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Electrocortical and Autonomic Alteration by Administration of a Pleasant and an Unpleasant Odor

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Abstract

The present study was designed to investigate whether there is a consistent response in ongoing EEG due to repetitive olfactory stimulation. Two odors of different hedonic quality were presented bilaterally to five male subjects at suprathreshold levels. A room-air blank served as the control stimulus. Each odor was presented six times to each subject in each of three sessions. Electrocortical activity, heart rate, skin conductance and breathing cycle were recorded continuously. EEG variables assessed were difference scores of absolute power in the frequency bands theta, alpha1, alpha2 and beta1 at eight locations. Phenylethyl alcohol was rated pleasant, while valeric acid was judged unpleasant. Within 8 s after stimulus release, valeric acid increased alpha2 power, whereas phenylethyl alcohol did not. No further frequency bands were affected by olfactory stimulation. These findings suggest that smelling an unpleasant odor leads to a cortical deactivation. Chem. Senses 20: 505–515, 1995.

Introduction

A variety of studies have shown effects of odors on cognition (Richardson and Zucco, 1989; Lorig and Roberts, 1990), emotion (Ehrlichman, 1987; Van Toller, 1988; Ehrlichman and Bastone, 1992a; Miltner *et al.*, 1994), attention (Warm *et al.*, 1991), memory (Ehrlichman and Bastone, 1992a) and sleep (Badia *et al.*, 1990). Further evidence that odors can affect electrocortical activity in the cortex is provided by the fact that olfactory evoked potentials can reliably be recorded from scalp sites (Kobal and Hummel, 1991). Up to now, however, reported results of odor presentation on frequency and spatial domains of ongoing EEG have been inconsistent. Also, little is known about the relationship between hedonic quality and electrocortical activity (Van Toller, 1988; Ehrlichman and Bastone, 1992a; Van Toller *et al.*, 1993).

When assessing spontaneous EEG responses to olfactory stimulation, three main questions can be addressed. (i) Is there a consistent response in the EEG due to odor administration? (ii) Are some brain regions more involved in the perception and processing of odor stimuli than others? (iii) Are there any electrophysiological variables that can discriminate between odors of different hedonic quality?

The first authors to demonstrate a response in electrocortical activity due to odor administration were Sem-Jacobsen *et al.* (1953) and Moncrieff (1962). Sem-Jacobsen *et al.* (1953) recorded depth EEG in a single

person. When an electrode was placed near the olfactory bulb, waves of higher frequency appeared when the person smelled an odor and ended as soon as the odor was removed. Moncrieff (1962) recorded EEG from eight electrodes in five subjects while they smelled different odors. By visually inspecting the data, this author found a suppression of the alpha rhythm for both pleasant and unpleasant odor stimuli. The disappearance of alpha waves was seen in about one fourth of all trials and for different locations. Therefore, both reports revealed a cortical activation upon olfactory stimulation, characterised in awake subjects as a suppression of alpha rhythm and an increase of beta waves (Lindsley and Wicke, 1973; Davidson *et al.*, 1990). These findings are not surprising, because a blocking of alpha waves has also been demonstrated for visual and acoustic stimulation (Lehmann, 1971; Kaufman *et al.*, 1992; Michel *et al.*, 1994).

Further evidence that olfactory stimulation is associated with a cortical activation is provided by Lorig *et al.* (1990, 1991) and Schwartz *et al.* (1992). Lorig *et al.* (1990) presented three different concentrations of lavender oil and spiced apple using an olfactometer. EEG was recorded during 15 s from four electrodes. These authors reported a trend to fewer alpha waves during presentation of odors and more left posterior theta activity during presentation of the lowest concentrations of both odors as compared with the medium concentrations. In a further study, Lorig *et al.* (1991) assessed zero crossings in the alpha band at nine locations for 10 s during the administration of five different levels of galaxolide. There was no main effect of concentration. However, during the lowest concentration, which was undetected, subjects had markedly fewer alpha waves as compared with the no-odor control. Schwartz *et al.* (1992) analysed EEG data obtained while subjects smelled three odorants in flasks at suprathreshold and subthreshold concentrations. When subjects detected the suprathreshold concentration, significant EEG alpha blocking was observed in anterior, central and posterior regions. When subjects smelled the subthreshold concentration, significant alpha blocking was found in the central region and especially in the right hemisphere.

