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Original Paper

Neuropsychobiology

Neuropsychobiology 2009;59:80–86
DOI: 10.1159/000209859

Received: July 19, 2007
Accepted after revision: June 29, 2008
Published online: March 27, 2009

Effects of Incense on Brain Function: Evaluation Using Electroencephalograms and Event-Related Potentials

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Key Words
Electroencephalograms · Alpha activity · Event-related potential, no-go · Incense

Abstract
To evaluate the effect of the odor of incense on brain activity, electroencephalograms (EEGs) and event-related potentials (ERPs) in a push/wait paradigm were recorded in 10 healthy adults (aged 23–39 years) with normal olfactory function. EEG was recorded from 21 electrodes on the scalp, according to the International 10-20 system, and EEG power spectra were calculated by fast Fourier transform for 3 min before and during odor presentation. ERPs were recorded from 15 electrodes on the scalp before, during and after exposure to incense with intervals of 10 min. In a push/wait paradigm, two Japanese words, 'push' as the go stimulus and 'wait' as the no-go stimulus, appeared randomly on a CRT screen with equal probability. The subjects were instructed to push a button whenever the 'push' signal appeared. Fast alpha activity (10–13 Hz) increased significantly in bilateral posterior regions during incense exposure compared to that during rose oil exposure. The peak amplitudes of no-go P3 at Fz and Cz were significantly greater during incense inhalation. The latencies of go P3 and no-go P3, and the amplitude and latencies of no-go N2 did not change by exposure to the odors of both incense, rose and odorless air. These results suggest that the odor of incense may enhance cortical activities and the function of inhibitory processing of motor response.
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Introduction

Many kinds of aromas have been used in daily life since ancient times because of the psychological effects of fragrances on mood, stress, and working capacity [1, 2]. Many studies have elucidated the mechanisms for the effects of odor on brain function in humans. Recently, neuroimaging studies by functional MRI, positron emission tomography, and magnetoencephalography have revealed that the orbitofrontal, pyriform, and the superior temporal cortices processed olfactory information [3–8]. Electrophysiological studies have shown that various odors affected spontaneous brain activities and cognitive functions, which were estimated as electroencephalograms (EEGs) and event-related potentials (ERPs) such as the contingent negative variation (CNV) and P300 [9–18]. The administration of sedative essential oils increases the alpha and theta rhythms, and decreases the amplitude of CNV and P300, while the presentation of stimulative oils decreases slow waves and increases the amplitudes of CNV and P300 [1, 2, 9, 12, 13, 15, 17]. In-

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0302–282X/09/0592–0080$26.00/0

