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## Cognitive and Physiological Moderators of Daily Smokers' Early Neural Attentional Biases to Smoking and Nonsmoking Cues

Patrick John Hammett  
*College of William & Mary - Arts & Sciences*

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Cognitive and Physiological Moderators of Daily Smokers' Early Neural  
Attentional Biases to Smoking and Nonsmoking Cues

Patrick John Hammett

St. Paul, Minnesota

Bachelor of Arts, The Colorado College, 2008

A Thesis presented to the Graduate Faculty  
of the College of William and Mary in Candidacy for the Degree of  
Master of Arts

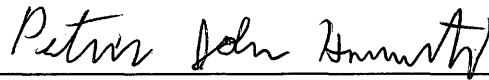
Department of Psychology

The College of William and Mary  
August, 2013

# APPROVAL PAGE

This Thesis is submitted in partial fulfillment of  
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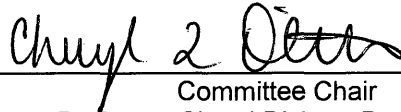
Master of Arts



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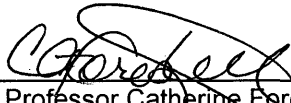
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Committee Chair

Professor Cheryl Dickter, Psychology  
The College of William and Mary



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Professor Catherine Forestell, Psychology  
The College of William and Mary



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Professor Pamela Hunt, Psychology  
The College of William and Mary

## COMPLIANCE PAGE

Research approved by

Protection of Human Subjects Committee

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Student Name (printed)

Cheryl L. Dickter

Faculty Research Advisor (printed)

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## ABSTRACT

The present study examined the relationship between “light daily” smokers’ implicit attentional biases to smoking stimuli, as measured by ERP difference scores, and several cognitive and physiological individual difference variables. Specifically, it sought to determine whether the constructs of physiological nicotine dependence, cigarette craving, “primary” dependence motives, including tolerance and automaticity, and “secondary” dependence motives, including social goals and affiliative attachment, moderated attentional biases across the early exogenous ERP components P1, N1, P2, and the endogenous ERP component N2. The present study also compared responses to stimuli containing only objects (i.e., inactive) to those that contained human/object interaction (i.e., active). In accordance with our hypotheses, reward-oriented craving emerged as a strong predictor of smoking-related attentional biases across the exogenous ERP components. Several “secondary dependence” motives were also positively correlated with these exogenous ERP components. Analyses of the endogenous N2 ERP component indicated that physiological nicotine dependence was positively correlated with smoking-related attentional biases at the N2 component. The presence of these significant moderator variable and smoking-related attentional biases were more consistent across the inactive stimuli as opposed to the active stimuli, indicating that these relationships may occur to a greater extent for inactive compared to active smoking stimuli. These findings indicate that subjective craving and secondary dependence motives are related to attentional biases to smoking stimuli, particularly those attentional biases occurring very early in processing. Contrary to past findings, physiological nicotine dependence was the only significant moderator of attentional biases at the endogenous N2 component, indicating that this construct may play a role in higher order cognitive processing.

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## **Cognitive and Physiological Moderators of Daily Smokers' Early Neural Attentional Biases in Response to Smoking and Nonsmoking Cues**

Tobacco addictions pose serious health risks, contributing to over 440,000 deaths annually in the United States and making cigarette smoking the leading cause of preventable death (American Cancer Society, 2009). While most age groups in the U.S. have experienced declines in rates of smoking, college students remain one of the few demographics to maintain consistent levels of smoking over the past decade (Centers for Disease Control and Prevention, 2009), despite the fact that many young adults report explicitly negative views towards smoking (Elders, Perry, Eriksen, & Giovino, 1994; Richardson, Green, Xiao, Sokol, & Vallone, 2010; DeBernardo et al., 1999). As research indicates that more than 50% of young people who experiment with cigarettes become regular smokers within two to three years (McNeil, 1991; Elders et al., 1994), it is important to understand the behavioral and biological mechanisms that initiate and perpetuate this addictive behavior.

Several theories have been put forth to explain the mechanisms responsible for addictive behavior. The cue-reactivity theory (Carter & Tiffany, 1999; Tiffany, 1995; Drummond, Tiffany, Glautier, & Remington, 1995; Wikler, 1965) posits that classical conditioning that results from repeated pairings of a drug (the unconditioned stimulus) with drug-related paraphernalia (the conditioned stimuli) can lead to increased cognitive and physiological reactivity to drug cues that are similar to that induced by the drug itself. This heightened cue reactivity to drug-related stimuli may also be explained through the lens of the classic opponent process model, in which an addict's cognitive and physiological responses to substance-related stimuli mimic withdrawal-like symptoms

(Siegel, 1975), or through an appetitive model, in which these responses map on to more positively-reinforcing symptoms (Stewart, de Wit, & Eikelboom, 1984; Carter & Tiffany, 1999). In a related vein, the incentive-sensitization theory posits that repeated exposure to substance-related paraphernalia results in a heightened dopaminergic response to these stimuli, increasing the relative salience of such cues in the environment (Robinson & Berridge, 1993). Furthermore, this theory posits that enhanced substance-related cue reactivity occurs independently of the physiologically addictive properties of the drug and that this effect may be permanent, persisting even despite changes in actual drug intake behavior. Thus, the substance-related environmental cues to which a fledgling smoker is regularly exposed bolster the addictive effects of the drug itself.

One manifestation of these heightened neural responses to drug-related environmental cues among smokers is attentional bias, or sensitivity to the detection of drug-related stimuli in the environment (Franken, 2003; Field & Cox, 2008). Attentional biases have been associated with an increase in smoking-related cognitions and a reduction in the availability of general cognitive resources (Franken, 2003). Such effects are thought to promote smoking maintenance and reduce the success of quit attempts because they occur independently of an individual's "wanting" or "desire" for a cigarette, thereby perpetuating addictive behavior regardless of the motivational state of the smoker (Robinson & Berridge, 1993, 2000; Waters et al., 2003; Williams, Mathews, & MacLeod, 1996). Thus, smokers who are motivated to quit as well as those who have already attained this goal are inherently at a heightened risk for relapse due to the ubiquity of substance-related environmental triggers.

Several core behavioral paradigms are used to assess attentional biases among substance users. One of these paradigms is the addiction Stroop task (Cox, Fadardi, & Pothos, 2006), in which a participant is presented with a series of substance-related and neutral words written in a colored font. The participant is asked to focus on and report the color of the font, with a slower reaction time indicative of enhanced semantic processing of the word itself, which reflects implicit attentional bias to the word. A second commonly used paradigm is the computerized visual dot probe task (Ehrman et al., 2002), in which the participant is asked to focus attention on the center of the screen while two types of cues, one substance-related and the other neutral, are simultaneously presented on opposing sides of the screen. Following a brief presentation of these stimuli, (e.g. 200 ms – 2000 ms) a visual probe appears on one side of the screen in place of one of the formerly presented cues. The participant is asked to indicate, as quickly as possible, which side of the screen this dot appears, with faster reaction times expected if the participant has attended to the stimulus presented on the same side as the probe. A comparison of reaction times to probes following substance-related and neutral cues gives a measure of attentional bias, with faster times indicative of increased bias. The use of these implicit measures of attentional bias confers several advantages when compared to self-reported measures of substance-related biases. First, implicit tasks cannot be easily manipulated by the participant and are not subject to self-presentational concerns, as are self-report measures. Furthermore, implicit tasks tend to be more sensitive to underlying subconscious processes about which the participant is unaware (Wright, Aquilino, & Supple, 1998).

