EFFECTS OF SLEEP DEPRIVATION, IRRITABILITY, AND NEGATIVE AFFECT ON AGGRESSION

by

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(Under the Direction of Amos Zeichner)

ABSTRACT

Sleep deprivation and aggression are common problems in this society. Aggression is a complex behavior which is influenced by intrapersonal (e.g., physiology and personality) and situational (e.g., alcohol and sleep deprivation) variables. Following Berkowitz's (1990) Cognitive-Neoassociationistic model of aggression, it was hypothesized that acute partial sleep deprivation (APSD) would serve as an aversive event that would increase negative affect and override cognitive inhibition and, therefore, increase aggressive responding during the Response Choice Aggression Paradigm (RCAP) task. It was also expected that this relationship would be mediated by trait irritability and by state negative affect. Two hundred and forty undergraduate men were recruited from the departmental research participant pool and screened for factors that may contribute to potential damaging effects from APSD. Participants also completed the Profile of Mood States (POMS), the Positive and Negative Affect Schedule (PANAS), the Caprara Irritability Scale (CIS), the Sleep Habits Scale (SHS), and the Brief Michigan Alcoholism Screening Test (B-MAST). Of those screened, eighty-nine were invited to participate in the laboratory session. Half were assigned to the experimental APSD group and were instructed to limit their sleep to 4 hours the night prior to the session, and half were

assigned to the control group and instructed to sleep their "normal amount." At the laboratory, participants completed the POMS and PANAS and participated in the RCAP. Seventy-three participants were included in analyses. Participants in the experimental group scored higher at the laboratory session on the Tension-Anxiety and Fatigue-Inertia subscales and lower on the Vigor-Activity subscale of the POMS and the Positive Affect subscale of the PANAS than at the screening session. It was found that participants in the experimental group evinced higher aggression (i.e., higher shock frequency and flashpoint duration). The effect of APSD on shock frequency was mediated by subjective fatigue. Additionally, trait irritability was positively correlated with the proportion of highest shocks administered but could not be tested as a mediating variable between APSD and aggression. Results were consistent with the Cognitive-Neoassociationistic model of aggression and demonstrate potential deleterious effects of sleep deprivation in our society.

INDEX WORDS: Acute Sleep Deprivation, Aggression, Response Choice Aggression Paradigm, Irritability, Negative Affect

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DEDICATION

This project is dedicated to my father, Al Bartolucci, whose love, patience, and support have made me want to be the next Dr. A. Bartolucci; to my mother, Lieve Bartolucci whose example has been the inspiration that led me into the helping professions; to my little sister, Lindey Bartolucci, who reminds me that even though the road less traveled may be harder, it's definitely worth the trip; and, finally and most deservedly, to my husband, Jason Graham, without whose encouragement I never would have made it through this process of obtaining my Ph.D.

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CHAPTER 1

Introduction

Aggression and violence have been acknowledged as widespread problems in our society. These behaviors take many forms including domestic violence, animal and child abuse, sexual assault, school shootings, and gang wars. Researchers have attempted to define and categorize aggression as well as to identify factors that contribute to the decision to act aggressively. Several models have been proposed, two of the most recent of which are described below. The current project seeks to add to the literature by applying the cognitive-neoassociationistic model (Berkowitz, 1990) to clarify the relationship between acute partial sleep deprivation, another common problem in this society, irritability, and aggressive behavior. The literature review includes a discussion of the different types of aggression, models of aggression, and the intrapersonal and situational variables that influence aggressive behavior. Irritability is conceptualized as an intrapersonal variable, and sleep deprivation as a situational variable.

Types of Aggression

Aggression has been defined as a behavior intended to harm another person who is motivated to avoid the potential injury (Anderson & Bushman, 2002; Berkowitz, 1993; Bushman & Anderson, 2001; Kingsbury, Lambert, & Hendrickse, 1997; Mawson, 1999). Theorists have traditionally divided aggression into different types depending on goal (e.g., instrumental vs. hostile; Kingsbury, Lambert, & Hendrickse, 1997), timeline of aggressive decision (premeditated vs. impulsive; Stanford, Houston, Villemarette-Pittman, & Greve, 2003), and target (e.g., displaced aggression; Marcus-Newhall, Pedersen, Carlson, & Miller, 2000). Although other

types of aggression have been identified (e.g., indirect and verbal), the focus of this review is on physical aggression.

Researchers have attempted to dichotomize aggression by proximate goal. Instrumental aggression is defined as an aggressive act with the goal of obtaining any of a range of rewards. Such reinforcement may either be material or social (Kingsbury, Lambert, & Hendrickse, 1997). For example, a gang member may harm another individual in order to obtain money or to prove something to his peers. The important part of the definition is that the goal is something other than harm of another person (Bushman & Anderson, 2001). Hostile aggression, on the other hand, has the single proximate goal of injuring another person and is typically preceded by a negative affect state such as anger (Kingsbury, Lambert, & Hendrickse, 1997).

Another way of conceptualizing proximate goal is to examine the time used to make the decision to act aggressively. As with hostile aggression, impulsive aggression involves a surge of emotion (typically negative) and a "loss of behavioral control" (Stanford, Houston, Villemarette-Pittman, & Greve, 2003, p. 774). Premeditated aggression is perceived as planned, determined ahead of time, and purposeful. Psychiatric inpatients who committed planned aggressive acts have been shown to differ from nonviolent controls in measures of past aggression and personality pathology such as impulsivity, anger, hostility, psychotic symptoms, and neuroticism, which indicates that aggressive individuals may have more difficulty modulating their emotions (Stanford, Houston, Villemarette-Pittman, & Greve, 2003). Authors have argued that the hostile/impulsive vs. instrumental/premeditated dichotomy is no longer useful in the current state of the research (Bushman & Anderson, 2001) and have proposed a different model to explain why humans act aggressively.

Displaced aggression is a construct that has fallen in and out of favor in the aggression literature. As the name implies, the aggressor's behavior is not due to the provocation performed by the target, but is in response to earlier provocation by another individual or situation (Marcus-Newhall, Pedersen, Carlson, & Miller, 2000). Variables that include type of provocation, degree of resemblance between original and subsequent sources of provocation, and characteristics of the setting of the displaced aggression have been found to affect the degree of aggression directed at the target. Reportedly, a higher level of initial provocation resulted in a lower degree of displaced aggression, perhaps because the aggressor assigned more positive valence to the target in comparison to the original source of provocation. This was true whether the original provocation was performed by a person or was situational. Targets that resembled the source of the original provocation were recipients of higher levels of displaced aggression, as were those who applied more provocation (Marcus-Newhall et al., 2000).

Theories of Aggression

As research has shown, aggression is a complex construct that includes many components. Theories of human aggression originally focused on single factors. For example, one of the earliest theories of aggression postulated that frustration is the primary instigator of aggressive behavior (Dollard et al, 1939, as cited in Berkowitz, 1998). As the interplay of different variables became apparent, the models grew in complexity. Current models of aggression integrate situational, physiological, cognitive, and emotional variables.

Cognitive-neoassociationistic Model

The basic assumption of the cognitive-neoassociationistic model is that negative affect leads to feelings of anger and resultant emotional aggression (Berkowitz, 1990). An associative network exists between these processes and memories, cognitions, and inclinations to act

aggressively (Berkowitz, 1993, 2001). Therefore, negative affect leads to physiological and cognitive reactions associated with both flight from the situation and an aggressive response to the situation (Berkowitz, 1990, 1993). These reactions are a result of automatic associations and direct the individual's initial impulses (Berkowitz, 2001). Although Berkowitz (1993) proposes that this network, or some of its components, may be activated by all types of negative affect, certain types of negative affect may activate more of the network than others. However, it is important to note that, for the purposes of his model, Berkowitz (1990) does not differentiate between irritation, anger, and annoyance because it is uncertain whether they produce different consequences.

The associative process is set in motion by an aversive event, which causes physical and/or emotional discomfort that produces negative affect (Berkowitz, 1990, 1993, 2001). Negative affect leads to feelings of anger and inclinations to act aggressively (Berkowitz, 1990, 2001) because it generates an impulse to action in accord with one's current emotional experience (Berkowitz, 1993). Not everyone who feels angry acts aggressively because cognitive processes operate to determine how initial physiological reactions, cognitions, and memories combine to affect the emotional state (Berkowitz, 2001). For example, pre-existing beliefs and experiences can intensify or weaken anger (Berkowitz, 1990). Cognitive processing controls the focus of an individual's attention and activates inhibitors (Berkowitz, 1993). Anger may be augmented by pre-existing negative affect and cognitions related to aggression. When higher-order cognitive processing does not engage (Berkowitz, 2001), the likelihood of emotional aggression may be maximal (Berkowitz, 1993, 1994). Experience of anger may be attenuated if the individual is cognizant of his/her emotions and their appropriateness and, consequently, restrains them (Berkowitz, 1993). In previous studies, distraction of participants'

attention from their cognitive processes increased likelihood of aggression (Berkowitz, 1994, 2001). Those participants whose anger and impulses were made salient were less hostile, possibly because they redirected their resources to reduce negative affect (Berkowitz, 1993).

One requirement for aggression is the availability of a target. If a target is available and the inhibition produced by cognitive activity is absent, aggressive tendencies are more likely to be expressed (Berkowitz, 2001), and even an ambiguous target may be judged unfavorably (Berkowitz, 1990, 2001) because he/she becomes the target of anger-related cognitions (Berkowitz, 1993, 1994). Berkowitz (1993) found that, indeed, participants were likely to act aggressively toward individuals who were not responsible for their discomfort.

General Aggression Model

The General Aggression Model (GAM; Anderson & Bushman, 2002) was developed from the General Affective Aggression Model (GAAM). The GAAM proposed that aggressive behavior arises from activation of cognitions related to aggression, experience of anger, and increase of arousal (Lindsay & Anderson, 2000). The GAM goes beyond other models proposed thus far and combines the cognitive-neoassociationistic model, social learning theory, script theory, excitation transfer, and social interaction theory. The GAM posits that an individual integrates perceptions, pre-existing beliefs, and behavioral scripts to determine his/her affective and behavioral responses.

When an individual confronts a situation that includes the potential for aggressive responses, he/she experiences three potential determinants of his/her internal state. These are: (a) person and situational inputs, (b) cognitive, affective, and arousal routes through which the inputs act, and (c) the outcomes of the process. Person inputs include personality, or the total of an individual's knowledge structures including beliefs and attitudes, physiologic, and genetic

factors. Some examples include values, goals, family history, affective predispositions, and self-esteem. Situational factors include provocation, frustration, pain, administration of alcohol or other drugs, and incentives (Anderson & Bushman, 2002). Pain and discomfort have been found to increase aggressive responding. In fact, Lindsey & Anderson (2000) found that pain and trait hostility predict state hostility, which increases likelihood of aggression.

These input variables combine to result in an internal state that is composed of cognitions, affect, and arousal, all of which interact to determine the behavioral response. Cognitions may include scripts about what one should do in that situation, memories, and thoughts, which may have aggressive content. Affect encompasses mood, emotion, and nonverbal behavior, particularly facial expression. Physiological arousal may serve as a motivator for action, may be misinterpreted as a negative emotion (e.g., anger), or may be aversive in itself. The individual receives information from immediate appraisal, an automatic process that is below awareness and which results in a situational inference or the activation of a personality trait. Immediate appraisal may prompt the individual to become aware of his or her situational goal (Anderson & Bushman, 2002).

Other variables may determine whether the individual responds aggressively. Opportunity has been demonstrated as a strong predictor of aggression. The presence of anger may also prompt aggression through the reduction of inhibition, interference with the dissipation of aggressive cognitions, a cued aggressive response from previous experience, or increased arousal.

Intrapersonal Variables

As the models above illustrate, intrapersonal variables as well as interpersonal response style affect how an individual responds in potentially aggressive situations. A person's

aggressive response is determined by basic physiological characteristics such as inherited predisposition and neurobiology, both of which are determined, in part, by gender. Trait characteristics such as self-esteem, impulsivity, irritability, and the ability to integrate and analyze information in order to control goal-directed behavior (executive cognitive functioning; Hoaken, Shaughnessy, & Pihl, 2003) help determine how a person will interpret situational cues and how he or she will react physiologically. Beliefs about others' intentions (e.g., hostility) and whether aggression is an adaptive response (e.g., catharsis) also increase or decrease the likelihood of aggressive behavior. All of these intrapersonal variables interact to determine whether an individual will respond aggressively to provocation.

Physiological Variables

As with susceptibility to cancer or heart disease, the expression of aggressive tendencies has been found to have genetic and physiological components in at-risk individuals. Although individuals reared in high-risk environments (e.g., in alcoholic families) are more likely to display aggressive behavior, genotypes have been shown through twin studies to contribute significantly to aggressive temperament. In their review, Lee & Coccaro (2001) concluded that the tendency to engage in aggressive and/or criminal behavior may be partially inherited. An earlier study by the same research team (Coccaro, Bergeman, Kavoussi, & Seroczynski, 1997) found that, in male twin pairs, the proportion of variance in aggressive behavior which could be explained through expression of genetic predisposition was 47% for physical aggression, 40% for indirect physical aggression (defined as aggression toward objects rather than people), and 28% for verbal aggression. While researchers have attempted to define the exact locus of the "aggression gene," the genetic map has proven too complicated thus far (Lee & Coccaro, 2001).

