

Tobacco Smoking and the Resting Maternal Brain: A Preliminary Study of Frontal EEG

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Tobacco smoking has been attributed to a wide range of detrimental health consequences for both women and their children. In addition to its known physical health effects, smoking may also impact maternal neural responses and subsequent caregiving behavior. To begin investigating this issue, we employed electroencephalography (EEG) to examine resting neural oscillations of tobacco-smoking mothers ($n = 35$) and non-smoking mothers ($n = 35$). We examined seven EEG frequency bands recorded from frontal electrode sites (delta, theta, alpha, alpha₁, alpha₂, beta, and gamma). While no between-group differences were present in high-frequency bands (alpha₂, beta, gamma), smokers showed greater spectral power in low-frequency bands (delta, theta, alpha, alpha₁) compared to non-smokers. This increased power in low-frequency bands of tobacco-smoking mothers is consistent with a less aroused state and may be one mechanism through which smoking might affect the maternal brain and caregiving behavior.

INTRODUCTION

Maternal smoking is a significant public health concern, affecting both mothers and their developing children. While tobacco smoking is the leading cause of preventable disease in the United States, secondhand smoke exposure may result in acute and chronic illnesses in children [1]. Furthermore, studies have documented the physiological effects of tobacco smoking during pregnancy on fetal development and infant outcomes [2-4]. A focus of recent interest has centered on how tobacco smoking may impact other qualities of the dyadic rela-

tionship. Neurobiological models of addiction and parenting have suggested that the increased rates of child neglect by addicted mothers may be associated with both the diminishment of natural rewarding properties of caregiving and the diminished abilities to properly regulate stress and negative affect [5,6]. Supportive of this notion in tobacco addiction specifically, a positive correlation between maternal tobacco smoking and child neglect was recently reported [7]. Furthermore, relative to non-substance-using mothers, maternal substance use has been associated with a decreased neural response to infant

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†Abbreviations: EEG, Electroencephalography; EO, Eyes Open; EC, Eyes Closed; FTND, Fagerstrom Test for Nicotine Dependence; AUDIT, Alcohol Use Disorders Identification Test; FFT, Fast Fourier Transform.

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socio-emotional signals, where a significant proportion of the mothers in the substance-using sample smoked tobacco [8]. However, the extent to which there exist neurophysiological differences between tobacco-smoking and non-smoking mothers that may underscore these prior findings is unknown. Therefore, in this study we examined resting neural oscillations in tobacco-smoking and non-smoking groups of mothers.

There has been increasing interest toward the examination of neural oscillations that constitute the EEG signal. These oscillations provide insight into the coordination of multiple systems in the brain [9,10] as well as between-group and within-subjects differences in engagement of these systems, which may relate to aspects of motivation [11], anxiety [12], and cognition [13,14]. In particular, low-frequency oscillations may reflect emotion and motivational processes underscored by subcortical brain structures, with high-frequency oscillations likely reflecting more cognitive control processes underscored by cortical structures [15]. Consequently, examination of these neural oscillations could prove valuable in understanding how tobacco smoking may influence maternal brain function and consequently behavior.

Past research has begun to investigate the impact of tobacco smoking on neural oscillations [16,17]. This approach is valuable given that shifts in the spectral power of different neural oscillations may be indicative of cortical arousal or activation, which may capture the stimulating and sedating actions of tobacco smoking behavior at an electrocortical level [18-20]. For instance, the immediate consequence of smoking or nicotine administration on neural oscillations has been associated with an 'activated' or 'aroused' EEG profile — marked by increased activity in higher frequency bands (e.g., beta) and decreased activity in lower frequency bands (e.g., delta and theta) [16,21-23]. This 'aroused' smoking-associated EEG profile may reflect higher cortical activation associated with the stimulating properties of tobacco [18], acting as positive reinforcement for future use [18,22]. Reciprocally, the reinforcement and dependence-producing properties of tobacco smoking may also depend on issues such as withdrawal, where smoking deprivation has resulted in the opposite EEG profile (i.e., EEG deactivation) [19].