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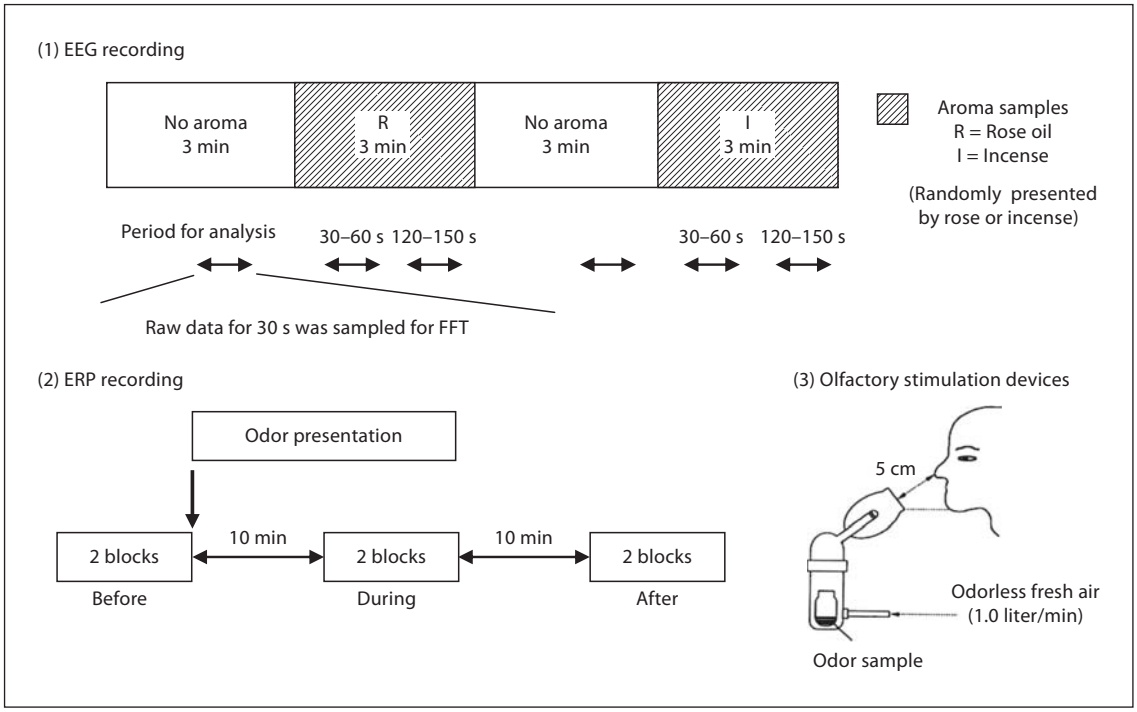

Fig. 1. Experimental protocol of EEG and ERP measurements. FFT = Fast Fourier transform.

cense is often used in Japanese religious ceremonies and has recently been the subject of studies on aromatherapy because of its supposed calming effects. The effect of incense on brain functions, however, has not been examined objectively. In this study, we verified the effect of incense on basic brain activities using EEGs and ERP.

Methods

Subjects

Fifteen right-handed healthy volunteers (8 males and 7 females) aged 23–39 years (mean ± SD: 29.6 ± 6.2) participated in this study. None of the subjects had olfactory diseases, smoked, or abused drugs. Informed consent was obtained from each subject after full explanation of the study. This study received the approval of the Committee of Medical Ethics of the National Rehabilitation Center for Persons with Disabilities.

EEG and ERP Measurement

EEGs were recorded using NeuroFax (Nihon Koden, Tokyo, Japan) from 21 shallow cup Ag electrodes placed on the scalp at Fp1, Fpz, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, Oz and O2 according to the International 10-20 System. The impedance was kept at less than 5 kΩ. All electrodes were referenced to the ipsilateral earlobe electrode. In order to reject blink artifact epochs, bipolar electrooculograms (EOGs) were recorded from electrodes placed below the right eye and its outer canthus. The bandpass filter was from 0.5 to 120 Hz for EEG and EOG, and the sampling rate for digital conversion was 500 Hz.

In a push/wait paradigm, the visual stimuli consisted of two Japanese words: '押せ' (push) as the go stimulus and '待て' (wait) as the no-go stimulus. These stimuli were randomly presented to the subjects for 400 ms, with a 50% probability of occurrence and an interstimulation interval of 1.7 s. The subjects were instructed to push a button as quickly and precisely as possible whenever the push signal appeared. ERPs were recorded from fifteen sites (F3, Fz, F4, C3, Cz, C4, P3, Pz, P4, T3, T4, T5, T6, O1 and O2) referred to linked earlobes by a Pathfinder (Nicolet, USA). The impedance was kept at less than 5 kΩ. The bandpass filter was from 0.5 to 30 Hz, and the sampling rate for digital conversion was 500 Hz. The analysis was done 100 ms before and 900 ms after stimulation. The rejection level was ±96 μV on EEG and EOG. Sixty artifact-free trials with errors in less than 10% of total trials were averaged in a block, and two blocks were recorded to confirm reproducibility and averaged off-line.

