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The Effects of Acute Abstinence from Smoking and Performance-Based Rewards on Performance Monitoring

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Abstract

Rationale—Abstinence from smoking disrupts performance in multiple cognitive domains, and such cognitive effects may serve to maintain smoking behavior. Rather than having specific effects on a narrow domain of processing, abstinence may disrupt more general cognitive control processes and/or motivation.

Objectives—The present study tested the prediction that overnight abstinence from smoking would disrupt a general performance monitoring system indexed via the error-related negativity (ERN). A secondary aim was to determine the extent to which performance-based monetary rewards improved the ERN among smokers and whether the effect of reward was diminished during abstinence.

Methods—The ERN was assessed during a flanker task among 25 heavy, non-treatment-seeking smokers both when smoking as usual and after overnight abstinence; reward and no-reward trial blocks occurred within each session.

Results—As predicted, mean ERN amplitude was reduced during abstinence. The ERN was enhanced by reward; this effect did not vary with smoking abstinence.

Conclusion—This study provides novel data that suggest acute abstinence from smoking disrupts a neurophysiological index of a general performance monitoring system that is involved in a range of cognitive functions. The ERN may be a useful complement to narrow-band cognitive studies of abstinence and interventions designed to target cognition in addiction. Because the ERN was concurrently sensitive to abstinence and performance-based incentives, it may be particularly useful for examining the interplay of cognition and motivation in smoking and smoking cessation.

Keywords

error-related negativity; performance monitoring; smoking; nicotine; abstinence; reward processing

Abstinence from smoking disrupts basic cognitive processes and executive functions. Smokers commonly report concentration difficulties during abstinence (Hughes & Hatsukami, 1986), and standard assessments of withdrawal include cognitive items (e.g., Minnesota Nicotine Withdrawal Scale; Hughes & Hatsukami, 1986, Wisconsin Smoking

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Withdrawal Scale, Welsch et al., 1999). From a negative reinforcement perspective (Baker, Brandon, & Chassin, 2004), smoking is maintained by the amelioration of withdrawal. Thus, the cognitive effects of abstinence may be important in maintaining daily smoking and in the return to smoking after longer periods of abstinence.

In addition to self-report of general cognitive function, lab studies demonstrate that acute abstinence impairs performance in several cognitive domains, including attention (Domier et al., 2007; Leventhal, Waters, Moolchan, Heishman, & Pickworth, 2010), working memory (Jacobsen et al., 2005; Kelemen & Fulton, 2008; Mendrek et al., 2006; Merritt, Cobb, & Cook, 2012), and inhibitory control (McClernon et al., 2008; Powell, Pickering, Dawkins, West, & Powell, 2004). Unfortunately, it is unclear from theory or empirical work whether one or all of these domains should be the focus of future work. The choice is complicated further by the multi-faceted nature of each cognitive domain. As highlighted in critical reviews of the literature on the effects of nicotine, some aspects of attention (Kassel, 1997), memory (Heishman, Kleykamp, & Singleton, 2010), or inhibitory control (see de Wit, 2009) may be more relevant than others. Although theory, meta-analysis, and large-scale empirical studies of comprehensive cognitive batteries may ultimately address the question, most studies of acute abstinence are much more modest and consider an understandably narrow portion of the cognitive range.

An alternative is to focus on more generic or general measures that are relevant to multiple cognitive domains. Many cognitive tasks focus on errors, whether manifested as failures to inhibit or lapses in attention, and related performance measures. Importantly, cognitive neuroscientists have identified a neurophysiological index of a generic performance monitoring system. The *error-related negativity* (ERN) is a negative deflection of electrocortical activity that occurs within 150ms of making an error (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993). Critical for the present discussion, the ERN occurs in a range of paradigms, including widely used tasks for studying attention (Carter et al., 1998), inhibitory control (Liotti, Pliszka, Perez, Kothmann, & Woldorff, 2005), and working memory (Horowitz-Kraus & Breznitz, 2009). Thus, the ERN may be as a useful tool for examining the effects of smoking abstinence on a facet of cognitive performance that is relevant to a range of cognitive processes (e.g., Hajcak et al. 2012). At present, the ERN has been examined within the field of addiction research including alcohol (Bartholow et al., 2012; Easdon et al., 2005; Ridderinkhof et al., 2002; Schellekens et al., 2010), smoking (Franken, van Strien, & Kuijpers, 2009; Luijten, van Meel, & Franken, 2011), cocaine (Franken et al., 2007; Sokhadze et al., 2008), and delta-9-tetrahydrocannabinol (THC; Spronk et al., 2011). Despite growing interest in the ERN in the context of addiction, no study has explicitly tested whether the ERN is reduced by acute abstinence from smoking. Testing this prediction was the primary aim of the current investigation.

