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Resting-state EEG, impulsiveness, and personality in daily and nondaily smokers ☆

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HIGHLIGHTS

- Resting EEG in non-deprived smokers may be a biomarker for nicotine use severity.
- Smokers report higher impulsiveness and lower conscientiousness than nonsmokers.
- Sensory hedonia may play a role in nondaily smoking.

ABSTRACT

Objectives: Resting EEG is sensitive to transient, acute effects of nicotine administration and abstinence, but the chronic effects of smoking on EEG are poorly characterized. This study measures the resting EEG profile of chronic smokers in a non-deprived, non-peak state to test whether differences in smoking behavior and personality traits affect pharmaco-EEG response.
Methods: Resting EEG, impulsiveness, and personality measures were collected from daily smokers ($n = 22$), nondaily smokers ($n = 31$), and non-smokers ($n = 30$).
Results: Daily smokers had reduced resting delta and alpha EEG power and higher impulsiveness (Barratt Impulsiveness Scale) compared to nondaily smokers and non-smokers. Both daily and nondaily smokers discounted delayed rewards more steeply, reported lower conscientiousness (NEO-FFI), and reported greater disinhibition and experience seeking (Sensation Seeking Scale) than non-smokers. Nondaily smokers reported greater sensory hedonia than nonsmokers.
Conclusions: Altered resting EEG power in daily smokers demonstrates differences in neural signaling that correlated with greater smoking behavior and dependence. Although nondaily smokers share some characteristics with daily smokers that may predict smoking initiation and maintenance, they differ on measures of impulsiveness and resting EEG power.
Significance: Resting EEG in non-deprived chronic smokers provides a standard for comparison to peak and trough nicotine states and may serve as a biomarker for nicotine dependence, relapse risk, and recovery.
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1. Introduction

The majority of smokers meet DSM-IV criteria for dependence, with daily use of multiple cigarettes being the most common pattern of use and rapid relapse being the most likely outcome of attempts at cessation (Hughes et al., 2004; Donny and Dierker, 2007; Zhu et al., 2012). Nevertheless, a significant proportion of smokers do so intermittently and do not show signs of nicotine dependence, an established diagnostic feature in smoking literature and randomized clinical trials that is absent from the DSM-IV/5 diagnostic lexicon (Coggins et al., 2009; Baker et al., 2012). The remarkable ability of intermittent smokers to use a

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highly addictive substance without transitioning to nicotine dependence may depend on processes that would be of great interest in both prevention and treatment development. Consequently, the neurophysiological and personality factors that differentiate these two groups of smokers have received increasing interest (Shiffman et al., 2009, 2012; Kvaavik et al., 2014; Rass et al., 2014). The electroencephalogram (EEG) is a measure of synchronized neural activity that is particularly promising as a sensitive measure of the acute and chronic effects of nicotine use (Lerman et al., 2009). To date, however, most studies have focused on the effects of acute nicotine intoxication, rather than on effects of chronic use on EEG, and none of these studies have contrasted daily and intermittent (non-daily) smokers. It is important to consider the resting state EEG of non-deprived smokers to more thoroughly understand whether nicotine's effects on functioning represent transient changes due to acute drug effects, reflecting amelioration of withdrawal or reversal of pre-existing deficits, or more lasting changes (due to chronic smoking).

Nicotine use is likely to impact oscillatory activity that is reflected in the scalp EEG. Regular nicotine use can alter the distribution or functionality of brain nicotinic acetylcholine receptors (nAChR), which play a significant role in neuronal communication within and across brain areas (Kadoya et al., 1994; Ghatan et al., 1998; Mansvelder et al., 2006; Bertrand, 2010). nAChR modulation of the velocity of action potential conduction, which alters the functional timing of electrical activity, may affect excitatory and inhibitory neuronal networks involved in thalamocortical transmission, sensorimotor interaction (e.g., gamma synchrony), and memory formation (e.g., theta synchrony) (Forgacs and Bodis-Wollner, 2004; Mansvelder et al., 2006; Kawai et al., 2007; Bertrand, 2010; Rutishauser et al., 2010). Preclinical studies have found that systemic low dose nicotine administration decreases alpha oscillatory power and enhances beta and gamma power in the cortex and VTA of nicotine naïve and experienced freely moving rats (Lenoir and Kiyatkin, 2011; Lenoir et al., 2013); higher doses produced a decrease in theta, delta, alpha, and beta-1 power in nicotine naïve rats (Ferger and Kuschinsky, 1997). Several studies have measured non-spontaneous EEG activity by presenting stimuli at different frequencies to evoke EEG synchrony. A study of auditory evoked EEG response in anesthetized, nicotine-naïve rodents showed enhanced high frequency (40 Hz) response following nicotine administration, attenuation of this enhanced response during administration of nicotine and NMDA antagonist MK801 or nicotine receptor ($\alpha4\beta2$) antagonist DHβE, and a reduced response with administration of MK-801 alone (Sivarao et al., 2013).

Human studies of resting EEG report that smoking or nicotine administration typically produces a decrease of slow wave EEG power (i.e., delta, theta, low alpha) and an increase of high frequency power (i.e., alpha, beta, gamma) in nicotine-deprived smokers (e.g., Mansvelder et al., 2006; Domino et al., 2009; Fisher et al., 2012). In contrast, smoking abstinence and withdrawal have been associated with a shift of the normal EEG distribution to more power at lower frequencies relative to higher frequencies, referred to as EEG deactivation or slowing. Nicotine's direct and indirect effects on dopaminergic neurons may further influence glutamatergic and GABAergic activity, resulting in changes to oscillatory activity in cortical circuitry (Ford et al., 2007; Weinberger and Dostrovsky, 2011). Dopamine receptor binding has been positively correlated with cigarettes per day and nicotine dependence scores (Weerts et al., 2014). Haloperidol antagonism of dopamine D_2 receptors was found to partially block EEG-activating effects of nicotine (i.e., shifting EEG distribution to having more power at higher frequencies relative to lower frequencies) and alter smoking behavior (Caskey et al., 1999; Walker et al., 2001). Differences in the DRD2-A1 allele (i.e., D_2 expression) have been associated with a greater reduction of EEG power during smoking abstinence (Gilbert et al., 2004). Because scalp recorded EEG is primarily generated by post-synaptic potentials in the cortex, this technique should be well suited to detect changes in oscillatory activity at different frequency ranges.

