Smoking’s Acute Effects on Emotional Response in Adolescent Smokers

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DISSERTATION

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<td>Description</td>
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</tr>
<tr>
<td>ANS</td>
<td>Autonomic nervous system</td>
</tr>
<tr>
<td>$\beta$</td>
<td>Standardized beta</td>
</tr>
<tr>
<td>bpm</td>
<td>Beats per minute</td>
</tr>
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<td>CES-D</td>
<td>Center for Epidemiologic Studies Depression Scale</td>
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<tr>
<td>CO</td>
<td>Carbon monoxide</td>
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<tr>
<td>CReSS</td>
<td>Clinical Research Support System</td>
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<tr>
<td>dB</td>
<td>Decibels</td>
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<tr>
<td>EMG</td>
<td>Electromyography</td>
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<td>HLM</td>
<td>Hierarchical Linear Modeling</td>
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<td>HR</td>
<td>Heart rate</td>
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<tr>
<td>HZ</td>
<td>Hertz</td>
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<tr>
<td>IAPS</td>
<td>International Affective Picture System</td>
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<tr>
<td>$\mu$S</td>
<td>microSiemens</td>
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<td>mFTQ</td>
<td>Modified Fagerstrom Tolerance Questionnaire</td>
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<tr>
<td>ms</td>
<td>Milliseconds</td>
</tr>
<tr>
<td>mV</td>
<td>Millivolts</td>
</tr>
<tr>
<td>mV*s</td>
<td>Millivolt seconds</td>
</tr>
<tr>
<td>ns</td>
<td>Nonsignificant</td>
</tr>
<tr>
<td>n</td>
<td>Number subgroup</td>
</tr>
<tr>
<td>$p$</td>
<td>p-value</td>
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<tr>
<td>PNS</td>
<td>Peripheral nervous system</td>
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<td>$r$</td>
<td>Pearson’s bivariate correlation</td>
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<td>SC</td>
<td>Skin conductance</td>
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<td>SD</td>
<td>Standard deviation</td>
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<td>SJC</td>
<td>Shiffman-Jarvik Withdrawal Questionnaire – Craving Subscale</td>
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<tr>
<td>SJWQ</td>
<td>Shiffman-Jarvik Withdrawal Questionnaire</td>
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<td>SER</td>
<td>Startle eyeblink response</td>
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<tr>
<td>SNS</td>
<td>Sympathetic nervous system</td>
</tr>
<tr>
<td>( t )</td>
<td>Student’s t-value</td>
</tr>
<tr>
<td>V</td>
<td>Volt</td>
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SUMMARY

Smoking is a problem behavior most often initiated in adolescence, and previous research indicates that smoking might influence change in affect in adolescent smokers similarly to adults. Most of these studies rely on self-report, however, and the use of psychophysiological indices for the evaluation of emotional response could enhance the validity and reliability of these findings. Therefore, the goal of the current study was to determine any affective changes that adolescent smokers may experience after smoking and what might moderate these effects. Participants completed two study sessions during which they smoked a cigarette ad libitum or relaxed for an equivalent period of time. Then, they viewed a series of positive, neutral, and negative visual stimuli while providing continuous psychophysiological measures of startle eyeblink response (SER), skin conductance (SC), and heart rate (HR). They also reported smoking behavior, changes in craving after smoking, and baseline nicotine dependence and depression symptoms. Overall, the findings are mixed: smoking decreased SC response and increased HR, as anticipated. In contrast, smoking increased SER magnitude and amplitude as well as SC level and had no effect on SER latency. These findings can be understood through both the stress-coping and incentive salience theories of addiction. Future studies might incorporate a more comprehensive picture of the relationship between craving, affect regulation expectancies, and smoking’s acute effects on psychophysiology, as previous research has established strong associations between these factors. Given that the current study is part of a longitudinal project, it will also be valuable to see how acute response to smoking in the lab relates to future smoking behavior and changes in nicotine dependence and mood symptoms over time.
I. INTRODUCTION

Smoking is a problem behavior that most often begins in adolescence, with 13.5% of eighth graders reporting that they have taken at least a puff of a cigarette during their lifetimes. Indeed, over the last 40 years, cigarettes have remained the most popular regularly-used substance by high school students (Miech, Johnston, O’Malley, Bachman, & Schulenberg, 2015). Despite our knowledge of this epidemiology, we have yet to fully understand the developmental trajectory of smoking behavior. In adults, motivation to smoke often involves an affect regulation component, as smokers seek to alleviate the negative affect associated with nicotine withdrawal (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004). Previous research indicates that this might be true of young, light smokers as well (Kassel et al., 2007), and that smokers can become dependent on nicotine earlier than originally thought (DiFranza, 2008). Understanding the relationship between the development of smoking behavior and emotion regulation is of great importance to future prevention and intervention efforts within this population.

A. Smoking and Emotion Regulation

1. Smoking to decrease negative affect

The long-standing stress-coping model of addiction suggests that desire to decrease negative affect may serve as a dominant motive for substance use (Wills & Shiffman, 1985). Indeed, there is evidence that many adolescents, like adults, smoke to relieve stress and negative affect (Kassel, Stroud, & Paronis, 2003; Koval & Pederson, 1999; Siqueira, Diab, Bodian, & Rolnitzsky, 2000). Longitudinal studies of adolescents show that those who develop regular smoking behavior perceive greater levels of stress in their lives and do not effectively utilize adaptive coping strategies, perhaps making them
more likely to view smoking as a viable coping resource (Dugan, Lloyd, & Lucas, 1999).

Piasecki and Baker (2000) speculate that perhaps smoking replaces severe, unexpected periods of negative affect with smaller, more predictable episodes. Indeed, some have described smoking as part of a vicious cycle (Parrott, 1999), whereby stress and negative affect precipitate smoking, and eventually, consistent smoking behavior results in nicotine dependence. The nature of nicotine dependence dictates that there will be some manifestation of physiological and/or psychological withdrawal after abstinence of even brief duration, and that the smoker will smoke again to alleviate these symptoms, thus perpetuating the cycle. Baker and colleagues (2004) illustrate a similar model of addiction in which withdrawal-based learning results in sensitivity to the interoceptive cues of negative affect, leading to further substance use.

In a three-year study of adolescents, however, Wills, Sandy, and Yaegar (2002) found a unidirectional relationship between negative affect and smoking, such that negative affect precipitated smoking behavior and not that smoking “caused” stress. Further, whereas nicotine withdrawal does increase self-reported negative affect, it does not always appear to affect involuntary emotional regulation, as indexed by the startle eyeblink response (SER; Piper & Curtin, 2006). Though it has become increasingly clear that young smokers might share motives for smoking with their adult counterparts, it remains unclear whether smoking actually reduces subjective stress and negative affect among newer smokers (Kassel et al., 2003). Further, stress-coping models of addiction acknowledge that increasing positive affect motivates substance use as well (Wills & Shiffman, 1985).
2. Smoking to increase positive affect

Researchers often understand the positive effects of smoking through the incentive salience theory of addiction (Robinson & Berridge, 1993). This posits that the loss of control seen in addiction arises from the attribution of increased salience to drug-related stimuli through repeated associations. This process might be especially relevant for stimulants like nicotine, as they act on areas of the brain that increase appetitive states, which may in turn maintain smoking behavior (Stewart, de Wit, & Eikelboom, 1984). In addition, the act of smoking carries a particular set of visual and behavioral cues that eventually become associated with the positive subjective effects of smoking via classical conditioning. Eventually, the association of smoking’s acute effects with these cues is strong enough that exposure to them alone mimics the subjective effects of smoking. Previous meta-analyses (e.g., Carter & Tiffany, 1999) have found evidence of this phenomenon via both self-report and objective measures.

These theories can be particularly helpful when considering how individuals move from initiating to maintaining smoking behavior. Barr and colleagues (2008) found that giving non-smokers a single dose of transdermal nicotine increased their response to rewards that were unrelated to smoking. They concluded that this effect may help illustrate reinforcement of early smoking behavior. Indeed, dependent and non-dependent smokers do not differ on response to music rewards after smoking, indicating that withdrawal relief does not influence this experience (Perkins & Karelitz, 2013). Light smokers (i.e., those who smoke less than 20 cigarettes a day) also spend more time looking at smoking-related (vs. neutral) visual cues than heavy smokers or non-smokers (Hogarth, Mogg, Bradley, Duka, & Dickinson, 2003). These kinds of cues often prompt smokers to focus on stimuli-induced internal states and emotional memories (Janes et al., 2010). Thus, it seems that positive reinforcement might be especially important when
individuals experience negative mood due to stress (Dagher, Tannenbaum, Hayashi, Pruessner, & McBride, 2009; Vinci, Kinsaul, Carrigan, & Copeland, 2015).

Among heavier smokers, nicotine administration alone often results in increased positive affect (Lindsey et al., 2013), and therefore, increased self-administration (Perkins, Grobe, Caggiula, Wilson, & Stiller, 1997). As individuals become more dependent on nicotine, however, incentive salience is less clearly related to hedonic experience. That is, it is possible to have strong cravings and smoke without experiencing any anticipated or actual positive subjective effects (Glautier, 2004). Incentive salience theory conceptualizes the associative learning process as happening outside of conscious thought and executive function. Given this potential disconnect between drug-seeking behavior and the ability to describe resulting subjective experience, it is important to find reliable ways of measuring affect that do not rely solely on self-report.