Although an aroused EEG profile has typically been observed following tobacco and nicotine consumption across frequency bands, research has suggested the need to examine different bandwidths of alpha, namely α_1 (lower alpha frequency) and α_2 (higher alpha frequency), that may be differentially sensitive to tobacco and nicotine effects [24]. In non-smokers, acute administration of nicotine has been found to increase α_2 activity [25]. Following tobacco smoking, only increases in α_2 have been observed in current smokers [16], with another study also evidencing an increase in α_2 as well as a decrease in α_1 activity [24]. Taken together, these data suggest that the alpha bandwidth may be too broad with respect to differential sensitivity of tobacco and nico-

tine effects, and therefore it is recommended to examine separately α_1 and α_2 , in addition to alpha, when investigating the impact of tobacco smoking on alpha activity [24].

The current study presents a spectral analysis of neural oscillations recorded at rest from maternal tobacco smokers and non-smokers. This approach advances prior work as, to our knowledge, no study has examined the influences of tobacco smoking on resting brain activity in recent mothers. This neurophysiological approach may begin to provide insight into potential mechanisms that may underscore differences in caregiving responsiveness [7,8]. Given the prior literature presented here in non-parent smokers and non-smokers [16,21-23], we hypothesized that tobacco-smoking mothers would evidence increased activity in high-frequency bands (α_2 , beta, gamma) and decreased activity in low-frequency bands (e.g. alpha, α_1 , theta, delta).

METHODS

Participants

Women were recruited at approximately three months postpartum from the local community as a part of a larger study of parenting and addiction, and were compensated \$50. Mothers were screened for self-reported use of tobacco (Fagerstrom Test for Nicotine Dependence; FTND [26]), alcohol (Alcohol Use Disorders Identification Test; AUDIT; [27]), and illicit substances (Addiction Severity Index Lite; [28]), as well as measures of trait anxiety (State-Trait Anxiety Inventory [29]) and depression (Beck Depression Inventory Second Edition [30]). In addition to these self-reported measures, and as part of the larger study of parenting and addiction, urine toxicology was assessed at another study visit following this EEG visit to confirm self-reported substance-use. From the larger study sample, 35 participants were identified as tobacco smokers and 35 were non-smokers, with neither group using any other illicit substances. These maternal tobacco smokers and non-smokers were matched across a series of demographic variables, including maternal age, education (in years), and ethnicity (Table 1). All women completed a neuropsychological screening to assess for any recent neurological issue that may impact the EEG. In the maternal tobacco smokers, four women reported a neurological issue, and in the non-smoking group, five reported a similar issue (e.g., five had previously lost consciousness; two reported past headaches due to vehicle accidents, two reported head injuries in their childhood). No discernible EEG abnormalities were observed associated with these injuries.

Apparatus

Net Station 4.2.1 recorded the continuous EEG (sampling rate: 250 Hz; high impedance amplifiers of 0.1 Hz high pass, 100 Hz low pass). A 128 Ag/AgCl electrode

Table 1. Demographic characteristics for maternal smokers and non-smokers. Maternal age and education are presented as mean (standard deviation).

	Smoking Mothers		Non-Smoking Mothers		<i>t</i>	<i>p</i>
Maternal Age (years)	25.80 (4.96)		26.91 (5.80)		1.47	.15
Education (in years)	12.10 (1.14)		12.66 (2.86)		1.01	.32
Did not report	4		6			
	<i>N</i>	%	<i>N</i>	%	χ^2	<i>p</i>
Maternal Race					2.05	.73
African American (AA)	20	57.14	20	57.14		
Caucasian	8	22.86	7	20		
Hispanic	6	17.14	6	17.14		
Asian American	0	0	1	2.86		
Caucasian/AA	0	0	11	2.86		
Did not report	1	2.86	0	0		

sensor net (Electrical Geodesics, Inc; Tucker, 1993) was placed on the participant's head, with the electrodes spaced evenly from nasion toinion and from left to right ear. Before fitting, the net was soaked in a warm potassium chloride solution for 10 minutes that acted as the electrolyte. Cz was used as the reference electrode during EEG recording. Impedances were checked prior to the recording and kept below 40 k Ω .