Studies using these behavioral paradigms have demonstrated the existence of attentional biases in users of various types of drugs. For example, using the Stroop task, researchers have shown that cocaine, heroin, and alcohol users show enhanced attentional bias to drug-related stimuli relative to control groups that did not engage in drug or heavy alcohol use (Franken, Kroon, Wiers, & Jansen, 2000, Hester, Dixon, & Garavan, 2006, Townshend & Duka, 2001). With respect to smoking, studies using dot-probe tasks have demonstrated that daily smokers exhibit attentional biases to smoking-related compared to neutral visual stimuli (Waters et al., 2003; Ehrman et al., 2006) which occur very early in processing (i.e., 200 ms) (Bradley, Field, Mogg, & Houwer, 2004). Using behavioral tasks, research has shown that substance-related attentional biases may be mediated by cognitive and physiological factors. Physiological nicotine dependence is one potential moderator of attentional bias towards smoking-related cues. Behavioral research, however, has failed to demonstrate a relationship between self-reported scores of nicotine dependence, as measured by the Fagerstrom Test for Nicotine Dependence (FTND), and attentional biases using visual dot-probe tasks (Waters et al., 2003). However, fMRI studies have noted dependence-related fluctuations in smoking-related cue reactivity at the neural level (McClernon, Kozink, & Rose, 2008; Smolka et al., 2006). One such study noted that dependence-related cue reactivity was found in cortical regions specifically associated with attention allocation, imagery, and motor preparation, suggesting that higher degrees of nicotine dependence are associated both with selective attentional processes and implicit, automated drug-taking behavior (Smolka et al., 2006).

Intrusive drug-related cognitions, like those associated with drug craving, may also help to facilitate attentional biases to drug-related cues within one's environment (Kavanagh, 2005). Investigations of this moderator among daily smokers has demonstrated that increasing subjective craving by nicotine deprivation results in increased attention to smoking-related compared to neutral cues among daily smokers (Field & Cox, 2008; Mogg & Bradley, 2002; Zack, Belsito, Scher, Eissenberg, & Corrigan, 2001). Past research indicates that these cravings likely map onto two distinct subtypes which vary in influence as a function of an individual's level of smoking experience (Carter & Tiffany, 1999). The first subtype, which is more typical of light and occasional smokers, is associated with the positively reinforcing aspects of cigarettes and assesses the urge to smoke as a means of inducing the euphoric sensations associated with nicotine (Stewart et al., 1984; Shiffman, Kassel, Paty, Gnys, & Zettler-Segal, 1994; Shiffman, Dunbar, Scholl, & Tindle 2012). The second subtype assesses the urge to smoke as a means of alleviating the negative sensations associated with nicotine withdrawal, and is more typical of highly experienced smokers (Shiffman et al., 1994; Shiffman, 1993).

Other research has focused on the degree to which people are motivated to smoke by physiological versus social motives for smoking (Piper et al., 2004; Piper et al., 2008). Motives within the first subtype pertain to the physiologically-based addictive properties of cigarettes, and are hence labeled "primary dependence" motives (PDMs) (e.g., tolerance, craving, automaticity). Motives within the second subtype pertain to the sensory, social, and environmentally-based reasons for smoking, and are labeled "secondary dependence" motives (SDMs). Past research has demonstrated that "light

daily” college smokers are more strongly motivated by these SDMs (e.g. affiliative attachment, social goals, positive reinforcement) than PDMs (Piper et al., 2008; Piasecki, Piper, Baker, & Hunt, 2011; Shiffman et al., 1994). Other than the recent fMRI findings linking cigarette craving to smoking-related cue reactivity (McClernon et al., 2008; Smolka et al., 2006), little research has explored the relationships between these potential cognitive and physiological moderators and neural measures of attentional bias.

Another potentially important component of the cognitive processing associated with attentional biases that has largely been overlooked is the context depicted in the visual cues. Examining how variation in the context in which the stimuli are depicted affects responses is important as it represents the variety of contexts in which smokers may encounter smoking cues in their environment; either alone or in a social smoking environment. Although research has demonstrated that stimuli containing a human component elicit greater neural processing than stimuli containing inanimate objects (Bentin, Allison, Puce, Perez, & McCarthy, 1996), stronger attentional biases have been shown to occur when smoking-related are depicted alone (inactive cues), rather than when they are presented with humans interacting with them (active cues) (Forestell, Dickter, Wright, & Young, 2012; Dickter & Forestell, 2012). As such, an aim of the present study was to further elucidate the influence of stimulus type on smoking-related attentional biases, specifically with respect to the relationship between potential cognitive and physiological moderating variables and these attentional biases.

While behavioral measures provide a measure of attentional bias superior to self-report measures, event-related potentials (ERPs) provide several advantages over implicit behavioral measures. First, ERPs are a sensitive measure of attentional biases due to



their high temporal resolution when examining visual cue reactivity across multiple presentations of stimuli, allowing for the precise chronological measurement of the neural processes associated with selective visual attention. Additionally, unlike behavioral measures that rely on a button press, ERPs are not under the conscious control of the participant (Ito, Thompson, & Cacioppo, 2004) and thus provide a more pure measure of the underlying neural events associated with attention to smoking-related cues.

Physiological research on attentional biases among smokers has primarily focused on the P300 ERP component, although the methodology employed to examine these attentional biases has varied. One popular paradigm involves the use of categorization-related decision tasks like the visual “oddball” task. This task involves the presentation of a series of categorically-related and unrelated stimuli which participants categorize by means of a button-press. Due to the heightened cognitive load associated with this task, the resulting P300 likely reflects the processing of working memory in addition to any selective attention-related cognitive processing (Ito & Urland, 2003; Campanella et al., 2002; Donchin, 1981). Studies using this paradigm have provided evidence for enhanced P300 reactivity in smokers in response to smoking-related compared to neutral cues, particularly following nicotine deprivation (Littel & Franken, 2007, 2011). Similar attentional biases have been noted when using passive-viewing tasks, as studies have shown that daily smokers exhibit enhanced P300 amplitudes to smoking-related compared to neutral stimuli (Warren & McDonough, 1999; McDonough & Warren, 2001).

Although the P300 component is a well-documented measure of attentional bias for smokers responding to smoking-related visual stimuli, research mapping out the

sensitivity of earlier ERP components to smoking-related stimuli is sparse, specifically with respect to the P1, N1, P2, and N2 components. The earliest of these ERP components, P1, N1, and P2, are classified as exogenous components and are associated with the subconscious processing of the basic sensory characteristics of a stimulus. By examining cue reactivity across these early components, assess responses that occur independently of higher-level cognitive processes that may pertain more to motivational and emotional processing. Individual difference variables, like levels of prior smoking experience or nicotine dependence, may be associated with automatic attentional biases across these early components.

Research on the earliest of these components, the P1, has shown that its amplitude may vary as a function of the basic physical qualities of a stimulus (Hillyard, Vogel, & Luck 1999), and may also reflect selective attention to visual stimuli (Clark & Hillyard, 1996; Luck, Woodman, & Vogel, 2000). Research within the substance-abuse domain has demonstrated that alcoholics exhibit increased P1 amplitudes in response to alcohol-related as compared to neutral visual stimuli (Petit et al., 2012), providing evidence that heavy-substance users' attention to these cues is enhanced very early on in processing, a phenomenon which occurs automatically and outside the individual's consciousness. Previous research on the N1 has demonstrated that amplitude shifts at this component may reflect the relative novelty of a stimulus (Hillyard, Mangun, Woldorff, & Luck, 1995; Kok, 1997), as well as basic attentional selectivity (Clark & Hillyard, 1996). In addition to the evidence of attentional bias manifested in these components, the P1 and N1 components have also been found to be modulated by the degree of self-relevance present in the stimuli of interest (Keyes, Brady, Reilly, & Foxe, 2010; Fields &