On the other hand, several studies revealed an increase of alpha or theta rhythms during the presentation of odors. Sawada *et al.* (1992) presented three sedative odors on strips for 3 min and recorded EEG from two parietal sites. These authors found an increase of relative power in the alpha band (8–14 Hz), and further inspection revealed that this increase was mainly related to the lower alpha band (8–11 Hz). Klemm *et al.* (1992) offered seven odors using an olfactometer and recorded EEG from 19 electrodes during 2 min stimulation periods. They found two different responses to the odors, an early theta increase which habituated and a later theta increase that ended as soon as odor delivery stopped. Moreover, there was a significant increase in theta voltage over the left anterior region for all odors, although individual variability made it difficult to find any results on the group level. No relationships between changes of theta voltage and subjective reportings were documented. Van Toller *et al.* (1993) presented six odors on smelling strips and found a systematic increase of alpha amplitude during the first 2.56 s of the administration of several odors. These authors extracted similarities in response patterns for the different odors, using 28 electrodes and alpha amplitude (8–14 Hz). They found two regions, one behind the central sulcus and the other located occipitally, where alpha amplitude responded. Furthermore, they correlated odor ratings with the electrocortical responses. They found changes in the alpha band at four electrodes anterior to the central sulcus to correlate with subjective ratings. Taken together, the findings of Sawada *et al.* (1992), Klemm *et al.* (1992) and Van Toller *et al.* (1993) suggest that olfactory stimulation is associated with cortical deactivation. The response, however, seems to differ between odors and subjects.

Several factors might be responsible for these inconsistent findings in spatial and frequency domains of ongoing EEG. First, cortical activity may change as a function of time. Therefore, the time frame assessed after stimulation is crucial. Secondly, effects of a single exposure to odors certainly differ from effects of repeated administration. Therefore, the number of averaged trials might be a further reason for inconsistent findings. Thirdly, the inter- and intra-individual variability of responses to odors makes it difficult to extract common findings and increases the occurrence of unreliable effects. Furthermore, several methodological limitations could explain these inconsistent findings, such as differences in EEG-recording systems, reference sites, analysing procedures, techniques of presenting odors and population of subjects. In addition, the high fluctuation of electrocortical activity (Burgess and Gruzelier, 1993), the partly poor statistical power in the studies reported and the lack of a theoretical foundation increases the incidence of spurious findings.

However, two hypotheses can be generated with respect to the localised processing of odor stimuli. First, the right hemisphere seems to predominate in olfactory processing (Richardson and Zucco, 1989; Zatorre *et al.*, 1992) and odor memory (Richardson and Zucco, 1989; Jones-Gotman and

Zatorre, 1993). Therefore, olfactory stimulation may suppress alpha rhythm in the right hemisphere, because less alpha is associated with higher activation (Lindsley and Wicke, 1974). Secondly, odors can induce an emotional experience (Ehrlichman and Bastone, 1992b). There are, however, at least two different theories on the lateral processing of emotion (Gainotti, 1989). Several authors claim the right hemisphere to be superior for the control of emotional processing, irrespective of the valence of emotion (Levy, 1983; Wittling and Roschmann, 1993). Others advocate the view that the processing of negative emotions is associated with higher activity in the right hemisphere, while positive emotions are related to higher activity in the left hemisphere (Ahren and Schwartz, 1985; Davidson and Tomarken, 1989; Davidson *et al.*, 1990; Jones and Fox, 1992). Therefore, an odor rated as unpleasant is expected to lead unequivocally to a higher activation of the right hemisphere (Ehrlichman, 1987). On the other hand, an odor rated as pleasant may increase activation in the right or the left hemisphere.

Some support for lateralised processing based on the evoked potential paradigm is provided by Kobal *et al.* (1992). These authors found shorter latencies and smaller amplitudes after presentation of unpleasant odors to the left nostril as compared with presentation to the right nostril. However, shorter latencies and smaller amplitudes were found when vanillin, an odor rated as pleasant, was presented to the right nostril. These authors interpreted their findings in concordance with the view that positive emotions are processed predominantly by the left hemisphere and negative emotions by the right.

The present study was designed to assess the effects of repeated odor administration on frequency and spatial domains of ongoing electrocortical activity. Two odors, one assumed to induce a pleasant and the other an unpleasant experience, were presented to five male subjects for 30 s. A room-air blank served as the control condition. Each odor was presented six times to each subject in each of the three sessions. Frequency analysis of raw EEG was computed and the pre-post stimulus differences were analysed for eight electrodes along the mid-sagittal plane for theta, alpha1, alpha2 and beta1. Heart rate, skin conductance and respiration rate were also recorded, because autonomic variables are indicators of arousal and have been found to respond to odor presentation (Van Toller *et al.*, 1983; Schwartz *et al.*, 1986; Miltner *et al.*, 1994).