B. Psychophysiology as a Measure of Emotion

While it might seem relatively simple to describe acute mood states, the measurement of emotion remains a challenge in current research. Mauss & Robinson (2009) outline a model of emotion that involves experience of a situation, appraisal of this situation based on one’s history and context, and a series of subjective and objective responses. However, there is a possible disconnect between subjective and objective emotional response that limits the interpretation of self-report when assessing the affective consequences of both acute smoking effects and nicotine withdrawal (Piper & Curtin, 2006). Indeed, self-report is the most common form of assessment, despite the bias sometimes associated with describing one’s own affective experience. Correspondingly, employment of a multidimensional approach that draws upon
psychophysiological indices of affect (e.g., Stritzke, Patrick, & Lang, 1996) for the evaluation of emotional response in drug research offers clear advantages with respect to enhancing the validity and reliability of these findings.

1. **Startle Eyeblink Response.**

   Psychophysiology has a long history of serving as an indicator of emotional response. The startle eyeblink response (SER) in particular first received interest in the 1930s, when Landis and Hunt (1939) observed a full-body reaction to a pistol shot. The fastest and most reliable element of this response was the sudden closure of the eyelids, and further research showed that this action remained even when a stimulus failed to engage the full-body startle response. Later research using rats found that startle probes presented in the context of affective stimuli (in this case, a light and buzzer that had previously been associated with a toy pistol shot) evoked a larger full-body reaction, and therefore a larger SER, than control stimuli (Brown, Kalish, & Farber, 1951). Building on this work, Davis and colleagues (1987) described the fear-potentiated SER in humans, which was first elicited in classical conditioning studies. Lang and colleagues (1990) subsequently hypothesized that SER would vary similarly by affective valence and arousal level. In this case, valence refers to the likelihood of assuming either an appetitive (approach) or defensive (avoidance) behavioral stance, whereas arousal refers to general energy level. In support of this theory, studies have found that the amygdala, which plays a central role in maintaining vigilance and detecting threats to the self, helps modulate the SER under perceived threat as well (Davis, 1989; Koch & Schnitzler, 1997).

   In accordance with this research, perhaps the most salient pattern to emerge is the
impact of negative affect on SER. Masterson and Crawford (1982) organized negatively-valenced animal behaviors into a “defense motivation system,” arguing that such behaviors comprise a set of natural defensive reactions. Accordingly, whole-body startle has been shown to vary as a function of affective state in rodents (Davis & Astrachan, 1978; Greenwald, Bradley, Cuthbert, & Lang, 1998; Grillon & Davis, 1997; Hamm, Greenwald, Bradley, & Lang, 1993; Schmid, Koch, & Schnitzler, 1995). This response mirrors the SER in humans, and so might originate from the same type of defensive system.

Given this history, one might predict that SER would be heightened among those experiencing negative affect, which is associated with both fear and the desire to defend oneself. Researchers often induce such affect by presenting participants with a series of images that vary in emotional content (i.e., positively, neutral, and negative). In relation to neutral images, the SER has been shown to increase significantly for negative images (Bradley Cuthbert, & Lang, 1988; Cook, Hawk, Davis, & Stevenson, 1991; Lang, Davis, & Öhman, 2001; Vrana Spence, & Lang, 1988), and decrease significantly for positive images (Bradley et al., 1988; Vrana et al. 1988). Both of these effects appear to increase with general arousal level (Lang, 1995), especially among children and adolescents (McManis, Bradley, Berg, Cuthbert, & Lang, 2001). The SER is just one part of a “behavioral cascade” that results from exposure to an aversive stimulus (Lang et al., 2001), however. Skin conductance (SC) and heart rate (HR) better capture overall arousal level.

2. Skin conductance and heart rate.

SC is a measure of sweat gland response that, along with HR, is largely affected
by the autonomic nervous system (ANS), which modulates peripheral functions (Öhman, Hamm, & Hugdahl, 2000). It can be divided further into the sympathetic (activation) and parasympathetic (relaxation) branches (Mauss & Robinson, 2009). SC predominantly reflects activity from the sympathetic nervous system (SNS), and so tends to increase monotonically with general arousal level (Lang & Bradley, 2010). In contrast, HR integrates input from the peripheral nervous system (PNS) as well as the SNS. There is evidence that HR can decrease even as other indices of SNS activity increase (Bradley & Lang, 2000; Lang et al., 1997b; & Libby, Lacey, and Lacey, 1973), and indeed, many theories of emotion integrate the valence of a situation to explain this. The eye is the primary pathway into the brain for mammals, so images and text are particularly evocative of a strong emotional response (Lang & Bradley, 2010). As SER is potentiated, SC increases with arousal, and HR decelerates before accelerating to prepare for action (Lang et al., 2001).

Whereas specific patterns of SER characterize exposure to differentially-valenced stimuli, SC and HR are thought to provide more direct measures of general physiological arousal (Lang et al., 1990). SC appears to be least influenced by valence, as it increases monotonically with arousal level (Lang, Bradley, & Cuthbert, 1998), though some studies suggest that SC changes most when one is viewing negative images, as compared with neutral or positive images (McManis et al., 2001). HR, however, might be able to distinguish among different emotions (Ekman, Levenson, & Friesen, 1983; Levenson, 1992). For example, previous studies have shown that HR decelerates when viewing unpleasant pictures and accelerates when viewing pleasant pictures (Lang et al., 1998), though it also accelerates when processing fearful text, as compared with neutral text.
(Vrana, Cuthbert, & Lang, 1989). It is unclear whether differences in understanding images versus encoding text might explain these discrepant results.

C. Acute Smoking and Nicotine Effects on Psychophysiology

These psychophysiological measures become particularly salient when considering smoking’s acute effects on affect, as the competing subjective and objective effects of nicotine create a paradox that has been observed over decades of smoking research. Nicotine is a stimulant that increases HR, perhaps even more in adolescents than adults (Corrigall, Zack, Eissenberg, Belstoio, & Scher, 2001). However, its effect on various other indices of emotional response is unclear. On one hand, studies have shown that smoking decreases emotional response as indicated by SER (Cinciripini et al., 2006; Mueller, Mucha, & Pauli, 1998), SC response (Gilbert & Hagen, 1980), HR (Mueller, Mucha, & Pauli, 1998), and tolerance of electric shock (Nesbitt, 1973), though the latter findings have been questioned (Shiffman & Jarvik, 1984). Further, greater negative affect in abstinent smokers has been indicated by increased SER, relative to nonsmokers (Grillon, Avenevoli, Daurignac, & Merikangas, 2007).

Other researchers have questioned these results by demonstrating increased SER in response to smoking-related sentences (Elash, Tiffany, & Vrana, 1995) and immersive, three-dimensional visual smoking (vs. neutral) cues (Muñoz, Idrissi, Sánchez-Barrera, Fernández-Santaella, & Vila, 2013). Further, nicotine administered via nasal spray increases SER amplitude in women who are not nicotine-deprived (Robinson et al., 2007). Despite these mixed results, however, most smokers over the last few decades have claimed that smoking relaxes them (Frith, 1971; Ikard, Green, & Horn, 1969; Ikard & Tompkins, 1973). Subjective measures have indicated that recency of smoking is
inversely associated with negative affect, as smoking successfully reduces experience of negative emotion (Kassel et al., 2007). In addition, smoking can increase subjective feelings of head rush and decrease both positive (Perkins, Jetton, & Keenan, 2003) and negative affect (Gilbert et al., 2008).

Previous studies suggest that several factors might be important moderators of nicotine’s effects on emotional response, because they often interact with discrete mood states to predict smoking behavior. Perhaps most intuitively, nicotine dependence seems to motivate smoking behavior (Bold, Yoon, Chapman, & McCarthy, 2013; Bolt et al., 2009; Edwards & Kendler, 2011), particularly among those for whom smoking is a more automatic behavior or provides significant relief of negative affect (Piper et al., 2004; 2008). Nicotine dependence is also associated with greater negative affect as a result of nicotine withdrawal, regardless of how much time has passed since smoking a cigarette (Robinson et al., 2011). However, a recent study showed that nicotine dependence did not significantly moderate situational (e.g., mood) effects on smoking behavior (Shiffman & Rathbun, 2011). Therefore, its role in moderating nicotine’s effects on emotional response is unclear.

In addition to nicotine dependence, mood symptoms often further motivate smoking behavior. In fact, nicotine dependence and depressive symptoms often co-occur. Longitudinal studies have shown that depression symptoms correlate with future smoking and development of nicotine dependence among adolescents (Fergusson, Lynskey, & Horwood, 1996; McKenzie, Olsson, Jorm, Romaniuk, & Patton, 2010). Further, analyses from another study within the larger program project indicate that those with higher scores on depression measures also score higher on nicotine dependence.
across multiple assessments over months and years (Dierker et al., 2015). Even after controlling for nicotine dependence, depressive symptoms are associated with greater negative affect and, therefore, continued smoking behavior (Brodbeck, Bachmann, Brown, & Znoj, 2014). Previous studies have shown that anxiety symptoms are associated with more severe mood symptoms of withdrawal as well (Leventhal, Ameringer, Osborn, Zvolensky, & Langdon, 2013), often resulting in a greater compulsion to stave off withdrawal symptoms by smoking.