Kuperberg, 2012). However, there is little research within the addiction literature investigating smoking-related attentional biases in these early components. An attentional bias study utilizing both Stroop and ERP measures examined smokers' responses to smoking-related and non-smoking words. This study found that smokers exhibit content-related ERP differences at N1 and P1, specifically noting increased negativity and positivity in response to smoking-relevant words at both of these components, respectively (Fehr, Wiedenmann, & Herrmann, 2006). Another study demonstrated enhanced P1 amplitudes in response to cigarette-related vs. neutral and emotionally-valenced cues among daily smokers, providing preliminary evidence for automatic, bottom-up neural processing of smoking-related stimuli as early as 100ms post stimulus-onset (Versace et al., 2011). While the P2 component has been associated with selective visual attention (Wastell & Kleinman, 1980; Carretié, Martín-Loeches, Hinojosa, & Mercado 2001; Cuthbert, Schupp, Bradley, Birbaumer, & Lang, 2000), there are no studies within the cigarette addiction literature specifically assessing smoking-related attentional bias at this component. The endogenous N2 component is thought to reflect higher-level cognitive processes, and is commonly associated with evaluative and decision-making processes (Dickter & Bartholow, 2010; van Veen & Carter, 2002). It has also been shown to vary as a function of the emotional relevance of a stimulus as well as selective visual attention (Wastell & Kleinman, 1980; Carretie et al., 2001; Cuthbert et al., 2000). Studies have revealed associations between N2 amplitude shifts and the "negativity" of a given stimulus, such that more negatively-valenced cues typically result in reduced N2 amplitude (Carretie et al., 2001). Within the addiction literature, a study by Warren and McDonough (1999) noted differences in smokers' neural responding to

smoking vs. neutral stimuli at the N2 component that were not seen among non-smokers. This research on smokers' implicit physiological responding to substance-related stimuli suggests that differences in cue reactivity may indeed be seen at this early attentional component. Furthermore, given that N2 reflects more complex cognitive phenomena like emotional relevance and conflict detection, individual difference variables pertaining to social and affiliative motivations for smoking may exhibit a heightened effect on attentional biases across this component.

The aim of the current study was to build on past behavioral and neural smoking-related cue reactivity research by examining whether attentional biases across the early attentional ERP components of P1, N1, P2, and N2 to active and inactive smoking-related stimuli were moderated by smoking-related cognitive or physiological phenomena. Specifically, the current study sought to explore how physiological nicotine dependence, subjective cigarette craving, and several other physiological and social smoking motives, all of which may be the products of emerging cigarette addiction, are related to early attentional neural processing of smoking cues compared to non-smoking cues. Although analyses of the P300 component have demonstrated that both visual oddball tasks and passive viewing tasks may both be reliable measures of smoking-related attentional biases (Warren & McDonough, 1999; McDonough & Warren, 2001; Littel & Franken, 2007, 2011), the present study elected to employ a passive viewing task to examine cue reactivity at early ERP components. This task was chosen because the resulting ERP response is a purer measure of the attentional bias to the visual stimuli in question as it is not contaminated by any task-related cognitive process (Kayser et al., 1997).

As past research has linked higher levels of nicotine dependence to heightened cortical reactivity in response to smoking-related imagery in brain regions thought to facilitate attention allocation and automated drug-taking behavior (Smolka et al., 2006), it was hypothesized that smokers with higher levels of physiological nicotine dependence as well as higher levels of physiologically-based PDMs (Piper et. al., 2004) would report heightened attentional biases to smoking-related stimuli at these components. In addition, based on addiction theories that posit that substance-related cognitions and the resulting cravings may be an implicit process (Tiffany, 1990) and evidence that subjective craving results in increased implicit attention to smoking-related cues in daily smokers (Field & Cox, 2008; Mogg & Bradley, 2002; Zack et al., 2001), it was hypothesized that smokers with higher levels of cigarette craving would exhibit heightened attentional biases to smoking-related stimuli across the exogenous components of P1, N1, and P2. Research investigating the influence SDMs on smoking-related attentional biases is lacking, and as such these constructs were investigated in an exploratory manner, and no specific hypotheses were offered with regard to their moderating influence on attentional biases across the exogenous components.

By examining the endogenous N2 component, the researchers wanted to explore whether the aforementioned moderators were also reflected in more higher-order cognitive processes, specifically those associated with conflict detection, emotional relevance, and motivational processes. As physiological nicotine dependence and PDMs are viewed as relatively low-level, implicit addiction process, the authors offered no specific predictions with regard to their moderating effects on smoking-related attentional bias at the N2 component. Furthermore, no previous studies within the nicotine addiction

literature have examined these specific constructs as they pertain to attentional bias at the neural level. Therefore, these factors were added as exploratory predictors in analyses of attentional bias at this component. However, past research has noted the influence of cigarette craving on neural smoking-related cue reactivity, specifically in brain regions associated with incentive motivation (Smolka et al., 2006). Incentive salience theory also posits that drug-related subjective craving, a result of the increased motivational salience of drug-related cues, results in increased substance-related cue reactivity (Robinson & Berridge, 1993). Due to this construct's association with cue-related motivational processing, it was hypothesized that higher levels of cigarette craving would be associated with heightened attentional biases to smoking-related cues at the N2 component. The SDMs, due to their associations with social, emotional, and environmental motivations for smoking, constructs which are more in line with the higher-order cognitive processes typically reflected in the N2 component, were hypothesized to be predictive of smoking-related attentional biases at this component. Specifically, it was predicted that higher scores on these measures would result in heightened attentional biases to smoking-related stimuli at this component.

## **Method**

### **Participants**

Twenty-one (14 male) self-identified daily smoking students at the College of William and Mary were recruited by way of an online, university-wide database, or through campus flyers. All participants were compensated for their time with credit for an introductory psychology course, or with \$15. Most of the participants were White ( $n = 17$ ), and the remaining participants were of Asian descent ( $n = 4$ ). Participants had an

average age of 20.38 years (SD = 2.42). All participants indicated that they were right-handed and had not previously experienced serious head injury. All procedures involved in this experiment were approved by the college's Protection of Human Subjects Committee, and each participant provided written consent prior to testing (see Appendix A)

## **Materials**

### *Stimuli*

The visual stimuli presented in this experiment consisted of a total of 40 color photographs depicting both smoking and nonsmoking cues. Half of all pictures presented were categorized as “active”, in that they depicted a person interacting with the stimulus, and half were categorized as “inactive”, in which the stimulus was presented alone. Each control stimulus was selected based on similarities in shape, color, and object position to each smoking stimulus. For example, an “inactive” smoking stimulus containing a package of cigarettes was matched with a neutral stimulus containing a package of dental floss of similar size, shape, and color. An “active” smoking stimulus might contain a hand grasping a package of cigarettes and a lighter, and the matched neutral stimulus would contain a hand grasping a pen and notebook pad, again matched according to size, shape, and color. All images were successfully pilot-tested in a previous study (Forestell et al., 2012), with average accuracy rates for the smoking and neutral stimuli at 98%.

### *Questionnaires*

A demographic questionnaire was used to measure participants' age, gender, race, and family income, and a general smoking questionnaire assessed current smoking habits

as well as age of smoking initiation. In addition, several standardized questionnaires were completed to measure nicotine dependence, cigarette craving, and motivations for smoking, which are described below.

The Fagerström Test of Nicotine Dependence (FTND) measured smokers' physiological dependence on nicotine (Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991) (see Appendix B). This brief questionnaire consists of the following six items: time to the first cigarette of the day, level of difficulty refraining from smoking, importance of the first morning cigarette, smoking frequency, importance of smoking in the morning, and determination to smoke. Scores range from 0 to 10, with higher scores indicating a greater level of dependence. Reliability of this questionnaire is .78, and Cronbach alpha levels for internal consistency range from 0.56 - 0.70 (Etter, Duc, & Perneger, 1999; Haddock, Lando, Klesges, Talcott, & Renaud, 1999; Payne, Smith, McCracken, McSherry, & Antony, 1994; Pomerleau, Carton, Lutzke, Flessland, & Pomerleau, 1994).