The following predictions were made. (i) There is a consistent EEG response due to odor administration. As the presentation of an odor is first of all a sensory stimulation, we predicted a decrease of alpha power upon odor administration. (ii) Within 8 s after stimulus release, we expected recognition of the odor and an appraisal of its hedonic quality (Ehrlichman and Batone, 1992a) and the induction of an emotional experience. Given that the hedonic valence of valeric acid would be negative, we expected higher activation of the right hemisphere during the detection and processing of valeric acid, thus leading to less alpha power in the right hemisphere. For phenylethyl alcohol higher values in the alpha bands were expected on the right side. (iii) Odor presentation can affect autonomic variables. Because a negatively valenced state has been associated with higher skin conductance and higher heart rate (Ekman *et al.*, 1983; Stemmler, 1989; Hubert and De Jong-Meyer, 1991), we expected increase in both variables after presentation of valeric acid and a decrease after phenylethyl alcohol. Furthermore, valeric acid was expected to consistently induce skin conductance responses.

Materials and methods

Subjects

Five right-handed males with a mean age of 24 years were recruited through advertisement. However, one subject was excluded from the analysis (see below). The remaining subjects were able to detect odors and to discriminate between odors with respect to their hedonic quality. Subjects were invited to take part in a study designed to assess physiological variables during odor smelling. The names of the odors were not mentioned. Mean laterality quotient of handedness, assessed using a German adaptation of the Edinburgh Inventory (Oldfield, 1971), was 86 (range 78–100). None of the subjects knew of any familial neurological disturbances. Subjects reported being in good health; they were not on medication; they were non-smokers and did not drink any coffee or alcohol on the days of assessment. Participation was compensated with sFr 150 (about $120.-).

Odorants

Phenylethyl alcohol (2-phenylethanol) and valeric acid (pentanoic acid) were used as odorants. Charcoal-filtered room air (room-air) served as the blank control. The main criterion for selecting these odorants was their ability to induce a pleasant or unpleasant odor perception. Phenylethyl alcohol, a widely used fragrance similar to the odor of roses, was expected to be rated as pleasant. Valeric acid, an odor related to sweat or cheese, was predicted to be judged as

unpleasant. Phenylethyl alcohol is believed to preferentially stimulate the olfactory system, whereas valeric acid may—at least at high concentrations—stimulate intranasal trigeminal nerve structures (Doty *et al.*, 1978).

Given the variability of threshold concentrations (Devos *et al.*, 1990), the concentration of odorants presented to the subjects was adjusted to a level of about 20–100 times above the odorant's group threshold concentration. At this concentration, odors were expected to be clearly recognisable without stimulating trigeminal nerve structures. The concentration of the odorant was estimated, based on the knowledge of the vapour pressure, the dilution ratio of odorant-saturated nitrogen flow and diluting air flow, and finally, confirmed by headspace measurement (Neuner-Jehle and Etzweiler, 1991). The headspace method enables a quantitative determination of the odorant coming out of the sniffing funnel. A microfilter is simply immersed into the gas stream within the smelling funnel. Due to the small size of the microfilter (2 mm diameter) there is ample space within the smelling funnel (25 mm diameter) so that the odorous gas flow can pass it without being disturbed by the filter. A defined volume of headspace is sucked through the filter and the adsorbed odorant is quantitatively determined after desorption from the filter by a solvent. For quantification of the odorant, gas chromatography was applied using the external standard method. The specifications of odor stimuli given in Table 1 were obtained by taking the mean of at least three independent headspace samples whose extract was quantified twice by gas chromatography.

Table 1 Specifications of odor stimuli

Specifications	Phenylethyl alcohol	Valeric acid
Analytic purity (%)	≥99.5	≥99.3
Odor note	floral, rose	cheesy, sweat
Concentration in air[1] (ng/l)	417	98
Concentration (vol./22°C, ppm)	0.076	0.023

[1]Measured at the sniffing funnel.