The only two studies that measured resting EEG in non-deprived chronic smokers were underpowered to find effects due to small sample sizes and sparse electrode montages (Knott and Venables, 1977; Pickworth et al., 1997). Most previous studies of nicotine effects have focused on the pharmaco-EEG profile of acute smoking or nicotine administration. In deprived smokers, acute administration produces a shifting from slow wave EEG activity associated with relaxed wakefulness or drowsiness (i.e., delta, theta, low alpha) to high frequency activity associated with arousal or intense mental/emotional activity (i.e., high alpha, beta, gamma), similar to studies of other stimulants (Cook et al., 1995; Knott et al., 1999; Domino, 2001; Pickworth et al., 2003; Mansvelder et al., 2006). Overnight and longer abstinence (3–31 days) from smoking produces consistent reduction in EEG power that is associated with decreased alertness, worsened cognitive performance, and concurrent withdrawal symptoms (e.g., increased craving for cigarettes, negative affect, appetite dysregulation) (Gilbert et al., 2004). Some argue that these changes in neural activity represent a normalization of the withdrawal state, rather than enhancement of electrocortical activation, citing evidence that EEG activation does not surpass that of non-smokers and that non-smokers show no change in slow-wave activity following nicotine administration (Knott, 2001). Other studies found increased dominant alpha frequency or regional power following nicotine administration in non-smokers, possibly reflecting activation of approach/motivational circuitry (Foulds et al., 1994; Fisher et al., 2012).

Measuring resting EEG in cigarette smokers with different use and dependence profiles in comparison with non-smoker controls could reveal a potential biomarker for nicotine dependence, yet the effects of smoking and nicotine dependence on resting EEG have not been thoroughly investigated. Several studies suggest that resting EEG changes persist past early withdrawal states. In two studies, Gilbert and colleagues found that altered EEG power spectra did not resolve after 31 days of abstinence (Gilbert et al., 1999, 2004). Additionally, Gilbert et al. found that higher baseline scores on Fagerström Tolerance Questionnaire (Gilbert et al., 1999) and the Fagerström Test for Cigarette Dependence (Gilbert et al., 2004) were associated with larger quit-related decreases in EEG power. In studies of other drugs, changes in specific frequency bands have been associated with individual factors, such as drug use frequency, dependence, family history of alcoholism, and elevated feelings of euphoria (Parvaz et al., 2011). Finn and Justus (1999) found that alpha EEG power was reduced in non-alcoholic offspring of an alcoholic parent, suggesting decreased alpha power may reflect risk for alcohol use disorder and possibly other substance use disorders (Finn and Justus, 1999). EEG studies of abstinent alcohol-dependent participants suggest that a greater presence of higher frequency beta activity, representing hyper-arousal of the central nervous system, corresponds with quantity and frequency of alcohol intake and can distinguish between 'low' and 'moderate' drinkers and between abstinent and relapse-prone alcoholic individuals (Saletu-Zyhlarz et al., 2004; Parvaz et al., 2011).

Both resting EEG response and smoking behavior may be associated with addiction-related personality traits. Approach-related (e.g., extraversion, novelty seeking, and impulsivity) and avoidance-related (e.g., neuroticism and harm-avoidance) traits have been associated with smoking initiation, progression, and persistence behaviors (Terracciano and Costa, 2004; Munafo et al., 2007; Iacono et al., 2008; de Wit, 2009). A behavioral measure of impulsive decision-making, the delay discounting task,

has demonstrated a greater preference for smaller, more immediate rewards over larger, delayed rewards (i.e., steeper discounting) in smokers vs. non-smokers. Preference for immediate reward has been positively correlated with impulsivity, substance abuse history, and higher smoking rates and dependence (Bickel et al., 1999; Kirby et al., 1999; Mitchell, 1999; Reynolds, 2004; Ohmura et al., 2005; Heyman and Gibb, 2006; Johnson et al., 2007; Sweitzer et al., 2008; Rezvanfard et al., 2010). Resting EEG findings measuring the association between cortical arousal and personality traits (e.g., introversion/extraversion and impulsiveness) have been mixed (Stough et al., 2001; Tran et al., 2001; Knyazev et al., 2002; Schmidtke and Heller, 2004; Houston and Stanford, 2005; Koehler et al., 2011). In one study, greater beta and gamma power were correlated with higher impulsiveness and addiction severity in participants with Internet addition (Choi et al., 2013).

Understanding resting EEG differences in non-deprived smokers is important for more accurately interpreting the acute effects of smoking or nicotine administration, acute and long-term nicotine withdrawal, and success of smoking intervention or cessation. The purpose of this study was to investigate resting EEG response in smokers (daily, nondaily) and non-smokers, and measure personality characteristics and impulsiveness as potential factors influencing EEG response and smoking status. Smokers were hypothesized to have altered EEG power spectra and higher scores on measures of impulsiveness and sensation seeking compared to non-smokers.

2. Methods

2.1. Participants

Thirty non-smokers, 31 nondaily smokers, and 22 daily dependent smokers were recruited from the local community surrounding Indiana University and paid for participation. Participants were recruited as part of a study on nondaily smokers; additional methods and results are reported elsewhere (Rass et al., 2014). Groups were classified according to the following criteria. Non-smokers (1) smoked <10 cigarettes in their lifetime; and (2) had not smoked in the past month. Nondaily smokers (1) smoked for $\geqslant$3 years; (2) smoked <27 days per month for the past 6 months; and (3) in the preceding 90 days, smoked on $\geqslant$10 days or smoked $\geqslant$20 cigarettes. Daily smokers (1) smoked daily for $\geqslant$12 months; and (2) showed at least moderate dependence (scored $\geqslant$4 on the Fagerström Test for Cigarette Dependence, FTCD; Agrawal et al., 2011; Fagerström, 2012). Ex-smokers and participants currently attempting to quit were not eligible for the study. Participants were excluded for a history of electroconvulsive therapy, neurological illness or serious head trauma (including loss of consciousness >5 min), current anxiety disorder or major depression, use of psychotropic medications, and current/past drug abuse/dependence (excluding nicotine) based on DSM-IV criteria. Additional exclusion criteria included marijuana use of more than once per week and consumption of more than 14 alcoholic drinks per week for males and 7 drinks per week for females. All non-smokers were right-handed; two nondaily smokers and three daily smokers were left-handed, and one nondaily smoker and one daily smoker were ambidextrous. Participants received detailed information about the study protocol and gave oral and written informed consent. The Indiana University Institutional Review Board approved the study.

