his attitude toward the partner. Following this, a debriefing discussion was held and the subject was thanked and paid; those in the alcohol condition were driven home.

Measures of aggression. Two measures of physical aggression and one measure of verbal aggression were taken: (a) The average shock intensity the subject was "delivering" to his partner, (b) the duration of time the subject depressed each shock button, and (c) the subject's score on the post-task attitude questionnaire.

Ethical considerations. For ethical reasons due to the unusual demands put on the subject in this experiment the following steps were taken: (a) All subjects were "pure" volunteers and not coerced in any way to participate in the experiment, (b) the possibility of pressing only shock button number 1 (hardly noticeable shock) was clearly stated, (c) during the debriefing discussion the subject was assured that he did not shock anyone and that his behavior was perfectly normal and comparable to other subjects' behavior in the same condition, and (d) several weeks following the experiment each subject received a letter explaining his role in the study, outlining the findings and reiterating the statement made in (c).

RESULTS

As subjects were randomly assigned to each of the six experimental groups, analyses of variance were performed to assess variance differences between groups on the following variables: Age, years of formal education, socio-economic status (after Blishen, 1967), usual quantity of alcohol consumed, score on the Locus of Control questionnaire and the personality scales of aggression, succorance, defendence, lying and social desirability. No significant differences between groups were found on any of these variables.

Alcohol Manipulation

BAC level. All subjects in the alcohol group achieved BAC levels well within the "social drinking" experience. BAC levels ranged between .075% and .120% ($\bar{M}_{BAC}$ = .085%) on the first reading, and between .080% and .120% ($\bar{M}_{BAC}$ = .089%) on the second reading. No identifiable BAC levels were found in the subjects of the placebo group.

"How drunk" scale. Subjects in the alcohol group indicated an average "drunk" score of 3.16 on the pre-task rating and 3.03 on the post-task rating. Placebo subjects averaged 2.87 on the first rating and 2.75 on the second rating. Two-tailed t tests performed to compare the means of the alcohol and placebo groups found the differences to be equally nonsignificant on the first, $t(46) = 0.74$, as

well as on the second rating, $t(46) = 0.79$.

Analyses of variance for repeated measures were performed for comparing drug groups and intent conditions. Significant results were further analyzed by the Tukey Honestly Significant Difference test (Tukey, 1962).

Physical Aggression

Shock intensity. The shock intensity score for each subject was obtained by computing an average of all shock choices each multiplied by its respective level. Across intent conditions alcohol subjects administered significantly higher shocks than either the placebo subjects, $Q = 36.00$ ($k = 3$, $df = 66$), $p < .01$, or the sober subjects, $Q = 41.53$ ($k = 3$, $df = 66$), $p < .01$. Placebo subjects chose higher shocks than the sober subjects, $Q = 5.63$ ($k = 3$, $df = 66$), $p < .01$. When exposed to neutral intent, alcohol subjects delivered higher shocks than either the placebo subjects, $Q = 27.09$ ($k = 3$, $df = 66$), $p < .01$, or the sober subjects, $Q = 32.69$ ($k = 3$, $df = 66$), $p < .01$. Placebo subjects chose higher shocks than sober subjects, $Q = 5.60$ ($k = 3$, $df = 66$), $p < .01$. When exposed to malicious intent, alcohol subjects delivered higher shocks than either the placebo subjects, $Q = 23.89$ ($k = 3$, $df = 66$), $p < .01$, or the sober subjects, $Q = 26.11$ ($k = 3$, $df = 66$), $p < .01$. No significant difference was found between placebo and sober subjects in this condition.

Intent effects within each drug condition were compared by two-tailed t tests. Alcohol, placebo, and sober subjects, all chose higher shock intensities under malicious intent than under neutral intent, $t(22) = 3.40$, $p < .005$; $t(22) = 5.66$, $p < .005$; $t(22) = 8.08$, $p < .005$, respectively. The computation of three single-degree of freedom contrasts revealed that this difference was significantly larger in the sober than in the alcohol group, $t(66) = 3.27$, $p < .002$. Means and standard deviations for this measure are shown in Table 2.

Insert Table 2 about here

Shock duration. No significant difference was found between neutral and malicious intent in the alcohol group. Moreover, the duration of shock intensities 2 through 5 were significantly longer than the duration used for intensity 1 (all $p < .01$). Subjects in the placebo group applied shocks of significantly longer duration under malicious intent than under neutral intent, $t(22) = 2.40$, $p < .01$. Subjects in the sober group behaved similarly, $t(22) = 1.59$, $p < .05$. Subjects in both these groups used significantly shorter durations for shock intensities 2 through 5 than for intensity 1 (all $p < .05$). Under the neutral intent condition alcohol subjects applied longer shock durations than either the

placebo, $Q = 3.35$ ($k = 3$, $df = 66$), $p < .05$, or the sober subjects, $Q = 3.85$ ($k = 3$, $df = 66$), $p < .01$. No significant difference was found between placebo and sober subject in this condition. Under the malicious intent condition no significant shock duration differences were found between any of the six groups. Mean shock durations for each shock intensity are shown in Figure 1.