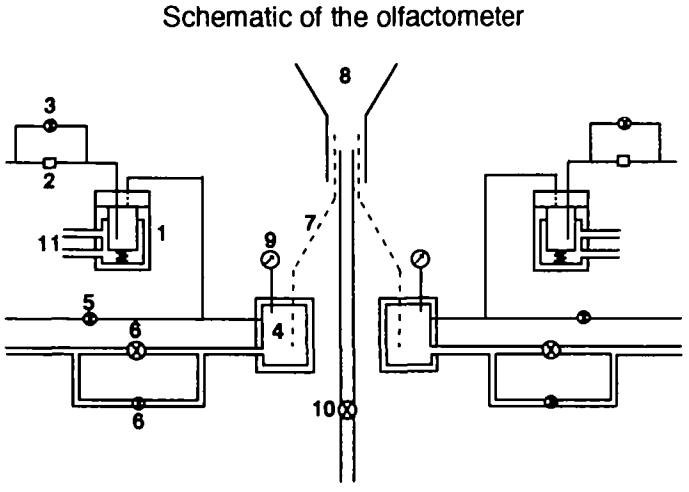

Figure 1 Schematic of olfactometer. (1) Sample container; (2) flow regulator (10–100 ml/min); (3) needle valve; (4) mixing chamber, (5) air dilution (0–10 l/min); (6) needle valves; (7) fused silica capillary, on-off switching device (1–256 ml/min odorous gas mixture), (8) sniffing funnel; (9) pressure sensor; (10) purified, humidified air adjustable to 8 l/min flow; (11) cryostatic cooling.

Odor delivery

An olfactometer from Givaudan-Roure Research Ltd (Dübendorf, Switzerland) was used as the odor delivery device. A schematic of the olfactometer is provided in Figure 1. This device is based on the dynamic dilution approach (Neuner-Jehle and Etzweiler, 1991). All material used to expose odorants is made from stainless steel or quartz to prevent decomposition of compounds. An odorant-saturated gas flow is generated by allowing carrier gas (pure nitrogen) to pass over a large surface coated with the odorant in the sample container (1). The nitrogen flow is controlled either by the flow regulator (2) between 10 and 100 ml/min or by adding nitrogen up to 500 ml/min using a needle valve (3). The flow into the mixing chamber (4) is set and measured before starting the investigation and then controlled at the end, using commercially available flow measuring devices. During the measurement, control is only performed by observing the head pressure at the inlet of the flow regulator. A flow up to 500 ml/min is used for weak odorous substances when no dilution is required. In the present study, however, the odor-saturated nitrogen had to be diluted in the mixing chamber with odorless air, purified by activated charcoal and filtered by a microfilter. The flow regulator (2) keeps the flow constant even when the pressure in the mixing chamber is changed. The dilution ratio is set by turning a needle valve (5). Measuring of the flow is performed at the exit of the mixing chamber after opening two needle valves (6) by means of a rotameter. Dilution is set and kept constant from the beginning to the end of the investigation. The flow through the fused silica capillary (7) into the sniffing funnel (8) is adjusted by turning two valves (6). Two valves are needed because of the technical problems to cover the whole dynamic range required for a precise adjustment of a flow between 0 and 10 l/min. Closing the valves (6), increases the pressure inside the mixing chamber,

simultaneously resulting in a higher flow through the fused silica capillary. The flow through the capillary is based on a calibration curve, which relates the pressure in the mixing chamber to the flow through the capillary. The actual pressure is read constantly by a pressure sensor (9) whose signal is converted into the flow reading through the capillary. Using a capillary of 330 mm length and 0.31 mm diameter, a flow from 1 to 256 ml/min can be regulated by altering the pressure from 20 to 800 mbar. By means of an on/off switching device (7), the odorous gas flow is delivered instantly to the sniffing funnel, where the final dilution with odorless air takes place. This flow is regulated by a valve (10) to 8 l/min and constantly displayed by a rotameter.

The time the stimulus requires from its onset at the olfactometer to its arrival at the subject's nose (positioned at the sniffing funnel) is less than 0.5 s. However, the time lag until the subject perceives the odor may be somewhat longer, depending on his breathing cycle.

Odor rating

Odors were rated in a training session held before the experimental sessions and then again before recording in each of the three experimental sessions. Dimensions rated were sleepiness-arousal, unpleasant-pleasant and low-high odor intensity on a nine-point scale.

EEG data

EEG was recorded from 17 electrodes according to the International 10–20 system (F3/4, F7/8, Fz, C3/4, Cz, T3/4, T5/6, P3/4, Pz, O1/2) with Hellige electrode cream and an elastic electrode cap (Blom and Anneveldt, 1982). Cz was used as recording reference, and F1 and F2 were used as ground. Signals were digitised at a rate of 512 Hz and 12 bit, and transmitted by an optical fibre to a ProScience Lab TEAM computer (Dimpfel *et al.*, 1993). Electrode impedance was kept below 10 kOhm. Additionally, ECG and horizontal EOG were recorded for detection of artifacts.