2.2. Procedure

2.2.1. Interviews

To determine eligibility, participants completed phone screens, which included questions about smoking behavior and the FTCD questionnaire. Study eligibility was confirmed in the laboratory using the demographics and screening module of the Structured Clinical Interview for Axis-I disorders (SCID-I; First et al., 1997), with follow-up questions from additional modules when necessary. Study self-report questionnaires were mailed to eligible individuals, and participants completed them prior to or after the lab session. Daily smokers were allowed to smoke prior to the EEG recording; their blood-nicotine levels stabilized during 30–45 min of set-up (Benowitz et al., 1988).

2.2.2. Smoking dependence

Several standard and widely used self-report measures of nicotine dependence were administered prior to the EEG procedure. Nicotine dependence was measured using the *Nicotine Dependence Syndrome Scale* (NDSS; Shiffman et al., 2004). Smoking motives were measured using the *Wisconsin Index of Smoking Dependence Motives* (WISDM; Smith et al., 2010). The *Physical Anhedonia Scale* assessed capacity to experience pleasure from natural reinforcers (i.e., physical sensation), with higher scores indicating greater anhedonia (PAS; Chapman LJ, Chapman JP. Revised physical anhedonia scale. Unpublished test. 1978). Current nicotine withdrawal (*Wisconsin Smoking Withdrawal Scale*, WSWS; Hendricks et al., 2006) and *breath carbon monoxide* (CO; piCO+, Bedfont Scientific Ltd.) were measured before and after the testing session.

2.2.3. Electrophysiological assessment

2.2.3.1. Procedure. The electroencephalogram (EEG) was sampled continuously (1000 Hz sampling rate, 0.1–200 Hz bandpass filter) from 34 Ag/AgCl electrodes that were mounted in a cap (EasyCap, GmbH) and referenced to the nose (Gilbert et al., 2000). Resting EEG was recorded in a sound-attenuated room for three minutes during eyes closed and three minutes during eyes open conditions. Two electrodes that were placed above and below the participant's left eye recorded bipolar vertical electrooculogram (vEOG). Neuroscan SynAmps I digitized the EEG. Electrode impedances were maintained at <10 kOhm.

2.2.3.2. EEG off-line processing. The recordings were segmented into two-second epochs, baseline corrected for the entire epoch, and corrected for ocular artifacts (Gratton et al., 1983). Epochs with voltage exceeding ±100 μV at any site were automatically excluded from further analyses. Power spectra used to measure signal power (in μV^2) were calculated by applying Fast Fourier Transform (FFT) to the EEG. The transformed epochs were averaged and then exported as the average value for each frequency band: Delta (1.5–3.5 Hz), Theta (4–7.5 Hz), Alpha (8–12.5 Hz), Beta (13–25 Hz) and Gamma (30–45 Hz) (Barry et al., 2011). Power values were averaged across 28 electrode sites: left anterior (F7, F3, FT7, FC3, C3), left posterior (P7, P3, PO7, PO3, O1), right anterior (F8, F4, FT8, FC4, C4), right posterior (P8, P4, PO8, PO4, O2), midline anterior (FPz, Fz, FCz, Cz) and midline posterior (CPz, Pz, POz, Oz). Participants with fewer than 20 accepted epochs were excluded from analysis: (a) three non-smokers and three nondaily smokers from the eyes closed condition and (b) two non-smokers and one daily smoker from the eyes open condition. Participants with EEG power >4 *SD* from the sample mean in at least one frequency condition were excluded from analysis: (a) two non-smokers and one nondaily smoker from the eyes open condition.

2.2.4. Personality and impulsiveness measures

The *NEO 5-Factor Personality Inventory* (NEO; Costa and McCrae, 1992; Terracciano et al., 2008) measured personality characteristics. The *Barratt Impulsiveness Scale-11* (BIS; Patton et al., 1995)

and the *Sensation Seeking Scale-V* (SSS; Zuckerman et al., 1978) measured factors related to impulsiveness.

The *Delay Discounting Task* measured temporal impulsivity and future-oriented decision-making. In this task, participants were asked to make hypothetical choices about money. Stimuli and procedures from a previous study (Ahn et al., 2011) were used. In order to familiarize participants with the task, participants completed a practice block of six trials where they were offered a choice between receiving a smaller, immediate reward (e.g., $30 now) or a larger, delayed reward (e.g., $60 in 8 months). Immediately afterwards, the discounting task began with an initial choice between an immediate reward ($400 now) and a delayed reward ($800) at one of six different delays: 2 weeks, 1 month, 6 months, 1 year, 3 years, and 10 years. Each delay block consisted of six trials, and order of the delays was randomized for each participant. Across repeated trials within the same delay, the initial amount of money available immediately ($400) was adjusted based on a participant's choice with the goal of identifying an indifference point using an adjusting amount procedure (Green and Myerson, 2004) continued for six trails within each delay. Once an indifference point was identified at a given delay, the smaller, immediate amount was reset to $400 and the process was repeated using a new delay to the larger amount ($800).

2.3. Data analysis

2.3.1. EEG analysis

One-way analysis of variance (ANOVA) with between-subjects factor of group was run for each EEG frequency. Effect size estimates for analysis of variance were determined with partial η^2 (partial η^2 = .01 is a small effect size, .06 is a medium effect size, and .14 is large effect size) (Kittler et al., 2007). Eyes closed and eyes open conditions were analyzed separately.