Given the importance of rewards in leading models of addiction (Koob & Le Moal, 2008; Robinson & Berridge, 1993, 2000, 2003, 2008), a secondary aim was to evaluate the degree to which abstinence reduced or eliminated the effect of reward on the ERN. Both preclinical and human behavioral pharmacology studies suggest that acute abstinence from nicotine or smoking disrupts the beneficial impact of rewards on performance (Dawkins, Powell, West, Powell, & Pickering, 2006; Epping-Jordan, Watkins, Koob, & Markou, 1998; Powell, Dawkins, & Davis, 2002; c.f. Kalamboka, Remington, & Glautier, 2009). Although cognitive and motivational processes related to addiction are typically studied in isolation, the ERN is notable as a measure that integrates these domains (Hajcak, 2012; Potts, 2011). Specifically, the ERN is grounded in theoretical models that incorporate reinforcement learning and dopamine (Holroyd & Coles, 2002). Empirical studies have demonstrated that the ERN is enhanced by performance-based incentives (Chiu & Deldin, 2007; Hajcak,

Moser, Yeung, & Simons, 2005; Pailing & Segalowitz, 2004). Moreover, results from recent pharmacological investigations lend support to Holroyd and Coles' theoretical appraisal of the role of the mesolimbic dopaminergic system and the ERN (de Bruijn et al., 2004; de Bruijn et al., 2006; Zirnheld et al., 2004). Thus, we predicted that the ERN would be enhanced by monetary incentives compared to no-incentive blocks, but that the effect of incentives on the ERN would be diminished during acute abstinence.

To test our predictions, we tested a community sample of dependent smokers during two visits to the laboratory: one while smoking as usual and one following overnight abstinence. During both visits, the ERN was assessed during a computerized flanker task that included incentive and no-incentive trial blocks. We predicted that the ERN would be reduced during abstinence and enhanced by incentives, with abstinence and incentives interacting such that abstinence would reduce the impact of incentives on the ERN.

Methods

Participants

Participants were 25 adult smokers (15 cigarettes per day; CPD) recruited from fliers posted in the community and from previous smoking studies conducted in our laboratory. Prospective participants were screened by telephone for the following exclusion criteria: current use of other tobacco products, nicotine replacement therapy (NRT), or smoking cessation medication; current use of antidepressants, antipsychotics, or antianxiety medications; actively trying to cut down or quit smoking; pregnancy; past-year diagnosis of or treatment for substance abuse or dependence, past diagnosis of a neurological disorder, lifetime history of schizophrenia or bipolar disorder, or head injury resulting in >10 minutes of loss of consciousness. Table 1 presents demographic and smoking characteristics of the sample.

Procedures

All procedures were approved by the Health Sciences Institutional Review Board and were in compliance with the Declaration of Helsinki. Participants completed two visits scheduled one week apart: 1) smoking as usual and 2) overnight abstinence. To rule out a variety of potential confounds associated with a fixed abstinence order, abstinence order was randomized and counterbalanced across participants (i.e., Abstinent First v. Smoking First). Upon arrival at the first visit, informed consent was obtained. To assess recent smoke exposure and ensure abstinence compliance, expired air carbon monoxide (CO) readings were obtained using a Vitalograph (Lenexa, Kansas) CO monitor before and after participants smoked one preferred brand cigarette (during the smoking as usual visit) or relaxed and read magazines for five minutes (abstinent visit).

Participants were taken to a chamber that was designed to minimize electrical noise and sound during the EEG recording (IAC, Bronx, NY; 2.5 × 2.3-m). Participants acclimated to the chamber while the electrode cap was fitted. Once impedance values were < 10 k Ω , participants were given task instructions. After the task, participants sat quietly for six minutes for collection of resting EEG data (these data were secondary and are not reported in this paper). Following resting EEG, participants completed self-report measures. Participants were remunerated for task performance and participation in the study.