In an analysis of several adoption studies, Miles and Carey (1997) found that an individual's inherited tendencies and environment contribute to whether an individual displays aggressive behavior. Fifty percent of the variance was explained by whether a parent had acted aggressively. Thus, inherited tendencies influence aggressive behavior even when environment is controlled, as it usually is with adoptions (e.g., adoption agencies carefully screen families where the environment may be conducive to aggressive behavior such as with alcoholic parents). Miles and Carey (1997) also hypothesized that the relative influences of genotype and environment may vary over time, with environment more influential on aggressive behavior in juvenile populations and inherited tendencies toward aggressive behavior stronger in adult offenders.

Scarpa and Ollendick (2003) found that physiology and previous history interact. These researchers compared young adults who had a history of victimization and/or reported physical aggression, verbal aggression, anger, or hostility with those who did not. Victims and non-victims who had reported aggression had lower resting heart rate, although the difference between non-victims who had and had not reported engaging in aggression was not significant ($p = .087$). Non-victims who were aggressors had higher resting heart-rate variability than those who were not, and victims who were also aggressors demonstrated higher levels of cortisol after a stressor task. These findings support a theory that individuals prone to antisocial temperament have lower resting heart rate and higher heart-rate variability and thus are more likely to act aggressively due to "boldness of character" (p. 335) and emotional dysregulation. This study demonstrated that physiology and history may interact to affect likelihood of aggressing.

Brain imaging studies have revealed differences in brain activity between those who aggress and those who do not. Harmon-Jones and Sigelman (2001) identified the left prefrontal

cortex as the location of anger, which they conceptualize as an "approach-related emotion" (p. 798). Greater EEG activity was found in that region in participants who experienced insults and reported anger than in those who did not. PET scans have further clarified the role between aggressive personalities and/or history and brain activity. In their review, Lee & Coccaro (2001) reported studies that implicated the prefrontal cortex, which is innervated by serotonergic neurons. Other serotonin-rich areas of the brain such as the superior parietal gyrus, the left angular gyrus, and corpus callosum showed lower rates of metabolism in murderers. Another PET study found lower metabolism in the orbital frontal, adjacent ventral medial, and cingulate cortex in participants diagnosed with a personality disorder. Giancola (2000) reviewed several neurophysiological and neuroimaging studies that identified the dorsolateral region of the prefrontal cortex as less activated in individuals who had displayed aggressive behavior. He theorizes that, because it filters signals from the amygdala and hypothalamus, both of which have been implicated in aggression, lower activity in that area allows more aggressive impulses to influence behavior, although the exact mechanism is not known.

Although other neurotransmitters and hormones such as testosterone and cholesterol have been implicated in aggressive behavior, low rates of serotonin metabolism have been most consistently linked with aggression. Low levels of the serotonin cerebrospinal fluid metabolite 5-hydroxyindoleacectic acid (5-HIAA) have been found in impulsive violent offenders convicted of arson and in violent criminal offenders (Lee & Coccaro, 2001). Lower responsivity to fenfluramine, which causes release of serotonin, and, in turn, activation of the limbic system and release of prolactin, has been correlated with aggression.

Gender

In general, research has found that women are more likely to aggress by indirect or relational means than through physical aggression (e.g., Green, Richardson, & Lago, 1996; Richardson & Green, 1999). Several meta-analyses have found that the situational and emotional factors which lead men and women to behave aggressively differ. In their meta-analysis examining provocation, Bettencourt and Miller (1996) found that men and women respond aggressively to different types of provocation. Men tended to respond aggressively under conditions of negative feedback regarding intelligence, frustration, or physical attack. Women aggressed when provoked by insult or negative evaluation. Men were more aggressive than women under neutral conditions than when provoked. Female judges who participated in the meta-analysis were also more likely to perceive provocation as lower and danger of retaliation as higher than the male judges. In a later meta-analysis that examined provocation along with presence of violent cues, Bettencourt and Kanahan (1997) replicated the earlier finding that gender differences are larger when the available means of aggression are physical rather than verbal, when the target is of a different gender than the participant, and when participants are not provoked. Under conditions of provocation, men were more aggressive than women when violent cues were present. Bettencourt and Kanahan (1997) also examined measures of reactivity. Under neutral conditions with violent cues, men with high reactivity were more aggressive than women with high reactivity while the level of aggressive responding was equal when participants were rated as having low emotional reactivity. These studies show that under specific provoking situations and with violent cues present, aggressive responding among women is increased. The authors argued that these results are consistent with the cognitive-neoassociationistic model, which indicates that, due to socialization, women may

require a higher level of stimulation to access the option of responding aggressively, particularly if the available response is uncharacteristic of women (i.e., physical).

In another meta-analysis, Knight, Guthrie, Page, and Fabes (2002) examined emotional arousal and gender differences in aggression. Arousal was defined as activation of the autonomic nervous system, which is excitatory and prepares the organism for action. Emotional regulation is the process by which this response can be controlled through emotional and physical manipulation. These researchers examined studies and coded them according to whether they produced no, small, moderate, or large changes in baseline emotional arousal. Gender differences in aggression were largest under small to moderate arousal and smallest under conditions of no arousal or high arousal. Men who were not emotionally aroused may have responded less aggressively, and therefore more similarly, to women. Women who were highly emotionally aroused may have been less able to self-regulate, and their inhibitions against responding aggressively may have been overridden. As mentioned above, gender differences were smaller for verbal and relational aggression than for aggressive responding by physical means or electric shock.

Astin, Redston, and Campbell (2003) hypothesized that, similar to the arousal/regulation theory, the reasons that men and women aggress differ with regard to social consequences. They postulated that when men behave aggressively, the reason is instrumental, or to exert control. Women perceive their own aggressive behavior as loss of control, or expressiveness. Men obtained higher scores than women on a measure of instrumentality, but both genders had lower scores on expressiveness. Relative to men, women viewed their own aggression as more morally acceptable. Archer (2004) found similar results for men and women on instrumentality and

expressiveness. In contrast to women in the previously cited study, women in this study viewed aggression as more effective rather than morally acceptable.

Zeichner, Parrott, and Frey (2003) found that, although women waited longer to aggress, they behaved more consistently during the Response-Choice Aggression Paradigm, a laboratory measure of aggression (see below), than men. In other words, while men alternated between shocking and not shocking, women were more likely to shock on subsequent trials. Consistent with previous research, men scored higher on measures of physical aggression such as shock intensity, proportion of the highest shock used, and shock frequency than women. This study also supports previous research in that women may perceive their own aggressive behavior as more justifiable because they wait longer to aggress and only respond to higher levels of provocation. Consequently, once they decide to engage in aggression, women may be less likely to disengage and therefore will shock more consistently in the laboratory.

Personality

Self-esteem.

Although the popular assumption has been that low self-esteem is a personality characteristic that predicts aggressive behavior, researchers have found the opposite. Baumeister and Boden (1998) propose that individuals most likely to engage in violent behavior can be described as "…arrogant, conceited, egotistical, narcissistic, or otherwise enamored of themselves" (p. 114) and that "the combination of a highly favorable view of self and an external, unfavorable appraisal is the main cause of aggression" (p. 114). They further propose that lack of self-control is the "final proximal cause" of most aggressive acts (Baumeister & Boden, 1998, p. 125).

Narcissism as a personality trait (rather than a personality disorder) has been described as "an inflated and extremely positive self-view that is unstable and insecure at the same time" (Stucke & Sporer, 2002, p. 510), "grandiosity and exhibitionism, which denotes excessively favorable views of self" (Baumeister & Boden, 1998, p. 119), and "a complex trait that includes inflated views of self, intrapsychic and interpersonal strategies for maintaining these inflated self views, and poor relational functioning" (Twenge & Campbell, 2003, p. 262). One of these interpersonal strategies may be aggression, particularly when individuals with an unrealistically high self-concept are challenged by the fact that they are not as attractive, competent, or talented as they perceive themselves to be (Baumeister & Boden, 1998; Bushman & Baumestier, 1998). Studies that used different types of ego threats including negative evaluation of an essay (Bushman & Baumeister, 1998), criticism of intelligence and creativity (Stucke & Sporer, 2002), and social rejection (Twenge & Campbell, 2003) demonstrated that narcissists became aggressive when their self-concept was threatened. All of the above cited studies also showed that narcissists directed their aggressive responding toward the source of the ego threat and did not displace it when they had the opportunity to aggress toward a third party.

Impulsivity and sensation-seeking.

Impulsivity has also been shown to be a personality characteristic associated with aggression. Baumeister and Boden (1998) propose that it is the loss of self-awareness that causes failure of self-regulation because individuals cease to monitor their actions, as when alcohol is consumed. They also state that this acquiescence, or the choice not to act with self-control, is promoted in some cultures.

Another trait related to impulsivity is sensation-seeking. Joireman, Anderson, and Strathman (2003) defined sensation seeking as looking for a variety of new, exciting activities

and taking risks to be able to engage in them. The components of sensation-seeking are boredom susceptibility, disinhibition, experience seeking, and thrill and adventure seeking. Joireman, Anderson, and Strathman (2003) also examined consideration of future consequences (CFC). The results of their study indicated that CFC mediated the relationship between impulsivity and aggression, that anger mediated the relationship between individual differences and aggression, and that hostile cognitions and anger partially mediated the relationship between sensation-seeking and aggression. Disinhibition predicted physical aggression, and boredom susceptibility predicted verbal aggression. Indeed, boredom proneness has been found to predict anger expression and hostility in previous studies (e.g., Rupp & Vodanovich, 1997).

The personality traits of narcissism and impulsivity appear to be related through lack of social problem-solving ability. McMurran, Blair, & Egan (2002) hypothesize that lack of social problem-solving ability, rather than impulsivity, is the true culprit and that it develops because impulsive individuals fail to learn effective, non-aggressive coping strategies in childhood. D'Zurilla, Chang, and Sanna (2003) also found that social problem solving mediated the relationship between self-esteem and aggression.

Irritability.

Irritability, which has been found to be related to impulsivity (Stanford, Greve, & Dickens, 1995) has been defined as "a stable tendency of the individual to react offensively to minimal provocation" (Caprara, Renzi, Alcini, D'Imperio, & Travaglia, 1983, p. 346). The reaction has also been described as impulsive or rude (Caprara et al., 1986) and may develop gradually from an early age (Caprara, Barbarinelli, Pastorelli, & Perugini, 1994). According to Caprara's theory, impulsive individuals are in a constant state of readiness to react to danger and are unable to reduce their own experience of frustration or take control of their agitation when

faced with real or imagined danger (Caprara et al, 1985). Consequently, they tend to be more likely to perceive events as stressful and to overreact to them (Caprara et al., 1985). Indeed, it was found that irritable participants shock at higher levels, and researchers speculated that this results from participants' inability to regulate negative emotions when frustrated.

Physiologically, irritable participants have been found to have higher systolic blood pressure and faster heart rate than participants who are not irritable (Caprara et al., 1985). As measurements were comparatively high at both baseline and after provocation, it is likely that these sympathetic nervous system indicators reflect the person's constant state of readiness for fight or flight. Consequently, irritable persons may be at higher risk to develop hypertension (Caprara et al., 1985), which has also been linked to hostility and heart disease in aggressive men (Hall & Davidson, 1996).

Irritability has been well-established as a variable that predicts aggression. Caprara and Renzi (1981) found that frustrated highly irritable participants shocked more frequently than low irritable participants. Caprara and colleagues (1983) found similar results, except that highly irritable participants (males) administered higher shocks only after provocation. This result was replicated by Caprara and colleagues (1986). In a study of nicotine-deprived and irritable and non-irritable men, Parrott & Zeichner (2001) found a main effect for irritability on shock intensity and duration and determined that, as urges to smoke increased, deprived and irritable participants had higher scores on all four indices of aggression. Giancola (2002) also found in a study of the effects of irritability, alcohol, and provocation on aggression that irritability was correlated with aggression for all participants and that provocation was the most significant elicitor of aggression. He surmised that alcohol disrupted the inhibitory system, and thus participants' irritability was expressed as aggressive behavior.

Executive Cognitive Functioning.

The construct of executive cognitive functioning (ECF) can be conceptualized as lying at the other end of the dimension of impulsivity, as persons with high ECF are more likely to inhibit inappropriate or risky behaviors (Giancola, 2000). Giancola and Zeichner (1994b) found that participants who scored lower on the Conditional Association Task (CAT), a neuropsychological test which activates the dorsolateral region of the prefrontal cortex, were more aggressive than those who scored higher on the CAT. Hoaken, Shaughnessy, and Pihl (2003) found that individuals in the lowest ECF quartile of their sample selected higher shock intensities than individuals in the highest ECF quartile. Participants with low ECF also responded to provocation with higher levels of aggression. However, instead of impulsivity, these researchers hypothesized that participants with low ECF responded aggressively due to "some interruption of social information processing" and "inappropriate selections" of responses (p. 26).