The relationship between nicotine withdrawal and mood symptoms is an important one, because negative affect appears to be a main symptom of nicotine withdrawal (Hughes, 2007). There is mixed evidence that depression specifically is a nicotine withdrawal symptom. Some smoking cessation studies have documented increased scores on depression measures during abstinence (Allen, Hatsukami, & Christianson, 2003; Pomerleau, Namenek Brouwer, & Pomerleau, 2001), whereas others have not (Brown et al., 2001; Hall et al., 1996; Kinnunen, Doherty, & Garvey, 1996; Levin et al., 1994). Depression often co-occurs with smoking behavior (Lasser et al., 2000), so smoking cessation can provoke a recurrence of symptoms (Covey, Glassman, & Stetner, 1997). In addition, numerous studies have documented increased anxiety in the first few weeks of smoking cessation (Gilbert et al., 1998; 2002; Hughes, 1992; Jorenby et al., 1996; Ward, Swan, & Jack, 2001). Given that many previous studies have compared nicotine-deprived smokers to non-nicotine-deprived smokers (e.g., Cinciripini et al., 2006; Mueller et al., 1998) or to those who do not smoke at all (e.g., Grillon et al., 2007), it can be difficult to parse which of nicotine’s effects on negative affect are due to relief of nicotine withdrawal versus genuine improvement in mood overall. Thus, it is
important to examine smoking’s effects on various mood states to facilitate a more nuanced understanding of this phenomenon.

D. Summary

We still have yet to fully understand the relationship between the development of smoking behavior and emotion regulation. Extant literature indicates that, like adults, adolescents may smoke to relieve stress and negative affect. Other studies have shown that positive reinforcement also influences the development of smoking behavior. It remains unclear, therefore, how smoking affects mood among newer smokers, especially given the interpretative problems inherent with self-report measures (e.g., inconsistent understanding and reporting of mood state across individuals). Previous research has identified SER as a relatively objective indicator of emotional response, with distinct patterns associated with differentially-valenced stimuli. Further, SC and HR provide options for indexing general arousal. Smoking is often used as a strategy for coping with negative affect, and smokers generally report a reduction of negative emotion subsequent to smoking. However, research on nicotine’s acute effects on psychophysiological indices of emotional response is mixed. Findings from our own lab indicate that smoking a single cigarette reduces emotional response in adolescents, as indicated by generally increased SER latency and reduced SC (Kassel et al., 2015). That is, we have found that smoking decreases various indices of emotional response in the absence of visual stimuli, as compared with measurements taken at baseline. It is unclear whether this decrease in emotional response is specific to negative affect, however, and knowledge of such effects is crucial to furthering our understanding of the processes governing development of smoking behavior and factors that might moderate these changes.
E. Aims of the Current Study

Therefore, the goal of the current study was to determine the affective changes derived from smoking by adolescent smokers. We also examined smoking’s effects on craving, nicotine dependence, and depression symptoms as potential moderators of nicotine’s effect on emotional response. Participants completed two study sessions during which they smoked a cigarette ad libitum or relaxed for an equivalent period of time. Then, they viewed a series of positive, neutral, and negative visual stimuli while providing continuous measures of SER, SC, and HR. They also reported subjective mood throughout the session. We hypothesized that the standard affective pattern for psychophysiological indices would be present such that SER magnitude and amplitude, as well as SC level and response, would be highest in response to negative slides, whereas SER latency and HR would be lowest. We also anticipated that smoking would decrease emotional response specific to negative images, as indicated by lower SER magnitude, amplitude, and SC and greater SER latency and HR. In contrast, we anticipated that smoking would increase response to positive slides. Further, we examined whether this change would be moderated by smoking’s effects on craving, baseline nicotine dependence, or baseline depression symptoms.
I. METHOD

A. Participants

The current study draws from a cohort of adolescents who participated in a larger program project that examined the social and emotional contexts of smoking using various methodologies. Participants from 16 area high schools were recruited via survey, which gathered information about their smoking history, intended substance use, demographic background, and parental history of smoking. Based on their responses, they were oversampled for current and past smoking behavior (i.e., having smoked at least one cigarette in their lifetimes). Participants were excluded only if they were former regular users (i.e., smoked 100 cigarettes total during their lives but no cigarettes in the last 30 days) or former occasional smokers (i.e., smoked less than 100 cigarettes but none in the past 12 months); current high rate regular users (smoked 5 or more cigarettes daily in the past 30 days); or provided inconsistent smoking information during screening. Study staff invited eligible students, as well as their parents, to participate in the initial study and subsequent follow-up assessments. As a result, 1263 9th and 10th graders enrolled in the overall program project. All participants and parents provided informed assent and consent, respectively, prior to participation. To protect participant confidentiality, parents were told that both smokers and non-smokers were participating in the current study, though they were aware that smokers would have the option of smoking during one of the study sessions.

A subgroup of 217 adolescents participated in the lab-based study from which the current sample originates within an average of 6 weeks of completing baseline measures from the larger program project. They completed two experimental sessions separated by
6-10 weeks (average inter-session interval: 56 days), and participants who reported smoking at least one cigarette in the last two weeks without a desire to quit qualified as “smokers.” They were offered the chance to smoke ad libitum at one of their study visits. Of the total sample, 122 qualified as smokers, and 97 chose to smoke when offered the chance. The goals of the current study were to determine whether 1) the affective changes derived from smoking by adolescent smokers are specific to negative visual stimuli, and 2) these effects are moderated by nicotine dependence or depressive symptoms. Therefore, analyses include only the 97 participants who smoked during one of their study sessions (see Figure I for complete flowchart). Of note, those who smoked during the study smoked more frequently in the last 30 days than those who chose not to smoke (smoking frequency: \( t(120) = 3.26, p = 0.001 \)); they did not differ on any other demographic or smoking behavior variables.

**B. Procedure**

Participants completed two experimental sessions which were most often scheduled between 10am and 7pm. They employed an identical procedure, with two exceptions (see Figure II for session timeline). First, participants were randomized to either the smoking or relaxation condition during the first session such that session order was counterbalanced. This design allowed for within-subject comparisons of emotional response after smoking versus not smoking. Second, different slides were presented during the second visit to avoid habituation effects, though they were matched to the first set on indices of emotional valence and arousal. This matching procedure was successful, based on valence and arousal ratings from participants at the end of study session (valence: \( ts(194) \leq 0.868, ps = ns \); arousal: \( ts(194) \leq 0.541, ps = ns \); see Table I
for these ratings). All procedures were approved by the Institutional Review Board of the University of Illinois at Chicago.

Upon arrival at the lab, all participants provided an expired breath carbon monoxide (CO) reading (Vitalograph EC 50 CO monitor, Vitalograph, Lexington, KY) to verify self-reported smoking recency before completing questionnaires; there were no abstinence requirements. Then, participants were fitted with electrodes to measure SER, SC, and HR and underwent baseline psychophysiological measurements. During the smoking session, they were offered the chance to smoke as much or as little of a cigarette as they wanted, choosing from among eight popular brands, half of which were mentholated. Given the likelihood that many participants would not be in possession of their own cigarettes, we chose these particular brands, including mentholated versions, to increase the desirability of cigarette options. While no formal analyses were conducted, it appears that we were successful in this goal, as no participant cited range of cigarette options as the reason he or she chose not to smoke. Research assistants told participants, “I’m now going to give you the opportunity to smoke a cigarette if you wish. We can offer you any of these eight brands. Would you like to smoke?” If they said they wanted to smoke, the research assistant told them, “Keep in mind you can smoke as much or as little of the cigarette as you wish.” This ad libitum administration method was deemed the best approach for assessing the effects of smoking on emotional response, given that this sample is comprised of light smokers who exhibit considerable variability in their smoking patterns. This procedure was also thought to maximize ecological validity, and has been used in several other studies of adolescent smokers (e.g., Corrigall et al., 2001; Kassel & Shiffman, 1997; Kassel & Unrod, 2000; Zack, Belsito, Scher, Eissenberg, &
Corrigall, 2001).

Those who smoked were instructed to light the cigarette before placing it in a CReSS device (Clinical Research Support System Smoking Topography Machine, Plowshare, Baltimore, MD), which examines aspects of smoking topography, including both puff volume and duration (see Kassel et al., 2007 and Veilleux et al., 2011 for these results). When they were finished smoking, they removed the cigarette butt from the machine and provided a CO reading to assess CO boost attributable to smoking the cigarette. During the non-smoking session, participants were offered an affectively “neutral” magazine and asked to sit and relax for nine minutes. This was provided in lieu of a behavioral task to avoid inadvertently inducing any pronounced emotional states during the non-smoking condition. Following the smoking or relaxation period, all participants completed a second psychophysiological assessment (i.e., SER, SC, and HR) and set of self-report questionnaires. At this time, they viewed the IAPS slide presentation and provided psychophysiological assessment throughout, as described below. Following this presentation, all participants completed a third set of self-report questionnaires. At the end of the session, they were debriefed and paid $45 for their participation, for a total of $90 for completing both.

C. Self-Report Measures

1. Smoking behavior.

At each session, participants completed a questionnaire about their current smoking behavior. Total number of cigarettes smoked during their lifetime and total number of cigarettes smoked in the last 30 days were of particular interest in the current study. Both capture individual differences in smoking behavior that could affect how
participants experience smoking a single cigarette during the study sessions. More specifically, total lifetime cigarettes illustrates participants’ exposure to smoking over time whereas total number of cigarettes smoked in the last 30 days indicates participants’ more recent smoking behavior.