Insert Figure 1 about here

Verbal Aggression

Although alcohol subjects scored higher on the post-task measure of verbal aggression than the two other groups, this difference did not reach statistical significance. No significant interactions were found although across drug groups, subjects exposed to malicious intent were significantly more aggressive than subjects exposed to neutral intent, $F(2,66) = 5.49$, $p < .02$.

Heart Rate

Analyses of variance for repeated measures revealed that alcohol and placebo subjects' baseline heart rate in both intent conditions was significantly lower than any of the subsequent readings, all $p < .01$. In the sober group only one in-task reading was significantly higher than baseline. No other significant interactions were found. Mean

heart rate on separate readings for the three drug groups across intent conditions are shown in Table 3.

Insert Table 3 about here

Correlations between Dependent Measures

Pearson product-moment coefficients were computed between dependent measures. In the alcohol/malicious intent group, a strong negative correlation was found between shock intensity and shock duration, $r = -.74$, $p < .001$. Similar results were found in the alcohol/neutral intent group, $r = -.68$, $p < .01$. However, a strong positive correlation was found between shock intensity and shock duration in the sober/neutral intent group as well as in the sober/malicious intent group, $r = .72$, $p < .001$, $r = .63$, $p < .001$, respectively.

Strong negative correlations between verbal aggression and the "how drunk" scores of the placebo/malicious intent subjects were found for both pre- and post-task ratings, $r = -.69$, $p < .001$, $r = -.55$, $p < .001$, respectively.

Correlations between Tone Stimulation and Dependent Measures

In order to assess the relationship between tone loudness and the subjects' response pattern, shock intensity and shock duration were correlated with the tone stimulus. As shown in Table 4, a strong relationship was found between tone and shock intensity in both alcohol and sober/malicious intent groups.

Insert Table 4 about here

Psychological Tests

Of all psychological tests administered only Jackson's aggression and defendence scales correlated with shock duration. In the placebo/malicious intent group the aggression scale correlated strongly with shock duration, $r = .68$, $p < .001$. In the sober/neutral intent group, both the aggression and defendence scales correlated with this dependent measure, $r = .67$, $p < .001$, $r = .60$, $p < .001$, respectively. In the sober/malicious intent group aggression correlated negatively with shock duration, $r = 0.60$, $p < .001$.

Debriefing

Debriefing discussions revealed that no subjects were aware of the deceptions or the purpose of the experiment. Subjects expressed their hope that they had not hurt their partner excessively. Whereas some subjects in the placebo group said their drinks were somewhat weak, others complimented the research assistant for his drink-mixing skills.

DISCUSSION

The findings of the present study demonstrate the importance of alcohol and instigator-intent in the determination of aggressive behavior. The fact that subjects who

were administered alcohol behaved more aggressively than either the placebo or sober subjects in choosing shock intensities implicates alcohol as a facilitator of aggressive behavior. Additional data demonstrating the unique aggressive response pattern of the inebriated subjects is found upon examination of shock duration at separate shock intensities. Whereas alcohol subjects applied longer durations to higher shock intensities, placebo and sober subjects displayed a reversed pattern.

More important, however, is the finding that under both intent conditions the intoxicated subjects were equally aggressive on the measure of shock duration. That is, regardless of whether the instigator's intent was neutral or malicious, alcohol subjects responded to him forcefully, with relatively long shock durations. It is important to note, however, that on the shock intensity measure inebriated subjects were more aggressive when exposed to malicious intent than in the neutral intent condition. One way to account for this finding is to assert that the intoxicated individuals were somewhat aware of the instigator's neutral intent. As a result, they may have chosen the shock intensities according to an "eye for an eye" response strategy which ultimately resulted in a lower overall aggression score. Simultaneously, however, these subjects may have expressed their anger by the use of relatively long shock durations.

In contrast, under the malicious intent condition, inebriated subjects engaged in "all out" retaliation.

Consistent findings concerning the effect of the instigator's intent were found in both the placebo and sober groups. On both measures of physical aggression these groups were more aggressive when exposed to malicious intent than when under neutral intent. Nevertheless, the differential responses of the sober subjects to different instigator-intent proved to be significantly greater than the differential response displayed by the inebriated subjects.