Intelligence.

The general concept of intelligence has been implicated in aggression. For example, Giancola and Zeichner (1994a) found that participants who scored lower on I.Q. tests chose higher intensity shocks. As with low ECF, low I.Q. may interfere with the ability to interpret internal and external cues in an aggressive situation (Giancola, 2000; Giancola & Zeichner, 1994a).

Hostility.

One way to conceptualize the social information processing deficits that seem to be found in aggressive individuals is the trait of hostility. As Hall and Davidson (1996) proposed, "hostile individuals are prone to enhanced physiological reactivity to stressors" (p. 378) and may hold a

"hostile other schema…a tendency to view others as potential sources of mistreatment and provocation" (p. 379). Tiedens (2001) also mentions a "hostile attribution bias" (p. 234), which is the view of others as planning to behave in a hostile manner toward the participant. Hall and Davidson (1996) found that participants who scored higher on a hostility questionnaire rated interviewers as more aggressive than did non-hostile participants and independent raters who viewed the interviews. With regard to bias in memory, Tiedens (2001) found that previously aggressive participants who were directed to feel anger remembered more information about certain scenarios in response to cues that implied hostility on the part of the actor than participants in other emotional conditions and nonaggressive participants. Hostile individuals appeared to rely on anger-related cues for memory. Thus, a hostile style appears to affect how individuals perceive and remember neutral situations.

Hostility may also affect speed of information processing. Tiedens (2001) stated, "it appears that anger encourages spontaneous judgments and activates thoughts of hostility and malevolence in aggressive people" (p. 248). "Spontaneous" may be interpreted as "fast." Indeed, Bond, Verheyden, Wingrove, and Curran (2004) found that, when presented with sentences describing an angry reaction to a scenario, both angry and aggressive participants read the sentences faster. The researchers interpreted this finding as indicating that angry cognitions and interpretations were more accessible in these participants. Thus they were able to anticipate the aggressive outcome and as such, needed minimal time to comprehend the scenario. Combined with the findings cited above, it appears that individuals who are hostile and angry anticipate an aggressive outcome, selectively attend to those cues, and react based on expectations rather than processing relevant social cues.

Catharsis

A popular theory of aggression, based on the Freudian idea of thanatos, or death impulses, posits that individuals act aggressively in order to release negative emotion before it increases to injurious levels (Bushman, Baumeister, & Stack, 1999). No evidence for this "hydraulic model of anger" (p. 368) has been found in research. Bushman, Baumeister, and Stack (1999) found that acting aggressively toward an inanimate object led to higher levels of subsequent aggression directed toward another individual. Participants also acted more aggressively after reading that catharsis is an effective way to release anger. In fact, participants who thought that they would be able to act aggressively toward someone who had angered them expressed greater desire to hit a punching bag than those who thought that the available target would be a different individual. Thus, contrary to the hydraulic model of anger, acting aggressively toward an inanimate object, which would release the anger, did not decrease subsequent aggressive behavior.

A catharsis theory proposed by Mawson (1999) holds that aggression is a drive much like eating and sexual activity, and that the three may represent different components of the same spectrum of stimulation-seeking behaviors (SSB). An SSB is defined as an action that allows an organism to receive stimulation from external sources through the sensory organs. The drive toward SSB is caused by arousal, which activates brain catecholaminergic systems. The neurotransmitters noradrenaline and dopamine are released, and the individual (or animal) may feel agitated or experience other negative emotions. The neurotransmitter systems are brought into balance through activation of the serotonin (5-HT) and acetylcholine (ACh) systems. The stimulation-induced behavioral inhibition model theorizes that SSBs, including eating, sex, and aggression, provide the necessary activation. High arousal requires high-intensity SSB

(Mawson, 1999). According to this theory, negative emotion is best eliminated through substitution of a positive emotion.

Translated into mood states, the stimulation-induced behavioral inhibition model indicates that individuals may aggress in order to generate a positive mood state. However, Bushman, Baumeister, and Phillips (2001) found the opposite. They found that individuals expect to feel better after aggressive behavior, but subsequent emotions differ according to how an individual typically expresses anger. In general, participants who accepted the catharsis model felt more anger after aggressing than those who did not. Experienced anger depended on gender and prior beliefs. In some cases, participants reported positive affect. However, this finding is not consistent enough to support the stimulation-induced behavioral inhibition model.

Situational Variables

An individual's behavior occurs in the context of an environment, and several environmental variables have been found to affect aggressive responding. One of the most widely researched variables in this area is alcohol intoxication, which acts at the physiological, emotional, cognitive, and environmental levels. A situational variable which has not been researched in aggression to date is sleep deprivation, which seems to have similar effects to alcohol, for example, on driving performance (Williamson & Feyer, 2000).

Alcohol

The association between alcohol and aggressive behavior has been well established. Alcohol appears to increase aggression through an individual's pre-existing traits. With regard to variables already discussed, alcohol has been shown to increase the likelihood of aggressive responses in individuals who are irritable (Giancola, 2002), high in trait anger (Parrot & Giancola, 2004; Parrott & Zeichner, 2002), and have moderate to low ECF (Giancola, 2000).

Alcohol may augment group differences. For example, as noted above, women tend to eschew use of direct aggression in public and in the laboratory. Giancola and Zeichner (1995) found that intoxicated women demonstrated longer shock durations than sober women. As women may be trained from an early age not to aggress, alcohol may loosen culturally-induced restraints.

Alcohol has also been found to affect social information processing in that intoxicated individuals are more likely to focus on hostile cues in a threatening situation. Steele and Josephs (1990) labeled this effect "alcohol myopia" and described it as "a state of shortsightedness in which superficially understood, immediate aspects of experience have a disproportionate influence on behavior and emotion" (p. 923). Moderate doses of alcohol can significantly and consistently reduce psychological distress by "screening out" (p. 929) inhibiting thoughts, particularly when the intoxication is paired with a distraction. Thus, individuals may focus on aggressive cues and ignore thoughts about the potential consequences of acting aggressively. Interestingly, when Zeichner, Allen, Petrie, Rasmussen, and Giancola (1993) tested the process of attention allocation in social drinkers under varying conditions of threat, they found that under the condition of high threat, intoxicated participants allocated more attention to salient information, which was thought to cause psychological arousal. Carey (1995) found that alcohol increased the likelihood of self-focused answers on a stem-completion task and remarked that when an individual is intoxicated, "information would be processed in terms of its self-relevance" (p. 251).

Alcohol acts at different stages of the models described above. Intoxication may increase the experience of negative affect, which leads to increased physiological arousal. Alcohol contributes to the cognitive part of the model by interfering with ECF. Thus, an individual may

focus on cues to aggress and ignore potential negative consequences. As Giancola (2000) noted in his review of ECF and alcohol, the physiological effects of alcohol are apparent in the dorsolateral aspect of the prefrontal cortex, in which glucose metabolism is decreased after acute consumption of alcohol. Finally, as has been well-established, environments in which alcohol is consumed serve as cues for aggressive behaviors.

Sleep Deprivation

An aversive event that may produce negative affect and subsequent anger and aggression is acute partial sleep deprivation, which has been shown to have strong effects on mood and performance after one night (Pilcher & Huffcutt, 1996). In fact, sleep deprivation has been found to have similar effects to alcohol on driving performance (Bonnet, 2005; Williamson & Feyer, 2000). In order to determine how sleep deprivation may influence levels of aggression, it is important to understand the definition of sleep deprivation, the difference between partial and total sleep deprivation, the emotional and cognitive effects, and the physiological theories behind those effects.

Partial sleep deprivation (PSD) is a reduction in total sleep time (TST) without regard to which specific stages are interrupted (in contrast to slow-wave sleep reduction or REM sleep reduction, in which a specific stage of sleep is targeted; Bonnett, 1994). Both PSD and total sleep deprivation (TSD), which involves depriving participants of sleep for longer than 24 hours, can either be short-term (i.e., for one day) or long-term. PSD is "the most common form of sleep deprivation seen in the real world" (Bonnett, 1994, p. 58) because individuals rarely obtain recommended TST (Pilcher & Huffcutt, 1996). Although the ideal amount of sleep varies by individual, the average minimum has been determined to be approximately eight hours (Van Dongen, Maislin, Mullington, & Dinges, 2003). Experimental effects such as lowered

performance and alertness have been seen after sleep reduction of two hours (Carskadon & Roth, 1991), and achieving less than four or four and a half hours of sleep has been described by sleep experts and their participants as "painfully uncomfortable" after one night (Carskadon & Roth, 1991, p. 156). Subjective sleepiness and reduction in performance level have been shown to result from sleeping more than one, but less than four hours on a single night (Dinges et al., 1997), and participants in one study expressed discomfort at between six and six and a half hours of sleep. In studies of long-term PSD, reported sleepiness increased after the first night of reduced TST (Dinges et al., 1997).

Effects of sleep deprivation include changes in mood, physiological discomfort, and performance deficits on memory and attention tasks. One of the most frequently reported results is complaints by participants of a change in mood. According to Pilcher and Huffcutt (1996), effects on mood are even greater than would be expected from self-reported data. An early study (Roth, Kramer, & Lutz, 1976) found a "shift in mood" (p. 136) correlated with sleep stages and dream content. Participants also obtained higher scores on a self-report measure of aggressive feelings after one night of TSD. In a study of long-term TSD, Angus, Heslegrave, and Myles (1985) found increased reports of fatigue, sleepiness, reduced positive mood, and increased negative mood, particularly during the initial 24 hours of the study. Although Dinges and Kribbs (1991) did not specify mood as a variable, they found that that sleep deprivation may affect the motivation to perform rather than performance itself. Bonnett (1994) reported that sleep deprivation leads to sleepiness, fatigue, irritability, disorientation, and negative mood. Hill, Welch, and Godfrey (1996) reported increases in scores on the Tension-Anxiety, Depression-Dejection, Anger-Hostility, and Confusion-Bewilderment subscales of the Profile of Mood States questionnaire in participants who endorsed an external locus of control. Dinges and colleagues

(1997) stated that participants in studies of long-term PSD to 4.5-6.5 hours reported "headaches, fatigue, forgetfulness, reduced concentration, irritability, and difficulty awakening" (p. 268).

Sleep deprivation has also been reported to affect performance, typically through "lapsing," or "inability of a sleepy person to sustain a stable attention on performance" (Dinges & Kribbs, 1991, p. 106). The most susceptible types of tasks are those involving higher-order cognitive processing and sustained attention, and longer time to complete tasks rather than increased errors are reported (Dinges & Kribbs, 1991). Sleep may also cause the brain to become more susceptible to habituation, which can disrupt concentration (Dinges & Kribbs, 1991). Bonnet (1994) reported concentration difficulties in subjects across a range of studies, as did Dinges and colleagues (1997). With regard to which processes are most susceptible to sleep deprivation, Pilcher and Huffcutt (1996) reported in a meta-analysis that motor tasks are affected least, followed by cognitive tasks. Mood scores show the most profound disruption with reported increases in negative affective states. PSD has a greater effect on general functioning and mood than long-term or short-term TSD.

Although effects of sleep deprivation have been noted, the physiological mechanisms underlying them remain unclear. One theory proposes that amount of time awake past a point when an individual requires sleep for optimal physiological functioning rather than TST is responsible for negative consequences of sleep deprivation. Van Dongen, Maislin, Mullington, and Dinges (2003) speculated that stable neurobehavioral functioning can only be maintained for 15.84 ± 0.73 hours. Once that maximum is reached, lapses in alertness and other "costs" occur (p. 125). According to their calculations, humans should sleep, on average, 8.16 hours per night. Another theory (Pilcher & Huffcutt, 1996) states that the effects of PSD result from alteration of the circadian rhythms (i.e., daily fluctuations in temperature and other metabolic functions that

cause individuals to feel sleepy or alert). However, this theory has not been tested. A theory which has been disproved is that PSD affects daytime functioning through alteration of sleep stages. Devoto, Lucidi, Violani, and Bertini (1999) demonstrated through multiple regression analyses that TST, but not amount of slow-wave sleep, was the best predictor of daytime sleepiness. In fact, when at least five hours of sleep are obtained, slow-wave sleep is not affected (Devoto et al., 1999). Rather, it is rapid-eye-movement sleep and stage 2 sleep, non slow-wave sleep which is characterized by sleep spindles and k-complexes on an EEG, that are affected (Bonnet, 1994).