2. Shiffman-Jarvik Withdrawal Questionnaire – Craving Subscale.

To assess craving, participants completed the six-item craving subscale of the Shiffman-Jarvik Withdrawal Questionnaire (SJWQ; Shiffman & Jarvik, 1976), which assesses withdrawal symptoms in general. Summing individual items created a total score for the craving subscale (SJC), and the measure demonstrated adequate internal consistency with the current sample (coefficient alpha = 0.52). Of interest in the current study is how craving changed after smoking. Therefore, all analyses include craving change as calculated by subtracting pre-cigarette craving from post-cigarette craving ratings during the smoking session.

3. Modified Fagerstrom Tolerance Questionnaire.

To assess nicotine dependence, participants completed the seven-item version of the Fagerstrom Tolerance Questionnaire that has been modified for use with adolescent smokers (mFTQ; Prokhorov, Koehly, Pallonen, & Hudmon, 1998; Prokhorov, Pallonen, Fava, Ding, & Niaura, 1996), as part of a battery given by the larger program project. Unlike the original scale, the mFTQ includes continuous response choices for all items except when asking whether individuals smoke soon after waking in the morning (scored as yes/no). Scoring for this last item is rescaled to compare to the original (Prokhorov et al., 1996). Items from the original scale, and therefore the modified version, correlate to physiological measures of dependence, containing items that assess several aspects of
smoking behavior (Fägerstrom, 1978; Heatherton, Kozlowski, Frecker, & Fägerstrom, 1991). Summing individual items created a total score, and the measure demonstrated adequate internal consistency with the current sample (coefficient alpha = 0.66). In adolescents, the clinical cut-off is 3, such that those scoring at or above this level are likely experiencing nicotine dependence.

4. Center for Epidemiologic Studies Depression Scale.

To assess depression symptoms, participants completed the Center for Epidemiologic Studies Depression inventory (CES-D; Radloff, 1977). The CES-D is a 20-item measure that assesses the frequency with which an individual has experienced depressive symptoms during the last week, from 0 (rarely or none of the time) to 3 (most or all of the time). Research indicates that the CES-D provides a valid and reliable way to measure depressive symptoms in adolescents (Lewinsohn, Rohde, & Seeley, 1998; Radloff, 1991; Roberts, Andrews, Lewinsohn, & Hops, 1990). Summing individual items created a total score, and the measure demonstrated strong internal consistency with the current sample (coefficient alpha = 0.89). In adolescents, the clinical cut-off is 22 for males and 24 for females, such that those scoring at or above this level are likely experiencing depressive symptoms.

D. Physiological Measures

1. Emotional stimuli.

Emotional stimuli were presented as a series of 36 slides from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1997a; Lang, Öhman, & Vaitl, 1988). The IAPS slides were developed to provide a set of normative emotional stimuli as a tool for investigating emotion (Lang et al., 1988). These images have been
previously rated and normed on dimensions of valence and arousal (Lang et al., 1997a). Within the current study, positive slides included scenes of food, children, and animals; negative slides included scenes of aimed guns and threatening animals; and neutral slides included pictures of household objects, plants, and neutral faces. Positive and negative slides used here were equal in rated arousal, and both were higher in arousal than neutral slides. No slides containing nudity or scenes of death were shown to ensure that images were age-appropriate.

2. **Startle eyeblink response.**

During the slide presentation, the SER was elicited by presenting white noise bursts (95 dB for 50 ms) via Telephonics headphones (Telephonics, Farmingdale, NY). On eight of the trials for each slide type, the acoustic startle probe stimulus occurred at a variable point during the 6-second slide viewing period (3, 4, or 5 seconds after slide onset; cf. Stritzke et al., 1995). One of the four slides within each content block was presented without a startle stimulus. In addition, each block included three startle stimuli that were presented during the 12-19-second interval between slide presentations. These random inter-slide aural stimuli served to reduce the temporal predictability of the startle and to de-emphasize a specific association between slide viewing and startle stimuli (see Vrana et al., 1988). Each presentation began with six neutral (habituation) slides, which allowed participants to habituate to the startle stimulus and prevented habituation from disproportionately affecting emotional response to any one type of slide.

The eyeblink component of the startle response was measured unilaterally by recording electromyography (EMG) activity from the orbicularis oculi muscle beneath the left eye. The raw EMG signal at each site was amplified (gain = 5,000) and filtered
(below 90 Hz and above 250 Hz) via a BIOPAC bioamplifier (BIOPAC Systems, Inc., Goleta, CA). Data were then recorded and displayed using BIOPAC Systems’ AcqKnowledge (Version 4.0) data acquisition software, as installed on a Pentium IV computer. This software logged 1,000 samples per second, which were integrated over a time constant of 10ms (i.e., over 10 samples). Trained research assistants completed computer-assisted scoring to obtain all SER variables. That is, after all filters were applied, the data was visually inspected to find peak response 21-120 ms after stimulus onset (as suggested by Balaban, Losito, Simons, & Graham, 1986 for acoustic startle probes). SER magnitude is quantified as the difference between maximum response during the slide and the average of the response just before the slide (most often zero). SER amplitude is quantified as SER magnitude without the non-response trials (Blumenthal et al., 2005). Finally, SER latency is quantified as the onset of the SER in relation to the beginning of the slide.

3. **Skin conductance level and heart rate.**

SC was assessed continuously throughout the presentation of the IAPS slides. Electrodes were filled with lubricating jelly and positioned on the hypothenar eminence of the nondominant hand. These electrodes were then connected to a BIOPAC isolated SC coupler that provided a constant voltage of 0.5 V. The raw SC signal was amplified (gain = 10), recorded, and displayed as described above. SC level was obtained directly from the signal, whereas a high pass filter at 0.05 Hz isolated SC response. Both were averaged across the 6-second slide-viewing period to yield final values. HR was recorded by electrodes placed on the right and left inner forearms. The bioamplifier noted every R-wave, which represents the electrical stimulus as it passes through the
main portion of the heart’s ventricular walls. This allowed the software to calculate HR as well as the R-R interval. The raw HR signal was amplified (gain = 1,000), recorded, and displayed as described above. Second-by-second values were averaged across the 60-second slide-viewing period to provide a single value for each slide in the current study. Future analyses will address how HR changes in real time (i.e., affective chronometry) with respect to acceleration and deceleration, as this is another potentially important component of nicotine’s effects on attentional and emotional factors.

E. Data Management

Each study session yielded measurements of six primary dependent variables: SER magnitude, SER amplitude, SER latency, SC level, SC response, and HR. Following active data collection and scoring via Acqknowledge, this program exported discrete values into a spreadsheet to facilitate data management. Observations of psychophysiological variables throughout the slide presentation were collapsed on slide type, such that there was a single value for positive, neutral, and negative slides for each participant in each session. SER magnitude and amplitude, as well as SC response, underwent natural log transformation to reduce skewness and kurtosis (skewness ≥ 3.19, kurtosis ≥ 11.89). Separate files were created for session- (e.g., SER magnitude) and person-level (e.g., mFTQ) data to facilitate hierarchical linear modeling (HLM). These data files were then imported into HLM7 (Scientific Software International, Skokie, IL) for analysis.

F. Statistical Analyses

Participant demographics (e.g., gender) and background information (e.g., smoking behavior) were summarized by mean and frequency, as appropriate. To
address the primary aims of the study, separate models were fitted for each dependent variable. For example, SER magnitude was entered as the session-level outcome, with session order (i.e., smoked during first session vs. smoked during second), smoking condition, slide type, and the interaction of smoking condition and slide type entered as session-level predictors. Then, the slope parameters from each session-level predictor became the person-level outcomes, with baseline measures (e.g., nicotine dependence) entered as between-subjects variables. Note that change in craving was only entered in person-level equations for smoking condition and the smoking condition by slide type interaction, as this value was derived from the smoking session only. Smoking behavior variables were also entered as covariates. All categorical variables were dummy-coded and continuous variables were grand-mean centered. Finally, all non-significant predictors were excluded from the final model.

Given the number of tests involved in this set of analyses, alpha adjustment was considered to avoid type I error. There has been considerable discussion of this question in the literature, and a particularly lively debate (Hewes, 2003; O’Keefe; 2003a, 2003b; Tutzauer, 2003) resulted in a loose consensus that the alpha level should be adjusted downward in specific circumstances that are not present here (e.g., in the presence of disparate claims that support a single theory). Therefore, the threshold for significance is maintained at the usual alpha = 0.05 level.
II. RESULTS

A. Participant Characteristics

Table II illustrates general characteristics for all participants. Participants were between 15 and 16 years old, half were male, and most were Caucasian and non-Hispanic/Latino. At their first visit, most smokers reported smoking at least 100 cigarettes in their lifetimes. Moreover, they reported smoking on a little over half the days in a month and an average of three cigarettes a day, for a total of about 75 cigarettes in the last 30 days. Smoking decreased craving overall, $t(96) = 11.92, p < 0.001$, and scores on the mFTQ and CES-D indicated low levels of nicotine dependence and depression, respectively.