The findings discussed so far suggest that under the influence of alcohol, subjects tend to differentiate less between types of intent when responding aggressively than do non-intoxicated individuals. The correlations between the instigating tone and the shock intensity responses indicate that under both types of intent, the inebriated subjects' responses were closely affected by the instigating tone levels. Furthermore, these strong correlations were found for both alcohol groups but only in the malicious intent condition for the placebo and sober groups. This may be indicative of an instrumental response strategy employed by nonintoxicated subjects when exposed to neutral intent. This may have been an attempt to persuade the instigator to lower his tone levels. Accordingly, there was no clear relationship between the instigator's predetermined stimuli and these subjects' responses.

Alcohol did not seem to interact with intent conditions or to have an overall significant effect on verbal aggression. However, across drug groups the instigator's intent manipulation resulted in significantly higher verbal aggression scores in the malicious intent condition than in the neutral intent condition. One can speculate that verbal aggression is more amenable to the effects of situational antecedents than is physical aggression.

An interesting finding concerning the effect of expectancies associated with the consumption of alcoholic beverages was demonstrated by comparing the placebo to the sober subjects on the shock intensity measure. When exposed to neutral intent, placebo subjects were significantly more aggressive than sober subjects. This may be due to the social permissiveness associated with social drinking discussed in earlier studies (Sobell & Sobell, 1975). The fact that this difference disappeared when instigation was applied with malicious intent may have been due to the justification both groups had for responding aggressively. It appears that subjects' heart rate was also affected by their expectancies associated with the drinks they consumed. The significant interaction between the separate readings and the drug conditions indicated a continuous increase in heart rate throughout the task for the alcohol and placebo subjects. Since only small increases in heart rate have been commonly associated with alcohol ingestion (Wallgreen &

Barry, 1970, p.156), the thought of administering shocks while being drunk may have caused the observed increase in heart rate. However, the use of heart rate as a measure of arousal is lacking. A multi-function index (e.g. Galvanic Skin Response, blood pressure and heart rate) would have been more adequate but beyond the present study's possibilities.

Three other results are deserving of some discussion. First, the correlations computed between the dependent measures indicate that only the sober subjects paired shock intensities with comparable lengths of shock durations. In contrast, a different response pattern was displayed by the inebriated subjects as both measures of physical aggression were negatively correlated with one another. Since the sober subjects were most aware of the instigator's intent, the difference between their and the inebriated subjects' response patterns may be due to the clear retaliatory message the sober subjects wanted to convey to the malicious instigator. Another finding of some interest in this study are the negative correlations between verbal aggression and the "how drunk" scores of the placebo/malicious intent subjects. Whereas no definite explanation of this finding is readily available, it is possible that the strong placebo responder may have felt that he shocked his instigator excessively and thus decided to present more positive attitudes toward

him on the attitude questionnaire. The fact that no such correlations were found in the placebo/neutral intent group seems to support this explanation. The third finding of some interest is the fact that the relationship between some personality measures and shock duration was found only in the nonintoxicated groups. Whereas alcohol could be implicated as a situational modifier of traits as reflected by the administered personality measures, the contradicting directionalities of these results prevent any firm conclusion.

The findings of the present study conform to the conclusion of a previous study carried out in this laboratory (Zeichner & Pihl, Note 1). Ingested alcohol seems to affect the intoxicated person's information processing ability. In the present study, information concerning the antecedents of a given situation (i.e. instigator-intent) had little differential effect on the inebriated subjects' aggressive responses. These subjects' response strategy resembles the angry-aggressive responses displayed by the intoxicated subjects in the Zeichner & Pihl study. In contrast, individuals who did not ingest alcohol in the present study, appeared better able to differentiate between types of intent, and to attribute their discomfort to their instigator's malice. The inebriated subjects' differential response on the shock intensity measure could have been due to their partial awareness of the instigator's intent. However, noting

the relatively small differences between the intoxicated subjects' behavior under the different intent conditions, one can hypothesize that a slightly larger alcohol dose may produce a total lack of intent differentiation leading to indiscriminant aggressive responsiveness.

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Footnotes

1. Beer being the popular beverage during the summer in Canada, this limit was set to avoid heavier drinkers. In actuality, however, none of the participating subjects drank on the average in excess of 10 beers per week.

2. Five different loudness levels were used. The tones were set so that one level was six dBA below the subject's aversive threshold whereas the other four were louder in increments of six dBA. This produced one nonaversive and four aversive tone stimuli. An earlier study in the laboratory (Zeichner & Pihl, 1978) indicated that all levels were distinguishable from one another.

