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Letter to the Editor

The Effect of Smoking on Brain Wave Activity in Middle-Aged Men Measured by Electrocorticography

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Dear Editor-in-Chief

The approximately 4,000 chemicals generated during smoking, such as carbon monoxide, nicotine, and tar, cause serious health diseases including cancer, vascular disease, kidney disease, and sudden death (1-3). The direct and indirect consequences of smoking are widely known, but the smoking population is not decreasing, and smoking is becoming an even more serious public health concern (3-5).

Nicotine causes physiological dependence by affecting catecholamine secretion in the central and peripheral nervous systems. Nicotine also interacts with chemical toxins and binds to specific receptors in the central nervous system, resulting in functional changes in the central nervous system and stimulation of the pleasure center within the cerebrum. This extreme pleasure is identical to that observed during other forms of drug use, such as heroin and morphine use (6, 7).

The effect of smoking on the central nervous system is well known. However, an objective understanding of the specific brain regions that are stimulated is lacking. Electrocorticography is a noninvasive method of investigating this brain activation efficiently (8).

Brain waves are classified into α- and β-waves Alpha (α)-waves (8–12.99 Hz) neurophysiologically reflect the steady state of the brain; the psychological status is most efficient during α-wave activity, and their presence, as during meditation for example, implies that stress and anxiety are decreased, and the brain is relaxed. Beta (β)-waves (13–29.99 Hz) are considered the "activity brainwave"; these waves range 13–20 Hz during daily activity, while high β-waves (20 Hz–29.99 Hz) imply stress, excessive concentration, and anger. High β-waves predominate during learning while in an arousal state or when concentrating on certain events. Brain neurons use a significant amount of energy during arousal, concentration, anxiety, and chaos, and further decision making is compromised if β-wave stimulation persists, resulting in the loss of ideal concentration. Accordingly, the present study investigated the effect of smoking on brain wave activity by examining the change in α- and β-waves before and after smoking.

This study included 16 men over 40 years of age who smoked for over 10 years. The subjects were asked to stop smoking 2 hours before undergoing electrocorticography. Electrocorticography was performed at baseline and repeated within 5 minutes after the subject smoked one cigarette. Subjects were asked to stop smoking 2 hours be-

fore measurement because the half-life of nicotine within the brain is 2 hours; the second measurement was conducted within 5 minutes of smoking because nicotine remains in the brain for 20–40 minutes. A stable electrocorticograph was maintained in all subjects using an 8-channel device (PolyG-I; Laxtha Inc., Seoul, Republic of Korea). Subjects kept the eyes closed during testing. Ten electrodes were placed, including a reference electrode, ground electrode, and the following eight measuring electrodes: left prefrontal (Fp1: Frontal Lobe Pre-1), right prefrontal (Fp2: Frontal Lobe Pre-2), left frontal lobe (F3: Frontal Lobe-3), right frontal lobe (F4: Frontal Lobe-4), left temporal lobe (T3: Temporal Lobe-3), right temporal lobe (T4: Temporal Lobe-4), left parietal lobe (P3: Parietal Lobe-3), and right parietal lobe (P4: Parietal Lobe-4). Signals greater than 60 Hz were removed from the electrocorticogram using a notch filter, and the bandwidth was set at 0.6–46.0 Hz. Subject data were analyzed with the paired *t*-test using SPSS Window version 18.0 (Chicago, IL, USA). Statistical significance was set at $P < 0.05$.

Brain wave activity in the 16 subjects before and after cigarette smoking is shown in Table 1. Prior to initiating smoking, the baseline brain wave activity was measured. All 16 subjects exhibited increased α-waves during steady state and increased β-waves during arousal. Following cigarette smoking, α-waves decreased ($P < 0.05$) and β-waves increased after smoking ($P < 0.05$).

These finding indicate that the pleasant feeling experienced after smoking results from the arousal state caused by increased β-wave activity (9). In addition, we found that smoking immediately affects the brain, as we observed an immediate decrease in the steady state and increase in the arousal state following smoking. Presumably, these changes may prove harmful to brain health if smoking is continued for a long duration.

Table 1: Alpha (α)- and beta (β)-wave activity before and after smoking during electrocorticography

Brain wave	Electrode position	Before smoking	After smoking	*t*	*P*
α-wave	FP1	0.524 ± 0.111	0.367 ± 0.146	3.837	0.006**
	FP2	0.512 ± 0.085	0.347 ± 0.107	6.121	<0.001***
	F3	0.609 ± 0.094	0.467 ± 0.143	3.281	0.013*
	F4	0.619 ± 0.130	0.506 ± 0.135	2.642	0.033*
	T3	0.661 ± 0.067	0.498 ± 0.166	3.937	0.006**
	T4	0.570 ± 0.054	0.430 ± 0.115	3.934	0.006**
	P3	0.830 ± 0.022	0.734 ± 0.082	3.892	0.006**
	P4	0.828 ± 0.003	0.759 ± 0.060	3.271	0.014*
β-wave	FP1	0.138 ± 0.020	0.129 ± 0.021	2.616	0.035*
	FP2	0.170 ± 0.034	0.142 ± 0.039	3.392	0.012*
	F3	0.138 ± 0.020	0.210 ± 0.044	-4.329	0.003**
	F4	0.130 ± 0.038	0.197 ± 0.035	-5.180	0.001**
	T3	0.154 ± 0.032	0.241 ± 0.073	-3.960	0.005**
	T4	0.185 ± 0.036	0.270 ± 0.047	-3.949	0.006*
	P3	0.077 ± 0.013	0.138 ± 0.036	-4.859	0.002**
	P4	0.079 ± 0.006	0.123 ± 0.035	-3.848	0.006**

Data are presented as the mean ± standard deviation

Significance at $*P < 0.05$, $**P < 0.01$, and $***P < 0.001$ as determined by the paired *t*-test.

Electrodes were placed at the following locations: left prefrontal (Fp1: Frontal Lobe Pre-1), right prefrontal (Fp2: Frontal Lobe Pre-2), left frontal lobe (F3: Frontal Lobe-3), right frontal lobe (F4: Frontal Lobe-4), left temporal lobe (T3: Temporal Lobe-3), right temporal lobe (T4: Temporal Lobe-4), left parietal lobe (P3: Parietal Lobe-3), and right parietal lobe (P4: Parietal Lobe-4).

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