B. Correlations Between Person-Level Predictors

Relationships between all person-level predictors were examined to prevent multicollinearity in subsequent HLM analyses. Scores on the mFTQ were significantly correlated with total number of cigarettes smoked in the last 30 days, $r(96) = 0.53, p < 0.001$, and total number of cigarettes smoked during their lifetime, $r(96) = -0.52, p < 0.001$. After some consideration of the distribution of self-report measures, the mFTQ and CES-D scores were transformed into dummy-coded categorical variables. That is, participants were separated into those who scored under the clinical cut-off and those who scored at or over this number. In addition to addressing issues of multicollinearity, it allows a more clinically meaningful interpretation of any moderator effects. Total number of cigarettes smoked in the last 30 days and total number of cigarettes smoked during their lifetime were also significantly correlated, $r(96) = -0.46, p < 0.001$. Given this correlation as well as the fact that many reported both not smoking in their lifetime
and smoking in the last 30 days, only total number of cigarettes smoked in the last 30 days is included as a covariate in subsequent analyses (see Table III for final HLM equations).

C. Startle Eyeblink Response

At the session level, session order, smoking condition, and slide type were all significant predictors of SER magnitude. SER magnitude decreased across sessions, $\beta = -0.48, p < 0.001$, and this effect was more pronounced among those with depressive symptoms, $\beta = 0.38, p < 0.05$. Smoking increased SER magnitude, $\beta = 0.05, p < 0.05$, and participants showed significantly variable response to slides, $\beta = 0.06, p < 0.01$. That is, SER magnitude was greatest for negative slides, and this effect was more pronounced among those with depressive symptoms, $\beta = 0.06, p < 0.05$. No other session-level or person-level predictors were significant, $\beta s \leq 0.04, ps = ns$. See Figure III for a graph of SER magnitude by smoking condition and slide type.

Similarly, session order, smoking condition, and slide type were all significant predictors of SER amplitude at the session level. SER amplitude decreased across sessions, $\beta = -0.53, p < 0.001$, and this effect was more pronounced among those with depressive symptoms, $\beta = 0.49, p < 0.01$. Smoking increased SER amplitude, $\beta = 0.09, p < 0.05$, and participants showed significantly variable response to slides, $\beta = 0.08, p < 0.001$. That is, SER amplitude was greatest for negative slides, and this effect was more pronounced among those with depressive symptoms, $\beta = 0.08, p < 0.05$. No other session-level or person-level predictors were significant, $\beta s \leq 0.06, ps = ns$. See Figure IV for a graph of SER amplitude by smoking condition and slide type.

Finally, slide type was a significant predictor of SER latency at the session level,
$\beta = -1.32$, $p < 0.001$, such that SER latency was lowest for negative slides. No other session-level or person-level predictors were significant, $\beta_s \leq 0.30$, $ps = ns$. See Figure V for a graph of SER latency by smoking condition and slide type and Table IV for a summary of all significant effects for SER.

**D. Skin Conductance and Heart Rate**

At the session level, smoking condition was a significant predictor of SC level such that smoking increased SC level, $\beta = 3.70$, $p < 0.05$. This effect was more pronounced among those with greater total number of cigarettes smoked in the last 30 days, $\beta = 0.06$, $p < 0.01$. Despite individuals having a statistically similar response to all slides, $\beta = 0.01$, $p = ns$, there was also a significant interaction between smoking condition and slide type, $\beta = 0.30$, $p < 0.05$, such that smoking increased SC level most for negative slides. No other session-level or person-level predictors were significant, $\beta_s \leq 0.01$, $ps = ns$. See Figure VI for a graph of SC level by smoking condition and slide type.

Conversely, smoking condition was a significant predictor of SC response such that smoking decreased SC response, $\beta = -0.37$, $p < 0.05$. Despite individuals having a statistically similar response to all slides, $\beta = -0.08$, $p = ns$, there was also a significant interaction between smoking condition and slide type, $\beta = 0.08$, $p < 0.05$, such that smoking decreased SC response least for neutral slides. No other session-level or person-level predictors were significant, $\beta_s \leq 0.06$, $ps = ns$. See Figure VII for a graph of SC response by smoking condition and slide type.

Finally, session order, smoking condition, and slide type were significant predictors of HR at the session level. HR decreased across sessions, $\beta = -0.08$, $p < 0.05$. 

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In addition, smoking increased HR, $\beta = 9.59, p < 0.001$, and participants showed significantly variable response to slides, $\beta = -0.23, p < 0.01$. That is, HR was lowest for negative slides. This effect was attenuated among those with nicotine dependence, $\beta = -0.43, p < 0.01$, and more pronounced among those with depressive symptoms, $\beta = 0.27, p < 0.05$. These main effects were qualified by an interaction, $\beta = -0.21, p < 0.01$, such that smoking increased HR most for negative slides. No other session-level or person-level predictors were significant, $\beta s \leq 0.10, ps = ns$. See Figure VIII for a graph of HR by smoking condition and slide type and Table V for a summary of all significant effects for SC and HR.
III. DISCUSSION

The goal of the current study was to determine the affective influence of smoking on adolescent smokers. We also examined change in craving after smoking, nicotine dependence, and mood symptoms as potential moderators of nicotine’s effect on emotional response. We hypothesized that the standard affective pattern for psychophysiological indices would be present such that SER magnitude and amplitude, as well as SC level and response, would be highest in response to negative slides, whereas SER latency and HR would be lowest. We also anticipated that smoking would decrease emotional response specific to negative images, as indicated by lower SER magnitude, amplitude, and SC and greater SER latency and HR. In contrast, we anticipated that smoking would increase response to positive slides. Further, we examined whether this change would be moderated by smoking’s effects on craving, baseline nicotine dependence, or baseline depression symptoms.

A. Smoking’s Effects on Emotional Response

1. Evidence of decreased negative affect

Smoking’s effects on SC response and HR conform to well-documented patterns in the literature (e.g., Gilbert & Hagen, 1980; Mueller et al., 1998) and are best understood using a stress-coping framework. That is, smoking decreased acute arousal, especially for negative and neutral slides, and this indicated at least some relief of negative affect. In addition, previous research has shown that emotional arousal, particularly as measured by SC, often increases among abstinent smokers in tandem with subjective craving (Bailey, Goedeker, & Tiffany, 2009; Drobos & Tiffany, 1997; Saladin et al., 2012; Tong, Bovbjerg, & Erblich, 2007). It follows that because acute smoking in
the lab decreased craving here, it also decreased emotional arousal.

2. Alternate explanations

Surprisingly, smoking increased SER magnitude and amplitude as well as SC level. Further, smoking’s effect on SC level was greatest for negative slides and more pronounced among those who smoked a greater number of cigarettes over the last 30 days. The general finding that smoking increased emotional response can be understood using the incentive salience framework. That is, smoking a single cigarette in the lab might have increased subjective positive experience, and this possibly increased responsivity and arousal. Others have demonstrated similar effects with SER (Elash et al., 1995; Muñoz et al., 2013), though the latter related these effects to the aversive aspect of craving, which decreased in the current study. Research focusing on gender differences in response to stress have linked increased SER to the hedonic effects of nicotine in men and women (Robinson et al., 2007) and decreased aggression in women (Verona & Curtin, 2007). As indicated by SC level, which is thought to be more stable than SC response, those who smoke more frequently might have been particularly sensitive to these effects. It is somewhat unclear why this would be particularly true for negative slides, though some studies suggest that SC changes most when one is viewing negative images, as compared with neutral or positive images (McManis et al., 2001).

A series of older studies on smoking’s attentional mediational effects on emotion may provide an alternative explanation for these results. In a series of studies, researchers applied the same logic of Steele & Josephs’ (1990) alcohol myopia model to smoking (Kassel, 1997). They found that smoking decreased self-reported anxiety and increased response times to a reaction time task only if participants smoked while
completing a distraction task; smoking had no effect (Kassel & Shiffman, 1997) or slightly increased anxiety (Kassel & Unrod, 2000) among those who smoked in the absence of a benign stimulus. This is compatible with Gilbert’s (1995) suggestion that smoking effectively relieves negative affect in the face of a moderate, as compared with intense, stressor. It is possible that these phenomena were present in the current study. That is, participants smoked alone in a room devoid of distraction during their session, and thus, could have experienced increased negative affect. It follows, then, that this would result in increased SER magnitude and amplitude as well as SC level.

B. Other Patterns of Emotional Response

SER magnitude and amplitude decreased across sessions, and this is indicative of habituation of the startle reflex through repeated presentations of the startle probe. Previous research has indicated that this pattern is normative in healthy participants both within (Bradley, Lang, & Cuthbert, 1993) and across sessions (Abel, Waikar, Pedro, Hemsley, & Geyer, 1998). This habituation was especially pronounced among those with depressive symptoms. Previous studies have found both increased (Allen, Trinder, & Brennan, 1999) and decreased SER (Kaviani, Gray, Checkley, Raven, Wilson, & Kumari, 2004) among those who have greater depressive symptoms. In the current study, it is possible that those with greater depression symptoms showed even greater SER magnitude and amplitude at baseline due to the novelty of the paradigm and the unanticipated volume and quality of the startle noise. Habituation to the task would have resulted in a steeper decline in SER magnitude and amplitude over time. Somewhat surprisingly, HR also decreased across sessions, indicating that it did not habituate over time.
With regard to affective patterns, SER magnitude and amplitude were both largest for negative slides, and SER latency and HR were lowest. This is consistent with the well-documented pattern of emotional response to valenced stimuli (Bradley et al., 1988; Cook et al., 1991; Lang et al., 2001; Vrana et al., 1988). The slide effects on SER magnitude and amplitude as well as HR were more pronounced among those with depressive symptoms. This is consistent with other studies that look explicitly at psychophysiology (e.g., Allen et al., 1999) as well as generally decreased reward responsiveness (Liverant et al., 2014) among those with depressive symptoms, and among those also experiencing nicotine withdrawal as well (Pergadia et al., 2014). We can speculate that preexisting mood symptoms exacerbated emotional response to negative stimuli as indicated by these measures. In addition, the slide effects on HR were attenuated among those with nicotine dependence. That is, those with nicotine dependence showed less emotional response for negative slides. It is possible that this is a result of greater sensitivity to the hedonic effects of smoking.