Table 1

Study Design and Placement of Subjects

	Neutral Intent	Malicious Intent
Alcohol	S_1	S_{37}
	.	.
	.	.
	S_{12}	S_{48}
Placebo	S_{13}	S_{49}
	.	.
	.	.
	S_{24}	S_{60}
Sober	S_{25}	S_{61}
	.	.
	.	.
	S_{36}	S_{72}

Table 2

Means and Standard Deviations for Shock Intensity

Drug Condition[a]	Neutral Intent	Malicious Intent
Alcohol		
M	3.99	4.39
SD	0.13	0.18
Placebo		
M	1.67	2.35
SD	0.45	0.36
Sober		
M	1.19	2.16
SD	0.22	0.30

[a] *N* = 72

Table 3

Mean Heart Rate for Drug Groups across Intent Conditions[a]

Heart Rate Reading	Alcohol	Placebo	Sober
Baseline	78.35	80.92	80.05
T_3[b]	85.37	88.16	83.87
T_9	87.53	88.00	81.91
T_{15}	87.42	88.60	80.45
T_{21}	90.00	88.30	80.45

[a] Measure expressed in beats per minute.

[b] Serial number of trial at which reading was taken.

Table 4

Correlations between Tone Loudness and Measures of Physical Aggression

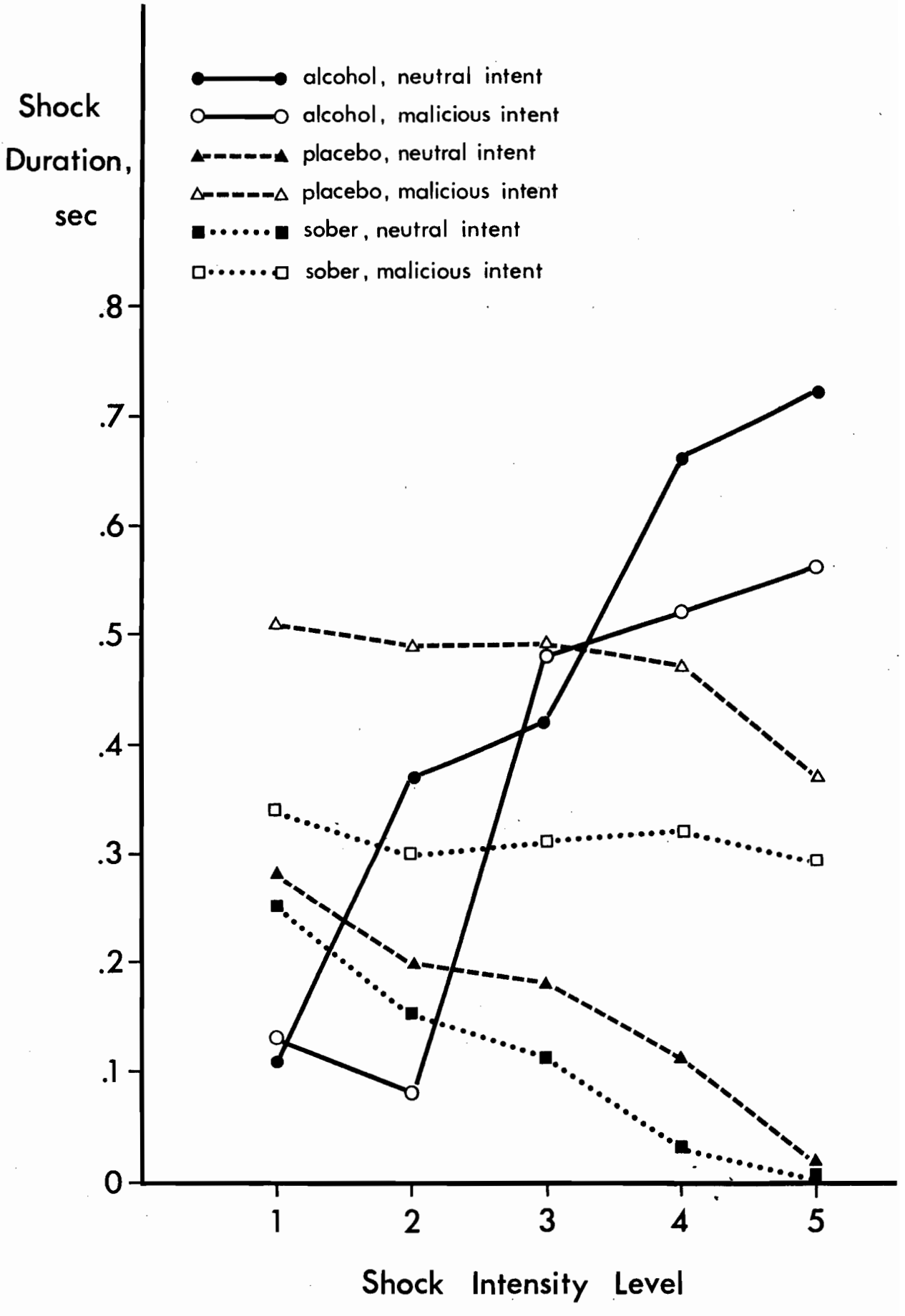
Drug/intent Condition	Shock Intensity (r)	Shock Duration (r)
Alcohol/neutral intent	.62**	.19**
Alcohol/malicious intent	.33**	.12*
Placebo/neutral intent	.07	.02
Placebo/malicious intent	.37**	.01
Sober/neutral intent	.07	.00
Sober/malicious intent	.47**	.04