The effects of sleep deprivation on the brain have been demonstrated through imaging studies. Cajochen, Foy, and Dijk (1999) note that, according to their EEG findings, the frontal cortical areas of the brain are affected by sleep deprivation. The prefrontal cortex (PFC), in particular, has been implicated more than the frontal cortex. Binks, Waters, and Hurry (1999) found that TSD did not affect intelligence and attention, both frontal cortex functions. While studies differ as to whether the PFC increases or decreases in activation, the weight of the evidence points to decreased activity (Bonnet, 2005). Imaging studies have shown that, during slow-wave sleep, the PFC evinces low metabolic rate and that sleep deprivation "leads to distinct PFC neuropsychological anomalies that are reversed after recovery sleep" (Horne, 1993, p. 414). Thomas and colleagues (2000) found decreases in glucose metabolism after sleep deprivation in the PFC and thalamus, both areas that control alertness and attention. The PFC serves as a gateway between urges and ECF and integrates sensory-motor and cognitive information. TSD also leads to behaviors similar to those found in individuals with deficits in the PFC such as distractibility, difficulty with divergent thinking, and childish sense of humor (Horne, 1993). Unlike other sleep deprivation-induced effects, these are less able to be overcome by increased

task-related effort. Other researchers (Chee & Choo, 2004; Drummond & Brown, 2001; Drummond, Gillin, & Brown, 2001) found that certain areas of the PFC and parietal lobes are activated after sleep deprivation, potentially to compensate for PFC deficits. One such deficit observed in the behavioral literature may be increased time to complete tasks (Drummond, Brown, Salamat, & Gillin, 2004).

There are several methodological issues to consider when using sleep deprivation as an independent variable. First, its effects do not appear to be influenced by other variables such as gender and age (Hill, Welch, & Godfrey, 1996). One does have to be aware, however, of the effects of circadian rhythm on performance. Dinges and Kribbs (1991) observed that performance was lowered immediately upon awakening from sleep and that performance of their participants on cognitive tasks was affected by the time of day. Dinges and colleagues (1997) reported that subjective sleepiness ratings were significantly higher at 10:00 a.m. than at 4:00 p.m. or 10:00 p.m. Another consideration is task length. Several researchers (e.g., Bonnett, 1994; Dinges & Kribbs, 1991) have noted that effects of sleep impairment become evident after 10 minutes of performance. Partial sleep deprivation has several advantages over total sleep deprivation as an independent variable. First, as it is a common problem in society (Pilcher & Huffcutt, 1996), experimental results will have greater applicability. Large average effect sizes (ranging from 2.04 to 4.10) have also been reported in a meta-analysis of sleep studies (Pilcher & Huffcutt, 1996), and PSD has a greater effect on mood than TSD. Sleep restriction for seven days only requires two nights of recovery sleep (Dinges et al., 1997). Thus, participants should recover quickly from one night of PSD. Finally, PSD has been shown to have strong negative effects on mood, which should allow it to serve as a manipulated aversive event for the current study.

Interestingly, extended wakefulness (17 to 19 hours) has been shown to have similar effects to the legal limit for alcohol on driving performance (Williamson & Feyer, 2000), as have both chronic partial and acute sleep deprivation (Powell et al., 2001). Specifically, "…changes in response speed, visual tracking, and driving commonly found during the first night of total sleep deprivation are equivalent to changes associated with legal intoxication" (Bonnet, 2005, p. 54). Sleep deprivation has also been found to augment the effects of alcohol on simulated driving performance (Rorhs, Beare, Jorick, & Roth, 1994). As they both act in the prefrontal cortex, these results are not surprising.

Although the direct effects of sleep deprivation on aggression have not been studied, PSD may increase aggressive behavior through activation of negative mood states and decrease of inhibition. As Berkowitz's (1990, 2001) cognitive-neoassociationistic model of aggression posits, an aversive event leads to negative affect and subsequent aggression. As sleep deprivation affects mood more than any other process (Pilcher & Huffcutt, 1996), it may serve as a strong aversive event. Also, as PSD has similar effects to alcohol on brain functioning and driving behavior, it may act similarly to alcohol in increasing aggression through diminished activation of the PFC, and thus lowered inhibition to behave aggressively.

Methodological Considerations

Several methods have been used to measure aggression both in and out of the laboratory. In their meta-analysis, Anderson, Lindsay, and Bushman (1999) found that both types of research appear to have external and internal validity. One of the best-supported methods to study direct physical aggression in the laboratory is to allow the participant to administer an electric shock to another person. Shock duration and intensity are the two most commonly obtained dependent variables from this method (Bushman & Anderson, 1998). Participants do

not administer a shock to another individual, and several "cover stories" have been used in previous research as deception. The original deception manipulation used by Berkowitz (1962, as cited in Bushman & Anderson, 1998) was the teacher-learner paradigm. This paradigm was also used by Milgram in his landmark study on human obedience to authority (Milgram, 1965). Taylor (1967, as cited in Berkowitz, 1998) modified it to eliminate the power differential and the possibility of viewing the shock as an altruistic means to help the "student" learn better and told his participants that they would be participating in a reaction-time competition where the winner would be able to shock the loser. In response to criticism that the participants were not being given the opportunity to refrain from aggressing, Zeichner, Frey, Parrot, and Butryn (1999) developed the Response-Choice Aggression Paradigm, a reaction time competition wherein the participant is given the option to aggress or to refrain from doing so following each trial. As the participant has a choice of whether or not to aggress, the measures of flashpoint, or initial shock, occurrence in the trial series, initial shock intensity, and flashpoint duration are added and have been shown to correlate moderately with other measures included in the paradigm. All the paradigm's aggression measures have been shown to co-vary with the physical aggression scale of the Buss-Perry Aggression Questionnaire (Zeichner, Frey, Parrot, & Butryn, 1999).

Summary and Hypotheses

Aggression is a complex behavior with cognitive, emotional, physiological, and behavioral aspects. One contributing variable which has not been researched to date is sleep deprivation, a common problem, as health behaviors are often eliminated to make time for other obligations or responsibilities (Bonnett, 1994). Research has shown that PSD can have emotional and physical consequences. One such consequence is a lowered tolerance for stressful events, or irritability (Caprara et al., 1994), and the resulting "irritation, annoyance, and anger"

(Berkowitz 2001, p. 325) can produce aggressive responses. PSD has also been shown to produce negative state affect (Dinges et al., 1997), which may in turn lead to aggressive responding (Berkowitz, 2001).

As with alcohol, sleep has been found to affect the functioning of the prefrontal cortex. The physiological basis for the hypotheses is that sleep deprivation will lead to lower levels of functioning of the prefrontal cortex, which may, in turn, lead to negative affect and aggressive behavior. Finding from studies on aggression and sleep deprivation differ on whether activation increases or decreases in the PFC. For example, while anger has been shown to increase PFC activation, participants who were aggressive scored lower on tasks that reflect ECF and PFC functioning (Giancola & Zeichner, 1994b).

While the GAM (Anderson & Bushman, 2002) is the most recent and integrative model, it was designed to explicate all areas of aggression as well as developmental influences. While instrumental aggression and knowledge structures are important, it was more parsimonious to use the cognitive-neoassociationistic model, which was designed to explain hostile aggression in the context of negative affect. Consequently, with Berkowitz's cognitive-neoassociationistic model, it was hypothesized that sleep deprivation of even a few hours would augment pre-existing tendencies toward negative affect, specifically irritability, which would increase the likelihood of aggressive behaviors.

According to the cognitive-neoassociationistic model, an aversive event elicits negative affect, which in turn leads to anger and potential aggressive behavior. For the purposes of this study, the aversive event was sleep deprivation. Tendency to experience negative affect was conceptualized as the trait of irritability as measured by the Caprara Irritability Scale (CIS; Caprara et al., 1985). State negative affect was measured by the Profile of Mood States (POMS;

McNair, Lorr, & Droppleman, 1971) and the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). Aggression was measured via the response-choice aggression paradigm (RCAP), which contributed an available target for the participants' aggression (Zeichner, Frey, Parrot, & Butryn, 1999). This paradigm yields seven measures of aggression: shock intensity, shock duration, proportion of highest shock, flashpoint latency (point at which first shock is used), flashpoint intensity, flashpoint duration, and shock frequency.

The current study had several aims with corresponding hypotheses. One purpose was to determine whether sleep has a similar effect to that of alcohol with regard to aggression. Consequently, the first hypothesis was that partial sleep deprivation would predict increases in measures of aggression as demonstrated by linear regression analyses. As irritability has been strongly linked with aggression, the second purpose of the study was to replicate previous findings that irritability and aggression are correlated. Accordingly, the second hypothesis was that irritability would be significantly correlated with some or all of the RCAP measures of aggression. Acute partial sleep deprivation has been demonstrated to increase negative affect, which may lead to aggression. In order to confirm that sleep deprivation increases negative affect, participants' mood states were assessed with the POMS and the PANAS at screening and at the laboratory sessions. The third hypothesis was that participants who were sleep deprived would have significantly higher scores on the Anger-Hostility, Fatigue-Inertia, and Tension-Anxiety subscales of the POMS at the laboratory session than at the screening session. Non-sleep-deprived participants were expected to have nonsignificant change in scores between sessions. As sleep deprivation increases negative affect, irritability was expected to act as a mediator between sleep deprivation and aggression. Accordingly, the fourth hypothesis was that the relationship between acute partial sleep deprivation and aggression would no longer be

significant when irritability was added to the regression model. The final purpose of the study was to determine whether state negative affect would act as a mediator between sleep deprivation and aggression. The fifth hypothesis was that state negative affect would mediate the relationship between acute partial sleep deprivation and aggression as demonstrated by a reduction in the relationship between sleep deprivation and aggression in a regression model when negative affect was included.

CHAPTER 2

Method

Participants and Experimental Design

Because, as reviewed above, women tend not to display direct aggression in laboratory situations, only men were recruited from the departmental research participant pool. Two hundred-forty undergraduate men participated in the screening sessions. Their mean age was 19.41 with a standard deviation of 1.11 (range 18 to 24). Participants indicated that they had obtained a mean of 13.77 years of education (*SD* = 1.19, range 12 to 19) and reported a mean income of 1.41 (*SD* = 1.59, range 1 to 9), or between 10,000 and 20,000 dollars/year. With regard to ethnic composition of the screening sample, 85.8% endorsed Caucasian race, 5.4% were African American/Black, 5.8% were Asian, 1.7% were Hispanic, and 1.3 % were Native American. Ninety-four percent of the screened participants endorsed being single, 5% were in a committed relationship, 0.5% were divorced, and 0.5% were married. Invitation to the laboratory session was based on a variety of criteria including expressed interest in participating in the laboratory session, a score of less than 6 on the B-MAST, and endorsement of fewer than a predetermined number of sleep disorder symptoms on the Sleep Habits Scale. Of the screening sample, 100 were invited and 89 agreed to participate in the subsequent laboratory session. Sixteen participants were eliminated from the laboratory sample and thus from subsequent analyses due to expressed suspiciousness with regard to the manipulation check (see below).

Participants in the laboratory sample endorsed a mean age of 19.15 (*SD* = 0.92), a mean of 13.61 years of education (*SD* = 1.06), and a mean income of 1.63 (*SD* = 2.04), or between

10,000 and 20,000 dollars/year. Eighty-nine percent were Caucasian, 4.0% were African American/Black, 5.3% were Asian, and 1.3% were Native American. Of the laboratory sample, 93.3% were single, 5.3% were in a committed relationship, and 1.3% were married.

The current study included two independent variables and one dependent variable. One of the independent variables was irritability, as measured by the Caprara Irritability Scale (CIS; Caprara et al., 1985). As irritability is a continuous variable, it was not dichotomized. The other independent variable was acute partial sleep deprivation (APSD). Participants in this condition were instructed to restrict their sleep time to 4 hours the night prior to the laboratory session. Control participants were instructed to maintain their normal bedtime. However, as researchers have noted, restriction of sleep by 2 hours to between 6 and 6.5 hours of sleep has been shown to lower performance and alertness and to increase subjective perception of discomfort (Carskadon & Roth, 1991; Dinges et al., 1997). Consequently, the two participants who endorsed sleeping 6 hours were expected to resemble APSD participants and were included with those who had restricted their sleep to 4 hours in the APSD group. Participants in both the APSD and control conditions were instructed to rise at their usual time. The total number of participants in the APSD group was 36; the total number of control participants was 37. The dependent variable was aggression, as measured by the Response-Choice Aggression Paradigm (RCAP; Zeichner et al., 1999).

Measures

Profile of Mood States (POMS)

The POMS (McNair, Lorr, & Droppleman, 1971) is a self-report measure of mood-affective states. It is comprised of 65 items by which participants indicate the degree to which they are experiencing a specific emotion. The Likert-type scale ranges from "0" (*not at all*) to

"4" (*extremely*). This scale has been shown to indicate the experience of six general mood-affective states: tension-anxiety, depression-dejection, anger-hostility, vigor-activity, fatigue-inertia, and confusion-bewilderment. It has internal consistency of .90, and the scales have test-retest reliabilities of .61 to .70. Internal consistency for the screening sample was $\alpha = 0.92$. The subscales had internal consistency of $\alpha = 0.72$ for Tension-Anxiety, $\alpha = 0.89$ for Depression-Dejection, $\alpha = 0.88$ for Anger-Hostility, $\alpha = 0.88$ for Vigor-Activity, $\alpha = 0.88$ for Fatigue-Inertia, and $\alpha = 0.67$ for Confusion-Bewilderment. The POMS was used to measure participants' mood at initial screening and at the beginning of the laboratory session. See Appendix A for questionnaire.