C. Limitations and Future Directions

Despite the strengths of the current study design, there are limitations. As with any laboratory study, external validity might be questioned. The relationship between emotion and smoking is influenced not just through the pharmacological effects of nicotine but through more general contextual factors as well. Another study within the larger program grant has examined mood immediately before and after a smoking episode using ecological momentary assessment (EMA). Results indicate that, after smoking, adolescents felt significantly higher positive and lower negative affect than they did at other random assessment timepoints (Hedeker, Mermelstein, Berbaum, & Campbell, 2009). The lab-based data presented here, as well as other
projects from our lab (Kassel et al., 2015), examine the next step in this emotion regulation process by indicating that these adolescent smokers actually do reap the anticipated affective changes from smoking.

As mentioned earlier, in the future, examination of HR changes within each slide might better illustrate how SNS and PNS influences interact in real time. A previously published article from our lab examined measures of heart-rate variability (i.e., respiratory sinus arrhythmia, RSA) both before and after smoking a single cigarette (vs. relaxing for an equivalent period of time). Heart-rate variability has been linked closely to PNS (Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000), and our results showed that smoking decreased RSA while increasing HR (Conrad, Gorka, & Kassel, 2015). It is possible we would see initial decreases in HR during each slide as well.

Future studies might incorporate a more comprehensive picture of the relationship between craving, affect regulation expectancies, and acute smoking’s effects on psychophysiology, as previous research has established strong associations between these factors (e.g., Robinson, Lam, Carter, Wetter, & Cinciripini, 2012). Given that the current study is part of a longitudinal project, it will also be valuable to see how acute response to smoking in the lab relates to changes in smoking behavior, nicotine dependence, and mood symptoms over time, as in vivo research indicates this might be a dynamic process over the course of weeks (Van Zundert, Ferguson, Shiffman, & Engels, 2012) and years (Selya et al., 2013). This relationship remains unclear and is an important piece of the theoretical puzzle surrounding escalation to continued and chronic cigarette smoking. Further elucidation of its nature may inform and refine future prevention and early intervention efforts.
## TABLE I.

PARTICIPANT RATING OF SLIDE VALENCE AND AROUSAL BY SESSION

<table>
<thead>
<tr>
<th></th>
<th>Valence</th>
<th></th>
<th>Arousal</th>
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<tbody>
<tr>
<td></td>
<td>Visit 1</td>
<td>Visit 2</td>
<td>Visit 1</td>
<td>Visit 2</td>
</tr>
<tr>
<td>Positive Slides</td>
<td>2.05 (0.44)</td>
<td>2.14 (0.45)</td>
<td>2.87 (0.83)</td>
<td>2.89 (0.75)</td>
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<tr>
<td>Neutral Slides</td>
<td>3.00 (0.32)</td>
<td>3.00 (0.30)</td>
<td>3.76 (0.76)</td>
<td>3.78 (0.81)</td>
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<tr>
<td>Negative Slides</td>
<td>4.12 (0.49)</td>
<td>4.14 (0.45)</td>
<td>2.78 (0.93)</td>
<td>2.74 (0.90)</td>
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### TABLE II.

PARTICIPANT CHARACTERISTICS

<table>
<thead>
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<th>General Characteristics</th>
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<tbody>
<tr>
<td>Age at baseline (years)</td>
<td>15.7 (0.6)</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>51 (52.6%)</td>
</tr>
<tr>
<td>Race (Caucasian)</td>
<td>77 (79.4%)</td>
</tr>
<tr>
<td>Ethnicity (not Hispanic/Latino)</td>
<td>81 (83.5%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Smoking Behavior</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of cigarettes smoked during lifetime</td>
<td></td>
</tr>
<tr>
<td>Did not smoke</td>
<td>39 (40.2%)</td>
</tr>
<tr>
<td>1 cigarettes</td>
<td>29 (29.9%)</td>
</tr>
<tr>
<td>2-5 cigarettes</td>
<td>16 (16.5%)</td>
</tr>
<tr>
<td>6-10 cigarettes</td>
<td>7 (7.2%)</td>
</tr>
<tr>
<td>11-20 cigarettes</td>
<td>3 (3.1%)</td>
</tr>
<tr>
<td>21-50 cigarettes</td>
<td>1 (1.0%)</td>
</tr>
<tr>
<td>51-99 cigarettes</td>
<td>1 (1.0%)</td>
</tr>
<tr>
<td>201-499 cigarettes</td>
<td>1 (1.0%)</td>
</tr>
<tr>
<td>Smoking frequency (out of the last 30 days)</td>
<td>17.64 (10.8)</td>
</tr>
<tr>
<td>Smoking quantity (cigarettes/day)</td>
<td>3.13 (3.0)</td>
</tr>
<tr>
<td>Total number of cigarettes smoked in the last 30 days</td>
<td>75.34 (96.8)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Craving (SJWQ)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-cigarette</td>
<td>22.0 (4.9)</td>
</tr>
<tr>
<td>Post-cigarette</td>
<td>15.62 (5.1)</td>
</tr>
<tr>
<td>Decrease in craving</td>
<td>6.35 (5.2)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Nicotine Dependence(mFTQ)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Average score</td>
<td>2.7 (1.4)</td>
</tr>
<tr>
<td>Below clinical cut-off</td>
<td>55 (56.7%)</td>
</tr>
<tr>
<td>At or above clinical cut-off</td>
<td>42 (43.3%)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mood Symptoms (CES-D)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Average score</td>
<td>18.4 (9.9)</td>
</tr>
<tr>
<td>Below clinical cut-off</td>
<td>68 (70.1%)</td>
</tr>
<tr>
<td>At or above clinical cut-off</td>
<td>29 (29.9%)</td>
</tr>
</tbody>
</table>

NOTE: Data are Mean (SD) or n (%). Clinical cut-off for mFTQ is 3. Clinical cut-off for CES-D is 22 for males, 24 for females.
TABLE III.
FULL HIERARCHICAL LINEAR MODEL

Session-level model: Dependent variable = π_0 + π_1(session order) + π_2(smoking condition) + π_3(slidi e type) + π_4(smoking condition x slide type) + e

Person-level model:
π_0 = β_{00} + r_0
π_1 = β_{10} + β_{11}(mFTQ) + β_{12}(CES-D) + r_1
π_2 = β_{20} + β_{21}(SJC) + β_{22}(mFTQ) + β_{23}(CES-D) + r_2
π_3 = β_{30} + β_{31}(mFTQ) + β_{32}(CES-D) + r_3
π_4 = β_{40} + β_{41}(SJC) + β_{42}(mFTQ) + β_{43}(CES-D) + r_4

NOTE: Random effects at the person level are assumed to have distribution of N (0, σ^2). Random effects at group level are assumed to have distribution of N (0, τ_00). Additional dependent variables are SER amplitude, SER latency, SC level, SC response, and HR. All person-level equations include total number of cigarettes smoked in the last 30 days as a covariate.
TABLE IV.
SUMMARY OF SIGNIFICANT PREDICTORS FOR STARTLE EYEBLINK RESPONSE

<table>
<thead>
<tr>
<th></th>
<th>SER Magnitude</th>
<th>SER Amplitude</th>
<th>SER Latency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Session Order</td>
<td>-0.48***</td>
<td>-0.53***</td>
<td></td>
</tr>
<tr>
<td>CES-D</td>
<td>0.38*</td>
<td>0.49**</td>
<td></td>
</tr>
<tr>
<td>Smoking Condition</td>
<td>0.05*</td>
<td>0.09*</td>
<td></td>
</tr>
<tr>
<td>Slide Type</td>
<td>0.06**</td>
<td>0.08***</td>
<td>-1.32***</td>
</tr>
<tr>
<td>CES-D</td>
<td>0.06*</td>
<td>0.08*</td>
<td></td>
</tr>
</tbody>
</table>

NOTE: All values are standardized betas ($\beta$). Session-level predictors are left-aligned and related person-level predictors are indented.

*p ≤ 0.05, **p ≤ 0.01, ***p ≤ 0.001
TABLE V.

SUMMARY OF SIGNIFICANT PREDICTORS FOR SKIN CONDUCANCE AND HEART RATE

<table>
<thead>
<tr>
<th></th>
<th>SC Level</th>
<th>SC Response</th>
<th>HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Session Order</td>
<td></td>
<td></td>
<td>-0.08*</td>
</tr>
<tr>
<td>Smoking Condition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cigarettes</td>
<td>3.70*</td>
<td>-0.37*</td>
<td>9.59***</td>
</tr>
<tr>
<td>Slide Type</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>mFTQ</td>
<td></td>
<td>-0.23**</td>
<td></td>
</tr>
<tr>
<td>CES-D</td>
<td></td>
<td>-0.43**</td>
<td></td>
</tr>
<tr>
<td>Smoking Condition x Slide Type</td>
<td>0.30*</td>
<td>0.08*</td>
<td>-0.21**</td>
</tr>
</tbody>
</table>

NOTE: All values are standardized betas ($\beta$). Session-level predictors are left-aligned and related person-level predictors are indented. Total cigarettes = total number of cigarettes smoked in the last 30 days. *$p \leq 0.05$, **$p \leq 0.01$, ***$p \leq 0.001$
1263 participants in the overall program project

217 participants in the lab-based study

95 participants did not qualify as smokers
• 60 indicated they were trying to quit
• 35 had not smoked in the past 2 weeks

122 participants qualified as smokers

25 did not smoke

97 did smoke
• 52 during Visit 1
• 45 during Visit 2

FIGURE I.
PARTICIPANT FLOWCHART
Figure II.