2.3.2. Self-report measures

One-way analysis of variance (ANOVA) with between-subjects factor of group was run for every dependent variable of self-report measures of smoking dependence, personality, and impulsiveness. Delay discounting was characterized using a single parameter hyperbolic model: $V = A/(1 + kD)$, in which a reward of amount A received after a given delay D is discounted at an individualized rate k to a subjective value V (Mazur, 1987). We used identical procedures from a previous study (Ahn et al., 2011) to estimate the discounting rate k. To briefly repeat them here, each participant's discounting rate was estimated by programming a customized R code that searches for the k value minimizing the root-mean-square error for all indifference points in six delays. Because the distribution of individual-subject k values was non-normal, a natural logarithm transformation was used to approximately normalize the distribution of k across participants. Participants with inconsistent performance across trials (positive $\ln(k)$) and a poor model fit (root-mean-square-error (RMSE) = 347, which was >4 *SD* from the sample mean) were excluded from analysis (n = 2 non-smokers). Indifference points were compared using a mixed model ANOVA with a within-subjects factor of delay (2 weeks, 1 month, 6 months, 1 year, 3 years, and 10 years) and between-subjects factor of group (non-smokers, nondaily smokers, daily smokers). ANOVAs at each delay tested significant interaction effects. Fisher's Least Significant Difference (*LSD*) tested significant main effects in post hoc analysis.

Exploratory Pearson correlations were used to examine relationships between significant resting EEG frequencies, smoking behavior among groups, and self-report measures.

3. Results

3.1. Demographics, smoking behavior, and subjective craving during the testing session (Table 1)

Groups did not differ by age or sex. Daily smokers completed less education than non-smokers (*LSD* p = .001). Daily smokers had higher carbon monoxide (CO) levels than nondaily smokers (*LSD* p < .001) and non-smokers (*LSD* p < .001) before and after the session. CO and withdrawal post-session compared to pre-session accounted for minimal differences in non-smokers compared to daily smokers (*LSD* p = .011). Daily smokers had a greater increase in craving than both nondaily smokers (*LSD* p = .001) and non-smokers (*LSD* p = .002).

Analysis of smoking self-report measures revealed that daily smokers initiated smoking earlier, smoked for a longer duration, and smoked more frequently relative to nondaily smokers. Daily smokers also reported higher levels of dependence and drive than nondaily smokers. On the NDSS measure of dependence, daily smokers reported significantly more drive ($F(1,51) = 89.70$, p < .001), tolerance ($F(1,51) = 13.06$, p = .001) and a trend for continuity ($F(1,51) = 3.47$, p = .068) than nondaily smokers. On the WISDM assessment of smoking motivation, daily smokers scored higher on primary dependence motives (i.e., automaticity, craving, loss of control, tolerance) than nondaily smokers (p's < .001). Daily smokers also scored higher on WISDM secondary dependence motives, associated with auxiliary features of dependence, with p's < .001 on affective enhancement, affiliative attachment, cognitive enhancement, cue exposure, sensory properties (p = .013), and weight control ($F(1,51) = 3.83$, p = .056) than nondaily smokers. Group differences on the PAS ($F(2,79) = 5.09$, p = .008) showed that nondaily smokers reported less physical anhedonia than non-smokers (*LSD* p = .002). One non-smoker was excluded from PAS analysis due scoring >4 standard deviations (*SD*) from the sample mean.

3.2. Resting state EEG (Fig. 1)

3.2.1. Eyes closed

A main effect of group for the alpha frequency ($F(2,74) = 3.254$, p = .044, partial η^2 = .081) showed a decreased response in daily smokers (M = 2.8, *SD* = 1.8) compared to non-smokers (M = 4.4, *SD* = 2.1), *LSD* p = .014. Differences between non-smokers and nondaily smokers (M = 3.5, *SD* = 2.4) did not reach significance, *LSD* p = .132. No differences were found for other frequencies.

3.2.2. Eyes open

A main effect of group for the delta frequency ($F(2,73) = 3.498$, p = .035, partial η^2 = .087) showed a decreased response in daily smokers (M = 2.7, *SD* = 1.0) compared to nondaily smokers (M = 3.6, *SD* = 1.2), *LSD* p = .014, and non-smokers (M = 3.5, *SD* = 1.6), *LSD* p = .038. A trend main effect of group for the theta frequency ($F(2,73) = 2.921$, p = .060, partial η^2 = .074) was driven by a reduced response in daily smokers compared to the other groups (nondaily smokers *LSD* p = .022; nonsmokers *LSD* p = .075). No differences were found for other frequencies.

3.3. Self-report questionnaires (Table 2, Figs. 2–5)

3.3.1. NEO-Five Factor Inventory

Post hoc analyses revealed that both daily smokers and nondaily smokers scored lower on conscientiousness than non-smokers (p = .028; p = .009).

Table 1
Demographics.