* $p < .05$

** $p < .001$

Figure Caption

Figure 1. Mean shock duration for five shock intensity levels.


alcohol, neutral intent
alcohol, malicious intent
placebo, neutral intent
placebo, malicious intent
sober, neutral intent
sober, malicious intent
Shock Duration, sec
.8
.7
.6
.5
.4
.3
.2
.1
0
1
2
3
4
5
Shock Intensity Level

CONCLUSION

The present two studies confirm the existence of a positive relationship between alcohol ingestion and human aggression. When exposed to certain situational variables, subjects who were administered alcohol displayed a higher level of aggressive behavior in comparison to individuals who ingested a placebo beverage or those who were sober controls. This general finding suggests that given a certain social context, the presence of alcohol may increase the probability for an aggressive interpersonal exchange to occur. This finding is in accordance with previous studies on alcohol-mediated aggression (Shuntich & Taylor, 1972; Taylor & Gammon, 1975).

Several issues of general interest are implicated by this finding. Firstly, the aggressive responses displayed by any of the subjects in the present studies did not appear related to any specific personality characteristic as measured by the various personality scales administered. This finding underscores the difficulty of identifying the aggressive drinker in terms of specific personality traits (Ruff et al., 1975). Secondly, due to the nature of the present population, the findings of the present studies offer external validation of earlier studies in this area. It has been suggested (Rosenthal, 1965; Stonner, 1976) that the overused student population may display behaviors in the

laboratory which are specific to that population's nature and knowledge of aggression research. The present studies, in using subjects who were recruited from a diverse background, and who were unfamiliar with the field of aggression research and other psychological experimentation, suggest that the observed behavior may have well been a valid representation of typical social behavior. Thirdly, different affective consequences of alcohol have been linked to social or solitary drinking (Pliner & Cappell, 1974). The present studies confirm that inspite of the fact that subjects in the alcohol condition drank their beverages alone, the ensuing affective state facilitated the occurrence of aggressive behavior.

The present studies attempted to elucidate two questions: (a) Does ingested alcohol modify a person's ability to use information concerning his behavior contingencies, and thus affect his aggressive behavior?, and (b) does ingested alcohol modify a person's ability to consider the instigator's intent in controlling his aggressive response?

The elucidation of these questions was facilitated by the following manipulations. Under the present particular experimental conditions, the alcohol dose that was administered gave rise to BAC levels ranging between .075% and .127%. More important, however, was the fact that the alcohol elimination phase had not begun prior to the completion of the task. This allows one to assume that the effect of

shock intensities. The nonintoxicated subjects seemed to use their responses instrumentally, evidently in an attempt to persuade their partner to lower the aversiveness of the behavior contingencies. This was done by applying short shock durations to higher shock intensities and vice versa. Moreover, an attempt to control the contingencies through the attenuation of their responses was specifically displayed by nonintoxicated subjects who scored low on the verbal aggression measure and paired short shock durations with low shock intensity.

Also seen in the first study, the correlations between the tone stimulations and the aggressive responses indicated that the inebriated subjects' behavior was less controlled by contingencies in comparison to the noninebriated subjects. In the inebriated subjects, significant positive correlations were found between the aggressive responses and the stimulus-tone. In contrast, in the noninebriated subjects, similar correlations were found between the aggressive responses and the contingency-tone stimulation. These findings were strongly supported by the regression analysis of the shock intensity measure. Thus, it appears that the intoxicated person's responses resemble stimulus-bound behavior, that is, they are controlled by the arousing aversive stimulation.