Autonomic variables and respiration

Heart rate, skin conductance and respiration were recorded with a Kölner Vitaport-System device (Becker, Karlsruhe, FRG) and stored on a RAM card. To assess heart rate, electrodes with solid-gel were placed in an Einthoven derivation, while beats per minute (bpm) were calculated through detection of R-spikes. Skin conductance was recorded with Ag/AgCl electrodes (0.8 cm^2) using Hellige electrode cream as electrolyte. They were placed at thenar and hypothenar sites on the left hand (Venables and Christie, 1973). Sampling rate was 4 Hz, while constant voltage was 0.5 V. The criterion for a skin conductance response was >0.01 μS. Because respiration irregularities can affect outcome variables (Schwartz *et al.*, 1986), the respiration cycle was assessed using a strain gauge attached around the upper abdomen.

Procedure

In the training session, the experiment and procedure were explained, and the odors were rated. Subjects gave informed consent, and several questionnaires including control variables and assessment of handedness were completed. Three experimental sessions, each 1 week apart, were held. In each session, six exposures to phenylethyl alcohol, six to valeric acid, always at the same concentrations, and four to the room-air blank were performed.

For the experimental sessions, a subject arrived between 8.30 and 9.30 a.m. at the laboratory. He completed a questionnaire for control variables and rated odors while he was seated in a comfortable semi-reclining chair in front of the olfactometer. Recording took place in a light-attenuated room with a temperature of 22±1°C, adjacent to the room with the recording equipment. Electrodes were attached, and the subject was orally instructed to relax and breathe normally through his nose. He was informed that he might or might not receive an odor in any trial. Every subject was video monitored during the whole session. Because the

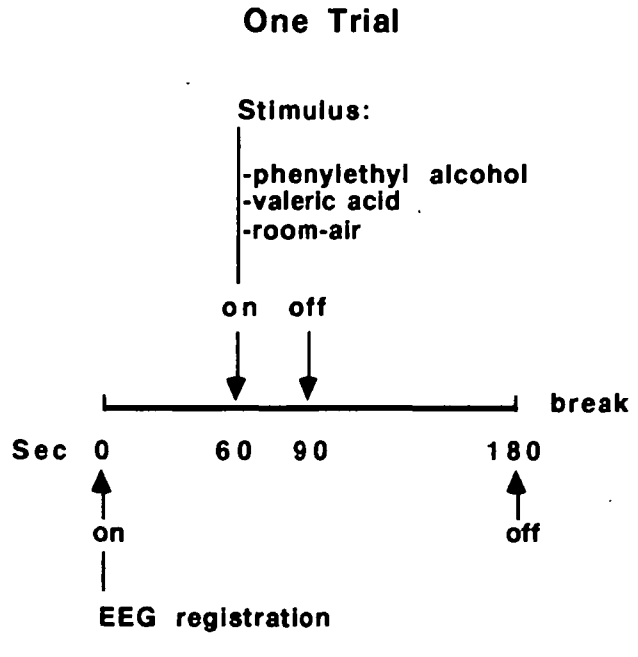

Figure 2 Structure of one trial.

on/off-switching device of the olfactometer generated a characteristic sound, white masking noise (about 70 dB) was delivered as background by room loudspeakers.

Each trial had the structure illustrated in Figure 2. A trial began with the interruption of the white noise. An order was given through an intercom system to the subject to sit upright with his nose at the sniffing funnel and to close his eyes. After a delay varying between 10 and 30 s in a random order, EEG recording was started (s 0). After exactly 60 s, one of the odorants, either valeric acid or phenylethyl alcohol, was released and a marker signal was set on the recording device. Odors were presented for 30 s. During the control situation, the same characteristic sound of on/off-switching was simulated without delivering an odor. After a total of 180 s of EEG recording, the subject was allowed to open his eyes and to lean back for about 30 s before starting the next trial. The aim of this procedure was to keep subjects alert. In order to prevent EMG artifacts from contaminating the EEG recording, the subject's head was comfortably supported during the trials, as the backrest of the reclining chair moved with the subject. After eight trials, a break took place, during which the subject was allowed to get up. Odors were presented in each session in the sequence: /phenylethyl alcohol (P)/control (C)/valeric acid (V)/P/V/C/V/P, before the break and /V/P/C/P/V/P/C/V after the break. One session took about 2.5 h.

Data reduction analysis

Subjective ratings of the odors were analysed using evaluation of the three experimental sessions and a Wilcoxon test.