	Non-smoker (n = 30)	Nondaily smoker (n = 31)	Daily smoker (n = 22)	Analysis	p
Sex Male n(%)	14 (47)	12 (39)	13 (59)	$X^2_{(2)}$ = 2.15	.342
Age	25.2 (4.3)	23.9 (4.4)	27.2 (5.3)	$F_{(2,80)}$ = 2.54	.085
Education (years)	16.6 (2.0)[a]	15.8 (1.6)	14.8 (1.6)[b]	$F_{(2,80)}$ = 6.13	.003
Ethnicity n(%)					
Caucasian	20 (67)	24 (77)	18 (82)		
Asian	8 (27)	5 (16)	3 (14)		
Black	2 (7)	1 (3)	1 (5)		
Biracial	0	1 (3)	0		
Age of smoking initiation		18.1 (1.7)[a]	16.3 (3.0) [b]	$F_{(1,51)}$ = 7.88	.007
Smoking duration (years)		5.6 (4.4)[a]	9.4 (6.4)[b]	$F_{(1,51)}$ = 6.69	.013
Cigarettes per day		3.6 (1.6)[a]	16.1 (6.1)[b]	$F_{(1,51)}$ = 120.81	<.001
Smoking days per week		3.2 (1.4)[a]	7.0 (0)[b]	$F_{(1,49)}$ = 160.93	<.001
Cigarettes per week		12.3 (9.0)[a]	113.0 (42.5)[b]	$F_{(1,49)}$ = 154.71	<.001
FTCD total score		0.5 (0.9)[a]	5.4 (1.3)[b]	$F_{(1,51)}$ = 272.52	<.001
NDSS total score		−2.6 (0.7)[a]	−0.1 (0.8)[b]	$F_{(1,51)}$ = 89.70	<.001
WISDM total score		26.9 (8.7)[a]	48.4 (8.0)[b]	$F_{(1,51)}$ = 84.00	<.001
PAS total score	12.4 (6.4)[a]	7.7 (5.0)[b]	10.4 (6.1)	$F_{(2,79)}$ = 5.09	<.008
CO ppm					
Pre	2.3 (1.4)[a]	3.7 (2.9)[a]	16.8 (10.8)[b]	$F_{(2,80)}$ = 45.66	<.001
Post	2.0 (0.8)[a]	2.8 (1.9)[a]	14.9 (8.8)[b]	$F_{(2,80)}$ = 57.49	<.001
Change score	−0.3 (0.8)[a]	−0.9 (1.3)	−1.9 (3.9)[b]	$F_{(2,80)}$ = 3.36	.040
WSWS					
Craving pre	1.0 (2.4)[a]	7.0 (6.5)[b]	11.5 (7.5) [c]	$F_{(2,80)}$ = 22.20	<.001
Craving post	0.8 (2.0)[a]	6.3 (6.1)[b]	15.0 (7.3) [c]	$F_{(2,80)}$ = 43.56	<.001
Craving change score	−0.2 (1.7)[a]	−0.7 (3.9)[a]	−3.5 (6.3)[b]	$F_{(2,80)}$ = 7.41	.001
Total pre	44.7 (17.4)	54.7 (23.4)	56.4 (25.4)	$F_{(2,80)}$ = 2.78	.109
Total post	49.6 (17.1)[a]	62.1 (23.8)[b]	65.7 (23.7)[b]	$F_{(2,80)}$ = 4.18	.019
Total change score	4.9 (11.8)	7.4 (17.8)	9.3 (18.6)	$F_{(2,80)}$ = 0.492	.613

Note. Values are $M(SD)$ unless otherwise noted. Superscript letters represent post hoc analysis, with differing letters indicating significant group differences (p's < .05). A cutoff of eight to ten CO ppm has been recommended to differentiate smokers from non-smokers (SRNT, 2002). Weekly smoking rate data are missing from two nondaily smokers.
Abbreviations: FTCD = Fagerström Test for Nicotine Dependence; NDSS = Nicotine Dependence Syndrome Scale; WISDM = Wisconsin Index of Smoking Dependence Motives; PAS = Physical Anhedonia Scale; WSWS = Wisconsin Smoking Withdrawal Scale.

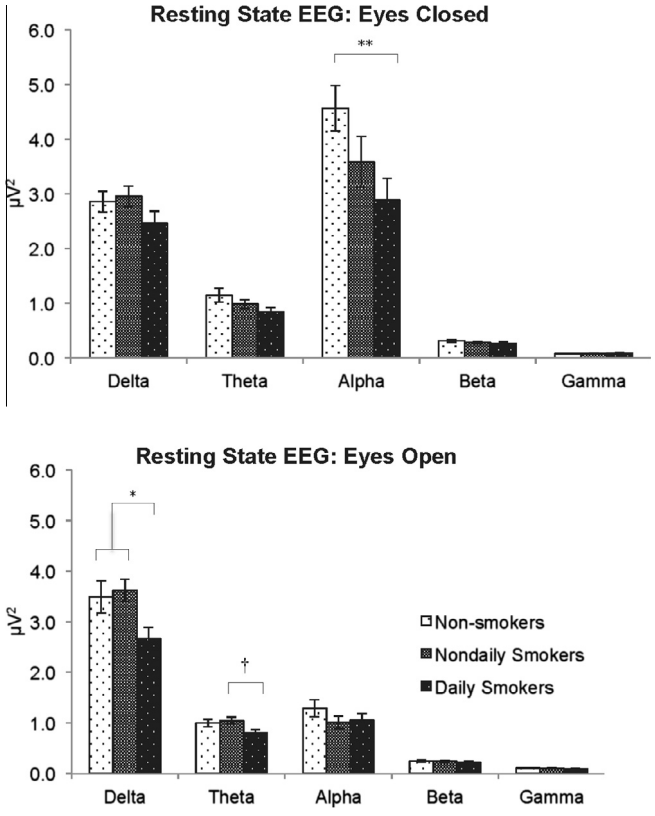

Fig. 1. Resting state EEG power spectra for the eyes closed and eyes open conditions across groups. Delta: 1.5–3.5 Hz; Theta: 4–7.5 Hz; Alpha: 8–12.5 Hz; Beta (13–25 Hz; Gamma: 30–45 Hz. Error bars represent ±1 SEM. The following symbols represent significance: $^{\dagger}p < .06$, $^{*}p < .05$, $^{**}p < .01$.

3.3.2. Barratt Impulsiveness Scale

Post hoc analyses revealed that daily smokers scored higher on the motor impulsiveness subscale than non-smokers (LSD p = .002) and nondaily smokers (LSD p = .014). Daily smokers scored higher overall than non-smokers (LSD p = .008).

3.3.3. Sensation Seeking Scale

Post hoc analyses revealed that higher SSS Total Score in daily smokers (p = .006) and nondaily smokers (p = .055) compared to non-smokers was driven by endorsing more disinhibition (p = .021; p = .004) and experience-seeking (p = .021; p = .031) items.