The Wisconsin Inventory of Smoking Dependence Motives (WISDM) is an electronic questionnaire assessing the motives behind cigarette use among adults (Piper et al., 2004) (see Appendix C). It has been validated against the following four major sets of clinical criteria: heaviness of smoking and nicotine self-administration, DSM-IV criteria for tobacco dependence (e.g., consequences of smoking), severity and duration of withdrawal symptoms, and likelihood or latency to relapse. This 68-item questionnaire contains between 4 and 7 questions for each of the following 13 motivational sub-scales: (1) Affiliative Attachment, (2) Automaticity, (3) Loss of Control, (4) Behavioral Choice-Melioration, (5) Cognitive Enhancement, (6) Craving, (7) Cue Exposure-Associative



Processes, (8) Negative Reinforcement, (9) Positive Reinforcement, (10) Social-Environmental Goals, (11) Taste and Sensory Processes, (12) Tolerance, and (13) Weight Control. The Taste and Sensory Properties sub-scale was not included in final analyses as it was not deemed of theoretical interest. Recent validation of these constructs has demonstrated that they may be categorized as either physiologically-based “primary dependence” motives, or more sensory, social, or environmentally-based “secondary dependence” motives (Piper et al., 2008). The Automaticity, Craving, Loss of Control, and Tolerance subscales load onto “primary dependence” motives factor, with the remaining subscales loading onto the “secondary dependence” motives factor.

The 10-item Questionnaire of Smoking Urges-Brief version (QSU-B) (Cox, Tiffany, & Christen, 2001) is an abbreviated version of the 32-item Questionnaire of Smoking Urges (QSU) developed by Tiffany and Drobes (1991) (see Appendix D). This questionnaire, which assesses nicotine-related craving, contains two distinct sub-scales with five items loading on to each factor. The first factor is associated the positively reinforcing aspects of cigarette use, i.e. the appetitive and pleasurable aspects of smoking, while the second factor is associated with the negatively reinforcing aspects of cigarette use, i.e. relief from negative affect and nicotine withdrawal. Taken together, these two measures provide a highly reliable measure of cigarette-related craving, with Cronbach alpha levels for internal consistency ranging from 0.87 to 0.89) (Cox et al., 2001).

#### *Carbon monoxide monitor*

A carbon monoxide BreathCO monitor (Vitalograph, Lenexa, Kansas) was used to assess recent tobacco smoke exposure.

## **Procedure**

Before beginning the experiment, all participants were screened for and subsequently eliminated from participation if they indicated that they had experienced past head trauma, or were left-handed. Participants then read and signed an informed consent statement. Participants were tested individually, and were told that the experiment was meant to assess cortical activation in response to visual cues. All participants were then seated 90 cm from the standardized position of a computer monitor, yielding a visual angle of about 6 degrees. Prior to attaching the electrodes, the experimenter cleaned the participant's face and forehead using an alcohol pad and exfoliating gel. After measuring the participant's head and affixing him or her with the properly sized electrode cap, the experimenter began attaching electrodes to the face and forehead. Electrode impedences were minimized using an electrode gel which was applied to individual electrodes using a blunt-tipped syringe. After this procedure was completed, the participant was asked to passively view a series of 40 smoking-related and 40 neutral primes, randomly selected. Each image was presented for an 8-second interval, followed by an inter-trial interval period that randomly varied between 6 and 8 seconds and a 2-second fixation cross. Following this portion of the experiment, the participant completed the computerized questionnaires and was debriefed. The entire procedure lasted for an average of one hour and thirty minutes.

### *Electrophysiological Recordings and Analysis*

EEG data were recorded with a DBPA-1 Sensorium Bioamplifier (Sensorium, Inc., Charlotte, VT, USA) with an analog high-pass filter of 0.01 Hz and a low-pass filter of 500 Hz (four-pole Bessel). The EEG was recorded from 74 Ag-AgCl sintered electrodes

in an electrode cap, placed with the expanded International 10–20 electrode placement system. All electrodes were referenced to the tip of the nose, and the ground electrode was placed in the middle of the forehead, slightly above the eyebrows. Eye movement and blinking were recorded from bipolar electrodes placed on the lateral canthi and periocular electrodes on the superior and inferior orbits, aligned with the pupils. Before data collection was initiated, all impedances were adjusted to within 0–20 kilohms. EEG was recorded continuously throughout the computer task, and was analyzed off-line by EMSE software (Source Signal Imaging, San Diego, CA, USA). Data were under-sampled at 500 Hz. The data were corrected for eye-movement artifacts, using independent component analysis (Jung et al., 2000). Channels containing extreme values ( $\pm 300$  mV) in more than 40% of the sweeps were spatially interpolated. All EEG data were filtered (FIR) at low-pass 20 Hz (Luck, 2005). The data were segmented between 200 ms prior to stimulus onset and 1000 ms post-stimulus onset. After baseline correction over the pre-stimulus interval, segmented data was averaged for each subject in each of the conditions (Fabiani, Gratton, & Federmeier, 2007; Luck, 2005). Sample-wide ERPs were identified from the grand-averaged waveforms.

## **Results**

### *Participant Characteristics*

Participants reported smoking an average of 6.64 cigarettes per day ( $SD = 4.28$ ), and had an average CO reading of 2.48 parts/million ( $SD = 2.86$ ). For a complete listing of participants' mean scores on the FTND, QSU – B, and WISDM questionnaires, see Table 1.

### *ERP Components*

Visual inspection of grand-averaged waveforms was used to identify epochs for the component amplitudes of interest, as well as to determine scalp locations where neural activation was maximal for the corresponding components. This inspection demonstrated that the stimuli yielded four different early attentional ERP components: P1, N1, P2, and N2. The P1 component was largest at the P1, P2, P3, P4, P5, P6, P7, P8, P9, P10, PO3, PO4, PO7, PO8, POZ and PZ electrodes. Neural activity across these 16 electrodes was averaged, and the P1 component was quantified as the average voltage between 52 and 160 ms at these electrodes. The N1 component was largest at the F1, F2, F3, F4, F5, F6, F7, F8, FC1, FC2, FC3, FC4, FC5, FC6, FCZ, and FZ electrodes. Neural activity across these 16 electrodes was averaged, and the N1 component was quantified as the average voltage between 76 and 216 ms at these electrodes. The P2 component was largest at the P1, P2, P3, P4, P5, P6, P7, P8, P9, P10, PO3, PO4, PO7, PO8, POZ, and PZ electrodes. The voltages at these 16 electrodes were averaged and P2 was quantified as the average voltage between 172 and 272 ms at these electrodes. The N2 component was largest at the F1, F2, F3, F4, F5, F6, F7, F8, FC1, FC2, FC3, FC4, FC5, FC6, FCZ, FPZ, and FZ electrodes. The voltages at the 17 electrodes were averaged and N2 was quantified as the average voltage between 220 and 328 ms at those electrodes.

ERP amplitude difference scores were then calculated for the P1, N1, P2, and N2 components by subtracting the mean amplitude for the neutral stimuli from the amplitude for the smoking stimuli across all participants, separately for active and inactive stimuli. These difference scores represent attentional bias for smoking compared to neutral stimuli, such that a positive score represented an attentional bias towards the smoking

stimuli and a negative score represented an attentional bias towards the neutral stimuli for the positive ERP components of P1 and P2. Conversely, for the negative ERP components of N1 and N2, a negative difference score was indicative of attentional bias towards the smoking stimuli whereas a positive difference score was indicative of attentional bias to the neutral stimuli. In order to explore relationships between ERP attentional bias scores and individual difference variables, these bias scores were subjected to a series of correlational analyses examining nicotine dependence as assessed by the FTND, subjective cigarette craving as assessed by Factors 1 and 2 of the QSU-B, and smoking motives as assessed by 12 motivational sub-scales of the WISDM. The Taste and Sensory Properties sub-scale of the WISDM was eliminated from these analyses as it was not deemed to be of theoretical interest. These analyses revealed several significant correlations (see Table 2), as described below. Variables that were significantly correlated with attentional biases were entered into further regression analyses in order to identify the unique contributions of these predictors to the overall variance in attentional bias scores.