Task

Participants completed a modified version of the Eriksen flanker task (Eriksen & Eriksen, 1974), which is common in studies of performance monitoring (Boksem, Tops, Wester, Meijman, & Lorist, 2006; Davies, Segalowitz, & Gavin, 2004; Franken, van Strien, Franzek,

& van de Wetering, 2007; Hall, Bernat, & Patrick, 2007). The flanker task was presented with E-Prime (ver. 1.2, PST Inc.) on a 43cm CRT monitor. On each trial, participants responded to indicate the direction of a central target arrow (< or >) via thumb-press on a response box (PST Inc.). The central arrow was flanked on both sides by either congruent (e.g., < <) or incongruent arrows (e.g., < >) with equal probability. Each stimulus was displayed for 150ms followed by a variable response window ranging from 1200 to 1600ms, after which visual feedback appeared for 300ms (see Figure 1). The intertrial interval ranged from 1000–1400ms.

Participants completed three practice blocks prior to administration of the task. Following an initial 20-trial practice, participants completed a 96-trial block that emphasized fast responding (while maintaining accuracy). From this speeded practice, a response deadline (mean RT + 0.5 SDRT) was calculated for each participant in each session (within a min of 400 and max of 800). A session-specific RT criterion was employed to reduce differences in error rates between smoking and abstinent sessions. The critical issue is that the magnitude of the ERN often varies inversely with error rate (Holroyd & Coles, 2002; Santesso, Segalowitz, & Schmidt, 2005). In the absence of the RT criterion, a reduced ERN during abstinence could reflect either a true reduction in the ability to monitor performance or simply the impact of differences in error expectancy. The RT deadline allowed us to focus on the former, as in other psychopharmacological studies of the ERN (e.g., de Bruijn et al., 2004, 2006; Spronk et al., 2011). Upon completion of the fast-responding practice block, participants completed a 20-trial reward practice that demonstrated the performance-based incentives and visual feedback used during reward blocks (see Fig. 1).

The actual task consisted of four test blocks (200 trials per block; 2 reward blocks and 2 no-reward blocks), with reward condition alternating between blocks and reward order counterbalanced across participants. Participants were given a short break between blocks. During reward blocks, positive and negative feedback was presented. A green square was presented for correct, fast responses and indicated the participant earned two points. Participants were informed that each point would be redeemed for \$.01USD and that they could earn up to 800 points for task performance (plus 200 points for completing the task). Incorrect fast responses were followed by a red square, indicating that no points were earned. During no-reward blocks, participants were told they would not earn points but should try their best, and ‘feedback’ consisted of a yellow square following correct and incorrect fast responses. In all task blocks, the phrase “Too Slow!” appeared in place of a colored square if the response occurred after the RT deadline.

Self-Report Measures

Following completion of the physiological data collection, participants completed self-report measures (presented in MediaLab, Empirisoft Corporation, New York, New York) to assess nicotine dependence (FTND; Heatherton, Kozlowski, Frecker, & Fagerstrom, 1991), craving (QSU-Brief; Cox, Tiffany, & Christen, 2001), and withdrawal (MNWS; Hughes & Hatsukami, 1986).

Psychophysiological Recording

EEG was recorded continuously from midline sites Fz, FCz, Cz, and Pz (as well as unexamined lateral sites (F3, F4, C3, C4, P3, P4) using a custom cap (ECI, Eaton OH). Ag-AgCl electrodes above and below the left eye provided vertical EOG activity, and an electrode on the forehead served as the ground. Impedance on all channels was kept below 10k Ω . Online electrocortical activity was referenced to the left mastoid. Neuroscan SynAmps2 bioamplifiers (gain of 10 in DC mode) continuously recorded the EEG (low-pass cutoff of 200 Hz) at 1KHz. Offline, signals were low-pass filtered at 30 Hz and re-

referenced to the average mastoids using Matlab (ver. 7.9) EEGLab software (ver. 7.2.9.20b). Eye blink activity measured at VEOG was removed from all scalp sites based on a variation of Semlitsch's algorithm (Semlitsch, Anderer, Schuster, & Presslich, 1986). Trials with RTs faster than 150ms (< 0.05%), omissions (< 0.61%), and EEG shifts of >100 μ V (< 4.8%) were excluded.

Data Reduction^{1, 2, 3}

Response-Locked ERN—Given the topography of the ERN, data from frontocentral midline electrode sites (Fz, FCz, Cz) were examined. Data were segmented into epochs from 200ms before until 1,000ms after each response. Baseline correction was applied in the time period of –200ms to 0 pre-response. Grand average waveforms for all valid correct and incorrect trials collapsed across congruency and reward were constructed to determine the electrode site and time window for assessing the ERN.³ Statistical analyses of ERN (error – correct) difference waveform amplitude suggested that ERN amplitude was maximal at site Cz in the window of 0–100ms.