Odor Administration

Incense (main component: agarwood) and rose essential oil (rose; not a component of incense) were used as the odor stimuli. The two stimuli were presented in a randomized sequence. Figure 1 shows the recording sequences in EEG and ERP. 0.05 g of rose oil spotted on filter paper or 2.0 g of incense sticks was placed inside an 80-ml sample chamber, and odorless air was pumped into the chamber at an air flow of 1 liter/min. The sample chamber was placed 5 cm in front of the subject's nose (fig. 1c). Each stimulus

was presented under standard conditions adjusted so that the odor intensity was established according to a subjective evaluation by the examiners with normal olfactory functions. This odor intensity was not too strong based on the subjects' answers to 'whether odor was too strong'. The subjects were seated in a comfortable chair in an electric-shielded room with the room temperature controlled at 24°C.

The subjects were instructed to close their eyes during the measurement. EEG was recorded under incense and rose exposure conditions. First, EEG was recorded for 3 min both before and during the first odor exposure. After resting for 3 min, EEG was again recorded before and during presentation of the second odor. The order of odor presentation was counterbalanced among participants.

For ERP recording, each subject was examined in three conditions: odorless fresh air (control), incense and rose odor. In each condition, two blocks were recorded before exposure, two blocks 10 min after exposure started, and 2 blocks 10 min after exposure stopped. The ERPs were recorded under conditions of odorless air, incense and rose oil on different days.

After the recordings, the subjects were asked to give their preference and impression of the odors presented.

Data Analysis

Three EEG segments were selected for each odor condition, 30 s before odor presentation (preexposure), 30–60 s (early exposure) and 120–150 s (late exposure) after starting the odor presentation. The mean power values (μV^2) of each segment were calculated by fast Fourier transform (window 2.05 s, step 1.03 s) for four frequency bands (4≤ theta <8 Hz, 8≤ alpha 1 <10, 10≤ alpha 2 <13 and 13≤ beta <30 Hz).

The rates of change in power (Δp) were calculated relative to the preexposure power [Δp (%) = power value at early or late exposure – preexposure power value]. The Δp was calculated for each frequency band during early and late exposure in the left frontal (Fp1, F3, F7), right frontal (Fp2, F4, F8), left posterior (P3, T5, O1) and right posterior (P4, T6, O2) regions.

Definition of Each Component of ERPs

Go N2 and no-go N2 were designated as the maximal negative wave between 200 and 350 ms after stimulation, and go P3 and no-go P3 as the maximal positive wave between 350 and 500 ms. A topographic analysis was carried out based on the amplitudes obtained from 15 electrodes at the latency of each component. A topographic map (TM) was generated from the interelectrode amplitudes obtained using a standard spline-interpolation algorithm.

Statistical Analysis

The SAS general linear model (GLM) procedure was used. Changes (preexposure vs. early exposure, preexposure vs. late exposure and early exposure vs. late exposure) in Δp of each frequency band in each brain region for each odor were examined using repeated time options (mean option and contrast option). Two-factor repeated ANOVA was performed to analyze Δp of each frequency band in each brain region with time (preexposure/early exposure/late exposure) and odor (rose/incense). Changes of ERP components at Fz, Cz and Pz were examined using two-factor repeated ANOVA with odor and time as within subject factors. Differences of latency or amplitude (before vs. during, during vs. after, and before vs. after) used repeated time option in the SAS GLM procedure.

To evaluate P3 TM before and after inhalation, significant probability maps were computed based on differences in T value. Significant difference of TM was defined as ±2 SD on the T value map.

Results

Odor Preference

The majority of subjects preferred the odor of incense (90%) to that of rose, and no subject disliked either odor. According to the subjects, the impressions imparted by the odor of incense were incense, fresh, calm, sweet and hometown, and those imparted by the odor of rose were rose, flower, perfume, grass, sweet and medicine.