Caprara Irritability Scale (CIS)

The CIS (Caprara et al., 1985) was developed to assess irritability, lowered ability to tolerate frustration and control emotional reactions in potentially harmful or aggressive situations. The modified version of the CIS used for this study was a 30-item true-false scale. Higher scores indicate higher trait irritability. The CIS has been shown to have an α coefficient of 0.81, test-retest correlation of 0.83, and a reliability coefficient of 0.90. Internal consistency for this sample was $\alpha = 0.73$. See Appendix B for questionnaire.

Sleep Habits Scale (SHS)

This scale was adapted for this study from the Insomnia Interview in Morin's (1993) treatment manual. No information on reliability or validity is available, as the interview has only been used clinically. The purposes of this questionnaire were to obtain basic demographic information, to allow participants to describe their sleep schedule (to aid in scheduling laboratory sessions), and to identify potential confounding factors such as use of alcohol, nicotine, and caffeine. The SHS also included questions to assess whether participants met the following

exclusion criteria: use of sleep medication or alcohol to fall asleep, rotating sleep schedule due to shift-work, potential for other sleep disorders (indicated by endorsement of clusters of listed symptoms to have accrued more than three times per week), and pre-existing and diagnosed medical or psychiatric problems. While no previous data regarding reliability and validity are available, the screening sample obtained an internal consistency of $\alpha = 0.73$. See Appendix C for questionnaire.

Positive and Negative Affect Schedule (PANAS)

The PANAS (Watson, Clark, & Tellegen, 1988) was developed to assess two dimensions of mood, namely positive affect, which is described as a state of "high energy, full concentration, and pleasurable engagement" (p. 1063) and negative affect, defined as "distress and unpleasurable engagement that subsumes a variety of aversive mood states, including anger, contempt, disgust, guilt, fear, and nervousness" (p. 1063). It has been found to have internal consistency ratings between $\alpha = 0.84$ and $\alpha = 0.90$, and internal and external validity have been well-established. The Cronbach's alpha for this sample was $\alpha = 0.89$ for the Positive Affect subscale and $\alpha = 0.79$ for the Negative Affect subscale. It was included as an additional, more general measure of state affect.

Brief Michigan Alcoholism Screening Test (B-MAST)

The B-MAST is an abbreviated form of the Michigan Alcoholism Screening Test, which was developed to aid in diagnosing alcoholism (Selzer, 1971). The B-MAST has been shown to have Pearson *r* correlations with the full MAST of .95 for diagnosed alcoholics and .96 for nonalcoholics (Pokorny, Miller, & Kaplan, 1972). A score of "6" is the identified lowest score for which an alcoholism diagnosis would be given per the B-MAST. Internal consistency for this sample was $\alpha = 0.49$. See Appendix D for questionnaire.

Aggression Paradigm

Aggression was measured using the Response-Choice Aggression Paradigm (RCAP; Zeichner et al., 1999). In this paradigm, participants believe they are competing against another (fictitious) participant in a reaction-time task and are given the opportunity to administer electrical shocks to their opponent. This paradigm has been found to be internally and externally valid. The seven measures obtained are as follows:

Shock intensity: The mean intensity of shocks chosen by the participant on the trials on which he/she chooses to shock. Range = 1-10.

Shock duration: Length of time each shock is administered.

Proportion of highest shock: Number of times the highest possible shock (i.e., 10) is chosen divided by total number of shocks chosen. For example, if the participant chooses to shock 10 of 20 trials and chooses to shock at level 10 three times, the proportion of highest shock would be 0.3.

Shock frequency: Total number of trials during which a participant chooses to shock his opponent.

Flashpoint latency: The total number of trials that occur before a participant chooses to shock.

Flashpoint intensity: The intensity of the first shock selected by the participant.

Flashpoint duration: The length of time of the first shock administered.

The instrument used to measure aggression is an aggression console, a white metal box mounted with electrical switches and light emitting diodes (LEDs). Ten shock push buttons labeled “1” through “10” are arranged horizontally on the console. Shocks are generated by a Precision Regulated Animal Shocker (Coulbourn Instruments, Allentown, Pa). The shock unit

features series resistance-regulation, which can never deliver more current than the total circuit resistance predicates. The set accuracy is controlled by a fixed series resistor. The unit does not require calibration. For added safety, a shock level tester is connected to the output in order to verify accuracy. In addition, electrodes are never placed to form a path across the chest, head, neck, or abdomen. A reaction time switch is located at the center of the console.

Procedure

Participants attended two sessions: a group screening session and a laboratory session. During the screening, participants completed the consent form, POMS, CIS, SHS, B-MAST, PANAS, and Permission to Contact form. The Research Participant pool study title was "Sleep, Mood, and Reaction Time." Participants were informed in the screening consent form that some of them would be invited to participate in the second part of the study and that they may be required to restrict sleeping time to 4 hours the night before the laboratory session. Participants who indicated on the B-MAST that they have a history of alcohol problems (B-MAST score > 6) were excluded. Other exclusion criteria included: regular use of alcohol as a sleep aid or sleeping pills (more than one night per week), falling asleep at inappropriate times or places (with the exception of in class) more than one time per week, difficulty falling or staying asleep more than three times per week, working at night or rotating shift, having sufficient level of symptomatology to indicate likelihood of a sleep disorder, and endorsement of a major medical condition (e.g., asthma, epilepsy), or current treatment of psychiatric disorder (e.g., depression, ADHD). Sleep disorder symptoms were examined in clusters, and participants who endorsed occurrence of two or more symptoms more than three times per week within a particular category were excluded from consideration for the laboratory study. In order to eliminate heavy

smokers, regular smokers who smoked twenty cigarettes per day or more were also eliminated from consideration, as nicotine is a stimulant and may interfere with mood and quality of sleep.

Participants invited to the laboratory phase of the experiment were randomly assigned to sleep-deprived and non-sleep-deprived groups by coin flip. Sleep-deprived participants were instructed to remain awake until 4 hours prior to their usual arising time on the day of the laboratory session. They were given sleep restriction instructions at scheduling as well as on the day prior to the laboratory session. Participants were instructed not to consume caffeinated beverages, alcohol, or nicotine within 12 hours of the scheduled laboratory session. Compliance with the sleep restriction procedure was monitored. Participants in the sleep-deprived group were required to call the laboratory and leave a message every half hour on an answering machine that digitally recorded the time of the message. They followed this procedure between their normal bedtime and just prior to going to sleep, and again upon awakening. Efforts were made to schedule participants for the laboratory session within 2 hours of their wake time. Participants who did not call at wake time were eliminated from eligibility to complete the laboratory session. Participants were informed that if they did not comply with the telephone calls, they were ineligible to complete the second part of the study. Control participants were asked to call immediately prior to sleeping and upon awakening. Answering machine recordings were checked prior to allowing participants into the experimental session.

When participants arrived at the laboratory, they were led to the experimental chamber, a sound-attenuated room adjacent to a similar chamber, the door of which was slightly ajar to imply the presence of another participant. They completed the consent form, the POMS, and the PANAS. Upon completion of questionnaires, participants were given instructions about the rules of the "competition." In order to portray the task as something other than an aggression task,

they were told that the study's purpose was to understand the relationship between personality, sleep, and reaction time. If participants asked whether their opponent was sleep deprived, they were informed that they were matched according to condition.

Per the instructions, the experimenter informed participants that when a yellow "press" light illuminates on the console, they were to depress and hold the RT key. Shortly after the RT key was depressed, a green "release" light illuminated, and participants were to release the RT key as quickly as possible. After a 3-second result-determination period, a green "win" light or a red "lose" light illuminated, informing participants about the outcome of that trial. Participants were told that they had the choice to deliver shocks to the opponent as "punishment" following trials they "win" or "lose" and were at liberty to do so as often as they desired throughout the task. Participants were told that the opponent had the same options. Participants were also told that they may refrain from administering any shocks during the 30 trials of the experiment. Shocks administered to the participants were accompanied by visual feedback via LEDs paralleling the level of each shock administered.

Participants were seated at the console. Following the explanation of the "rules," a sufficient amount of time lapsed to allow them to think that the competitor was receiving identical instructions. Next, the participants' pain thresholds were assessed. In order to further enhance the deception that another participant was present, a tape recording of a confederate reading a list of predetermined responses (e.g., "discomforting," "painful") was played through the intercom. Participants' pain thresholds were assessed by administering incrementally stronger shocks to the second and third fingers of the non-dominant hand. Shocks began at an imperceptible level and were increased to a level described by participants as "painful." During the competition, participants received shocks of 0.50 s at the level that they described as painful.

The entire competition consisted of 30 trials interspaced at 5-s intervals. The win-loss pattern was predetermined such that participants won half the trials and lost half of them as the task progressed. Initiation of trials, recording of the participants' responses, and shock administration were controlled by microcomputer. Upon completion of the trials, participants were thanked, debriefed, given research participation credit, and dismissed.

In order to verify the success of the deception, participants were asked a series of questions about their opponents prior to debriefing. Questions included, "What is your impression of your opponent Philip?", "Was he fair?", and "Was the task a good test of reaction time?" This manipulation check has proven successful in past studies. Participants who responded with suspicion were excluded from analyses.

CHAPTER 3

Results

While previous research has found that sleep deprivation may increase negative affect and negative affect, particularly irritability, can lead to aggressive behavior, the link between sleep deprivation and aggression has not yet been investigated. The purpose of the analyses was to determine whether the following model was true: that sleep deprivation leads to aggression, and that this relationship is affected by the individual's level of negative affect (see Figure 1). This research question may best be answered by a mediation model.

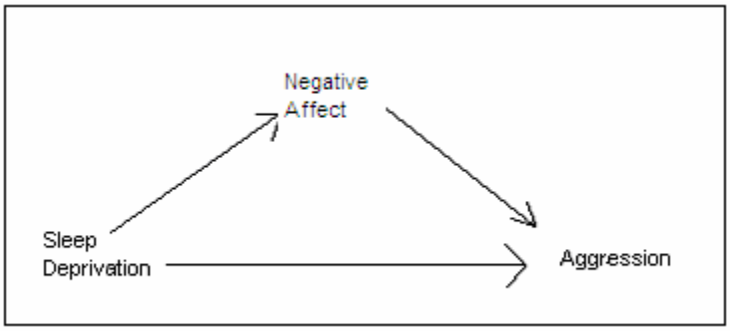

Figure 1. Mediation model of sleep deprivation, negative affect, and aggression

According to the method of determining mediation as specified by Baron and Kenney (1986), three regression analyses were performed: one to determine whether sleep deprivation relates to aggression, one to determine whether trait irritability and state negative affect relate to aggression, and one to determine whether trait and state negative affect and sleep deprivation relate to aggression.

Aggression and Sleep Deprivation

Because independent variables in regression equations can be either categorical or continuous, a simple linear regression was sufficient to determine the effect of sleep deprivation on aggression (Pedhauzer, 1997). The seven indices of aggression were considered separately in the regression equations, as no valid method of combining them has been demonstrated. It has been proposed that Shock Intensity, Shock Frequency, and Proportion of Highest Shock may represent direct measures of aggression (e.g., slapping or hitting). Shock Duration may demonstrate more indirect aggressive means (e.g., giving someone an anonymous negative evaluation). Finally, the flashpoint indices (i.e. Flashpoint Latency, Flashpoint Intensity, and Flashpoint Duration) may represent an individual's level of aggression upon engagement in an interaction.

Standardized coefficients and significance levels for condition on the seven indices of aggression are in Table 1. As can be seen in Table 1, Acute Partial Sleep Deprivation significantly affected Flashpoint Duration and Shock Frequency.

Aggression and Irritability

As Pedhauzer (1997) notes, when only one variable is regressed on another variable, a Pearson product-moment coefficient is equivalent to a single linear regression. Consequently, correlation coefficients were computed for measures of aggression and irritability. Trait irritability significantly correlated with Proportion of Highest Shock ($r = .24$, $p < .05$, one-tailed).

Table 1

Linear Regression Analysis Results for Effects of Acute Partial Sleep Deprivation on the Seven Measures of Aggression (N = 73)

Aggression Measures	*B*	*SE B*	β
Shock Intensity	-0.62	0.52	-.14
Shock Duration	-351.27	187.09	-.22
Proportion of Highest Shock	-0.04	0.06	-.09
Shock Frequency	-0.13	0.06	-.26*
Flashpoint Latency	1.36	1.55	.10
Flashpoint Intensity	-0.17	0.73	-.03
Flashpoint Duration	-321.48	148.47	-.25*

* $p < .05$.

Sleep Deprivation, Irritability, and Stated Negative Affect

In order to determine whether participants in the sleep-deprived group endorsed higher levels of negative affect than non-sleep-deprived participants, their scores on the six scales of the POMS, which were administered at screening and at the laboratory session, were compared. No differences between groups were noted for baseline screening scores, and analyses did not indicate any ceiling or floor effects. Paired-samples t-tests revealed that, as was hypothesized, sleep-deprived participants' scores increased on the Tension-Anxiety and Fatigue-Inertia subscales of the POMS between screening and laboratory sessions (see Table 2). They also experienced a reduction in scores on the Vigor-Activity subscale of the POMS and the Positive Affect subscale of the PANAS. Contrary to the third hypothesis, there was no significant change in the Anger-Hostility subscale, which is significantly correlated with the other negative

affect scales of the POMS. Also contrary to the third hypothesis, participants in the control group scored higher on the second administration of the Tension-Anxiety subscale of the POMS and the Negative Affect subscale of the PANAS. They also scored lower on the Fatigue-Inertia subscale (see Table 2).