3.3.4. Delay discounting

Repeated measures ANOVA showed a group × delay interaction ($F(10,390) = 2.680$, p = .042) and a main effect of delay ($F(5,390) = 473.524$, $p < .001$). Post hoc analysis showed a trend for higher indifference points in daily smokers than non-smokers (*LSD* p = .060). Follow-up ANOVAs found differences at ten years delay ($F(2,78) = 4.032$, p = .022) and marginal differences at three years delay ($F(2,78) = 3.030$, p = .054. At 10 years delay, daily smokers (*LSD* p = .012) and nondaily smokers (*LSD* p = .024) had higher discounting rates than non-smokers. At 3 years delay, daily smokers (*LSD* p = .032) and nondaily smokers (*LSD* p = .024) had higher discounting rates than non-smokers. There was a trend (p = .10) for steeper discounting rates for the smoking groups compared to non-smokers (Fig. 5). Based on existing literature, we also compared non-smokers with all smokers (combining daily and nondaily smokers) using an independent t-test analysis (Bickel et al., 1999; Mitchell, 1999; Baker et al., 2003; Reynolds et al., 2004). The analysis showed that smokers discounted delayed rewards more steeply than non-smokers ($t(79) = 2.030$, p = .046), which is consistent with the previous reports.

Table 2
Results from self-report measures and delay discounting.

	Non-smoker (n = 30)	Nondaily smoker (n = 31)	Daily smoker (n = 22)	$F_{(2,80)}$	p
NEO-Five Factor Inventory					
Neuroticism	21.3 (8.4)	24.5 (8.1)	21.4 (9.6)	1.35	.265
Extraversion	29.5 (8.0)	31.1 (7.0)	28.9 (6.3)	0.68	.512
Openness	29.9 (8.0)	32.0 (6.7)	32.7 (7.9)	1.30	.279
Agreeableness	30.1 (6.8)	30.2 (5.8)	28.9 (6.9)	0.30	.741
Conscientiousness	34.4 (6.2)[a]	29.8 (6.2)[b]	30.2 (7.7)[b]	4.24	.018
Barratt Impulsiveness Scale					
Attentional impulsiveness	15.9 (3.1)	16.7 (3.4)	16.9 (4.3)	0.61	.547
Motor impulsiveness	20.9 (3.6)[a]	21.6 (3.2)[a]	24.1 (4.1)[b]	5.48	.006
Non-planning impulsiveness	21.4 (3.6)	23.2 (5.2)	24.0 (4.7)	2.34	.102
Total score	116.3 (14.2)[a]	123.0 (17.7)	130.0 (22.1)[b]	3.78	.027
Sensation Seeking Scale					
Boredom	2.9 (2.1)	2.8 (1.9)	3.8 (2.2)	1.80	.173
Disinhibition	4.1 (2.1)[a]	5.7 (1.9)[b]	5.5 (2.6)[b]	5.01	.009
Experience Seeking	4.9 (2.1)[a]	6.1 (1.7)[b]	6.3 (2.4)[b]	3.56	.033
Adventure Seeking	5.8 (2.9)	6.2 (2.7)	7.0 (2.9)	1.09	.341
Total Score	17.8 (5.6)[a]	20.8 (5.6)[b]	22.6 (7.3)[b]	4.32	.017
Delay discounting (ln(k))	−4.7 (1.9)	−4.1 (1.5)	−3.8 (1.5)	2.33	.104

Note. Values represent *Mean*(*Standard Deviation*). Superscript letters represent post hoc analysis, with differing letters indicating significant group differences (p's < .05). Degrees of freedom for delay discounting were 2,78.

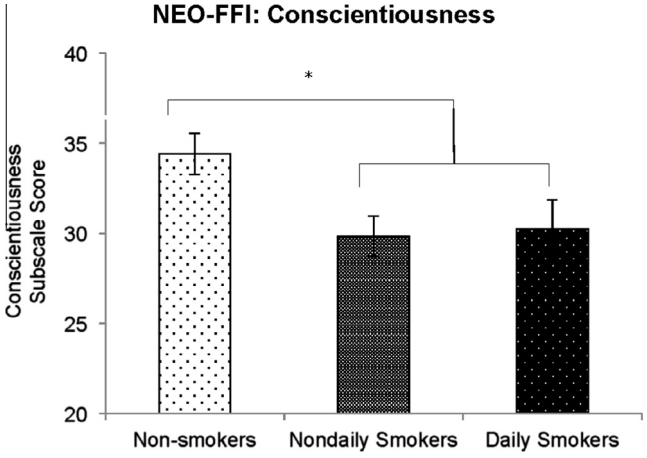

Fig. 2. Self-report measure of conscientiousness (scale range: 0–48) from the NEO Five Factor Inventory across groups. Error bars represent ±1 SEM. The following symbols represent significance: $^{*}p < .05$, $^{**}p < .01$.

3.4. Exploratory correlations

Pearson correlations explored the relationship between the resting EEG measures that differed between groups. Pearson correlation coefficients were computed for resting EEG eyes open (delta, theta) and eyes closed (alpha) condition and their relationship with smoking variables (smoking duration, cigarettes per week (smoking days per week × CPD), CO levels pre-session, FTCD total, NDSS total, WISDM total, and PAS total) for daily and nondaily smokers as well as personality and impulsiveness measures (BIS total, SSS total, NEO conscientiousness, and delay discounting