Design

Participants sat at rest approximately 70 cm away from a computer screen in a dimly lit room. The experiment consisted of two conditions — eyes open (EO) and eyes closed (EC), each lasting 1 minute. Each condition was presented three times in an alternating design (EO, EC, EO, EC, EO, EC) resulting in 3 minutes of EC data and 3 minutes of EO data. Our rationale for alternating these conditions was to ensure participants remained in an awake and alert resting state throughout the recording.

Data Analysis

Net Station 4.5 was used to pre-process the EEG data. EEG data (3 minutes of EO and 3 minutes of EC) were first segmented into two-second epochs, creating 90 EO trials and 90 EC trials. Manual visual inspection was then conducted to inspect signal quality of the EEG recorded at eye channels. Artifact detection was set at 200 μ V for bad channels and ocular artifact removal [32] was then applied using a blink slope threshold of 14 μ V/ms. Horizontal and vertical eye movements were also assessed ($> 150 \mu$ V). Channels that were identified with more than 40 percent of artifacts in trials were replaced through spline interpolation. EEG data were then re-referenced to the average reference and baseline correction was applied. After pre-processing, there were, on average, 78 EC segments (range 36-90) and 73 EO segments (34-90), $t(48) = -2.03$, $p = .048$. A Fast Fourier Transform (FFT) was run using MatLab ([33]) to examine seven EEG frequencies defined by prior research [25,34]; specifically, delta (1-4 Hz), theta (4-8 Hz), alpha (8-13 Hz), α_1 (8-10.5 Hz), α_2

(10.5-13 Hz), beta (13-30 Hz), and gamma (30-80 Hz). FFT analyses were conducted at electrode sites 25 and 124, located at left and right frontal scalp sites, respectively. These sites overlap with F3 (EGI electrode 25) and F4 (EGI electrode 124), used in the 10/20 electrode EEG studies to assess resting EEG and hemisphere effects [35-37], and are thought to reflect activity in dorsolateral prefrontal cortex [38-40]. A log-transform function was performed to normalize the spectral data (raw data are presented in Table 1 in the Appendix), and data from two participants within the maternal smoking group were removed as outliers following box-plot analysis across the multiple frequency bands. A repeated-measures ANOVA specifying the within-subjects factors of condition (EO, EC) and hemisphere (right, left) and the between-group factor of smoking status (smoking, non-smoking) was conducted separately on each frequency band. Effect sizes are presented as partial eta-squared (η^2_{partial}). A .01 η^2_{partial} represents a small, .06 η^2_{partial} represents a medium, and 0.14 η^2_{partial} represents a large effect size [41].

RESULTS

We first report results associated with anxiety, depression, alcohol use, and nicotine dependence scores in smokers and non-smokers (Table 2). Maternal smokers and non-smokers did not differ by anxiety or depression, and no correlations were found between depression or anxiety scores and neural oscillations within or between the maternal groups (p 's $> .05$). Evidence of alcohol use was present in smokers and non-smokers, and the difference in alcohol use was not statistically different. No correlations were found between AUDIT scores and neural oscillations within or between groups (p 's $> .05$), and the AUDIT scores representing alcohol use did not contribute as a covariate in the analyses (p 's $> .05$). From our sample of tobacco-smoking mothers, 28 completed all items of the FTND, and their FTND scores ranged from 1 to 8 (i.e., from low dependence to high levels of dependence). No

Table 2. Depression, anxiety, hazardous alcohol use and nicotine dependence in smoking and non-smoking mothers. Data are presented as means (standard deviations).