The increase in Tension-Anxiety scores across groups could be a result of answering the second questionnaire in an unfamiliar laboratory environment whereas the first administration of the questionnaire occurred in a classroom. With regard to the decrease in scores on the Vigor-Activity subscale and increase in scores on the Fatigue-Inertia subscale in the APSD group, it appears as though sleep-deprived participants experienced lowered energy and increased sleepiness than did the control participants. Indeed, independent samples t-tests revealed that participants who had obtained at least 7 hours of sleep scored significantly higher on the Vigor-Activity subscale of the POMS and the Positive Affect subscale of the PANAS than APSD participants. APSD participants' scores on the Fatigue-Inertia subscale of the POMS were higher than those in the Control group (see Table 2).

Intercorrelations among the six subscales of the POMS, the two subscales of the PANAS, and Trait Irritability were computed from the screening sample. Total Trait Irritability was significantly correlated with all subscales of the POMS with the exception of Vigor-Activity (see Table 3). It was also positively correlated with the PANAS Negative Affect subscale and negatively correlated with the PANAS Positive Affect Subscale.

Aggression, Sleep Deprivation, and Irritability

As trait irritability as measured by the Caprara Irritability Scale did not affect the same aggression measures as did acute partial sleep deprivation, the first requirement of Baron and

Kenney's (1986) mediation analysis was not satisfied. Therefore, a hierarchical regression analysis with APSD and trait irritability could not be computed.

Table 2

Paired-samples Test Results for Participants on POMS and PANAS Subscales (N = 73)

Subscale	Pre-PSD Mean (SD)	Post-PSD Mean (SD)	t
Tension-Anxiety			
APSD	2.56 (5.65)	4.64 (5.04)	-2.34*
Control	1.49 (3.91)	3.27 (5.03)	-2.63**
Depression-Dejection			
APSD	3.89 (5.18)	3.14 (4.19)	0.86
Control	3.97 (5.31)	3.30 (6.22)	0.85
Anger-Hostility			
APSD	3.26 (3.99)	3.34 (4.17)	-0.12
Control	3.27 (4.37)	2.78 (4.46)	0.78
Vigor-Activity			
APSD	15.11 (7.49)	9.86 (6.45)[a]	5.32†
Control	16.11 (5.13)	14.81 (6.21)[a]	1.80
Fatigue-Inertia			
APSD	8.83 (5.78)	14.36 (5.93)[b]	-5.41†
Control	7.22 (5.32)	4.08 (5.28)[b]	4.01**
Confusion-Bewilderment			
APSD	6.19 (3.12)	6.25 (3.30)	-0.11
Control	6.22 (3.14)	5.43 (3.11)	1.72
Positive Affect			
APSD	27.77 (8.69)	23.06 (8.27)[c]	4.30†
Control	28.35 (7.52)	28.22 (7.69)[c]	.16
Negative Affect			
APSD	12.26 (2.94)	13.43 (3.53)	-1.70
Control	10.84 (2.04)	12.16 (2.73)	-3.75†

* $p < .05$. ** $p < .01$. † $p < .001$. [a] $t = -3.29$, $p < .01$. [b] $t = 7.83$, $p < .001$. [c] $t = -2.74$, $p < .01$.

Table 3

Intercorrelations among POMS and PANAS Subscale Scores and Total Trait Irritability (N = 240)

Subscale:	1	2	3	4	5	6	7	8	9
1. Tension-Anxiety	--	.59**	.66**	-.02	.51**	.67**	-.03	.60**	.29**
2. Depression-Dejection		--	.78**	-.23**	.57**	.63**	-.15*	.67**	.37**
3. Anger-Hostility			--	-.13	.52**	.61**	-.11	.68**	.45**
4. Vigor-Activity				--	-.33**	.04	.74**	-.13*	-.12
5. Fatigue-Inertia					--	.55**	-.26**	.45**	.31**
6. Confusion-Bewilderment						--	.03	.56**	.26**
7. Positive Affect							--	.03	-.56**
8. Negative Affect								--	.36**
9. Total Trait Irritability									--

* $p < .05$. ** $p < .01$.

Aggression, Sleep Deprivation, and Negative Affect

As state negative affect has been found to be the expression of trait negative affect in several aggression studies (e.g., Lindsey & Anderson, 2000), the subscale scores of the POMS and PANAS were correlated with the aggression measures. Results are listed in Table 4. Significant coefficients indicated that the Vigor-Activity subscale score was negatively correlated with Shock Intensity and Shock Frequency and positively correlated with Flashpoint Latency. The Fatigue-Inertia subscale score was positively correlated with Shock Frequency and

Flashpoint Latency. Finally, the Negative Affect subscale score of the PANAS was positively correlated with Shock Frequency and negatively correlated with Flashpoint Latency.

Table 4

Pearson Product-Moment Correlations between Profile of Mood States and Positive and Negative Affect Schedule Subscale Scores and Aggression Measures (N = 73)

	Aggression Measures						
Subscale:	MSI	MSD	P10	PCS	FPL	FPI	FPD
1. Tension-Anxiety	.08	.00	.02	.04	-.10	-.08	.05
2. Depression-Dejection	.08	.09	.04	-.11	-.13	.03	- .15
3. Anger-Hostility	.08	.06	.17	.10	-.17	.01	-.10
4. Vigor-Activity	-.25*	.15	- .17	-.23*	.23*	-.21	-.10
5. Fatigue-Inertia	.16	.08	.10	.34**	-.27**	.03	.07
6. Confusion-Bewilderment	.00	.00	.00	.09	-.01	-.02	-.14
7. Positive Affect	-.10	.11	-.15	-.13	.02	-.10	-.08
8. Negative Affect	.13	.03	.05	.20*	-.23*	.10	.03

Note: MSI = shock intensity; MSD = shock duration; P10 = proportion of highest shock; PCS = shock frequency; FPL = flashpoint latency; FPI = flashpoint intensity; FPD = flashpoint duration; all correlations tested for significance using a one-tailed test.

* $p < .05$. ** $p < .01$.

Acute partial sleep deprivation predicted Shock Frequency, and Fatigue-Inertia was significantly correlated with Shock Frequency. Thus, a hierarchical regression analysis was used to determine whether Fatigue-Inertia mediated the relationship between APSD and Shock Frequency (due to the high intercorrelation among scores on the Fatigue-Inertia , Vigor-Activity,

and Negative Affect subscales, only Fatigue-Inertia was examined in the context of the regression equation). The unique proportion of variance accounted for by an independent variable above and beyond that of another independent variable can be determined by entering the variable of interest into a regression equation last (Pedhauzer, 1997). APSD was entered into the regression equation to predict Shock Frequency and was responsible for a significant portion of the variance (See Table 5). As mentioned previously, Fatigue-Inertia was significantly correlated with Shock Frequency. When both APSD and Fatigue-Inertia were entered into the equation, the effects of APSD virtually disappeared. Thus, as the fifth hypothesis posited, Fatigue-Inertia appears to mediate the relationship between APSD and Shock Frequency (see Figure 2).

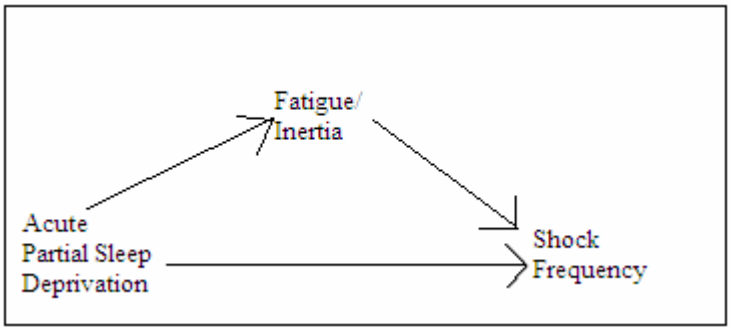

Figure 2. Mediation model of APSD, Fatigue-Inertia, and Shock Frequency

Sleep Disorder Symptoms, Negative Affect, and Aggression

During the screening session, participants completed the Sleep Habits Scale, which assessed sleep habits and sleep disorder symptoms. Average bedtime for weekdays was approximately 2:30 a.m. and average bedtime for weekends was approximately 3:00 a.m. Average arising time was 9:00 for weekdays and 11:30 for weekends. Participants endorsed napping on 1.81 days per week (*SD* = 1.57). They reported obtaining a mean of 7.36 (*SD* = 1.29)

hours of sleep on weeknights and 8.63 (*SD* = 1.56) hours on weekends. Average amount of sleep obtained the night prior to the screening session was 7.10 (*SD* = 1.82) hours.

Table 5

Summary of Hierarchical Regression Analysis for Variables Predicting Shock Frequency (N = 73)

Variable	*B*	*SE B*	β
Step 1			
Acute Partial Sleep Deprivation	-0.13	0.06	-.26*
Step 2			
Fatigue-Inertia	.01	.01	.37*
Acute Partial Sleep Deprivation	-.01	.07	-.01

* $p < .05$.

Surprisingly, 97.9 % of participants at the screening endorsed experiencing one or more symptoms of sleep disorders. The mean number of symptoms endorsed for the entire sample was 4.71 (*SD* = 2.61), and they indicated that they experience sleep disorder symptoms an average of 12.9 (*SD* = 9.68) times per week. Thus, it appears that symptoms of sleep disorders are extremely common and may interfere with obtaining good quality sleep in a majority of undergraduate men. These data were analyzed to determine whether regularly obtaining poor sleep or not sleeping enough would be associated with negative affect. Consequently, two variables were created, Sleep Symptom Frequency (SSFreq), or total number of sleep symptoms endorsed, and Sleep Symptom Severity (SSSev), or total times per week symptoms of sleep disorders occurred. As this was a post-hoc analysis, a Bonferroni correction was applied to

determine a new required level of significance for 30 comparisons ($p < .002$). As can be seen in Table 6, frequency and severity of sleep disorder symptoms were significantly positively correlated with trait irritability as well as with subjective experience of negative affect including tension, anxiety, anger, depression, and confusion.

Pearson product-moment correlations were also computed between poor sleep hygiene habits, sleep disordered symptomatology, and subjective experience of negative affect (Table 7). Significant coefficients indicated that use of alcohol as a sleep aid was positively correlated with number and severity of sleep symptoms and use of sleeping pills was positively correlated with subjective depression and anger.

Frequency and severity of sleep disorder symptoms were examined within the laboratory subjects to ascertain whether number of sleep symptoms endorsed and number of nights per week during which sleep symptoms were experienced were correlated with the aggression measures of the RCAP. Indeed, frequency and severity of sleep disorder symptoms were significantly correlated with Shock Frequency ($r = .27$, $p < .05$ and $r = .36$, $p < .01$, respectively) and Flashpoint Latency ($r = -.25$, $p < .05$ and $r = -.27$, $p < .05$, respectively).

Table 6

Correlations between POMS and PANAS Subscale Scores and Sleep Symptomatology (N = 240)

	Sleep Symptoms	
Scale:	SSFreq	SSSev
1. Tension-Anxiety	.31†	.24†
2. Depression-Dejection	.27†	.23†
3. Anger-Hostility	.27†	.17
4. Vigor-Activity	.00	.02
5. Fatigue-Inertia	.15	.13
6. Confusion-Bewilderment	.32†	.28†
7. Total Trait Irritability	.21†	.15
8. Positive Affect	.03	.00
9. Negative Affect	.32†	.24†

Note: SSFreq = number of sleep symptoms endorsed; SSSev = number of sleep symptoms experienced per week.

† $p < .002$

Table 7

Correlations between POMS and PANAS Subscale Scores, Sleep Symptomatology, and Poor Sleep Hygiene (N = 240)

	Poor Sleep Hygiene				
Scale:	Naps	Pills	ETOH	Nicotine	Caff
1. Tension-Anxiety	.01	.18	.12	.08	.07
2. Depression-Dejection	.02	.21†	.14	.04	.07
3. Anger-Hostility	.01	.27†	.14	.03	.05
4. Vigor-Activity	-.06	-.08	.05	-.06	.01
5. Fatigue-Inertia	.16	.14	-.02	.01	.02
6. Confusion-Bewilderment	.09	.03	.13	.01	.03
7. Total Trait Irritability	.09	.12	.18	.11	.08
8. Positive Affect	-.10	-.09	-.04	-.19	-.08
9. Negative Affect	.08	.15	.13	.05	.04
10. SSFreq	-.01	.19	.27†	.13	.09
11. SSSev	.07	.06	.27†	.17	.09

Note: Naps = number of naps per week; Pills = number of nights of sleeping pill use; ETOH = alcohol as a sleep aid; Nicotine = cigarettes smoke per day; Caff = caffeinated beverages after dinner; SSFreq = number of sleep symptoms; SSSev = number of sleep symptoms experienced per week.