The expectancy effect observed in an earlier study (Lang et al., 1975) did not interact significantly with the

aggressive responses observed in the present study. However, it seems that expectancies associated with the effect of alcohol were responsible for some of the placebo subjects' behavior. On the measure of verbal aggression, placebo subjects exposed to either of the contingency conditions were equally aggressive. As discussed elsewhere (Sobell & Sobell, 1975), experiencing a state of "drunkenness" may have encouraged these subjects to abuse their partner verbally. One ought to remember, however, that these subjects' response pattern may have been influenced by the fact that at the time this measure was taken, no more aversive contingencies were imminent. The interaction between this placebo response and the subjects' aggressive behavior was particularly evident in the placebo/correlated contingencies group.

The present study's findings offer some suggestive evidence concerning the unique style in which the inebriated individual may treat the antecedents to his aggressive response. Also in this study, the instigating stimulation was presented to the subjects as predetermined by the experimenter, whereas the contingency tones were described as being under the partner's control. Thus, although the stimulus tones did not have a provocative nature they may have been construed as such by the inebriated subjects. This speculation may harbor the possibility that intoxicated individuals may lose their ability to make correct attributions concerning

to high instigating tones and vice versa. When exposed to neutral intent, however, no such correlation was found. In contrast, the inebriated subjects displayed a positive relationship between instigating tone and aggressive response on both measures of physical aggression under both types of instigator-intent.

It appeared that under the effect of alcohol less differentiation between types of intent was maintained. In contrast, a clear differential response pattern was displayed by the nonintoxicated subjects. On both measures of physical aggression these subjects were more aggressive when exposed to malicious intent than when exposed to neutral intent.

The expectancy effect associated with alcohol ingestion (Lang et al., 1975) may have affected some of the present study's findings. When exposed to neutral intent, placebo subjects expressed more aggression through shock intensities then the sober subjects. Again, this response pattern may have been due to the social permissiveness associated with social drinking (Sobell & Sobell, 1975). The fact that the difference between the placebo and sober groups' responses disappeared when the instigation was applied with malicious intent may be due to the justification both groups had for responding aggressively (Brock & Buss, 1964).

The findings of the second study indicate that the drinker seems to be able to process some information concerning

events antecedent to a given situation. The inebriated subjects of the present study seemed to be aware of the instigator's intent and correctly attributed to him their discomfort. However, the ability of the inebriated subjects to clearly differentiate between intent types is somewhat obscured by these subjects' angry response pattern directed toward the neutral instigator. This response pattern bears resemblance to the response strategy employed by the inebriated subjects of the first study.

The findings of the present investigation may be summarized within the framework of the disinhibition model of alcohol and the social learning analysis of human aggression. The subjects who received alcohol in the present investigation were administered a moderate dose that had a stimulating effect on them similar to what has been observed in previous studies (Grennell, 1972; Kalant, 1970). Although the drinking was carried out in an objectively relaxed atmosphere, the mere fact that they were participating in an experiment may have induced some anxiety in the subjects. As the aversive stimulation began, the inebriated subjects seemed to attribute their discomfort to their partners. This is consistent with earlier findings concerning the effect of the arousal source on the aggressive response (Baron & Bell, 1975, 1976; Berkowitz et al., 1969). These conditions serve to trigger a set of previously learned aggressive

responses as mentioned earlier (Bandura, 1973, 1977b).

Although the purpose of the present investigation was not to test the Russell and Mehrabian (1975) model on the effects of alcohol on emotion-mediated behaviors, some probable internal events may have contributed to the expression of aggression by the inebriated subjects. Assuming the subjects experienced an initial displeasure and moderate arousal due to the task requirements, the combined effect of the ingested alcohol, the aversive stimulation, and the likely thoughts of hostility and dominance (Boyatzis, 1974, 1975; Kalin et al., 1965) may have facilitated overt aggression. In fact, the intoxicated subjects displayed a pattern of angry-type aggressive responses in contrast to the instrumental response pattern displayed by the nonintoxicated subjects.

It seems that the alcohol administered in the present investigation had a specific effect on cognitive processes facilitating aggressive behavior. Normally, when an individual is about to engage in aggressive behavior his responses are presumably controlled by anticipated consequences according to past experience (Bandura, 1969). Under the level of intoxication achieved by subjects in the present study, however, the cognitive processing involved in such an evaluation seemed to be disrupted. This may bear resemblance to earlier findings concerning cognitive performance

on different tasks (Jones & Vega, 1972). The taking of uncalculated risks by the inebriated subjects indicates that information concerning behavior contingencies was not used to control their aggressive behavior. This finding seems to be in accordance with studies examining other risk-taking behaviors in intoxicated individuals (Cutter et al., 1973; Teger et al., 1969).