Changes of EEG Power Values

Figure 2 shows Δp in the left frontal, right frontal, left posterior and right posterior regions of each frequency band under two odor exposure conditions. Comparing the two odors, the interaction of odors and time in Δp of alpha 2 activity in the left posterior was significant ($p < 0.05$), and Δp of alpha 2 activity increased significantly ($p < 0.05$) in the incense condition compared to that in the rose condition. Analyses of time dependences in each odor showed that during incense exposure, Δp of alpha 2 activity changed significantly in the left frontal region [$F(2, 14) = 5.09$, $p = 0.022$], increased during early exposure compared to preexposure ($p = 0.031$) in the right frontal region [$F(2, 14) = 4.82$, $p = 0.025$], increased during early exposure compared to preexposure ($p = 0.014$) in the left posterior region [$F(2, 14) = 5.83$, $p = 0.014$], increased during early exposure compared to preexposure ($p = 0.039$), increased during early exposure compared preexposure ($p = 0.071$) in the right posterior regions [$F(2, 14) = 6.61$, $p = 0.009$], and increased during early exposure compared to preexposure ($p = 0.015$). The Δp of beta activity showed no significant changes when exposed to both incense and rose.

Changes of ERP Components

The interactions between odors (incense, rose and odorless air) and time were found to be significant in the peak amplitudes of no-go P3 at Fz ($p = 0.012$) and Cz ($p = 0.046$), but the factor 'odor' was not significant in the repeated ANOVA. Repeated time option analysis in the SAS GLM procedure showed that the peak amplitudes of no-go P3 changed significantly at Fz [$F(2, 18) = 4.44$, $p = 0.027$], increased during exposure to incense odor