† $p < .002$

CHAPTER 4

Discussion

Sleep deprivation has been typically associated with effects such as lowered motivation (Dinges & Kribbs, 1991) and, thus, has not been tested as a potential factor in aggressive behavior. However, due to sleep deprivation's dampening effects on the prefrontal cortex, similar to those of alcohol, it may act indirectly to inhibit reduction of aggressive impulses, particularly under conditions of high provocation (Giancola, 2002). Following the cognitive-neoassociationistic model of aggression, it was hypothesized that acute partial sleep deprivation would influence aggression and be mediated by irritability and negative affect.

As was hypothesized, acute partial sleep deprivation significantly affected two measures of aggression: shock frequency and flashpoint duration. Also, trait irritability was correlated with the proportion of highest shock. As trait irritability was not correlated with either of the aggression measures affected by acute partial sleep deprivation, it could not be tested as a mediating variable between APSD and aggression. Another model with Acute Partial Sleep Deprivation as the independent variable, Shock Frequency as the dependent variable, and Fatigue-Inertia as the mediating variable was tested and found to be accurate. Thus, APSD appears to have caused participants to feel fatigued, which increased the frequency of administered shocks.

Previous studies that used trait irritability as a predictive variable in aggression paradigms have found it to be related to Mean Shock Intensity (e.g., Caprara et al., 1983; Caprara et al., 1986; Giancola, 2002) and to Mean Shock Duration and Proportion of Highest Shock (Parrott &

Zeichner, 2001). This study confirms previous research in that trait irritability was positively correlated with Proportion of Highest Shock. Previous studies have found that trait negative affect is expressed through state negative affect (e.g. Lindsey & Anderson, 2000). In this study, trait irritability appears to have been expressed through Fatigue-Inertia, with which it is correlated. The POMS subscales associated with subjective tiredness and energy level showed the most consistent effects on aggressive behavior. The Vigor-Activity subscale was not significantly correlated with irritability but was negatively correlated with Fatigue-Inertia and had the opposite effect on Shock Frequency and Flashpoint Latency as did Fatigue-Inertia. Thus, in the current study, irritability may have led to increased subjective fatigue in partially sleep-deprived participants. Additionally, participants who endorsed regularly disturbed sleep through number and frequency of sleep disorder symptoms obtained higher scores on Shock Frequency and demonstrated lower Flashpoint Latency.

The total mediation effect by the Fatigue-Inertia subscale of the POMS proved to be a novel finding, as low energy has typically been associated with less aggression. However, the measure of aggression affected was not a variable associated with severity, but, rather, with frequency. In other words, participants who felt tired took more frequent opportunities to act aggressively. These participants also shocked sooner, although this result was not associated with partial sleep deprivation. Perhaps individuals who are sleep-deprived and tired do not seek opportunities to act aggressively, but take advantage of those that are presented to them (e.g., road rage).

As was expected, participants in the acute partial sleep deprivation group scored higher on the second administration of the Tension-Anxiety and Fatigue-Inertia subscales of the POMS. It also decreased scores on the Vigor-Activity subscale. APSD did not, however, increase scores

on the Anger-Hostility subscale as expected. Thus, it appears that partially sleep-deprived participants felt more fatigued and less energetic at the laboratory session than at the screening session. Control group participants endorsed less fatigue than the APSD participants. As such, it is apparent that, at the laboratory session, there was a true difference in subjective discomfort due to limited sleep obtained. Interpretation regarding the increase in the Tension-Anxiety subscale scores is limited due to the increase in scores on that subscale in the control group. The difference in scores on that subscale from screening to laboratory session may be attributed to the change in environment in which the questionnaire was administered or other uncontrolled effects. While the screening session occurred in a classroom, the experimental session was held in a sound-attenuated chamber containing an unfamiliar apparatus. At the experimental session, prior to completion of the POMS and PANAS, participants signed a consent form that mentioned that they may be shocked (see Appendix G). Thus, higher levels of tension and anxiety may be better explained by anxious anticipation and environmental influences.

Although emotions such as anger, irritation, and annoyance are typically associated with aggressive acts, Berkowitz (1993) postulated that all types of negative affect may increase the likelihood of direct and indirect aggressive responding. In the present study, fatigue served as a mediating variable in the relationship between acute partial sleep deprivation and the aggression measure of shock frequency. This finding is consistent with the Cognitive-Neoassociationistic model of aggression (Berkowitz, 1993, 2001). Sleep deprivation served as the aversive event that produced negative affect. APSD may have also interfered with the activation of cognitive processes through its dampening effects on the prefrontal cortex. The negative affect and availability of a target resulted in increased aggressive responding in those who were partially

sleep deprived. In addition, as all types of negative affect may lead to aggressive acts, fatigue and sleepiness may have caused participants to act more aggressively.

As was mentioned above, flashpoint duration, or the length of time participants chose to administer the first shock, may be conceptualized as a measure of the severity of the first aggressive act in which an individual chooses to engage. While not as direct as flashpoint intensity, it indicates that a sleep-deprived person may choose to wait longer to disengage from an initial aggressive act than a non-sleep-deprived individual. One alternate explanation for increase in flashpoint duration may be slowed motor behavior due to APSD. However, motor tasks are the least affected by sleep-deprivation (Pilcher & Huffcutt, 1996), and in the present study, overall shock duration was not affected by APSD. Although the Anger-Hostility subscale of the POMS was not correlated with flashpoint duration, the POMS was administered at the beginning of the laboratory session, and feelings of anger may have been activated by the provocation and augmented by acute partial sleep deprivation.

Shock frequency has been hypothesized to be a direct index of aggression. Sleep-deprived individuals may take advantage of more opportunities to aggress than those who have obtained the recommended amount of sleep, and discomfort due to sleepiness appears to be at the root of this choice. Inhibition of aggressive responding may have been due to the effects of sleep deprivation on physiological processes that decrease aggressive impulses to the frontal cortex (Thomas et al., 2000).

The need for sleep varies widely. Some individuals require as many as 9 to 10 hours of sleep per night; others need as little as 6 or 7 (Morin, 1993). A recent review of studies indicated that young adults sleep an average of 7.5 hours on weeknights and 8.5 hours on weekends (Carskadon & Dement, 2005). While this is consistent with the screening data, many of the

participants in the present study regularly obtained less than optimal total sleep times, and over half indicated that their sleep may have been disrupted by sleep-disordered symptoms. Not surprisingly, number and severity of sleep-disorder symptoms were correlated with the Tension-Anxiety, Depression-Dejection, Anger-Hostility, and Confusion-Bewilderment subscales of the POMS. Furthermore, a recent study found that caffeine does not enhance mood in sleep restricted participants (James & Gregg, 2004), so this popular method of combating the effects of sleep deprivation is ineffective.

The present study has some limitations. As Dement (2005) notes, it is impossible for the researcher and participants in sleep deprivation studies to be blind to whether they are or are not sleep-deprived, and so the influence of some experimenter bias is possible. While every effort was made to ensure that participants were sleeping during the times they reported, no physiological measures were taken to ensure the accuracy of their self-report. Also, a possible environmental confound was discovered regarding change in scores on the Tension-Anxiety subscale of the POMS for both the experimental and control groups. POMS and PANAS subscale scores may have changed in both groups due to change of testing environment or other uncontrolled factors, as baseline mood ratings were often taken as much as a week before laboratory session. Finally, negative affect was only measured before and not during the RCAP, so emotions that may have been activated by provocation were not measured or analyzed.

In spite of its limitations, the current study makes a novel contribution to the literature on aggression with regard to the scope of factors that may lead to negative affect and, with them, aggressive actions. The present mediation models demonstrate that an individual's tiredness may increase the likelihood that a person will take the opportunity to act aggressively and may not quickly disengage from the interaction. As alcohol and sleep deprivation seem to act in

similar ways on the prefrontal cortex, the implications for individuals who do not obtain adequate sleep and imbibe alcoholic beverages are interesting areas for further exploration, particularly in light of the findings from the screening data wherein a vast majority of individuals endorsed symptoms of sleep disorders and concurrent maladaptive means used to address them (e.g., alcohol as a sleep aid or sleeping pills) were associated with measures of negative affect. Finally, the identification of acute partial sleep deprivation, an extremely common event in our society, as a valid aversive event in the Cognitive-Neoassociationistic model of aggression has implications for health education.

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APPENDIX A

Profile of Mood States

Below is a list of words that describe feelings people have. Please read each one carefully, then circle the number under the answer that best describes HOW YOU ARE FEELING RIGHT NOW using the following scale:

0 – Not at all
1 – A little
2 – Moderately
3 – Quite a bit
4 – Extremely

1. Friendly	0 1 2 3 4	16. On edge	0 1 2 3 4
2. Tense	0 1 2 3 4	17. Grouchy	0 1 2 3 4
3. Angry	0 1 2 3 4	18. Blue	0 1 2 3 4
4. Worn out	0 1 2 3 4	19. Energetic	0 1 2 3 4
5. Unhappy	0 1 2 3 4	20. Panicky	0 1 2 3 4
6. Clear-headed	0 1 2 3 4	21. Hopeless	0 1 2 3 4
7. Lively	0 1 2 3 4	22. Relaxed	0 1 2 3 4
8. Confused	0 1 2 3 4	23. Unworthy	0 1 2 3 4
9. Sorry for things done	0 1 2 3 4	24. Spiteful	0 1 2 3 4
10. Shaky	0 1 2 3 4	25. Sympathetic	0 1 2 3 4
11. Listless	0 1 2 3 4	26. Uneasy	0 1 2 3 4
12. Peeved	0 1 2 3 4	27. Restless	0 1 2 3 4
13. Considerate	0 1 2 3 4	28. Unable to concentrate	0 1 2 3 4
14. Sad	0 1 2 3 4	29. Fatigued	0 1 2 3 4
15. Active	0 1 2 3 4	30. Helpful	0 1 2 3 4

31. Annoyed	0	1	2	3	4	51. Alert	0	1	2	3	4
32. Discouraged	0	1	2	3	4	52. Deceived	0	1	2	3	4
33. Resentful	0	1	2	3	4	53. Furious	0	1	2	3	4
34. Nervous	0	1	2	3	4	54. Efficient	0	1	2	3	4
35. Lonely	0	1	2	3	4	55. Trusting	0	1	2	3	4
36. Miserable	0	1	2	3	4	56. Full of pep	0	1	2	3	4
37. Muddled	0	1	2	3	4	57. Bad tempered	0	1	2	3	4
38. Cheerful	0	1	2	3	4	58. Worthless	0	1	2	3	4
39. Bitter	0	1	2	3	4	59. Forgetful	0	1	2	3	4
40. Exhausted	0	1	2	3	4	60. Carefree	0	1	2	3	4
41. Anxious	0	1	2	3	4	61. Terrified	0	1	2	3	4
42. Ready to Fight	0	1	2	3	4	62. Guilty	0	1	2	3	4
43. Good-natured	0	1	2	3	4	63. Vigorous	0	1	2	3	4
44. Gloomy	0	1	2	3	4	64. Uncertain about things	0	1	2	3	4
45. Desperate	0	1	2	3	4	65. Bushed	0	1	2	3	4
46. Sluggish	0	1	2	3	4						
47. Rebellious	0	1	2	3	4						
48. Helpless	0	1	2	3	4						
49. Weary	0	1	2	3	4						
50. Bewildered	0	1	2	3	4						

APPENDIX B

Caprara Irritability Scale

Circle "T" if the statement is generally true for you or "F" if it is not.

T F 1. I easily fly off the handle with those who don't listen or understand.

T F 2. I am often in a bad mood.

T F 3. Usually when someone shows a lack of respect for me, I let it go by.

T F 4. I have never been touchy.

T F 5. It makes my blood boil to have somebody make fun of me.

T F 6. I think I have a lot of patience.

T F 7. When I am irritated I need to vent my feelings immediately.

T F 8. When I am tired I easily lose control.

T F 9. I think I am rather touchy.

T F 10. When I am irritated I can't tolerate discussions.

T F 11. I could not put anyone in his place, even if it were necessary.

T F 12. I can't think of any good reason for resorting to violence.

T F 13. I often feel like a powder keg ready to explode.

T F 14. I seldom strike back even if someone hits me first.

T F 15. I can't help being a little rude to people I don't like.

T F 16. Sometimes when I am angry I lose control over my actions.

T F 17. I do not know of anyone who would wish to harm me.

T F 18. Sometimes I really want to pick a fight.

T F 19. I do not like to make practical jokes.

T F 20. When I am right, I am right.

T F 21. I never get mad enough to throw things.

T F 22. When someone raises his voice, I raise mine higher.

T F 23. Sometimes people bother me just by being around.

T F 24. Some people irritate me if they just open their mouth.

T F 25. Sometimes I shout, hit and kick and let off steam.

T F 26. I don't think I am a very tolerant person.

T F 27. Even when I am very irritated I never swear.

T F 28. It is others who provoke my aggression.

T F 29. Whoever insults me or my family is looking for trouble.