The alcohol also had a partial disrupting effect on the way inebriated subjects processed information concerning the intent with which the instigating stimulation was delivered. Judging by the small degree of differential responses to different types of instigator-intent, one may speculate that a larger dose of alcohol may have eliminated the intent processing capability of the intoxicated subject entirely.

Furthermore, the ingested alcohol clearly introduced a stimulus-bound element into the intoxicated subjects' responses. These subjects' behavior did not appear to be controlled as much by the antecedents and consequences of their aggression as it appeared to be controlled by the instigating aversive stimulation per se.

The findings of the present investigation have several broad implications. It seems to be necessary to recognize that a certain disruption of cognitive functions crucial for the control of aggression takes place following the ingestion of sufficient alcohol. In spite of the fact that the

drinking individual is likely to assert his complete control over his behavior while intoxicated, no such claims should be taken at face value. During an episode of acute alcohol intoxication extrasituational restraints such as social norms, status requirements, responsibilities, attitudes, short-term and long-term consequences may lose their controlling function over the individual's behavior. Combined with the increase in arousal experienced by the drinker, the nonspecific depressing effect of alcohol may have an inhibiting effect on some higher cortical functions (Kalant, 1975). This may motivate the drinking individual to act without being able to adequately process the implications of his actions.

Perhaps this effect of alcohol on cognitive functions may help to explain the high incidence of assaultive and homicidal behaviors associated with the ingestion of alcohol. As observed in several studies of social drinkers (Boyatzis, 1974, 1975), the ingestion of alcohol is followed by an increase in boisterous and verbal aggressive behaviors. Under these circumstances, practical jokes, arguments, and a variety of other common mishaps such as pushing, drink-spilling, etc., may be perceived as initiated with an aggressive intent. The inebriated individual may not be able to differentiate between accident and intended offence. This erroneous processing of information may well contribute to

the exaggerated retaliation of the intoxicated individual often resulting in bodily harm to the victim. In addition, being primarily motivated by the unpleasant stimulation, the inebriated person seems to pay little attention to the probable consequences of his aggression such as injury to the self, arrest or self-defamation. It appears that the intoxicated individual selectively attends to what he perceives as hostile behavior of another person directed at him.

In summary, it appears from the present findings that alcohol acts both as a disinhibitor of approach behaviors and emotional expression, as well as a substance blocking cognitive functions crucial to accurate information processing. Thus, the probability that the intoxicated individual will respond aggressively to instigation is high as his behavior seems to be controlled by the instigation rather than by antecedent or consequent factors.

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APPENDIX I

STUDY I (BEHAVIOR CONTINGENCIES): INSTRUCTION TO SUBJECTS

"In this study we are trying to establish how the drinking of alcohol affects one's reaction-time and pain threshold. You have been randomly (by lottery) assigned to the group of subjects tested for reaction-time. Your counterpart (another subject) is assigned to the pain-threshold group. After you and the other subject (now sitting in another room) have filled out some questionnaires and have drunk an appropriate quantity of alcohol, we will begin.

As I will be explaining the experiment to you, the other subject will be watching us on a T.V. screen like the one on this table. Later you will be able to watch the other subject as I will be explaining the procedure to him. We use the T.V. in order to make sure that the two of you understand the experiment well.

Observe the control panel in front of you. The yellow "ready" light will indicate to you to get ready. Several seconds thereafter, the subject in the other room will administer to you an aversive noise. He will set the level of this noise according to a list previously made up by me. You will have to listen to this noise until the green "act" light comes on. Then, as fast as you can, press any one of these five shock buttons. By doing so, you will simultaneously turn the noise off and administer an electric shock of the indicated level (1-5) to the other subject.

You can choose any button you want. All turn the noise off and all deliver a shock when they are pressed.

Several seconds after you pressed the button, the other subject will respond to you by administering to you once more an aversive noise. This time, however, he can choose any noise level he wants. This will be accompanied by a red light indicating to you which noise level he chose for you. We follow this procedure in order to learn about his response to the shock.

This will end one trial. After a short pause we will repeat this procedure several times. I will come in and tell you when the experiment is over. You will not meet the other subject nor will you be told his identity. Do you have any questions so far?"

APPENDIX II

F TABLES FOR STUDY I

ANOVA table for shock intensity

Source	Sum of squares	Degrees of freedom	F	p
Drug	84.77	2	274.64	0.000
Contingency	12.17	1	78.84	0.000
Drug x contingency	2.28	2	7.39	0.001
Error	10.18	66		

ANOVA table for shock duration

Source	Sum of squares	Degrees of freedom	F	p
Drug	1.76	2	2.81	0.068
Contingency	1.11	1	3.54	0.064
Drug x contingency	2.14	2	3.42	0.039
Error	20.66	66		
Intensity [a]	0.23	4	0.62	0.648
Intensity x drug	4.81	8	6.57	0.000
Intensity x contingency	0.17	4	0.48	0.751
Intensity x drug x cont.	0.88	8	1.21	0.291
Error	24.15	264		

a In aposteriori tests the shock durations were analyzed for the five different shock intensities separately.