	Smoking Mothers	Non-Smoking Mothers	<i>t</i>	<i>p</i>
Depression Level	13.03 (9.92)	12.06 (7.60)	.45	.65
Missing Scores	1	0	1.38	.17
Anxiety Level	48.45 (3.40)	47.35 (3.03)	1.87	.07
Missing Scores	1	1		
Hazardous Alcohol Use	2.64 (4.78)	1.87 (1.45)		
Nicotine Dependence	4.32 (1.79)			
Missing Scores	5			

Note: Depression and anxiety scores did not reach levels suggested to detect clinically significant symptoms [53, 54].

correlations were found between FTND scores and neural oscillations.

Across all EEG frequencies, smoking status did not interact with condition, hemisphere, or their combination, F 's < 2.6, p 's > .11. Therefore, we first report the between-group effect of smoking status, which was found to be statistically significant for delta, theta, alpha, and alpha₁ frequency bands (Table 3). Specifically, spectral power in delta, theta, alpha, and alpha₁ was higher in smoking mothers as compared to non-smoking mothers. There was no between-group effect of smoking status modulating alpha₂, beta, and gamma frequency bands. Taken together, these results suggest that maternal smoking modulates low-frequency neural oscillations at rest.

As presented in Table 4, there was a main effect of hemisphere across all frequency bands, with larger spectral power in the right hemisphere as compared to the left hemisphere, irrespective of smoking status. With the exception of delta and beta, there was also a main effect of condition across the frequency bands. Greater alpha, alpha₁, alpha₂, and theta activity was found in the EC condition relative to the EO condition. In contrast, the EO condition elicited a greater gamma activity as compared to the EC condition. There was also an interaction between hemisphere and condition for alpha, alpha₁, and alpha₂ frequency bands — likely driven by larger spectral power in the right hemisphere in the EC condition (as compared to the left hemisphere and the EO conditions; Table 1, Appendix). Taken together, and consistent with prior literature, these analyses suggest that neural oscillations at rest may be differentially modulated by hemisphere, condition, and their interaction.

DISCUSSION

The detrimental health effects of tobacco smoking can be seen in a myriad of respects, both within smokers and individuals who are exposed to secondhand smoke. In addition to the known physical effects, smoking may also impact maternal responding to infants [7,8]. Existing neurophysiological differences associated with tobacco smoking may underlie these alterations in maternal response. Therefore, the current study analyzed neural os-

cillations measured using EEG to investigate whether tobacco smoking impacted resting brain activity in mothers. While tobacco smoking did not interact with condition (EO, EC), hemisphere (right, left), or their combination, there were main effects of hemisphere and condition modulating neural oscillations that were consistent with the lateralization of resting EEG to emotion processing [36,42] and variation in arousal and cortical processing of visual input [43]. Nevertheless, our central finding was that tobacco smoking modulated EEG spectral power in the low-frequency (delta, theta, alpha₁, and alpha) but not high-frequency (alpha₂, beta, and gamma) bands, and may be one mechanism through which smoking affects the maternal brain and caregiving behavior.

This increase in low-frequency power in maternal smokers as compared to non-smokers is contrary to past research, which instead suggested tobacco smoking might be associated with an activated or aroused EEG profile (i.e., a predominant decrease in lower frequencies, an increase in beta, as well as modulation of alpha activity) [16,21,23]. Critically, in these prior studies, participants were either acutely administered nicotine following brief or no abstinence, or participants smoked tobacco just prior to the EEG. In the current study, mothers were given no instructions regarding smoking behaviors to encourage regularity in their smoking patterns. Given the structure of the laboratory visit (approximately 20 to 30 minutes elapsed between participants arriving at the lab and the EEG recording), there was no immediate or acute nicotine or tobacco consumption. Consequently, this increased low-frequency band activity observed in maternal smokers may reflect more systemic long-term consequences of nicotine use and tobacco smoking in these women rather than any acute effects [19].

Increased low-frequency spectral power is consistent with a less activated or aroused EEG profile, which may be enhanced by continued tobacco smoking, reinforcing this behavior and maintaining the addictive process [19,22]. Furthermore, this decreased neural state resonates with the prior findings of a decreased neural response to infant socio-emotional signals in substance-using mothers, where a significant proportion of these mothers smoked tobacco [8]. Therefore, maternal tobacco smok-

Table 3. Log-transformed spectral power for EEG oscillations recorded from smoking and non-smoking mothers comparing between-group differences of smoking status averaged across condition (EO, EC) and hemisphere (left, right). Data are presented as means (standard deviations).