## **P1**

Correlational analyses revealed that attentional bias for the inactive attentional bias scores were significantly positively correlated with the QSU - B Factor 1 at the  $p < .05$  level (see Figure 1). For the active smoking stimuli at the P1 component, there was a marginal negative correlation with the number of cigarettes smoked daily ( $p > .08$ ), such that a greater attentional bias was found in those who smoked fewer cigarettes. A marginal positive correlation was also found with the Positive Reinforcement sub-scales of the WISDM, ( $p < .08$  level). A linear hierarchical multiple regression was then

performed to determine whether scores on the Positive Reinforcement scale significantly predicted attentional bias scores for active smoking stimuli after controlling for the variance accounted for by daily cigarette consumption. After controlling for this variable, the Positive Reinforcement sub-scale failed to reach significance ( $p < .08$ ).

## **N1**

For the inactive attentional bias scores, correlational analyses revealed a significant positive correlation with QSU - B Factor 1 and the Positive Reinforcement sub-scale of the WISDM at the  $p < .05$  level, as well as marginal positive correlation with Behavioral Choice-Melioration sub-scale of the WISDM at the  $p < .08$  level. A linear multiple regression was performed to determine whether scores on the smoking measures significantly predicted attentional bias scores for active smoking stimuli, selecting only independent variables that had been found to be significantly correlated with these bias scores. The regression model including the QSU - B Factor 1, and the Positive Reinforcement and Behavioral Choice-Melioration sub-scales of the WISDM as simultaneous predictors was significant,  $R^2 = .39$ ,  $F(4, 16) = 3.58$ ,  $p = 0.036$ . Analyses of the separate predictor variables indicated that the QSU - B Factor 1 significantly predicted N1 inactive attentional bias scores,  $t = 2.11$ ,  $p = 0.050$ . However, the Positive Reinforcement and Behavioral Choice-Melioration sub-scales of the WISDM did not achieve significance ( $p < .08$ ) (see Table 3 for Beta values).

The active attentional bias was marginally negatively correlated with the number of cigarettes smoked daily at the  $p < .08$  level, such that a greater attentional bias towards the smoking stimuli was found in those who smoked more cigarettes. The active attentional bias was also significantly negatively correlated with Factor 1 of the QSU-B

and the Behavioral Choice-Melioration and Positive Reinforcement sub-scales of the WISDM at the  $p < .05$  level, indicating that a greater attentional towards the smoking stimuli was found in individuals with higher scores on these measures. A linear hierarchical multiple regression was then performed to determine whether scores on the smoking measures significantly predicted attentional bias scores for active smoking stimuli after controlling for the variance accounted for by daily cigarette consumption, selecting only independent variables that had been found to be significantly correlated with these bias scores. The first level of the regression included the number of cigarettes smoked daily, and the second level included Factor 1 of the QSU-B and the Behavioral Choice-Melioration and Positive Reinforcement sub-scales of the WISDM. After controlling for the number of cigarettes smoked daily, the second level of the model predicted 57.7% of the variance,  $F(4, 16) = 5.45, p = 0.006$ , with  $R^2 \text{ change} = .40$ ,  $F\text{change} = 5.09, p = 0.012$ , indicating a significant change in variance over and above that accounted for by daily cigarette consumption. Analyses of the separate predictor variables at this level indicated that the QSU - B Factor 1 significantly predicted N1 active attentional bias scores at the  $p < .05$  level, while the Positive Reinforcement and Behavioral Choice-Melioration sub-scales of the WISDM did not reach significance (see Table 4 for Beta values).

## **P2**

The inactive attentional bias scores were significantly positively correlated with QSU - B Factor 1 and the Positive Reinforcement, Behavioral Choice-Melioration, and the Cue-Exposure-Associative Processes sub-scales of the WISDM at the  $p < .05$  level. Inactive attentional bias scores were also marginally positively correlated with the

Affiliative Attachment sub-scale of the WISDM. The Behavioral Choice-Melioration sub-scale was removed from the regression model due to high collinearity with the other scales. Based on these results, a linear multiple regression was conducted which included the QSU - B Factor 1, Positive Reinforcement, Cue Exposure - Associative Properties, and the Affiliative Attachment sub-scales as simultaneous predictors. Overall the model was significant,  $R^2 = .52$ ,  $F(4, 16) = 4.26$ ,  $p = 0.016$ . Analyses of the separate predictor variables indicated that the QSU - B Factor 1 marginally predicted inactive attentional bias scores (see Figure 2), whereas the Positive Reinforcement, Cue Exposure - Associative Properties, and Affiliative Attachment sub-scales of the WISDM did not achieve significance (see Table 5 for Beta values).

Analyses of active attentional bias scores did not reveal any significant correlations with the independent variables.

## **N2**

The inactive attentional bias scores were significantly negatively correlated with the FTND at the  $p < .05$  level.

Analyses of active attentional bias scores did not reveal any significant correlations with the independent variables.

## **Relationships between ERP Components**

To examine relationships between the ERP component attentional bias scores, correlational analyses were conducted (see Table 6). Analyses indicated that the P1 active attentional bias score was positively correlated with all other active components. The P1 inactive bias was positively correlated with N1 active, P2 active, and P2 inactive scores. The N1 active bias was positively correlated with both P2 active and N2 active



scores. N1 inactive scores were positively correlated with P2 inactive and N2 inactive scores. P2 active bias was positively correlated with N2 active bias, and P2 inactive bias was correlated with N2 inactive scores.

### **Discussion**

The present study investigated the moderating influence of subjective cigarette craving, physiological nicotine dependence, and PDMs and SDMs on smoking-related attentional biases across the early ERP components of P1, N1, P2, and N2. Analyses of the exogenous components of P1, N1, P2 revealed several significant relationships. In accordance with hypotheses, subjective craving, as measured by the reward-oriented sub-scale of the QSU - B, demonstrated a positive relationship with smoking-related attentional biases across the P1 and P2 components. Surprisingly, significant positive relationships were also found for several of the SDM sub-scales across the exogenous ERP components of P1 and P2. Contrary to hypotheses, physiological nicotine dependence, as measured by the FTND, was the only construct that was significantly positively correlated with smoking-related attentional biases at the endogenous N2 component. Across both the endogenous and exogenous components, these relationships were more consistently significant in response to the “inactive” rather than the “active” stimuli.

When interpreting the relationships observed between these moderating variables and attentional biases across the exogenous components of P1, N1, and P2, it is important to note that the reward-oriented craving sub-scale of the QSU-B emerged as the strongest single predictor of smoking-related attentional biases. Past research indicates that this construct is essentially inseparable from substance-related attentional biases in that they

form a mutually excitatory relationship, such that an increase in one inevitably results in a corresponding increase in the other (Field & Cox, 2008). While the existence of this relationship is well-documented both behaviorally and neurally (Mogg & Bradley, 2002; McClernon et al., 2008; Smolka et al., 2006), the use of ERPs in the present study demonstrated that this moderating relationship occurs early in visual processing, providing further links between this construct and automated cue-related processes. These findings indicate that craving affects the visual processing of smoking-related cues on a very basic, implicit level, even among “light daily” smokers. That this moderating relationship was only present for the positive reinforcement (i.e., reward) sub-scale of the QSU -B, which loads onto appetitive smoking urges, while the withdrawal-based sub-scale of the QSU – B was not a significant moderator, is also consistent with past research. Studies examining the motivational profiles of “light daily” versus “heavy” smokers have demonstrated that appetitive, or positively reinforcing urges to smoke are more typical of the former smoking demographic, as these individuals have not yet developed a level of nicotine dependence sufficient to induce strong withdrawal-based smoking urges (Shiffman et al., 1994; Shiffman et al., 2012). However, it must be noted that significant positive relationships between this moderating variables and smoking-related attentional biases were only present at the P1 and P2 components. While this construct was a significant predictor at the N1 component, this was a negative relationship, indicating that higher scores on this variable were associated with heightened attentional biases towards the neutral rather than the smoking stimuli. Further research across these exogenous components should work to elucidate why the direction of this relationship appears to switch across the positive and negative components.