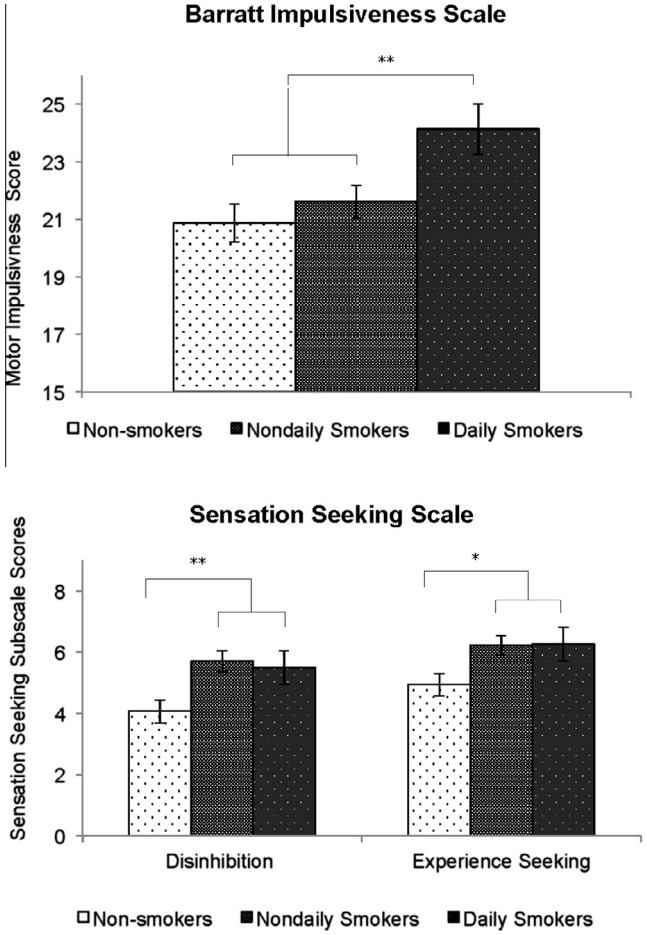

Fig. 3. Self-report measures of impulsivity from the Barratt Impulsiveness Scale (subscale range: 11–44) and the Sensation Seeking Scale (subscale range: 0–10) across groups. Error bars represent ±1 SEM. The following symbols represent significance: $^{*}p < .05$, $^{**}p < .01$.

ln(k)) for all groups. Delta and theta activity showed significant, negative correlations with cigarettes per week ($r = -.40$, $p = .005$; $r = -.31$, $p = .030$), CO levels pre-session ($r = -.38$, $p = .006$; $r = -.34$, $p = .017$), and FTCD total ($r = -.34$, $p = .016$; $r = -.29$, $p = .042$). Delta activity also showed significant, negative correlations with NDSS total ($r = -.34$, $p = .015$) and WISDM total ($r = -.29$, $p = .042$). A Bonferroni correction for the smoking variables set significance levels at $p < .002$, negating the significant exploratory correlations. No significant correlations were found between resting EEG measures and impulsiveness variables.

4. Discussion

The present study measured resting EEG response in non-deprived smokers and non-smokers. Compared to previous studies investigating EEG of smokers without acute drug administration or withdrawal effects (Knott and Venables, 1977; Pickworth et al., 1997), this study had a larger sample size, included nondaily smokers, included both eyes-closed and eyes-open recording conditions, and used a denser electrode montage. Attenuated EEG power in daily smokers compared to nonsmokers suggests alterations of neural synchrony that may reflect risk factors for nicotine use and dependence or effects of chronic nicotine use.

Daily smokers showed less alpha power during the eyes closed condition and less delta power during the eyes open condition than non-smokers. The findings of reduced alpha power in daily

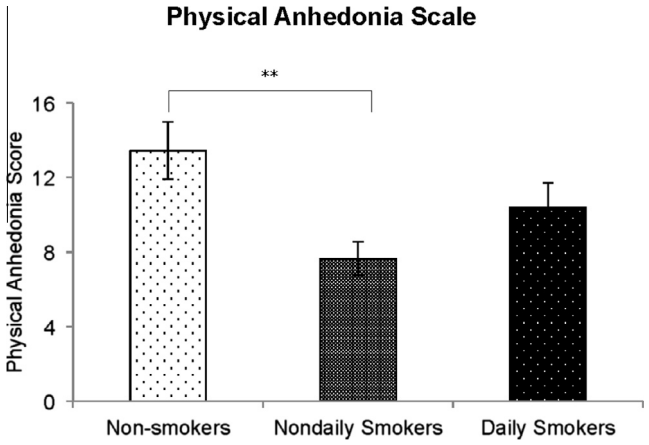

Fig. 4. Self-report measures of the physical anhedonia across groups (scale range: 0–61). Error bars represent ±1 SEM. The following symbols represent significance: $^{*}p < .05$, $^{**}p < .01$.

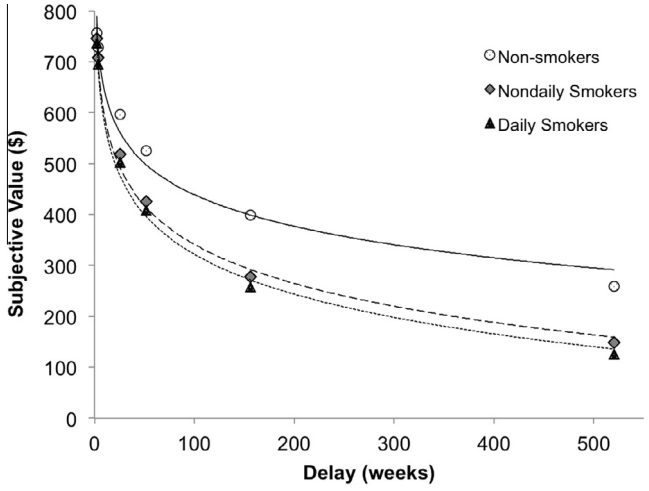

Fig. 5. Mean subjective values for non-smokers, nondaily smokers, and daily smokers of an $800 reward plotted as a function of time from the choice until the receipt of the reward. Trend lines show the best-fitting logarithmic function through the mean subjective values for the groups.

smokers are consistent with past findings of reduced EEG power during nicotine withdrawal, and may explain electrophysiological differences when compared to non-smokers. Without comparison to states of acute nicotine administration or abstinence, it is difficult to judge whether decreases in power reflect an early withdrawal state, premorbid differences in neural response, or neural changes due to chronic nicotine use. In order to avoid comparing an acute withdrawal (daily smokers) to groups who do not experience withdrawal (nondaily smokers) or do not smoke, non-deprived daily dependent smokers were compared to nondaily smokers and non-smokers. Acute nicotine effects were not present in nondaily smokers, who showed a non-significant trend of lower alpha (eyes closed) response than non-smokers that may be independent of nicotine state. Future studies testing daily and nondaily smokers at peak and trough nicotine states using a within-subjects design are needed to resolve nicotine effects on resting EEG in dependent and non-dependent individuals. Comparing effects of nicotine administration on smokers and non-smokers during EEG recording would demonstrate differential magnitude of change in alpha power between groups.