Behavioral Data—To assess task performance, we computed the percentage of correct responses for each test block. In addition, the individualized response deadline was analyzed for each smoke condition (smoking or abstinence) based on the values obtained during the fast-responding practice block (Mean RT + 0.5 SD). Valid trials were defined as those with reaction times < 150ms. All error trials and only correct trials with reaction times that did not exceed the individualized response deadline (i.e. correct fast responses) were analyzed. Slow responses were not included in primary task performance analyses.

Data Analytic Plan

For all measures Smoke Condition (smoking, abstinence) was the within-subjects factor. Reward (no reward, reward) and Accuracy (error, correct) were within-subjects factors for ERN and task performance analyses. Site served as an additional within-subjects factor for analysis of the response-locked ERN (Fz, FCz, Cz) and stimulus-locked N200 (Fz, FCz, Cz) and P300 (Cz, Pz).

Results

Smoking Variables

Results for select smoking variables are presented in Table 1. Consistent with the abstinence manipulation, expired air CO (ppm) was markedly lower during abstinence compared to smoking as usual, $F(1, 24) = 88.8$, $p < .001$, $d = 1.88$. The average reduction in expired air CO was 74% (Minimum = 53%, Maximum = 92%). Craving (QSU-Brief) and withdrawal (MNWS) were increased during abstinence, $F(1, 23) = 36.7$, $p < .001$, $d = 1.24$, $F(1, 23) = 7.2$, $p < .05$, $d = 0.55$, respectively.

¹Self-report data was missing from one participant during the abstinence visit due to computer failure.

²We had hoped to examine the impact of abstinence on the feedback-related negativity (FRN). Although all participants met the recommended minimum of six trials per condition for the ERN (Olvet & Hajcak, 2009), only 56% met the recommended minimum of 20 trials per condition for the FRN (Marco-Pallares et al., 2010). Given the small subsample ($n = 14$), the FRN was never analyzed.

³Grand average waveforms for all valid correct and incorrect trials were collapsed across congruency. This approach was favored for two reasons. First, we sought to maximize the number of error trials included in waveform computations for the purpose of stability. Second, participants committed very few errors on congruent trials during no reward blocks (Abstinent $M = 3.9$, $SD = 4.2$, Range = 0–15; Smoking $M = 3.3$, $SD = 3.2$, Range = 0–11) as well as reward blocks (Abstinent $M = 2.3$, $SD = 3.4$, Range = 0–14; Smoking $M = 1.9$, $SD = 2.3$, Range = 0–7).

Task Performance⁴

As planned, the tailored RT deadline resulted in comparable accuracy between abstinent and smoking sessions, $F(1,24)=1.24$, $p=.28$. Accuracy was modestly improved during reward compared to no reward blocks, $F(1,24)=12.53$, $p=.002$, $d=0.71$, but this pattern was not influenced by abstinence; Smoke Condition x Reward $F(1,24)=1.85$, $p=.19$. As illustrated in Table 2, accuracy was reduced on incongruent trials, $F(1,24)=203.50$, $p<.001$, but this effect did not interact with Smoke Condition, Smoke Condition x Congruency $F(1,24)=2.85$, $p=.11$.

Abstinence increased the RT response deadline relative to smoking as usual, $F(1,24)=4.28$, $p=.05$, $d=0.41$ (Table 2.). Similarly, mean reaction time was significantly slower during abstinence, Smoke Condition, $F(1,10)=20.63$, $p=.001$, $d=1.40$. Reaction times were significantly slower on error trials, Accuracy $F(1,10)=10.71$, $p=.008$, $d=0.99$. The Smoke Condition x Accuracy interaction was significant, $F(1,10)=29.78$, $p<.001$, indicating that the difference between error and correct reaction times was significantly larger during abstinence (Mean Difference=64.37, Std. Error=14.82, $p=.001$). Responses were significantly faster on congruent trials, Congruency $F(1,10)=31.18$, $p<.001$, $d=1.68$. Error and correct reaction times were not influenced by reward, Accuracy x Reward $F(1,10)=2.00$, $p=.19$ or congruency, Accuracy x Congruency $F(1,10)=0.30$, $p=.60$. The effect of reward was not significant, Reward $F(1,10)=2.77$, $p=.13$.