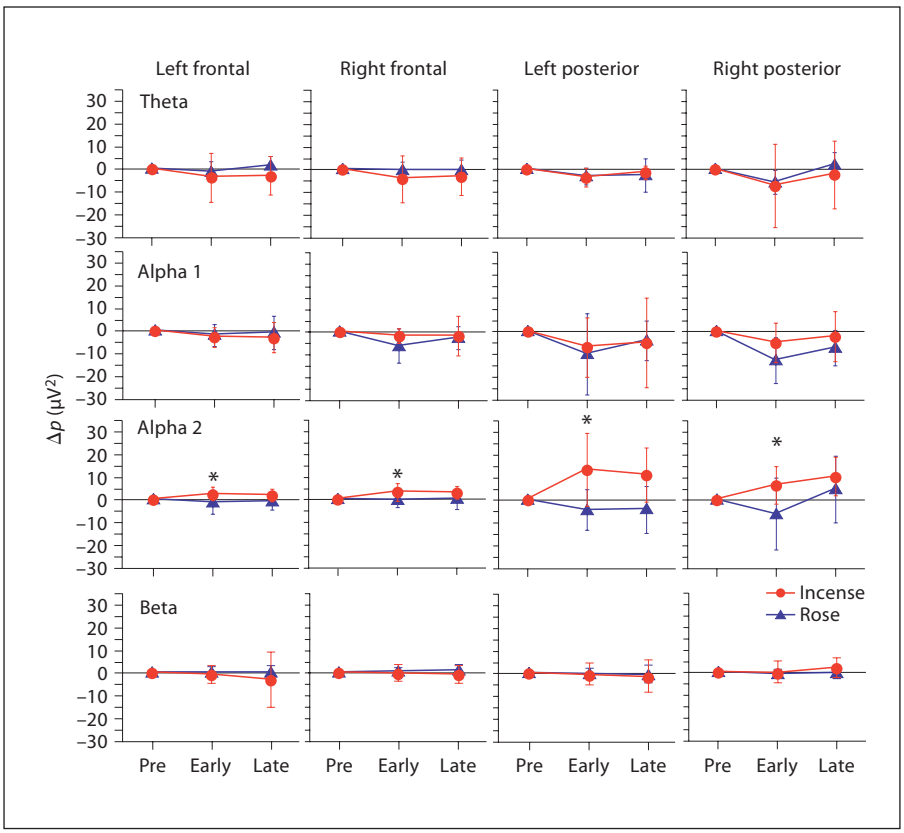

Fig. 2. Change in power (Δp) in bilateral frontal (left: Fp1, F7, F3; right: Fp2, F4, F8) and posterior (left: P3, T5, O1; right: P4, T6, O2) regions for each frequency band before and during exposure to incense. Δp is the power during early or late exposure, – that before exposure. Each panel shows the mean of Δp of all subjects. pre = 30-second period before odor presentation; early = 30- to 60-second period during odor exposure; late = 120- to 150-second period during odor exposure. Bar: mean ± SD. * $p < 0.05$ vs. pre.

compared to before exposure ($p = 0.006$), and at Cz [$F(2, 18) = 3.57$, $p = 0.049$], during exposure to incense odor compared to before exposure ($p = 0.003$), but did not change significantly during exposure to rose odor and odorless air. The amplitude of go P3 at Fz changed significantly during odorless air inhalation [$F(2, 18) = 4.16$, $p = 0.028$], decrease after exposure compared to before ($p = 0.034$). In the incense condition, the go P3 amplitude tended to increase during incense exposure, but it did not reach statistical change. The latencies of go P3 and no-go P3, and the amplitude and latencies of no-go N2 did not change significantly by exposure to the odors of both incense, rose and odorless air (tables 1, 2). Go P3 TM and no-go P3 TM showed no significant change on the T value map (fig. 3).

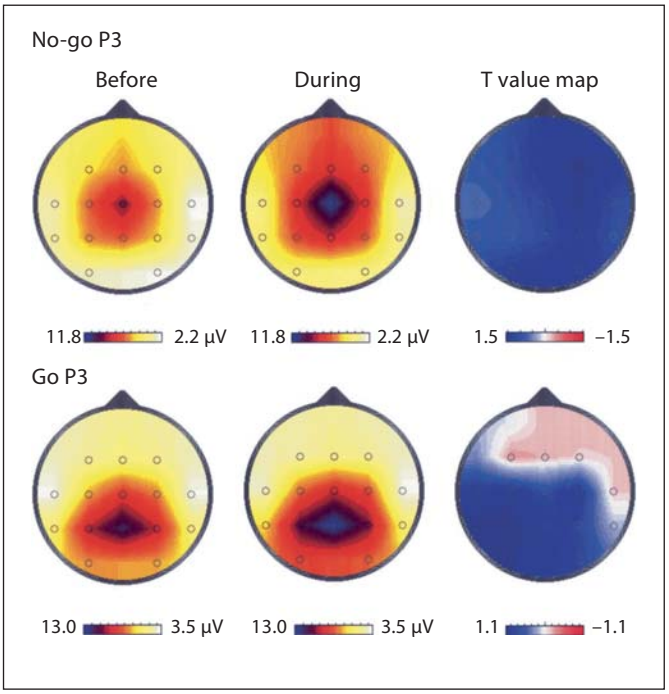

Fig. 3. Comparison of go and no-go P3 TM before and during incense exposure.

Discussion

The purpose of this study was to verify the effect of incense on basic brain activities using EEGs and ERP. Our study showed that alpha 2 activity (10–13 Hz) increased significantly during incense exposure in the pos-

Table 1. Change of go P3 before, during and after exposure to air, incense and rose (mean ± SD)