Daily smokers showed less delta and theta power during the eyes open condition and a trend for lower alpha power than nondaily smokers. Exploratory correlation analyses suggested that the electrophysiological responses were sensitive to smoking variables, with higher use/dependence correlating with lower EEG power. However, this association did not hold when a correction factor was applied. The differences between daily and nondaily smokers suggest a potential dose–response effect that merits further investigation. Including light and heavy daily smokers in the sample and recording smoking pack-years would control for effects of smoking duration across varying smoking rates and may elucidate the role of smoking rate and duration on resting EEG response. Adding a measure of evoked EEG response may provide an additional evaluation of neural synchrony capacity in daily and nondaily smokers. Crawford et al. (2002) measured auditory evoked synchrony and found enhanced gamma (40 Hz) response during both abstinent and peak nicotine states in chronic, heavy smokers compared to never-smokers. Crawford et al. (2002) attributed group differences to acute nicotine effects and chronic inhibition of monoamine oxidase on dopamine neurotransmission and gamma synchrony. Impaired gamma synchrony has been found in clinical populations with abnormalities in GABA and glutamate neurotransmission (e.g., Uhlhaas and Singer, 2006). Resting and evoked EEG measures may be sensitive to different smoking rates and states.

Self-report measures of sensation seeking and personality distinguished non-smokers from both daily and nondaily smokers. Non-smokers scored lower on disinhibition and experience seeking than both smoking groups, consistent with studies showing that sensation seeking is associated with increased drug abuse vulnerability and can differentiate smokers and non-smokers (Kelly et al., 2006; Perkins et al., 2008; Spillane et al., 2010). Lower conscientiousness in daily and nondaily smokers is consistent with research identifying low conscientiousness as a risk factor for smoking initiation and maintenance and high conscientiousness as a protective factor against smoking (Kashdan et al., 2005; Von Ah et al., 2005; Conner et al., 2009). Conscientiousness has been associated with better behavioral control, healthy coping strategies, and greater feelings of personal control (Terracciano and Costa, 2004; Kashdan et al., 2005; Terracciano et al., 2008; Conner et al., 2009). These results support that increased sensation seeking and low conscientiousness may be a risk for smoking behavior. In contrast, measures of impulsiveness differentiated non-smokers and nondaily smokers from daily smokers. Specifically, daily smokers reported greater motor impulsivity than the other groups. Motor impulsivity has been associated with more habit-driven rather than goal-directed behavior, and associated with risk-taking and impaired working memory and executive function (Hogarth et al., 2012). Motor impulsiveness may reflect greater dependence and potentiate continued smoking behavior. Additionally, delay discounting showed that smokers made more impulsive choices than non-smokers, but daily and nondaily smokers discounted delayed rewards at a similar rate.

As expected, nondaily smokers scored lower on all measures of smoking history and behavior, including smoking duration, measures of dependence (FTCD, NDSS, WISDM), and respiratory CO levels, and they started smoking at an older age. A greater increase in craving by daily smokers than nondaily smokers may represent a response to cues and expectations rather than abstinence time (Dar et al., 2005, 2010; Tiffany et al., 2009). One interesting result was from a self-report measure included to reflect experiencing decreased reward from natural reinforcers as a consequence of drug use and dependence. The Physical Anhedonia Scale (PAS) is a self-report measure assessing the ability to experience pleasure from typically rewarding physical stimuli, such as food, sex, and environment (Chapman LJ, Chapman JP. Revised physical anhedonia scale. Unpublished test. 1978). The PAS differs from past measures of anhedonia used in smoking research because it separates

sensory pleasure from social experiences and personal interests captured by broader scales of hedonic capacity (e.g., Snaith–Hamilton Pleasure Scale). Based on previous studies associating increased anhedonia with smoking onset, escalation, persistence of dependence, and poor cessation outcomes, we expected dependent, daily smokers to show greatest anhedonia (higher scores) (Leventhal et al., 2009; Audrain-McGovern et al., 2012). Smokers and non-smokers did not differ, which may indicate that the measure is not sensitive to smoking behavior. However, findings of lower anhedonia in nondaily smokers compared to non-smokers suggest that nondaily smokers may have a heightened capacity for sensory experiences. Low PAS scores suggest that nondaily smokers may retain reinforcement from natural reward and experience positive physical stimulation beyond the norm. Intermittent smoking may promote continued smoking due reduced development of tolerance to pleasurable sensory effects. Alternatively, nondaily smokers may be more prone to sensory satiation or saturation following one or fewer cigarettes or reduced adaption to aversive consequences of inhaling heat or smoke (e.g., throat irritation, nausea). Future studies could include questions targeting physical sensations of smoking, such as taste or throat hit. Smoking cessation treatment studies may consider a potential role for sensory substitution during initial intervention.

Study results must be considered within the context of an observational study. The study design does not allow for differentiation between possible premorbid conditions, such as smoking-related changes in neuronal structure and function and low neuronal excitability, that may drive smoking behavior and dependence. The sample consisted of young, relatively light smokers, leaving out older longer-term heavy smokers and non-dependent daily smokers. Younger smokers might be more resilient and have fewer nicotine-related changes due to having less experience with nicotine, resulting in smaller effect sizes and lack of significant group differences for other frequency bands. Measuring resting EEG in adolescents or adults during smoking initiation may reveal risk factors for future transition to daily, dependent smoking. Additionally, strict criteria excluding participants with comorbid psychiatric disorders, which commonly occur in smokers, controlled non-nicotine contributions to EEG response. This design reduced the potential for contamination to the EEG effect by the usual comorbidities found in smokers, while also reducing generalization to the typical smoker population. A larger sample size and greater variability in smoking history, comorbid diagnoses, gender, and ethnicity would increase statistical power to the address these factors.

In conclusion, resting EEG may be a useful marker of risk for nicotine dependence and use severity as well as for evaluating relapse risk and treatment efficacy (e.g., nicotine replacement therapies and smoking cessation pharmacotherapies) in chronic smokers. Vulnerability for initiation and maintenance for smoking behavior may be predicted by measures of impulsiveness and conscientiousness, but these personality measures do not seem to have a major influence on resting EEG. More research is needed to evaluate the role of physical (sensory) anhedonia in smoking behavior and its potential utility in smoking cessation treatment.

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