EEG data were analysed off-line. From the period up to 12 s before (pre-stimulus EEG) and up to 12 s after stimulus release (task EEG), 8 s of artifact-free raw EEG was selected. In most of the cases, those 8 s immediately before and 8 s after the onset of the stimulus could be evaluated. Eye blinks, which were detected by visual inspection of the horizontal EOG, occurred rarely. Using a Hanning window, 4-s sweeps were submitted to an FFT and then averaged. The measurement unit was microvolt squared. Data obtained were downloaded on a mainframe computer for statistical analysis, where difference scores were calculated by subtracting pre-stimulus EEG from task-EEG. Then, data were averaged within a session separately across all trials of phenylethyl alcohol, valeric acid and the room-air blank. Frequency bands considered were theta (4.75–6.75 Hz), alpha1 (7–9.5 Hz), alpha2 (9.75–12.5 Hz) and beta1 (12.75–18.5 Hz). Delta and beta1 were omitted, as artifacts in these bands were after all likely to occur due to the task. Electrodes analysed were frontal (F3/F4), central (C3/C4), parietal (P3/P4) and occipital (O1/O2). These eight locations were taken to represent a matrix, consisting of four levels in the anterior-posterior dimension and two levels in the lateral dimension. With this approach, the most interesting regions were covered and hemispheric asymmetries were expected to be revealed.

EEG data were analysed using repeated measures ANOVAs (BMDP 2V) with the factors condition (phenylethyl alcohol, valeric acid, room-air blank), hemisphere (left, right) and location (frontal, central, parietal, occipital). Session (session one, session two, session three) served as the between factor. Data used in the factor condition were means across all trials within one session (six trials phenylethyl alcohol per session, six valeric acid, four room-air blank). The use of the factor hemisphere, representing a two level factor of all right- or left-hemisphere locations, has been recommended in laterality studies (Pivik *et al.*, 1993). In order to reveal baseline differences, testing of pre-stimulus EEG was done with the same statistical model.

For heart rate and skin conductance, difference scores were calculated by subtracting an 8-s mean before the stimulus release from an 8-s mean after stimulation. As a skin conductance response is expected within 5 s after the onset of a stimulus, an increase in skin conductance represents a skin conductance response. Respiration cycle was assessed by counting zero crossings and analysed using difference scores between 30 s before and 30 s after stimulus onset. Data for heart rate, skin conductance and respiration

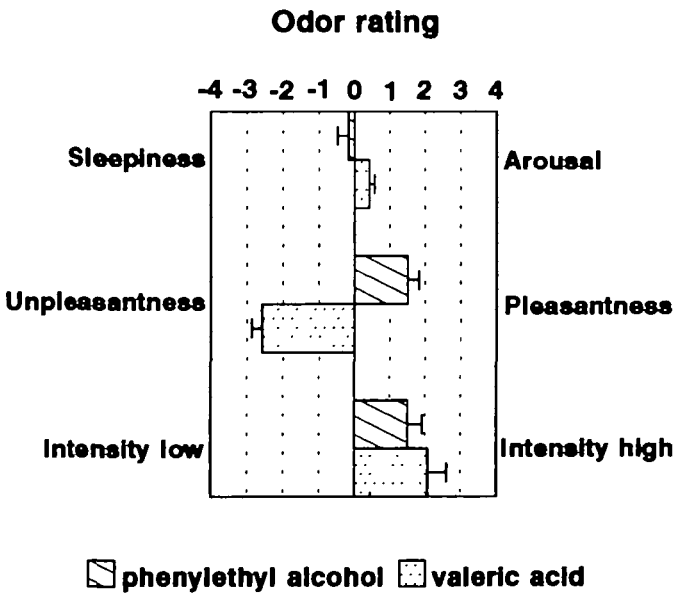

Figure 3 Mean (and SEM) of subjective ratings of phenylethyl alcohol and valeric acid.

cycle were averaged within a session across all trials of one condition. For autonomic data and respiration cycle, ANOVAs with the repeated measures factor condition and the between factor session were performed.

For all significance levels of the analysis of variance, Greenhouse–Geisser probabilities were considered where appropriate (Vasey and Thayer, 1987). If *P*-values reached the 0.05 level, the result was considered as significant, while the 0.10 level was taken to indicate a marginal effect. Planned *post-hoc* analysis was done by comparing two conditions.

Although all subjects were able to discriminate the two odors in the training session with respect to hedonic quality, one subject failed in the experimental sessions. Therefore, his data were excluded from further analysis.

Results

Odor rating

Figure 3 shows mean ratings and SEM (standard error of the mean) of the two odors. Ratings differed with respect to pleasantness (Wilcoxon, $P = 0.002$) and to some extent in the odor intensity dimension ($P = 0.043$). Valeric acid was always rated as unpleasant (≤ -1), whereas phenylethyl alcohol was rated as pleasant (≥ 1), with only one exception.