Session Timeline

0: Participant arrives
Informed consent
CO #1
SR #1

21: Electrode placement

35: SER/SC/HR

37: CO #2

43: CO #3
SER/SC/HR

45: SR #2

60: SR #3

80: SR #4

81: Debriefing and compensation

Smoke

Slide presentation

Slide viewing/rating

Note: Numbers represent minutes after participant arrival. CO = expired breath carbon monoxide reading; SR = self-report questionnaires; SER/SC/HR = psychophysiology reading; smoke = smoking/relaxation period
FIGURE III.
STARTLE EYEBLINK RESPONSE MAGNITUDE BY SMOKING CONDITION AND SLIDE TYPE

NOTE: Data points reflect intercepts for these measures. Error bars reflect standard error and may not be visible due to scale.
FIGURE IV.

STARTLE EYEBLINK RESPONSE AMPLITUDE BY SMOKING CONDITION AND SLIDE TYPE

NOTE: Data points reflect intercepts for these measures. Error bars reflect standard error and may not be visible due to scale.
FIGURE V.

STARTLE EYEBLINK RESPONSE LATENCY BY SMOKING CONDITION AND SLIDE TYPE

NOTE: Data points reflect intercepts for these measures. Error bars reflect standard error and may not be visible due to scale.
FIGURE VI.
SKIN CONDUCTANCE LEVEL BY SMOKING CONDITION BY SLIDE TYPE

NOTE: Data points reflect intercepts for these measures. Error bars reflect standard error and may not be visible due to scale.
FIGURE VII.

SKIN CONDUCTANCE RESPONSE BY SMOKING CONDITION AND SLIDE TYPE

NOTE: Data points reflect intercepts for these measures. Error bars reflect standard error and may not be visible due to scale.
FIGURE VIII.
HEART RATE BY SMOKING CONDITION AND SLIDE TYPE

NOTE: Data points reflect intercepts for these measures. Error bars reflect standard error and may not be visible due to scale.
CITED LITERATURE


depressive disorders and nicotine dependence in a cohort of 16-year-olds. 
*Archives of General Psychiatry, 53*(11), 1043–1047.


Clinical Psychology, 64, 1003–1009.


Lindsey, K. P., Bracken, B. K., MacLean, R. R., Ryan, E. T., Lukas, S. E., & Frederick, B. D. (2013). Nicotine content and abstinence state have different effects on subjective ratings of positive versus negative reinforcement from smoking. *Pharmacology Biochemistry and Behavior, 103*(4), 710–716.


ORIGINAL INSTITUTIONAL REVIEW BOARD (IRB) APPROVAL FOR PROGRAM PROJECT

UNIVERSITY OF ILLINOIS AT CHICAGO

Office for the Protection of Research Subjects (OPRS)
Office of the Vice Chancellor for Research (MC 672)
203 Administrative Office Building
1737 West Polk Street
Chicago, Illinois 60612-7227

Approval Notice
Initial Review (Response To Modifications)

January 4, 2005

Robin J. Mermelstein, PhD
Psychology
1747 W Roosevelt
Rm 558, M/C 275
Chicago, IL 60612
Phone: (312) 996-1469 / Fax: (312) 413-4122

RE: Protocol # 2004-0621
“Social-Emotional Contexts of Adolescent Smoking - Longitudinal Study”

Dear Dr. Mermelstein:

Your Initial Review (Response To Modifications) was reviewed and approved by IRB #2 by the Expedited review process on December 16, 2004. You may now begin your research.

Please note the following information about your approved research protocol:

Protocol Approval Period: December 16, 2004 - November 17, 2005
Approved Subject Enrollment #: 2800
Performance Sites: UIC, University of Chicago
Sponsor: NCI - National Cancer Institute
Research Protocol:
  a) Social-Emotional Contexts of Adolescent Smoking - Longitudinal Study
Recruitment Materials:
  b) Enrollment Script Parent Health Habits Version 3 12/07/2004
  c) Enrollment Script Student Health Habits Version 3 12/07/2004
  d) Invitation Letter Version 3 12/07/2004
e) "Piece by Piece" Making Health Connections (Research Project Fact Sheet) Pamphlet (No V#, No Date)

Informed Consents:
   a) Project 2 Family Talk Parental Consent Version 2 10/22/2004
   b) Consent Parent Questionnaire Version 2 10/22/2004

Assents:
   a) Project 2 Family Talk Teen Assent Version 2 10/22/2004
   b) Assent Electronic Diary Version 2 10/22/2004
   c) Assent Project 3 Smoking & Emotions Version 3 12/07/2004
   d) Assent Health Habits Version 3 12/07/2004

Parental Permissions:
   a) Project 3 Parental Permission Smoking & Emotions Version 3 12/07/2004
   b) Parental Permission Electronic Diary Version 3 12/07/2004
   c) Parental Permission Health Habits Version 3 12/07/2004

Other Document:

Please note the Review History of this submission:

<table>
<thead>
<tr>
<th>Receipt Date</th>
<th>Submission Type</th>
<th>Review Process</th>
<th>Review Date</th>
<th>Review Action</th>
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</thead>
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<tr>
<td>09/08/2004</td>
<td>Initial Review</td>
<td>Convened</td>
<td>09/23/2004</td>
<td>Deferred</td>
</tr>
<tr>
<td>11/03/2004</td>
<td>Response To Deferred</td>
<td>Convened</td>
<td>11/18/2004</td>
<td>Modifications Required</td>
</tr>
<tr>
<td>12/13/2004</td>
<td>Response To Modifications</td>
<td>Expedited</td>
<td>12/16/2004</td>
<td>Approved</td>
</tr>
</tbody>
</table>

Please remember to:

➤ Use your research protocol number (2004-0621) on any documents or correspondence with the IRB concerning your research protocol.

➤ Review and comply with all requirements on the enclosure, "UIC Investigator Responsibilities, Protection of Human Research Subjects"

Please note that the UIC IRB has the prerogative and authority to ask further questions, seek additional information, require further modifications, or monitor the conduct of your research and the consent process.

We wish you the best as you conduct your research. If you have any questions or need further help, please contact OPRS at (312) 996-1711 or me at (312) 355-2939. Please send any correspondence about this protocol to OPRS at 203 AOB, M/C 672.

Sincerely,
Jewell Hamilton, MSW  
IRB Coordinator, IRB # 2  
Office for the Protection of Research

Subjects

Enclosures:

1. **UIC Investigator Responsibilities, Protection of Human Research Subjects**
2. **Informed Consent Documents:**
   a) Project 2 Family Talk Parental Consent Version 2 10/22/2004
   b) Consent Parent Questionnaire Version 2 10/22/2004
3. **Assent Documents:**
   a) Project 2 Family Talk Teen Assent Version 2 10/22/2004
   b) Assent Electronic Diary Version 2 10/22/2004
   c) Assent Project 3 Smoking & Emotions Version 3 12/07/2004
   d) Assent Health Habits Version 3 12/07/2004
4. **Parental Permissions:**
   a) Project 3 Parental Permission Smoking & Emotions Version 3 12/07/2004
   b) Parental Permission Electronic Diary Version 3 12/07/2004
   c) Parental Permission Health Habits Version 3 12/07/2004
5. **Recruiting Materials:**
   b) Enrollment Script Parent Health Habits Version 3 12/07/2004
   c) Enrollment Script Student Health Habits Version 3 12/07/2004
   d) Invitation Letter Version 3 12/07/2004
   e) "Piece by Piece" Making Health Connections (Research Project Fact Sheet) Pamphlet (No V#, No Date)
6. **Other Document:**
7. **Form 310 - Protection of Human Subjects, Assurance Identification/Certification/Declaration**

cc: Gary E. Raney, Ph.D., Chairperson, Department of Psychology, M/C 285
CURRICULUM VITAE

Megan Conrad
Cincinnati VA Medical Center
3200 Vine Street
Cincinnati, OH 45208
Email: megan.conrad@va.gov
Preferred Email: mfconrad@gmail.com

EDUCATION
University of Illinois, Chicago, IL
Doctorate of Philosophy
2015
Master of the Arts
2011
Major: Clinical Psychology
Minor: Statistics, Methods, and Measurement

University of Illinois, Urbana-Champaign, IL
Bachelor of Science
2006
Majors: Psychology and Spanish

HONORS AND AWARDS
University of Illinois, Chicago, IL
DMPE Foundation Award for Excellence in Clinical Psychology ($500) 2014
Graduate Student Council Travel Award ($500) 2012-2014
Lavender Research Forum Student Paper Competition ($250) 2014

University of Illinois, Urbana-Champaign, IL
University Honors/Bronze Tablet Scholar 2006
Phi Beta Kappa Honor Society Member 2006
James Scholar in the College of Liberal Arts and Sciences 2003-2006