Error-Related Negativity

As depicted in Table 3, the number of epochs included in the analyses did not differ by Smoke Condition, $F(1,24)=0.46$, $p=.51$. The critical Smoke Condition x Accuracy interaction was not significant, $F(1,24)=0.39$, $p=.54$, indicating that the number of error and correct epochs did not vary by condition. A significant effect of accuracy revealed a greater number of correct compared to error epochs, $F(1,24)=726.08$, $p<.001$, $d=5.39$.

As reflected in Figure 2, error responses were more negative compared to correct responses, Accuracy, $F(1,24)=63.27$, $p<.001$, $d=1.59$. The predicted Smoke Condition x Accuracy interaction was statistically significant, $F(1,24)=5.35$, $p=.03$; as illustrated in Figure 3, the ERN (error minus correct) was smaller during abstinence (Mean error-correct difference = -5.25, Std. Error=0.80, $p<.001$) compared to smoking (Mean error-correct difference = -6.91, Std. Error=0.89, $p<.001$), although an ERN was present in both conditions. Additional comparisons indicated that the effect abstinence was not statistically significant on error trials (Mean smoke-abstinent difference = 0.66, Std. Error=0.94, $p=.49$) or correct trials (Mean smoke-abstinent difference = -1.01, Std. Error=1.23, $p=.42$). Although the ERN was larger at Cz than at Fz, Accuracy x Site linear $F(1,24)=34.7$, $p<.001$, the effect of abstinence on the ERN did not vary by site, Smoke Condition x Accuracy x Site (linear and quadratic) $F_s<1.60$, $p_s>.21$.

The hypothesized Reward x Accuracy interaction was also statistically significant, $F(1,24)=10.05$, $p=.004$. As depicted in Figure 3, separate tests demonstrated that the ERN (error minus correct) was larger during reward (Mean error-correct difference = -6.95, Std. Error=0.74, $p<.001$) compared to no-reward (Mean error-correct difference = -5.21, Std. Error=0.88, $p<.001$). The alternative set of follow-up comparisons revealed that reward

⁴supplemental RT analyses evaluated fast error trials (rather than all error trials) and fast correct trials. Mean reaction time (MRT) did not differ by Smoke Condition, $F(1,5)=3.28$, $p=.13$. RTs were faster for error trials, $F(1,5)=19.51$, $p=.007$, $d=1.80$, but did not interact with Smoke Condition, $F(1,5)=2.59$, $p=.17$. A main effect of congruency was present and indicated that RTs were significantly slower on incongruent trials, $F(1,5)=15.93$, $p=.01$, $d=0.99$. The Congruency x Accuracy, $F(1,5)=2.58$, $p=.17$ and Smoke Condition x Congruency x Accuracy interactions, $F(1,5)=2.72$, $p=.16$ were not significant. Reward had no effect on RTs, $F(1,5)=.01$, $p=.93$. Similarly, all interactions involving Reward were not significant, all F 's < 0.73, all p 's > .43.

significantly increased response-locked activity on correct trials (Mean reward-no reward difference = -1.25, Std. Error = 0.53, $p = .03$), but not on error trials (Mean reward-no reward difference = 0.49, Std. Error = 0.58, $p = .41$). The hypothesized Smoke Condition x Reward x Accuracy interaction was not significant, $F(1,24) = 0.11$, $p = .75$.

Supplementary Analyses

Stimulus-locked ERPs—Although stimulus-locked processing was not the focus of the present study, examination of the N2 (mean amplitude 180–280 ms after stimulus onset) and P3 (mean amplitude 300–750 ms after stimulus onset) can inform the interpretation of the response-locked ERN. Each stimulus-locked component was computed using correct trials and analyzed in separate Smoking Condition x Reward x Congruency x Site (Fz, FCz, Cz for the frontal N2 component; Cz, Pz for the P3 component) repeated measures ANOVA.

The N200 was largest at Cz, Site linear $F(1,24) = 12.78$, $p = .002$, and was influenced by smoke condition, Smoke Condition x Site linear $F(1,24) = 10.95$, $p = .003$, such that N200 amplitude was most negative at Cz during smoking compared to abstinence. Although a main effect of congruency was not observed, $F(1,24) = 1.58$, $p = .22$, there was a significant Smoke Condition x Congruency x Site linear interaction, $F(1,24) = 4.39$, $p = .05$. Specifically, the effect of congruency was most pronounced during abstinence at site Cz. In addition, a Reward x Site interaction emerged, linear $F(1,24) = 9.78$, $p = .01$, such that reward reduced N200 amplitude compared to the no reward condition at site Fz ($p = .02$), but not at FCz or Cz ($p = .15$ and $.83$, respectively). The effect of reward did not vary as a function of Smoke Condition $F(1,24) = 0.37$, $p = .55$, or Congruency $F(1,24) = 0.06$, $p = .80$. The Smoke Condition x Reward x Congruency interaction was not significant, $F(1,24) = 0.79$, $p = .38$.