Physiological nicotine dependence was not a significant moderator of smoking-related attentional biases across the exogenous components, a finding that runs contrary to hypotheses. This null result may have been due to relatively low dependence of the sample of “light smokers” used in the present study; that is, physiological nicotine dependence may have not be a strong enough factor to account for the kind of implicit attentional biases typically reflected in exogenous ERP components. It is also possible that because the FTND, the measure of physiological nicotine dependence used in the present study, suffers from relatively low internal consistency (Heatherton et al., 1991), it was not sensitive enough to detect a relationship between dependence and attentional bias. This issue may have been exacerbated in light of the population sampled for the present study, as research indicates that multiple factors may contribute to nicotine dependence among light, inexperienced smokers (Piper et al., 2004; Piper et al., 2008). Thus, constraining all dependence-based constructs to a single measure may have resulted in a lack of sensitivity to those specific constructs which may be most influential for creating and sustaining dependence among the smokers sampled.

Interestingly, the SDM sub-scales of Positive Reinforcement, Behavioral Choice-Melioration, Cue-Exposure-Associative Processes, and Affiliative Attachment, which were included in the exogenous ERP component analyses for exploratory purposes, demonstrated significant positive relationships with smoking-related attentional biases at the P1 and P2 components. As with the QSU – B Factor one, these constructs were predictive at the N1 component as well, but switched to a negative relationship at this component, indicating that higher scores on these constructs at the N1 resulted in heightened attentional biases towards the neutral stimuli. Again, further research should

investigate the difference in the direction of the relationships across these components. With respect to the findings at the P1 and P2 component, the presence of these significant relationships may partially be attributable to the present sample, as past research examining smoking motives among “light daily” smokers has demonstrated that these motives typically align with social and environmental reinforcers as opposed to habitual or withdrawal-based motives (Shiffman, 1993). These trends have been replicated in more recent research examining the validity of the WISDM for assessing smoking motives among daily and non-daily smokers (Piasecki et al., 2012; Shiffman et al., 2012). This research noted that PDMs were more strongly associated with higher levels of nicotine dependence as well as smoking motives born out of habit or automaticity, whereas SDMs were not associated with measures of strict physiological dependence. Thus, the strong correlations among SDMs and smoking-related attentional biases in the present study, and the lack of such correlations with the PDMs, may be due to the relatively low levels of daily smoking ( $M = 6.64$  cigarettes per day) and nicotine dependence ( $M_{FTND} = 1.74$ ) among the sample used.

Analyses of the relationships between moderator variables and attentional biases at the endogenous N2 component revealed that, contrary to hypotheses, physiological nicotine dependence as indexed by the FTND was the only variable that was significantly correlated with smoking-related attentional biases. This unexpectedly strong positive relationship may suggest that nicotine dependence plays a role in the higher-order cognitive processing of smoking-related stimuli. However, problems with the validity of the FTND, as discussed previously, prevent the researchers from drawing this conclusion with a high degree of certainty, and further research exploring the relationship between

nicotine dependence and smoking-related attentional biases at the exogenous ERP components is needed.

Another unexpected finding at the N2 component was the lack of significant correlations between the measures of subjective cigarette craving and SDMs and smoking-related attentional biases. One possible explanation for the lack of significant correlations between measures of craving and smoking-related attentional biases, and the inability of the present study to replicate the incentive-based motivational processing found in past neural cue reactivity studies (e.g., Smolka et al., 2006), was that the present study assessed craving using the 2 factor structure of the QSU – B, whereas the fMRI study by Smolka and colleagues (2006) used a uni-dimensional assessment of craving. While Cox and Tiffany (2001) indicate that this scale is appropriate for use both as a 2-factor and as a global measure of craving, subsequent investigations have confirmed the validity of the initial 2 factor structure (Clausius et al., 2012; Cappelleri et al., 2007). Furthermore, in the study by Smolka and colleagues (2006), ratings of subjective craving were measured during the actual fMRI task, whereas in the present study participants reported levels of craving only after viewing the cues. It may be the case that exposure to the smoking cues themselves caused heightened incentive motivation associated with this craving. Hence, simultaneous assessments of craving and cue reactivity may be needed in order to observe the higher-order processing biases associated with subjective craving.

While the primary aim of the present study was to examine relationships between attentional bias and individual difference variables, an additional goal was to explore the possibility that the type of stimulus might act as an additional moderator of these relationships. Past research has indicated that inactive cues appear to facilitate smoking-

related attentional biases to a greater extent than cues containing a human component (Forestell et. al., 2011; Dickter & Forestell, 2012; Forestell, Dickter, & Young, 2012). Analyses of the active and inactive stimuli in the present study revealed a similar trend across the exogenous and endogenous ERP components. While the present study did not solely examine the influence of this variable on smoking-related attentional biases as did these previous studies, but rather their influence on the relationships between smoking-related moderating variables and attentional biases, the present findings suggest that these relationships are similarly bolstered in response to inactive rather than active stimuli.

In contrast, after controlling for the variance accounted for by daily cigarette consumption, analyses of the active stimuli revealed only one significant moderating relationship, which was found at the N1 component. However, the presence of strong correlations between individual difference variables and attentional biases at the earliest exogenous components, P1 and N1, suggests that the presence of a human component may facilitate the relationships between moderating variables and smoking-related attentional biases very early in processing. The human interaction present in the “active” smoking stimuli may heighten the early attentional reactivity associated with implicit, automated drug-taking cognitive processes, perhaps indicating that presenting smoking cues in a social context bolsters these implicit drug-taking processes. This view is bolstered by recent addiction research demonstrating that college students at-risk for alcohol dependence demonstrate enhanced alcohol-related attentional biases when these cues are presented with a human component (Dickter, Forestell, Hammett, & Young, under review).

Because light smokers differ considerably from heavy smokers in their self-reported motives for smoking (Shiffman, 1993; Shiffman et al., 2012; Piasecki et. al., 2010), the generalizability of the present findings is limited. Continued focus on light smokers who are not dependent on nicotine is warranted given that many of these light smokers will eventually become addicted to nicotine. This is of concern in light of recent data which indicate that the proportion of occasional or “light smokers” within the general smoking population increased by 40% between 1996 and 2001 (Centers for Disease Control and Prevention, 2003; Shiffman et al., 2012).

In line with these population trend data, future research should continue to focus on smokers who are not yet addicted to nicotine in an effort to better understand the cognitive mechanisms that motivate these individuals’ smoking behavior. In this vein, further examination of the influence of social, environmental, and emotional motives for smoking is of particular importance among this demographic because, despite a lack of withdrawal symptoms among these non-dependent smokers, these individuals still experience high levels of relapse when trying to quit (Tindle & Shiffman, 2011), indicating that these factors may be affecting their ability to do so. Future research should also address the problems associated with current measures of nicotine dependence, like the FTND. Such research should utilize a multi-dimensional measure of dependence that is more sensitive to individual differences. Due to the excellent temporal resolution of EEG measures, this research may continue to benefit from the use of ERP measures which offer a way of assessing at precisely which stages of cognitive processing individual difference variables like craving, dependence, and social/environmental motives are most influential.

As drug-related cues in the environment are thought to help perpetuate substance-use behaviors and increase the likelihood of relapse amongst abstainers (Niaura, 1988), assessing smoking-related attentional biases among smokers trying to quit may be a valuable therapeutic tool for determining likelihood of relapse. By elucidating the cognitive processes underlying attentional biases to smoking-related environmental stimuli, the current study aimed to bolster existing research linking substance-related attentional biases and relapse behavior. The present study sought to add to this research by examining the moderating factors which may facilitate smoking-related attentional biases. By helping to elucidate these moderating relationships, the present research helps to inform intervention strategies by identifying specific constructs which may put individuals at increase risk for smoking-related attentional biases.