ANOVA table for heart rate

Source	Sum of squares	Degrees of freedom	F	p
Drug	1259.72	2	0.94	0.395
Contingency	518.48	1	0.77	0.382
Drug x contingency	2389.99	2	1.79	0.175
Error	44121.69	66		
Reading [a]	636.71	4	7.65	0.000
Reading x drug	513.01	8	3.08	0.002
Reading x contingen.	261.40	4	3.14	0.015
Reading x drug x con.	178.75	8	1.07	0.381
Error	5488.03	264		

a The heart rate scores for the five different readings were analyzed separately.

ANOVA table for verbal aggression

Source	Sum of squares	Degrees of freedom	F	p
Drug	2.53	2	0.27	0.762
Contingency	84.49	1	18.28	0.000
Drug x contingency	53.08	2	5.74	0.005
Error	304.99	66		

APPENDIX III

STUDY II (INSTIGATOR-INTENT): INSTRUCTION TO SUBJECTS

NEUTRAL INTENT [a]

"In this study we are trying to establish how drinking of alcohol affects one's reaction-time and pain threshold. You have been randomly (by lottery) assigned to the group of subjects tested for reaction-time. Your counterpart (another subject, who has been drinking in the other room) is assigned to the pain threshold group. As I will be explaining the experiment to you, the other subject' will be watching us on a T.V. screen like the one on this table. Later you will be able to watch the other subject as I will be explaining the procedure to him. We use the T.V. in order to make sure that the two of you understand the experiment well.

Observe the control panel in front of you. The yellow "ready" light will indicate to you to get ready. Several seconds thereafter, the subject in the other room will administer to you an aversive noise. He will set the level of this noise exactly according to a list previously made up by me. You will have to listen to this noise until the green "act" light comes on. Then, as fast as you can, press any of these five shock buttons. By doing so, you will simultaneously turn the noise off and administer an electric shock of the indicated level (1-5) to the other subject. You can choose any button you want.

All turn the noise off and all deliver a shock when they are pressed

After receiving the shock, the other subject will rate the degree of pain he experienced on a form. We follow this procedure in order to learn about his pain threshold.

This will end one trial. After a short pause we will repeat this procedure several times. I will come in and tell you when the experiment is over. You will not meet the other subject nor will you be told his identity. Do you have any questions so far?"

a Instructions for the MALICIOUS INTENT condition were the same apart from changing one sentence as follows:

"... Several seconds thereafter the subject in the other room will administer to you an aversive noise. He is entirely free to set this noise at any level he wishes..."

APPENDIX IV

F TABLES FOR STUDY II

ANOVA table for shock intensity

Source	Sum of squares	Degrees of freedom	F	p
Drug	89.77	2	504.78	0.000
Intent	8.46	1	95.14	0.000
Drug x intent	0.95	2	5.35	0.007
Error	5.87	66		

ANOVA table for shock duration

Source	Sum of squares	Degrees of freedom	F	p
Drug	2.29	2	2.35	0.103
Intent	1.69	1	3.47	0.067
Drug x intent	2.75	2	2.82	0.067
Error	32.13	66		
Intensity [a]	0.41	4	2.08	0.083
Intensity x drug	5.32	8	13.43	0.000
Intensity x intent	0.21	4	1.07	0.372
Intensity x drug x intent	0.51	8	1.29	0.248
Error	13.08	264		

a In aposteriori tests the shock durations were analyzed for the five different shock intensities separately.

ANOVA table for heart rate

Source	Sum of squares	Degrees of freedom	F	p
Drug	1996.54	2	1.02	0.364
Intent	60.05	1	0.06	0.805
Drug x intent	2706.79	2	1.39	0.256
Error	64230.58	66		
Reading [a]	2026.94	4	17.75	0.000
Reading x drug	953.46	8	4.17	0.000
Reading x intent	191.42	4	1.67	0.156
Reading x drug x intent	92.00	8	0.40	0.918
Error	7536.44	264		

a Heart rate scores for each of the five readings were analyzed separately.

ANOVA table for verbal aggression

Source	Sum of squares	Degrees of freedom	F	p
Drug	8.69	2	1.33	0.272
Intent	17.99	1	5.50	0.022
Drug x intent	11.08	2	1.69	0.192
Error	215.99	66		