P300. P300 amplitude did not differ by site, $F(1,24) = 2.09$, $p = .16$, or Smoke Condition, $F(1,24) = 0.81$, $p = .38$. Reward tended to increase P300 amplitude, $F(1,24) = 3.40$, $p = .08$, $d = 0.37$, and this effect was similar across sites, Reward x Site $F(1,24) = 1.89$, $p = .18$, and Smoke Condition, Reward x Smoke Condition $F(1,24) = .03$, $p = .86$. Although a main effect of congruency was not present, $F(1,24) = 0.90$, $p = .35$, a Congruency x Site interaction emerged, $F(1,24) = 5.38$, $p = .03$, indicating that the congruency effect on the P300 was most prominent at site Cz. The effect of congruency did not vary as function of Reward, $F(1,24) = 0.31$, $p = .58$, or Smoke Condition, $F(1,24) = 0.00$, $p = .99$. The Smoke Condition X Congruency x Reward interaction was not significant, $F(1,24) = 0.24$, $p = .63$.

Discussion

The present study tested the separate and combined effects of overnight abstinence from smoking and performance-based incentives on performance monitoring indexed via the error-related negativity (ERN). Consistent with our predictions, the ERN was significantly reduced during abstinence compared to smoking as usual with an effect that appeared component-specific; acute abstinence was not associated with a reduction in the stimulus-locked N200 and P300 brain potentials. ERN amplitude was greater during reward than no-reward blocks. These effects were independent; we found no evidence that abstinence from smoking reduced the impact of performance-based incentives on the ERN.

Despite the substantial interest in the role of cognition in nicotine dependence (e.g., Heishman, et al., 2010; Lerman, Perkins, & Gould, 2009; Sofuoglu, 2010), and in the cognitive neuroscience and psychopharmacology of the ERN (e.g., de Bruijn et al., 2004; de Bruijn et al., 2006; Johannes et al., 2001; Kramer et al., 2007; Olvet et al., 2010; Riba et al., 2005a; Riba et al., 2005b; Spronk et al., 2011; Tiegies et al., 2004; Zirnheld et al., 2004), the current study appears to be the first to demonstrate the sensitivity of the ERN to acute abstinence from smoking. The disruptive effect of abstinence on the ERN was moderate in

size in this sample of heavy smokers recruited from the community. This may have been due in part to the relatively strong abstinence manipulation, with participants abstaining for at least 12 hours and exhibiting substantial reductions in expired-air carbon monoxide and increases in craving and withdrawal symptoms. Still, it is important for future research to replicate the effect of abstinence on the ERN and to test the generalizability of this effect. That is, the effect of abstinence may be more pronounced in heavy, but not light smokers (e.g., Shiffman, Paty, Gnys, Elash, & Kassel, 1995) and may vary as a function of whether smokers are minimally deprived (e.g., two hours) versus prolonged periods of abstinence (Hendricks, Ditte, Drobles, & Brandon, 2006; Hughes, 2007).

The disruption of the ERN by smoking abstinence suggests that withdrawal disrupts a neurophysiological index of performance monitoring. Because the ERN tends to be inversely related to error rate (Holroyd & Coles, 2002; Santesso, et al. 2005), and because error rates tend to increase during abstinence, a tailored RT deadline was employed to equate accuracy across visits (e.g., de Bruijn et al., 2006; Spronk et al., 2011). As planned, accuracy was similar across smoking and abstinent visits, thereby increasing our confidence that the abstinence effect on the ERN reflects a true decrease in performance monitoring. A reasonable next step would be to examine linkages between the ERN and behavioral performance by taking the complementary approach of allowing error rates to vary between smoking and abstinence conditions.