## Appendix A

### Research Participant Consent Form

Psychology Department - College of William & Mary

**TITLE:** Affective Responses to Pictorial Cues among College Students

**INVESTIGATORS:** Chelsie Young, Graduate Student  
Pat Hammett, Graduate Student  
Catherine A. Forestell, Ph.D., Assistant Professor  
Cheryl Dickter, Ph.D., Assistant Professor

The purpose of this study is to examine your responses to a series of pictures and pictographs presented. This experiment involves the following steps:

First, several recording electrodes will be placed on your scalp, face, and forearms. These electrodes will record the tiny electrical activity in your brain and muscles as you view and respond to the stimuli presented in this study; the electrodes will not be used to harm you in any way. Electrode gel will be inserted into each electrode prior to recording, and will need to be washed out of your hair following the session. This gel easily washes out with water. Shampoo is available if you would like to use it. There are no known discomforts or risks associated with the response tasks in this experiment. It is possible that you will experience minor fatigue during set up of the experiment (cap administration and preparation) or after the experiment. It is possible that you have an allergic reaction to the gel, and you will be tested for this possibility prior to cap application. If you do experience fatigue during the experiment, please alert the experimenter and a break will be given as soon as possible. You will also be wearing earphones with disposable ear buds and will hear 100 dB tones.

- On a computer screen, you will see a series of trials in which a series of pictures followed by Chinese pictographs will be presented.
- You will complete a judgment task in which you will be asked to make responses on a keyboard by pressing one of two keys depending on the pleasantness of the pictographs presented.
- You will be asked to view a series of pictures to be recalled later, while hearing 100 dB tones.
- You will be presented with a series of questionnaires after the computer portion of the experiment.

Your privacy is important to us and we will make every effort to protect your privacy. An arbitrary code number has been assigned to you for this study. The link between this code number and information that could be used to personally identify you will be kept in a password-

protected database in a locked location. The results of this experiment will not be linked to any specific individual; we are only interested in group averages. No identifying information will ever be made public.

Please read the paragraph below and sign at the bottom.

The general nature of this study has been explained to me. I understand that I be participating in a judgment task on the computer while electrodes record my brainwaves. My participation in this study should take a total of about two hours. I understand that my responses will be confidential and that my name will not be associated with any results of this study. I know that I do not have to participate in this study and that if I do choose to participate, I may stop at any time without any penalty. I know that I may refuse to answer any question asked and I also understand that any credit for participation will not be affected by my responses or by my exercising any of my rights. I am aware that I may report dissatisfactions with any aspect of this experiment to the Chair of the Protection of Human Subjects Committee, Dr. Lee Kirkpatrick, 757-221-3997 or [lkirk@wm.edu](mailto:lkirk@wm.edu). I understand that I may contact Dr. Cheryl Dickter, Dr. Cathy Forestell, Chelsie Young, and Pat Hammett about this experiment to ask any questions or to obtain the results of this study after it is completed at 757-221-3722, [cdickter@wm.edu](mailto:cdickter@wm.edu), 757-221-3892 or [caforestell@wm.edu](mailto:caforestell@wm.edu), [cmyoung01@email.wm.edu](mailto:cmyoung01@email.wm.edu), or [pjhammett@email.wm.edu](mailto:pjhammett@email.wm.edu). I am aware that I must be at least 18 years of age to participate. My signature below signifies my voluntary participation in this project, and that I have received a copy of this consent form.

---

Signature

Date

---

Print Name

## Appendix B

**Items and scoring for Fagerstrom Test for Nicotine Dependence (FTND)**

<i>Questions</i>	<i>Answers</i>	<i>Points</i>
1. How soon after you wake up do you smoke your first cigarette?	Within 5 minutes	3
	6-30 minutes	2
	31-60 minutes	1
	After 60 minutes	0
2. Do you find it difficult to refrain from smoking in places where it is forbidden e.g. in church, at the library, in cinema, etc.?	Yes	1
	No	0
3. Which cigarette would you hate most to give up?	The first one in the morning	1
	All others	0
4. How many cigarettes/day do you smoke?	10 or less	0
	11-20	1
	21-30	2
	31 or more	3
5. Do you smoke more frequently during the first hours after waking than during the rest of the day?	Yes	1
	No	0
6. Do you smoke if you are so ill that you are in bed most of the day?	Yes	1
	No	0



42. I crave cigarettes at certain times of day.
43. I would feel alone without my cigarettes.
44. A lot of my friends or family smoke.
45. Smoking brings me a lot of pleasure.
46. Cigarettes are about the only things that can give me a lift when I need it.
47. Other smokers would consider me a heavy smoker.
48. I feel a strong bond with my cigarettes.
49. It would take a pretty serious medical problem to make me quit smoking.
50. When I haven't been able to smoke for a few hours, the craving gets intolerable.
51. When I do certain things I know I'm going to smoke.
52. Most of my friends and acquaintances smoke.
53. I love the feel of inhaling the smoke into my mouth.
54. I smoke within the first 30 minutes of awakening in the morning.
55. Sometimes I'm not aware that I'm smoking.
56. I'm worried that if I quit smoking I'll gain weight.
57. Smoking helps me think better.
58. Smoking really helps me feel better if I've been feeling down.
59. Some things are very hard to do without smoking.
60. Smoking makes me feel good.
61. Smoking keeps me from overeating.
62. My smoking is out of control.
63. I consider myself a heavy smoker.
64. Even when I feel good, smoking helps me feel better.
65. I reach for cigarettes when I feel irritable.
66. I enjoy the sensations of a long, slow exhalation of smoke.
67. Giving up cigarettes would be like losing a good friend.
68. Smoking is the easiest way to give myself a lift.

*WISDM-68 Scoring Key*

Item numbers Motive assessed

20, 36, 43, 48, 67	Affiliative Attachment
5, 19, 26, 41, 55	Automaticity
6, 28, 34, 62	Loss of Control
10, 21, 35, 38, 46, 49, 68	Behavioral Choice-Melioration
13, 15, 24, 39, 57	Cognitive Enhancement
11, 29, 37, 50	Craving
4, 17, 23, 40, 42, 51, 59	Cue Exposure-Associative Processes
7, 18, 25, 32, 58, 65	Negative Reinforcement
3, 8, 45, 60, 64	Positive Reinforcement
22, 30, 44, 52	Social-Environmental Goals
1, 12, 27, 33, 53, 66	Taste and Sensory Processes
9, 14, 47, 54, 63	Tolerance
2, 16, 31, 56, 61	Weight Control

*Note.* To calculate the scores of the subscales, take the mean of the items that load onto each subscale. The total scale score is the sum of all of the subscale scores, or a sum of the means for each subscale.

## Appendix D

**Questionnaire of Smoking Urges – Brief Version**

*Answers are yes/no, yes = 1, no = 2*

*A higher total score on each of these scales is indicative of greater craving*

*Factor 1*

1. I have a desire for a cigarette right now
3. If it were possible, I probably would smoke now.
6. I have an urge for a cigarette.
7. A cigarette would taste good now.
10. I am going to smoke as soon as possible.

*Factor 2*

2. Nothing would be better than smoking a cigarette right now.
4. I could control things better right now if I could smoke.
5. All I want right now is a cigarette.
8. I would do almost anything for a cigarette now.
9. Smoking would make me less depressed.

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Table 1

*Questionnaire Descriptive Statistics*

	<b>Minimum</b>	<b>Maximum</b>	<b>Mean</b>	<b>SD</b>
<b>QSU-B Factor 1</b>	5	35	18.67	9.76
<b>QSU-B Factor 2</b>	5	21	10.33	5.23
<b>FTND</b>	.00	6.00	1.95	1.82
<b>Affiliative Attachment</b>	1.00	5.80	2.13	1.37
<b>Automaticity</b>	1.00	5.00	2.42	1.24
<b>Loss of Control</b>	1.00	4.50	2.46	.95
<b>Behavioral Choice-Melioration</b>	1.00	5.14	2.59	1.05
<b>Cognitive Enhancement</b>	1.00	6.20	3.03	1.79
<b>Craving</b>	1.00	6.25	2.83	1.57
<b>Cue Exposure–Associative</b>	1.00	7.00	3.87	1.66
<b>Negative Reinforcement</b>	1.17	6.67	3.76	1.56
<b>Positive Reinforcement</b>	1.60	6.80	3.90	1.35
<b>Social-Environmental Goals</b>	1.00	7.00	3.86	2.00
<b>Tolerance</b>	1.00	5.80	2.70	1.34
<b>Weight Control</b>	1.00	6.00	2.17	1.44