CLINICAL EXPERIENCE (all services offered in English and Spanish)
Cincinnati VA Medical Center, Cincinnati, OH
Intern 2014-2015
Inpatient Psychiatric Unit and Partial Hospitalization Program
Supervisor: Connie Boehner, Ph.D.
  • Led daily therapy group and conducted individual therapy sessions
  • Performed psychodiagnostic and neuropsychological assessment upon request
  • Participated in daily interdisciplinary treatment team meetings
  • Attended meetings for VA Caregiver Support Program, Suicide Risk Reduction Committee, and Unit Planning Committee
  • Treatment modalities: CBT and interpersonal processing

Substance Abuse Residential Rehabilitation Treatment Program
Supervisor: Teri Bolte, Ph.D., ABPP
  • Co-led daily therapy groups and conducted individual therapy sessions
  • Participated in daily interdisciplinary treatment team meetings
• Co-developed and implemented tobacco-free policy
• Treatment modalities: mindfulness, interpersonal processing, DBT skills, and ACT

Psychiatric Evaluation Center
Supervisor: Rashidat Akinyemi, Psy.D.
• Conducted interviews to determine relevant DSM-5 diagnoses and make appropriate referrals
• Facilitated admission of patients in acute crisis to inpatient psychiatric ward
• Participated in regular interdisciplinary staff meetings

Psychology Psychotherapy Clinic for Long-Term Outpatient Therapy
Supervisor: David Greenwald, Ph.D.
• Provided therapy for individuals with various diagnoses
• Administered and interpreted MMPI at intake
• Tracked symptoms at each session via well-validated self-report instruments (e.g., BAI, PHQ-9)
• Provided case presentation to staff, interns, and post-doctoral fellows
• Treatment modalities: CBT and graduated exposure

Rush Pain Center, Rush University Medical Center, Chicago, IL
Extern 2013-2014
Supervisor: Patricia Merriman, Ph.D.
• Conducted semi-structured interviews to assess for psychological disorders
• Provided in- and outpatient therapy
• Treatment modalities: CBT and mindfulness

Bitch to Quit Smoking Cessation Program, Howard Brown Health Center, Chicago, IL
Extern 2012-2014
Supervisor: Alicia Matthews, Ph.D.
• Clinical trial funded by NIH (#5R01DA023935-02)
• Provided behavioral counseling for smoking cessation groups using treatment manual
• Conducted individual phone sessions during early abstinence
• Treatment modalities: CBT, MI, and 12-step

Office of Applied Psychological Services, University of Illinois, Chicago, IL
Assessment/Therapy Clinician 2009-2014
Supervisors: Gloria Balague, Ph.D.; Nancy Dassoff, Ph.D.; Jon Kassel, Ph.D.; Ellen Herbener, Ph.D.; Amanda Lorenz, Ph.D; and Alicia Matthews, Ph.D.
• Conducted semi-structured interviews and administered self-report questionnaires at intake
• Performed psychodiagnostic and neuropsychological assessment for adults and children
• Provided individual, couples, and family therapy
• Treatment modalities: CBT, ACT, and interpersonal therapy

PEER-REVIEWED PUBLICATIONS


**BOOK CHAPTERS**


**PRESENTATIONS AT SCIENTIFIC MEETINGS**


GRANTS
Collaborator $10,000 1/1/2012-12/31/2012
Effects of Race, Gender, and Sexual Orientation on Experience of Hate Crimes in Chicago
Co-Is: Alicia Matthews, Ph.D.; Paul Schewe, Ph.D.; Kyle Jones, M.A.
Institute for Research on Race and Public Policy
University of Illinois, Chicago, IL

RESEARCH EXPERIENCE
Howard Brown Health Center, Chicago, IL
Consultant 2011-2013
Text Messaging Intervention to Improve ART Adherence among HIV-Positive Youth
Funding: NIH #5R34DA031053-02
PI: Rob Garofalo, M.D., M.P.H.; Co-I: Lisa Kuhns, Ph.D., M.P.H.
- Programmed database for phone screening

Culturally Targeted Smoking Cessation for HIV-positive Men who Have Sex with Men
Funding: University of Chicago
PI: Alicia Matthews, Ph.D.
- Created databases for data entry and management and wrote accompanying codebooks
- Managed, scored, cleaned, and analyzed data for publication

Culturally Targeted and Individually Tailored Smoking Cessation Study: LGBT Smokers
Funding: PI: Alicia Matthews, Ph.D.; Co-Is: Andrea King, Ph.D.; Lisa Kuhns, Ph.D., M.P.H.
- Co-developed study design and protocol
- Programed databases for data collection
- Conducted analyses on pilot data to further adjust study design
- Served as liaison to Spanish-speaking community partners

University of Illinois, Chicago, IL
Research Assistant 2010-2014
Emotional Indices of Withdrawal in Young Adult Smokers
Funding: NCI #1PO1CA98262
PI: Jon Kassel, Ph.D.
- Programmed database for collection of self-report data
- Collected self-report, psychophysiological, and performance data during experimental sessions
- Managed, scored, cleaned, and analyzed data for publication

Project Coordinator 2009-2011
Negative Mood Regulations Expectancies Study
Funding: University of Illinois at Chicago
PI: Jon Kassel, Ph.D.
- Maintained study materials and IRB paperwork
- Collected self-report data during experimental sessions
- Trained and managed undergraduate research assistants in study procedures
University of Chicago, Chicago, IL
Follow-Up Coordinator 2007-2009
Determinants of Drug Preference in Humans
Funding: NIH #2M01RR000055-30
PI: Harriet de Wit, Ph.D.
  • Contacted participants via phone, email, and mail for follow-up assessments
  • Maintained online questionnaires for electronic administration
  • Managed, scored, cleaned, and analyzed data for publication

Research Assistant 2006-2009
Alcohol Stimulation and Sedation in Binge Drinkers
Funding: NIH #5R01AA013746-02
Alcohol Effects on Smoking Urge and Behavior
Funding: NIH #1R03AA015337-01A1
Efficacy of Naltrexone in Women’s Smoking Cessation
Funding: NIH #1R01DA016834-01A2
PI: Andrea King, Ph.D.
  • Co-developed study protocols and staff training manuals
  • Collected self-report, psychophysiological, biological, and performance data during sessions
  • Conducted individual sessions and follow-up interviews for clinical trial
  • Managed, scored, cleaned, and analyzed data for publication

TEACHING EXPERIENCE
University of Illinois, Chicago, IL
Teaching Assistant 2009-2014
Practicum in Interviewing (Undergraduate and Graduate classes)
Instructors: Gloria Balague, Ph.D.
  Evi Behar, Ph.D.
  Ellen Herbener, Ph.D.

Lab in Clinical Psychology
Instructor: Jon Kassel, Ph.D.
  Dan Conybeare, M.A.

Introduction to Research Methods in Psychology
Instructors: Evi Behar, Ph.D.
  Benjamin Storm, Ph.D.
  Jennifer Veilleux, M.A.

Instructor 2012
Colloquium on Teaching Psychology

INVITED TALKS AND GUEST LECTURES
Council of University Directors of Clinical Psychology Programs
Student Experiences of Preparation and Support During the Internship Application Process Jan 2014
Co-Presenters: Michael Williams, M.A., Casey Calhoun, M.A., & Brian Feinstein, M.A.

**University of Illinois, Chicago, IL**
Assessment of Trauma in Clinical Interviews
Practicum in Interviewing
Instructor: Gloria Balague, Ph.D.

Measuring Emotions
Laboratory in Clinical Psychology
Instructor: Dan Conybeare, M.A.

The Effects of Race, Gender, and Sexual Orientation on the Experience of Hate Crimes in Chicago
Co-presenters: Kyle Jones, M.A., Alicia Matthews, Ph.D., & Paul Schewe, Ph.D.
Institute for Research on Race and Public Policy

Non-Recursive Models
Latent Variable Models
Instructor: Maike Luhmann, Ph.D.

Stability of Emotional Response in Adolescent Smokers and Nonsmokers: A Longitudinal Analysis
Current Topics in Clinical Psychology
Instructor: Stew Shankman, Ph.D.

Design Issues in Alcohol Research
Introduction to Research Methods in Psychology
Instructor: Benjamin Storm, Ph.D.

Alcohol Response and Problem Drinking
Developmental Psychopathology
Instructor: Ellen Herbener, Ph.D.

**COMMUNITY LEADERSHIP ROLES**

**Cincinnati VA Medical Center**
Student Member
Training Committee

**Council of University Directors of Clinical Psychology**
Student Member
Board of Directors

**University of Illinois, Chicago, IL**
Panelist for McNair/MARC/RiSE Visiting Day @ UIC
Moderator: Gerry Smith

Panelist for Applying to Graduate Programs in Mental Health
Moderator: Yoko Mori, Ph.D.

2014-2015
2013-2015
2013
2012
Member of Research and Ally Development Subcommittees 2011-2014
Chancellor’s Committee on LGBT Issues

Member of Graduate Student Council 2011-2013
Committee on Graduate Studies

MEMBERSHIP IN PROFESSIONAL ORGANIZATIONS
Society for a Science of Clinical Psychology 2012-present
Research Society on Alcoholism 2012-present
American Psychological Association 2010-present
American Psychological Association of Graduate Students 2010-present
Association for Psychological Science 2010-present