EEG data

Analysis of pre-stimulus EEG revealed no significant effects for the factor condition and no interaction with the factor condition in any frequency band (all Ps > 0.16). For the difference scores (pre-post stimulus release) in the theta and beta1 band, no factor or interaction was significant (all Ps > 0.12). For alpha1, the interaction condition × session reached marginal significance [$F(2,18) = 3.07$, $P = 0.050$], indicating an unstable activity in this band across sessions. As no further factor or interaction became significant (all Ps > 0.22), no additional analysis was performed.

Difference scores of alpha2 power are depicted in Figure 4. Conditions differed significantly in the alpha2 band [$F(2,18) = 6.95$, $P = 0.007$]. Additionally, a marginal interaction between condition and location was observed [$F(6,54) = 2.92$, $P = 0.052$]. *Post-hoc* analysis revealed an increase of alpha2 power during valeric acid as compared with the room-air blank [$F(1,9) = 12.65$, $P = 0.006$], as well as marginally more alpha2 in comparison with phenylethyl alcohol [$F(1,9) = 4.38$, $P = 0.065$]. When comparing valeric acid with the room-air blank, there was a significant interaction between condition and location [$F(3,27) = 5.07$, $P = 0.016$]. Table 2 summarises the analysis of variance for the alpha2 band.

In order to reveal inter-individual variability, change scores pooled over all trials with valeric acid for each subject are shown in Table 3. Change scores are difference scores during valeric acid minus difference scores during the room-air blank. An increase of alpha2 power can be seen in all subjects at frontal and parietal locations. At occipital electrodes alpha2 power increased in all but one subject. Change scores were inconsistent at central locations.

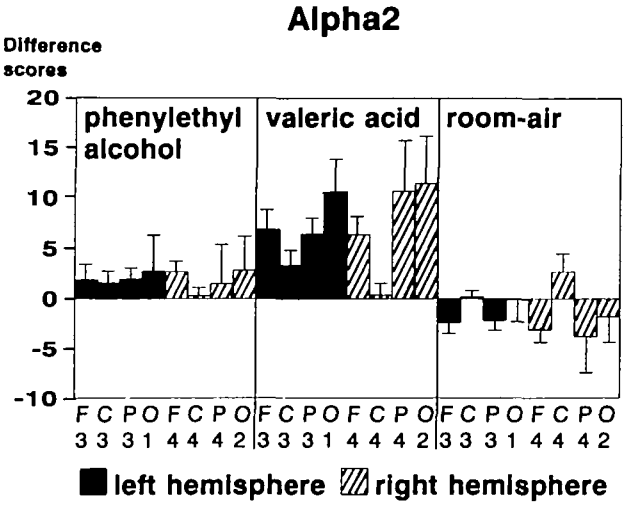

Figure 4 Difference scores (and SEM) of alpha2 power for the three conditions. Unit is microvolt squared.

Table 2 Summary of the analysis of variance for the alpha2 band (Greenhouse–Geisser corrected)

Factors and interactions	df	*F*-value	Probability
Condition	2	6.95	0.007
Session	2	0.41	0.68
Hemisphere	1	0	1
Location	3	1.74	0.18
Condition × session	4	0.47	0.76
Condition × hemisphere	2	0.17	0.85
Condition × location	6	2.92	0.052
Condition × session × hemisphere	4	0.25	0.85
Condition × session × location	12	0.30	0.98
Condition × hemisphere × location	6	1.19	0.32
Condition × session × hemisphere × location	12	0.40	0.96

Respiration

Neither pre-stimulus values ($P > 0.9$) nor difference scores in breathing cycle differed between conditions ($P > 0.6$, data not shown).

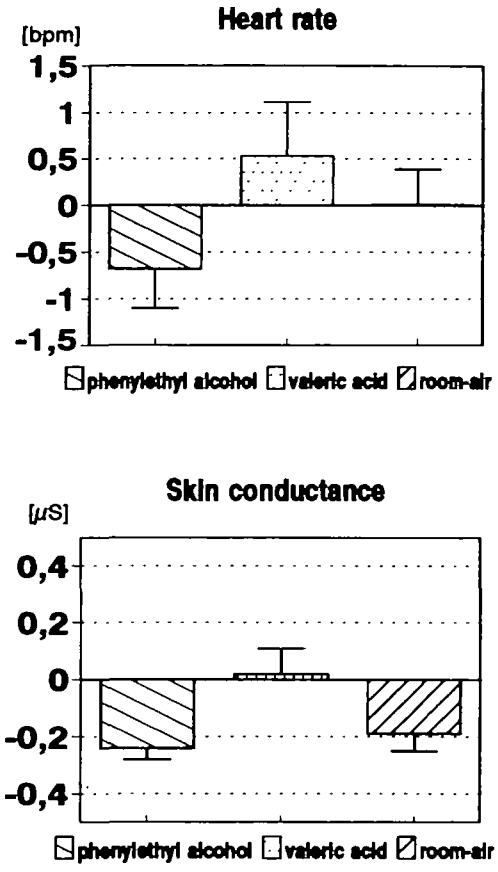

Figure 5 Difference scores (and SEM) of heart rate and skin conductance for the three conditions.