T F 30. It takes very little for things to bug me.

APPENDIX C

Sleep Habits Scale

1. Demographic Information:

Race:

_____ Caucasian
_____ African/Black
_____ Asian
_____ Hispanic
_____ Native American

Age: _____

Marital Status:

_____ Single
_____ Committed Relationship (living with someone, but not married)
_____ Married
_____ Divorced
_____ Widowed

Years of Education: _____

Income:

_____ $0 – 10,000
_____ $10,000-20,000
_____ $20,000-30,000
_____ $30,000-40,000
_____ $40,000-50,000
_____ $50,000-60,000
_____ $60,000-70,000
_____ $70,000-80,000
_____ $80,000+

2. Current Sleep-Wake Schedule

What is your usual **bed**time on Sunday? _____ o'clock

What is your usual **arising** time on Monday? _____ o'clock

What is your usual **bed**time on Monday? _____ o'clock

What is your usual **arising** time on Tuesday? _____ o'clock

What is your usual **bed**time on Tuesday? _____ o'clock

What is your usual **arising** time on Wednesday? _____ o'clock

What is your usual **bed**time on Wednesay? _____ o'clock

What is your usual **arising** time on Thursday? _____ o'clock

What is your usual **bed**time on Thursday? _____ o'clock

What is your usual **arising** time on Friday? _____ o'clock

What is your usual **bed**time on Friday? _____ o'clock

What is your usual **arising** time on Saturday? _____ o'clock

What is your usual **bed**time on Saturday? _____ o'clock

What is your usual **arising** time on Sunday? _____ o'clock

How often do you take naps? _____ days/week

On a typical night (past month), how long does it take you to fall asleep after you go to bed and turn the lights off? _____ hours _____minutes

How many hours of sleep per weeknight do you usually get? _____ hours

How many hours of sleep per weekend night do you usually get? _____ hours

How many hours of sleep did you get last night? _____ hours

Do you use sleeping pills? Yes No

How many nights per week? _____ nights/week

Do you use alcohol as a sleep aid? Yes No

How many nights per week? _____ nights/week

3. Eating/exercise/substance use:

How many times per week do you exercise? _____ times/week

Do you sometimes exercise prior to bedtime? Yes No

How many caffeinated beverages do you drink per day? ____ caffeinated beverages/day

How many caffeinated beverages do you drink after dinner? ____ caffeinated beverages

How many cigarettes per day do you smoke? ____ cigarettes/day

4. *Sleep problems*:

Do you ever fall asleep at inappropriate times/places? Yes No
Where? _____
How often? _____times/week

How many nights/week do you have a problem with falling/staying asleep? _____ days/week

When you have trouble falling/staying asleep, how long does it take you to fall asleep? _____ hours _____minutes

When you have trouble staying asleep, how often do you wake up? _____ times/night

When you have trouble staying asleep, how long are your periods of being awake? _____ hours _____minutes

Do you work at night or rotating shift? Yes No

Have you ever noticed one of the following? How many times per week does it occur?

___ Crawling or aching feelings in the legs (calves)	____ times/week
___ Inability to keep legs still at night	____ times/week
___ Leg twitches or jerks during the night	____ times/week
___ Waking up with cramps in legs	____ times/week
___ Snoring	____ times/week
___ Pauses in breathing at night	____ times/week
___ Shortness of breath	____ times/week
___ Choking at night	____ times/week
___ Morning headaches	____ times/week
___ Chest pain	____ times/week
___ Dry mouth	____ times/week
___ Falling asleep in the middle of sentences	____ times/week
___ Inability to move for a few seconds upon awakening	____ times/week
___ Vivid dreams as you are falling asleep	____ times/week
___ Falling asleep or feeling weak when you hear a funny joke or get very emotional	____ times/week
___ Sour taste in mouth, heartburn, reflux	____ times/week
___ Nightmares	____ times/week

___ Night terrors ____ times/week

___ Sleepwalking/talking ____ times/week

___ Grinding your teeth ____ times/week

5. *Medical/Psychiatric History:*

Current medical problems: _____

Are you currently receiving psychological
or psychiatric treatment for emotional or mental
health problems? Yes No

Which one(s)? _____

Have you or anyone in your family ever been treated
For emotional or mental health problems in the past? Yes No

Who, and which one(s)? _____

APPENDIX D

Brief Michigan Alcoholism Screening Test

1. Do you feel you are a normal drinker?	YES	NO
2. Do friends or relatives think you are a normal drinker?	YES	NO
3. Have you ever attended a meeting of Alcoholics Anonymous (AA)?	YES	NO
4. Have you ever lost friends or girlfriends/boyfriends because of drinking?	YES	NO
5. Have you ever gotten into trouble at work because of drinking?	YES	NO
6. Have you ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking?	YES	NO
7. Have you ever had delirium tremens (DTs), severe shaking, heard voices, or seen things that weren't there after heavy drinking?	YES	NO
8. Have you ever gone to anyone for help about your drinking?	YES	NO
9. Have you ever been in a hospital because of drinking?	YES	NO
10. Have you ever been arrested for drunk driving or driving after drinking?	YES	NO

APPENDIX E

Positive and Negative Affect Schedule

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you *feel* this way right now, that is, *at the present moment.* Use the following scale to record your answers.

1 very slightly **2 a little** **3 moderately** **4 quite a bit** **5 extremely**

_____ interested	_____ irritable
_____ distressed	_____ alert
_____ excited	_____ ashamed
_____ upset	_____ inspired
_____ strong	_____ nervous
_____ scornful	_____ determined
_____ guilty	_____ loathing
_____ scared	_____ attentive
_____ hostile	_____ jittery
_____ enthusiastic	_____ active
_____ angry	_____ afraid
_____ proud	_____ disgusted

APPENDIX F

SCREENING CONSENT FORM

I, _____ agree to take part in a research study titled "Sleep Habits, Personality, and Reaction Time," which is being conducted by Anne D. Bartolucci, M.S. under the direction of Amos Zeichner, Ph.D., both of the Psychology Department at the University of Georgia, and both of whom may be reached at 542-1173. My participation is voluntary; I can stop taking part at any time without giving any reason, and without penalty. I can ask to have information related to me returned to me, removed from the research records, or destroyed if it contains identifiable information.

The reason for this study is to determine how sleep patterns and personality affect reaction time. People sleep for different durations during the night, and the need for sleep varies widely. Individuals also have different ways of responding to situations. Both may affect how quickly they react.

The benefits I may expect from this study are 0.5 hours of research participation credit for participating in this screening session. If I meet the study requirements based on my answers today, I may be invited back to participate in a laboratory session, which will allow me to earn up to four additional hours of research participation credit.

The procedures for this study include this screening session, during which I will answer questions about myself and my sleep habits and indicate whether I am willing to be contacted for future participation. More about the laboratory session will be discussed with me at that time. I understand that this screening session will last approximately 30 minutes.

No discomfort or stress is anticipated during this phase of the research. I understand that, if invited to return for the laboratory session, I may be asked to restrict my sleep time the previous night to 4 hours, which may produce some discomfort. No risks are expected from this altered sleep pattern.

Any identifying information that is obtained in connection with this study will remain confidential unless required by law.

The researcher will answer any further questions about the research, now or during the course of the project, and can be reached by email.

My signature below indicates that I am between the ages of 18 and 25, that the researchers have answered all of my questions to my satisfaction, and that I consent to volunteer for the study. I have been given a copy of this form.

Anne D. Bartolucci, M.S.	_____	_____
Name of Researcher	Signature	Date
Telephone: 706-542-1173		
_____	_____	_____
Name of Participant	Signature	Date

Please sign both copies, keep one, and return one to the researcher.

Additional questions or problems regarding your rights as a research participant should be addressed to Chris A. Joseph, Ph.D., Human Subjects Office, University of Georgia, 612 Boyd Graduate Studies Research Center, Athens, Georgia 30602-7411; Telephone (706) 542-3199

APPENDIX G

LABORATORY CONSENT FORM

I, _____, agree to take part in the second phase of a research study titled "Sleep Habits, Personality, and Reaction Time," which is being conducted by Anne D. Bartolucci, M.S. under the direction of Amos Zeichner, Ph.D. of the psychology department at the University of Georgia. My participation is voluntary; I can stop taking part at any time without giving any reason, and without penalty. I can ask to have information related to me returned to me, removed from the research records, or destroyed if such is identifiable.

The reason for this study is to determine how sleep deprivation and personality affect reaction time. The need for sleep varies widely among individuals, as well as how it affects them when they do not sleep.

The benefit I may expect from this study is 1.0 hour of research participation credit. I will also learn about the process of psychological research and good sleep hygiene. I understand that this portion of the experiment will last approximately 1 hour. In order to make this study a valid one, some information about my participation will be withheld until after the study.

The procedures for this study include engaging in a reaction-time competition. I may administer shocks to my opponent after every trial, and he will have the same opportunity. Level of shocks will not exceed that identified by me as "painful." The shocks used here have been shown to have NO HARMFUL EFFECTS.

There are no risks foreseen for this research. Although causing momentary discomfort, the shocks I will be receiving have been repeatedly used at UGA in the past with NO REPORTED ADVERSE CONSEQUENCES. In order to make this study a valid one, some information about my participation will be withheld until after the study. I understand that in the unlikely event that I do experience undue psychological discomfort after participating in this study, the following resources are available to me. I understand that no financial or mental health assistance will be available other than my access to the University Health Center, which is contingent upon paying the associated student fees. If I need mental health services, I may call:

University of Georgia Psychology Clinic: (706) 542-1173
Counseling and Psychological Services at UHC: (706) 542-2273
Center for Counseling and Personal Evaluation: (706) 542-8508

Any information that is obtained in connection with this study and that can be identified with me will remain confidential unless required by law.

The researcher will answer any further questions about the research, now or during the course of the project, and can be reached by email at annebart@egon.psy.uga.edu

My signature below indicates that I am between 18 and 25 years old, that the researchers have answered all of my questions to my satisfaction and that I consent to volunteer for the study. I have been given a copy of this form.

Anne D. Bartolucci, M.S.	_____	_____
Name of Researcher	Signature	Date
Telephone: 706-542-1173		
_____	_____	_____
Name of Participant	Signature	Date

Please sign both copies, keep one, and return one to the researcher.

Additional questions or problems regarding your rights as a research participant should be addressed to Chris A. Joseph, Ph.D., Human Subjects Office, University of Georgia, 612 Boyd Graduate Studies Research Center, Athens, Georgia 30602-7411; Telephone (706) 542-3199; E-Mail Address IRB@uga.edu

APPENDIX H

DEBRIEFING STATEMENT

Thank you for participating in our study titled, "Sleep Habits, Personality, and Reaction Time," conducted by Anne D. Bartolucci, M.S., under the direction of Amos Zeichner, Ph.D.

You were informed that you were competing against another individual in a nearby room on a reaction time task. You were also informed that you and your opponent had the opportunity to administer a shock following each trial. Actually, you did not compete against another person. Your "opponent" was a computer program, and the task was "fixed" so that you would lose half the trials and win the other half, and there was no association between "winning" or "losing" a trial and being shocked. Furthermore, YOU DID NOT ADMINISTER ANY REAL SHOCKS WHEN PRESSING THE SHOCK BUTTONS. The true purpose of this task was to measure level of aggression, and we did so by looking at how "high" and how "long" you "shocked" your fictitious opponent and how your behavior was influenced by mood and personality, which were measured by the questionnaires you filled out, and by reduced amount of sleep. This type of deception was necessary in order to make the results of the study valid. Had you known that you were not competing against another participant and the nature of our research focus, you may not have behaved naturally and may have biased our results.

We know from previous studies conducted in this laboratory that people are more aggressive when they have endorsed being in certain mood states (e.g., angry or irritable) and when they endorse certain patterns of reacting to stressors. The purpose of this study was to find how these variables – stated mood and personality traits – interact with reduced sleep time to influence aggressive behavior. We believe that having less sleep than normal will increase aggression through personality and mood. In other words, if someone sleeps less and has a tendency to feel angry and irritable, he will be more likely to feel grumpy the next day and may have lower tolerance for stress, which will cause him to be more likely to behave aggressively.

Partial sleep deprivation is a common problem in our society, in which good health behaviors like sleeping enough and exercise are often pushed aside to make time for other obligations. Some effects of not sleeping enough include feelings of sleepiness during the day, grumpiness, and, over time, decreased ability and motivation to do the things that are important to you. Some good sleep habits to follow are:

- Sleep at least 8 hours per night. Most adults need between 7 and 9 hours of sleep.
- Wake up every day at the same time, even on weekends.
- If you take naps, keep them short (less than 30 minutes) and don't take them after 3:30 p.m.
- Don't drink caffeinated beverages after lunch time.
- Don't use alcohol to help you fall asleep; it may feel like it helps you get to sleep more quickly, but it disrupts your sleep during the second half of the night.
- If you suspect you may have a sleep disorder, seek help soon.

If you have any questions, please feel free to e-mail me (Anne).

Please help keep our procedure confidential! Please do not share the specifics of this experiment with your friends or other individuals who are or may be in the RP pool! Thank you!