Interpretation of the observed decrement in the ERN during abstinence may be a disruptive component of nicotine withdrawal. Nicotine, a potent dopaminergic agonist (Di Chiara & Imperato, 1988), may moderate the magnitude of the ERN. This interpretation is partially supported by recent neuropharmacological investigations in which dopaminergic antagonists tended to reduce the size of the ERN (de Bruijn et al., 2004; Zirnheld et al., 2004). More broadly, a withdrawal interpretation of the ERN data is consistent with evidence that abstinence is associated with deficits in various cognitive domains, including attention (Domier et al., 2007; Leventhal, Waters, Moolchan, Heishman, & Pickworth, 2010), working memory (Jacobsen et al., 2005; Kelemen & Fulton, 2008; Mendrek et al., 2006; Merritt, Cobb, & Cook, 2012), and inhibitory control (McClernon et al., 2008; Powell, Pickering, Dawkins, West, & Powell, 2004). Although recent research has examined the potential associations between the ERN and cognitive variables (i.e., Horowitz-Kraus & Breznitz, 2009; Larson & Clayson, 2011; Miller, Watson & Strayer, 2012), no study has directly examined the relationship between abstinence effects on the ERN and behavioral indices from multiple cognitive paradigms.

Alternatively, the smaller ERN amplitude during abstinence compared to smoking as usual could reflect a return to a low pre-existing baseline. Consistent with this interpretation, Luijten et al. (2011) found the ERN to be reduced among modestly deprived healthy young smokers compared to a non-smoker control group. However, in a similar comparison, Franken et al. (2009) did not observe a difference in the ERN between smokers and non-smokers.

Testing the nicotine withdrawal versus pre-existing baseline hypotheses is an interesting direction for future work. Given the difficulty of longitudinal work to examine pre-initiation differences, the next step may be to compare smokers during abstinence and smoking as usual (as in the present study) with non-smokers receiving placebo versus nicotine. Testing the influence of nicotine on ERN among non-smokers would also complement emerging work on drug effects on performance monitoring (i.e. cocaine; Franken et al., 2007; Sokhadze et al. 2008; alcohol; Bartholow et al., 2012; Easdon et al., 2005; Ridderinkhof et al., 2002, Schellekens et al., 2010; marijuana; Spronk et al., 2011; amphetamine; de Bruijn et al., 2004).

Extending previous work on the ERN and motivation (Chiu & Deldin, 2007; Hajcak et al., 2005, Pailing & Segalowitz, 2004, Potts, 2011; Potts, Martin, Kamp, & Donchin, 2010), reward increased ERN amplitude. However, the effect of reward on this index of performance monitoring was not influenced by abstinence. Thus, we failed to support the hypothesis that reward processing is attenuated during deprivation from cigarette smoking, a prediction based on both preclinical (Epping-Jordan et al., 1998; Koob & Le Moal, 2008, Robinson & Berridge, 1993, 2000) and human behavioral data (e.g., simple sorting speed; Dawkins et al., 2006).

There are a number of factors that may account for this discrepancy. It is possible that we lacked adequate power to detect the Smoke Condition x Reward interaction in this small sample. However, visual inspection of the means (Figure 3) suggests that this is not the case, as the mean ERN during abstinence/reward is, if anything, larger than the mean during smoking/no-reward. Alternatively, it could be that the effect of abstinence on reward responsivity on cognitive performance or neurophysiological indices is different than the measures typically employed in the preclinical (Epping-Jordan, et al., 1998) or behavioral pharmacology studies (e.g., simple sorting speed; Dawkins et al., 2006; Powell et al., 1996). However, there is evidence a methodological difference, the order of reward and no-reward conditions, may explain the disparities in results with human smokers. Specifically, in an extension of work with the sorting task (Dawkins, et al., 2006; Powell, Dawkins, & Davis, 2002), Kalamboka (Kalamboka et al., 2009) demonstrated that reduced reward responsivity during abstinence was not evident when the order of reward and no-reward conditions was fully counterbalanced. Indeed, their behavioral data for the four Smoking x Reward conditions (Kalamboka et al., 2009, Figure 1) parallel the pattern for the ERN in the present study. Together with Kalamboka et al. (2009), the present data suggest that the impact of abstinence from smoking on reward processing is not a broadly generalizable phenomenon at the behavioral or neurophysiological level in humans.

Viewed differently, the concurrent sensitivity of the ERN to both abstinence and reward may increase its utility for research that integrates motivation and cognition in smoking and addiction. In addition, the concurrent sensitivity of the ERN to both abstinence and incentives opens up other avenues for future work. The majority of attempts to quit smoking are unsuccessful (Cohen et al., 1989). Indeed, research has sought to identify cognitive variables that predict smoking abstinence (Patterson et al., 2010; Powell et al., 2004, 2010; Rukstalis et al., 2005; Schlam et al., 2011). As the ERN may influence behavioral adjustments that follow the commission of an error (Themanson et al., 2012), abstinence-induced decrements in ERN amplitude may represent a neurobiological predictor of smoking relapse. Provided this effect replicates, the ERN may have considerable utility in future work identifying cognitive and motivational predictors of smoking cessation.