	Before	During	After
Latency, ms			
Air			
Pz	385.7 ± 52.5	396.5 ± 44.2	393.3 ± 38.8
Cz	404.9 ± 65.1	404.2 ± 44.7	408.8 ± 66.5
Fz	398.7 ± 65.1	407.0 ± 41.1	387.7 ± 35.2
Incense			
Pz	378.2 ± 36.4	383.8 ± 24.0	375.0 ± 34.8
Cz	380.6 ± 37.0	394.4 ± 13.8	386.6 ± 29.0
Fz	374.2 ± 43.0	364.2 ± 42.7	376.3 ± 44.2
Rose			
Pz	396.3 ± 53.2	393.3 ± 41.6	386.1 ± 51.8
Cz	396.0 ± 63.2	406.7 ± 60.3	400.1 ± 50.0
Fz	393.6 ± 63.3	409.7 ± 58.0	404.9 ± 46.4
Amplitude, mV			
Air			
Pz	12.7 ± 6.2	12.4 ± 6.5	12.6 ± 6.0
Cz	10.4 ± 5.6	9.3 ± 3.8	10.8 ± 6.6
Fz	6.4 ± 3.34	5.0 ± 3.6	6.1 ± 4.7*
Incense			
Pz	11.7 ± 2.0	12.4 ± 3.1	12.8 ± 3.1
Cz	9.4 ± 1.7	10.2 ± 2.0	10.3 ± 1.9
Fz	6.3 ± 3.0	6.7 ± 2.9	6.7 ± 2.9
Rose			
Pz	11.9 ± 5.2	11.7 ± 6.1	12.4 ± 5.8
Cz	9.4 ± 5.3	8.9 ± 5.2	9.5 ± 5.3
Fz	6.4 ± 5.7	4.9 ± 3.0	5.6 ± 3.8

* $p < 0.05$ vs. before.

Table 2. Change of no-go P3 before, during and after exposure to air, incense and rose (mean ± SD)

	Before	During	After
Latency, ms			
Air			
Pz	414.5 ± 49.4	420.2 ± 49.6	432.5 ± 50.5
Cz	417.8 ± 30.0	434.5 ± 39.7	427.8 ± 36.0
Fz	424.8 ± 34.4	431.8 ± 41.1	430.3 ± 35.3
Incense			
Pz	399.2 ± 33.8	404.6 ± 32.2	406.6 ± 24.3
Cz	404.0 ± 24.1	416.4 ± 26.6	407.0 ± 15.2
Fz	392.8 ± 46.9	419.4 ± 27.7	419.4 ± 29.7
Rose			
Pz	400.7 ± 65.1	409.7 ± 51.7	421.5 ± 54.0
Cz	418.2 ± 32.0	415.2 ± 37.3	432.2 ± 46.4
Fz	419.0 ± 27.0	415.2 ± 37.3	427.5 ± 32.2
Amplitude, mV			
Air			
Pz	8.6 ± 5.5	9.7 ± 5.8	8.9 ± 6.0
Cz	10.3 ± 6.7	11.4 ± 6.9	10.5 ± 7.3
Fz	7.4 ± 4.6	7.4 ± 4.3	7.2 ± 4.6
Incense			
Pz	7.4 ± 3.5	8.8 ± 2.8	8.5 ± 3.1
Cz	9.2 ± 5.6	11.8 ± 4.5*	11.6 ± 4.8
Fz	6.2 ± 3.7	8.5 ± 3.7*	8.1 ± 3.2
Rose			
Pz	8.4 ± 4.9	8.0 ± 4.9	8.7 ± 5.7
Cz	10.7 ± 5.9	9.7 ± 5.4	10.0 ± 6.8
Fz	7.6 ± 4.2	6.5 ± 3.8	6.3 ± 5.1

* $p < 0.05$ vs. before.

terior regions, and no-go P3 amplitude at Fz and Cz increased during incense exposure.

Most of the past EEG studies on the effects of odors have demonstrated increased alpha activity by administration of several essential oils such as lavender, sandalwood and chamomile oils [1, 2, 9, 12, 13, 15, 17]. These substances are components of incense, and their aromas have a relaxing effect on brain activity. Alpha activity is attenuated under emotional tension and stress condition [19]. Therefore, an increase of alpha 2 activity without an increase of theta, slow alpha or beta activity during early incense exposure suggests that the olfactory stimulation by incense may increase the vigilance level and have a relaxing effect on brain activity.