Frequency (Hz)	Smoking Mothers	Non-Smoking Mothers	<i>F</i>	η^2_{partial}
Delta (1-4 Hz)	.20 (.24)	.04 (.31)	5.22*	.07
Theta (4-8 Hz)	-.40 (.26)	-.58 (.33)	6.11*	.09
Alpha (8-13 Hz)	-.38 (.34)	-.57 (.43)	4.08*	.06
Alpha ₁ (8-10.5 Hz)	-.31 (.39)	-.54 (.47)	4.71*	.07
Alpha ₂ (10.5-13 Hz)	-.52 (.34)	-.66 (.43)	2.37	.04
Beta (13-30 Hz)	-1.01 (.28)	-1.14 (.30)	3.24	.05
Gamma (30-80 Hz)	-1.59 (.33)	-1.61 (.36)	<i>F</i> <1	<.001

Note. **p* < .05.

ing may have detrimental downstream consequences on maternal responses to infant cues. To date, prior research in substance use has not typically examined how tobacco smoking may affect maternal behavior, although a positive correlation between maternal smoking during pregnancy and later child neglect has been reported [7]. A related study reported that mothers who had maltreated their children were also more likely to have smoked on the day of a lab visit than non-maltreating mothers, despite both groups being instructed not to smoke [44]. As a consequence, investigating effects of tobacco smoking on parenting is continually warranted.

In addition to our central findings on maternal tobacco smoking at an electrocortical level, it is worthwhile noticing that both smokers and non-smokers showed greater frontal activity in the right, as compared to the left, hemisphere. Hemispheric differences in EEG are consistent with investigations of variations in the frontal lateralization of resting EEG and emotional processing and motivational tendencies [36,45]. Critically, differences in hemisphere activation have also been observed in mothers while they engaged with infant emotional stimuli [35], suggesting the importance of examining hemispheric processes as they relate to caregiving and non-caregiving contexts and the necessity to more fully understand how lateralization of brain activity may relate to maternal behavior.

The present findings should be considered in light of their limitations and directions for future research. The novelty of this study reflects the consideration of multiple frequency bands in tobacco-smoking and non-smoking mothers. While it was a strength to allow mothers to smoke as normal, neither the time of last cigarette was reported nor biochemical markers of tobacco use were collected. Therefore, future research should address how these findings may be influenced by variation in prior to-

bacco smoking (e.g., activation/deactivation of the EEG profile) and if these findings may be associated to residual effects of tobacco/nicotine use or reflect a phenotypic difference. While it is worthwhile noting that spectral power did not correlate with self-reported levels of nicotine dependence, withdrawal symptoms and craving in smokers were not assessed, and these may have had significant effects on the EEG data [20,46]. Therefore, it will be important to establish an understanding of whether smoking cognitions and behaviors contribute to these increased low-frequency bands.

Examining the contribution of factors beyond smoking status that may influence the present results will be necessary. While the samples were matched for demographic characteristics and were comparable with respect to anxiety and depression, a multitude of factors related and unrelated to caregiving may be important to consider. This would include the potential for other psychiatric symptoms to influence EEG activity (e.g., [12,47]) as well as additional physiological factors, including lactation. Indeed, prolactin and oxytocin, which are associated with lactation [48,49], have been found to influence EEG activity [50,51]. Critically, levels of these hormones may vary across our maternal sample. Finally, we only examined neural oscillations recorded from two frontal electrodes consistent with prior EEG research [35-37], whereas evidence suggests that psychotropic-related changes may not be homogeneous across brain regions [20,52]. Therefore, in extending this work, it would be valuable to examine whether this increased power in low-frequency bands in mothers who smoke is represented across the scalp or is limited to frontal brain regions.