In sum, the present study suggests that acute abstinence from smoking is associated with a reduction in ERN amplitude. Reward improved ERN amplitude independent of abstinence. These results supplement the extant literature base that has documented reductions in ERN amplitude as a function of alcohol consumption (Bartholow et al., 2012; Easdon et al., 2005; Ridderinkhof et al., 2002), cocaine abuse/dependence (Franken et al., 2007; Sokhadze et al., 2008), smoking (Luijten et al., 2010), and THC (Spronk et al., 2011). Importantly, this is the first study to evaluate and demonstrate the effect of acute abstinence from smoking on a neurophysiological index of performance monitoring. If this finding is replicable, then performance monitoring may complement future work on both cognitive and motivational factors involved in the maintenance of smoking behavior.

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Fig. 1.

The trial structure of the flanker task. Flanker stimuli (top panel) were presented for 150ms, followed by a variable-duration blank screen during which responses were recorded. Feedback was informative during reward blocks (green for correct; red for incorrect), but not during no-reward blocks; responses that did not meet the individualized response deadline received “Too slow” feedback in both reward conditions. Feedback was followed by a variable-length inter-trial interval (ITI).

Fig. 2.
Grand average waveforms at midline electrode sites (Fz, FCz, Cz) by Smoke Condition, Reward (no reward, reward), and Accuracy (error, correct).

Fig. 3. Mean ERN (error minus correct) amplitude as a function of Smoke Condition and Reward at site Cz, where the Accuracy x Site interaction was maximal.

Table 1**Participant Demographics and Smoking Characteristics**

Sex, % Female	48
Race/Ethnicity, % Minority	20
Age, Mean (SD) Years	40 (11)
Cigarettes Per Day, Mean (SD)	22 (7)
FTND Score, Mean (SD)	5.6 (1.1)
Years Smoking, Mean (SD)	23 (12)

Table 2

Means and standard deviations for task performance variables

	Smoking Mean (SD)	Abstinence Mean (SD)
Response Deadline (ms)	484.52 (87.26)	530.80 (100.27)
Accuracy (% Correct)		
No Reward		
Congruent	.92 (.09)	.92 (.07)
Incongruent	.51 (.20)	.56 (.18)
Reward		
Congruent	.95 (.06)	.96 (.05)
Incongruent	.56 (.20)	.63 (.18)
Too Slow (%)		
No Reward		
Congruent	.05 (.05)	.04 (.03)
Incongruent	.19 (.08)	.17 (.08)
Reward		
Congruent	.02 (.03)	.01 (.01)
Incongruent	.17 (.10)	.13 (.08)
Correct Reaction Time (ms)		
No Reward		
Congruent	370.24 (44.47)	391.94 (50.32)
Incongruent	423.12 (57.02)	453.21 (65.44)
Reward		
Congruent	363.47 (41.89)	383.90 (48.24)
Incongruent	420.15 (57.58)	448.79 (61.41)
Error Reaction Time (ms)		
No Reward		
Congruent	387.65 (99.11)	461.59 (126.59)
Incongruent	420.44 (79.04)	487.81 (120.23)
Reward		
Congruent	376.64 (91.98)	423.90 (115.14)
Incongruent	427.26 (106.99)	463.38 (97.85)

Table 3

ERN Waveform Trial Averages

	Smoking Mean (SD)	Range	Abstinence Mean (SD)	Range
No Reward				
Incongruent Error	38.8 (19.4)	7–70	33.1 (20.9)	4–66
Congruent Error	3.3 (3.2)	0–11	3.9 (4.1)	0–14
Incongruent Correct	134.1 (27.6)	68–184	138.1 (25.0)	96–177
Congruent Correct	166.4 (17.7)	114–193	165.1 (14.2)	130–183
Reward				
Incongruent Error	36.6 (21.3)	8–80	30.6 (23.6)	5–81
Congruent Error	1.9 (2.3)	0–7	2.3 (3.4)	0–14
Incongruent Correct	138.9 (28.6)	61–182	141.0 (27.6)	87–184
Congruent Correct	171.5 (17.4)	125–194	169.6 (17.6)	125–192