Table 2

*Predictor Variable and ERP Attentional Bias Correlations*

	<b>P1 Active</b>	<b>P1 Inactive</b>	<b>N1 Active</b>	<b>N1 Inactive</b>	<b>P2 Active</b>	<b>P2 Inactive</b>	<b>N2 Active</b>	<b>N2 Inactive</b>
<b>Cigarettes Daily</b>	-.396*	.181	-.415*	-.185	-.044	-.224	-.245	-.244
<b>CO Level</b>	.098	.190	.183	.020	-.151	.235	.205	.061
<b>QSU-B Factor 1</b>	.329	.657**	.648**	.614**	.144	.639**	.256	.311
<b>QSU-B Factor 2</b>	-.084	.511	.302	.187	-.033	.131	.027	-.081
<b>FTND</b>	-.282	.085	-.215	-.323	-.017	-.271	-.160	-.474**
<b>Affiliative Attachment Automaticity</b>	.079	.281	.093	.262	.042	.404*	.045	.295
<b>Behavioral Choice- Melioration</b>	.265	.194	.492*	.422*	.043	.513**	.324	.353
<b>Cognitive Enhancement</b>	.012	-.174	.124	.091	-.367	.202	.202	.243
<b>Craving</b>	.127	-.050	.274	.290	-.095	.328	.169	.191
<b>Cue Exposure– Associative Processes</b>	.124	.156	.323	.288	-.054	.437**	.163	.221
<b>Loss of Control</b>	-.043	.053	-.045	.204	-.081	.256	-.047	.264
<b>Negative Reinforcement</b>	.122	.008	.347	.249	-.130	.357	.234	.210
<b>Positive Reinforcement</b>	.409*	.136	.656**	.436*	.100	.554**	.284	.282
<b>Social- Environmental Goals</b>	.125	.117	.248	.257	-.001	.386	-.015	.258
<b>Tolerance</b>	-.107	.010	-.093	-.058	.097	-.035	.073	-.117
<b>Weight Control</b>	.067	.310	.163	.242	-.238	.335	-.045	.176

\*\* Denotes a correlation with a significance level of  $p < .05$ .

\* Denotes a correlation with a significance level of  $p < .08$ .

Table 3

*N1 Inactive Attentional Bias Multiple Regression Model.*

Variable	B	SE B	Beta
QSU-B Factor 1	.286**	.136	.534
Positive Reinforcement	.432	.993	.111
Behavioral Choice- Melioration	.127	1.314	.026
F		3.582**	
R Square		.387**	

\*\* Denotes a correlation with a significance level of  $p < .05$ .

\* Denotes a correlation with a significance level of  $p < .08$ .

Table 4

*N1 Active Attentional Bias Hierarchical Multiple Regression Model.*

Variable	Level 1			Level 2		
	B	SE B	Beta	B	SE B	Beta
Cigarettes Smoked	-.545*	.274	-.415	-.292	.246	-.223
QSU-B Factor 1				.262**	.126	.456
Positive Reinforcement				1.244	1.037	.299
Behavioral Choice-Melioration				-.075	1.215	-.014
F		3.963*			5.447**	
R Square		.173*			.577	
R Change					.404**	

\*\* Denotes a correlation with a significance level of  $p < .05$ .

\* Denotes a correlation with a significance level of  $p < .08$ .

Table 5

*P2 Inactive Attentional Bias Multiple Regression Model.*

Variable	B	SE B	Beta
QSU-B Factor 1	.406*	.222	.461
Positive Reinforcement	2.090	1.401	.270
Cue Exposure	-.832	1.317	-.161
Affiliative Attachment	1.695	1.252	.328
F		4.258**	
R Square		.516**	

\*\* Denotes a correlation with a significance level of  $p < .05$ .

\* Denotes a correlation with a significance level of  $p < .08$ .



Table 6

*ERP Component Attentional Bias Correlations*

	1	2	3	4	5	6	7	8
<b>(1) P1 Active</b>	—							
<b>(2) P1 Inactive</b>	.323	—						
<b>(3) N1 Active</b>	.794**	.409*	—					
<b>(4) N1 Inactive</b>	.454**	.425**	.431**	—				
<b>(5) P2 Active</b>	.720**	.350	.619**	.228	—			
<b>(6) P2 Inactive</b>	.486**	.574**	.398*	.779**	.164	—		
<b>(7) N2 Active</b>	.521**	.229	.661**	.143	.536**	.127	—	
<b>(8) N2 Inactive</b>	.365	.202	.003	.734**	.142	.785**	.045	—

\*\* Denotes a correlation with a significance level of  $p < .05$ .

\* Denotes a correlation with a significance level of  $p < .08$ .

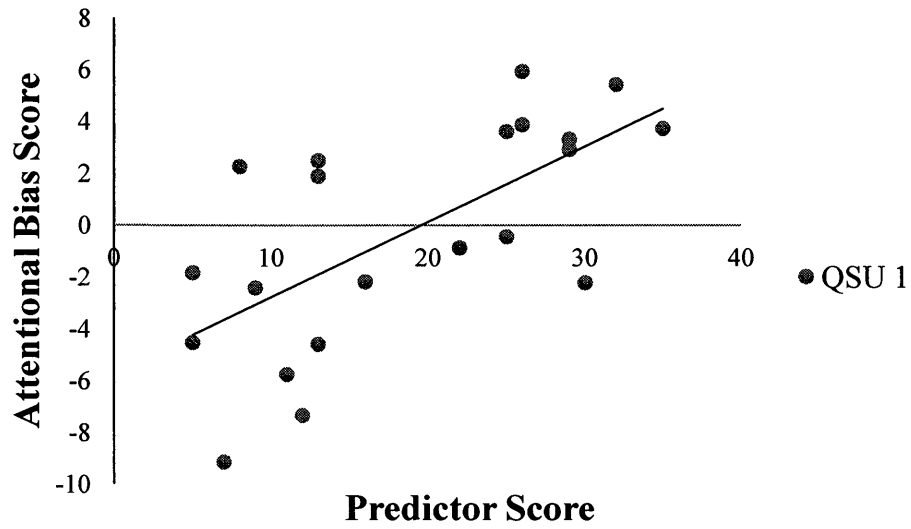


Figure 1. Scatterplot of P1 inactive attentional bias scores and QSU – B Factor 1 scores.

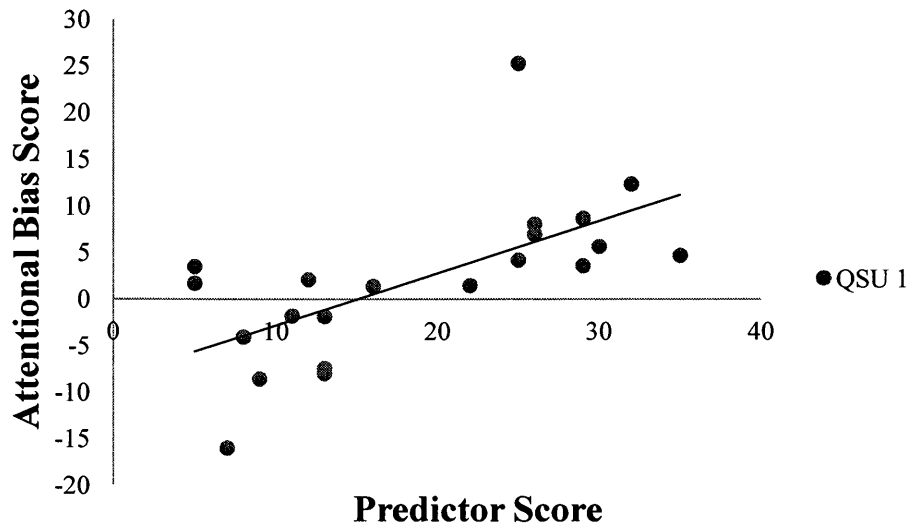


Figure 2. Scatterplot of P2 inactive attentional bias scores and QSU – B Factor 1 scores.