In conclusion, this preliminary study demonstrated key differences in neural oscillations between tobacco-smoking and non-smoking mothers at rest. The central finding indicated increased power in low-frequency bands,

Table 4. Log-transformed spectral power for EEG oscillations recorded from both smoking and non-smoking mothers comparing hemisphere (left, right), condition (EO, EC), and their interaction. Data are presented as means (standard deviations).

Frequency (Hz)	Hemisphere		<i>F</i>	Condition		<i>F</i>	<i>F</i>
	Left	Right		Eyes Closed	Eyes Open		
Delta (1-4 Hz)	.00 (.35)	.23 (.32)	32.83**	.13 (.29)	.10 (.31)	1.70	3.99
Theta (4-8 Hz)	-.63 (.33)	-.35 (.35)	69.69**	-.43 (.32)	-.54 (.31)	45.54**	1.40
Alpha (8-13 Hz)	-.61 (.42)	-.32 (.44)	91.02**	-.27 (.47)	-.68 (.37)	154.32**	42.71**
Alpha ₁ (8-10.5 Hz)	-.58 (.44)	-.27 (.48)	82.10**	-.23 (.51)	-.62 (.40)	122.26**	34.33**
Alpha ₂ (10.5-13 Hz)	-.74 (.40)	-.45 (.42)	85.60**	-.41 (.47)	-.78 (.35)	129.83**	25.42**
Beta (13-30 Hz)	-1.21 (.31)	-.95 (.34)	58.37**	-1.07 (.29)	-1.09 (.32)	2.54	1.51
Gamma (30-80 Hz)	-1.67 (.38)	-1.53 (.42)	8.54*	-1.66 (.33)	-1.53 (.38)	30.73**	<i>F</i> <1

Note: **p* < .05, ***p* < .001. Across all frequencies, η^2_{partial} ranged from .12 to .58 for hemisphere, from .03 to .70 for condition, and from .02 to .39 for their interaction.

which may reflect a 'less activated' EEG profile. These findings may manifest through changes in the neural processing of infant cues (e.g., [8]) as well as caregiving behaviors [7,44]. Beyond experimental research, extrapolation of these data further highlight the importance of smoking-cessation programs during pregnancy and the postpartum period.

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Appendix

Table 1. Average raw spectral power for EEG oscillations recorded from smoking and non-smoking mothers. Data are presented as means (standard deviations) in microvolts-squared per Hz for eyes open and eyes closed conditions in each hemisphere.

Frequency (Hz)	Smoking Mothers				Non-smoking Mothers			
	Eyes Closed		Eyes Open		Eyes Closed		Eyes Open	
	Left	Right	Left	Right	Left	Right	Left	Right
Delta (1-4 Hz)	1.78 (2.33)	2.53 (1.65)	1.62 (1.75)	2.73 (2.56)	1.38 (1.48)	1.89 (1.95)	1.28 (1.86)	1.96 (1.95)
Theta (4-8 Hz)	.41 (.31)	.86 (.56)	.31 (.23)	.59 (.38)	.30 (.26)	.64 (.97)	.25 (.23)	.50 (.71)
Alpha (8-13 Hz)	.66 (.59)	1.67 (1.57)	.25 (.24)	.47 (.34)	.52 (.64)	1.29 (1.8)	.20 (.2)	.43 (.85)
Alpha ₁ (8-10.5 Hz)	.84 (.86)	2.25 (2.59)	.31 (.34)	.59 (.48)	.58 (.74)	1.54 (2.72)	.25 (.30)	.57 (1.38)
Alpha ₂ (10.5-13 Hz)	.48 (.46)	1.09 (1.41)	.19 (.16)	.35 (.26)	.47 (.70)	1.03 (1.35)	.16 (.14)	.30 (.37)
Beta (13-30 Hz)	.10 (.11)	.17 (.12)	.10 (.12)	.19 (.24)	.07 (.05)	.14 (.13)	.07 (.05)	.13 (.16)
Gamma (30-80 Hz)	.03 (.06)	.04 (.05)	.04 (.07)	.08 (.14)	.03 (.03)	.04 (.04)	.04 (.05)	.06 (.09)