Our result showed that the increased alpha 2 activity was maintained up to late exposure (120–150 s) to incense. Olfactory receptors adapt to a continuous stimulus within several seconds [20], and recovery of the receptors from adaptation takes approximately 30 s [21]. The primary olfactory cortex, consisting of the pyriform, periamygdaloid, lateral and entorhinal cortices, habituates immediately to a continuous stimulus [7, 22]. Poellinger et al. [7] showed in a functional MRI study that the primary olfactory cortex was activated from 10 to 15 s after odor stimulation, followed by a decrease in activity within 30 s for a duration of 60 s. In contrast, activation of the orbitofrontal cortex, which is connected with the primary olfactory cortex, was sustained for 60 s after odor stimulation. In our study, the first odor did not influence the effect of the second odor on brain activity because of an adequate interval between the first and the second odor presentation. The increase of alpha 2 activity during late exposure (120–150 s) to incense suggests that the odor of incense affects not only the primary olfactory cortex, but also the adjoining cortices. The primary olfactory cortex is connected with the limbic cortex, hypothalamus, hip-

pocampus, insular cortex, and orbitofrontal cortex, which are related to emotion and memories [23, 24]. Therefore, it is possible that the odor of incense may induce strong emotional responses and memories. The individual preference for odors can influence EEG activity and affect psychological mood [12, 13, 16, 17]. Kline et al. [16] showed that alpha (8–13 Hz) activity increased during administration of a pleasant odor, and their results were in line with ours showing that alpha 2 activity increased significantly during presentation of incense, the aroma of which was preferred by the subjects to that of rose.

In our study, the no-go P3 amplitude increased significantly at Fz and Cz during inhalation of incense odor. The ERP components of go stimuli in a go/no-go task reflect an executive cognitive process. On the other hand, the ERP components of no-go stimuli reflect the electrical changes of the brain in relation to the inhibition process of motor response [25–28]. The sources for no-go P3 are located in the orbitofrontal and anterior cingulate cortices [28]. The orbitofrontal cortex has strong neural connections with the olfactory and the limbic regions, and the anterior cingulate cortices are linked through their involvement in behavior (e.g., mood) and inhibitory control processes [29, 30]. Damage to the orbitofrontal cortex reduces inhibitory control in affective processing in humans, and entails errors of perseveration and impaired response inhibition in animals [31]. The anterior cingulate cortex is involved in response inhibition, initiation, intention, and conflict monitoring [30]. Therefore, the increase of no-go P3 amplitude during inhalation of incense suggests that incense may enhance the function of inhibitory processing of motor response by activating the orbitofrontal and anterior cingulate cortices. In addition, almost all subjects preferred the odor of incense. Previous studies showed that EPR components changed with individual preference for odors. For instance, the amplitude of P300 and the early CNV increased with the inhalation of pleasant odors, and decreased with the inhalation of unpleasant odors [10, 12]. It is possible that a subject's preference may affect the cortices involved in the emotional pathway.

Agarwoods have been reported to contain several sesquiterpenoids. A recent study in mice revealed that several sesquiterpenoids isolated from oriental incenses are antagonists of dopamine D2 and serotonin 5-HT_{2A} receptor binding, and have sedative and analgesic effects on the central nervous system [32]. The dopaminergic and serotoninergic neurons in the nuclei in the brainstem and hypothalamus project to the nuclei of the basal ganglia and accumbens, amygdala, and prefrontal cortex [33]. Through these pharmacodynamic actions, the components of incenses may potentially act not only on the primary olfactory cortex but also on the mesocorticolimbic dopaminergic and serotoninergic systems. We did not demonstrate the pharmacological actions of incense because incense components in serum were not measured. This and the small study population are limitations of this study. Further studies of the effect of incense on brain functions are warranted.

Acknowledgments

We thank Nippon Kodo Co. for providing incense, and Dr. Satoru Shimizu for the statistical support. This study was supported by the Research Grant for Research on Brain Science H12-Brain-023 from the Japan Ministry of Health, Labour and Welfare.

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