Autonomic variables

Pre-stimulus values of heart rate and skin conductance did not differ between conditions ($P > 0.5$). Mean difference scores for heart rate and skin conductance are presented in Figure 5. The factor condition failed to reach significance ($P = 0.15$), although a small increase of heart rate was observed during valeric acid and a decrease during phenylethyl alcohol. Heart rate differed marginally between sessions [$F(2,9) = 3.67$, $P = 0.068$]. During the first session, valeric acid induced the highest increase as compared with the other sessions. On the other hand, phenylethyl alcohol showed the least decrease during the first session as compared with the following sessions.

Skin conductance differed marginally between conditions [$F(2,18) = 3.25$, $P = 0.089$]. *Post-hoc* comparison revealed that skin conductance was marginally higher during valeric acid as compared with phenylethyl alcohol [$F(1,9) = 4.55$, $P = 0.062$]. In order to clarify these changes in skin conductance, the skin conductance responses occurring within 8 s after stimulus onset were counted. A response was present in 55.6% of all trials with valeric acid, in 36.1% with phenylethyl alcohol and in 29.1% with the room-air blank.

Post-hoc analysis

Data of the present study suggest an association of skin conductance response with an increase of alpha power. In order to clarify this assumption, we conducted a *post-hoc* analysis, comparing difference scores of alpha2 power of those trials of valeric acid producing a skin conductance response ($n = 40$) with those which did not ($n = 32$). The

Table 3 Change scores, pooled over all trials with valeric acid for each subject Change scores are difference scores obtained during administration of valeric acid minus difference scores during presentation of the room-air blank. Unit is microvolt squared

Subject	Hemisphere	Location			
		Frontal	Central	Parietal	Occipital
Subject 1	Right	6.8	−13.2	4.3	−6.2
	Left	4.6	4.6	6.8	−11.8
Subject 2	Right	6.4	−0.2	20.6	15.0
	Left	8.3	3.3	12.4	15.7
Subject 3	Right	15.6	2.5	2.3	14.3
	Left	11.9	−3.5	9.8	20.4
Subject 4	Right	9.0	1.7	30.6	33.1
	Left	12.1	8.2	5.5	18.2

Mann–Whitney U test revealed no difference in alpha2 band at any of the eight electrodes (all $Ps > 0.22$).

Discussion

The present study addressed the question whether there is a consistent response in electrocortical activity due to olfactory stimulation. Repeated presentation of valeric acid, an odor rated as unpleasant, increased overall alpha2 power within 8 s after stimuli release. However, inspection of individual data revealed that this increase was consistent across subjects only at frontal and parietal locations. Thus, there seems to be a consistent response in the alpha2 band due to stimulation with an unpleasant odor. Moreover, this increase of alpha2 power was similar in three sessions, each 1 week apart, and thus represents no casual effect. These findings are in agreement with data reported by Klemm *et al.* (1992), Sawada *et al.* (1992) and Van Toller *et al.* (1993) that olfactory stimulation decreases cortical activity. However, data obtained in the present study are not consistent with the first hypothesis predicting lower alpha power. Because there is clear evidence that alpha rhythm is suppressed during visual (Lehmann, 1971; Michel *et al.*, 1994) as well as during acoustic stimulation (Kaufman *et al.*, 1992), one might have expected less alpha power during suprathreshold olfactory stimulation. Therefore, it is concluded that repetitive olfactory input is processed with less cortical activity than are visual and acoustic stimuli. This interpretation goes along with anatomical findings that olfactory processing occupies large subcortical areas (Greer, 1991).

There are other interpretations. Some odors are known for sedative effects, which may increase alpha rhythm (Sawada *et al.*, 1992). However, it remains unclear why valeric acid, an odor rated as unpleasant, should have the highest relaxing effects. Therefore, data obtained in the present study question the interpretation of more alpha activity in terms of relaxation. On the other hand, an increase of alpha power upon olfactory stimulation could be interpreted as a cortical activation if subjects were drowsy before stimulation. Because theta power did not change, there is no support for this interpretation.

The second prediction of a lateralised processing of odorous stimuli was not supported. Because the factor hemisphere did not reach significance in the alpha bands for any odor, no difference of hemispheric activation could be found. Thus, no electrocortical activity commonly seen during emotional experience could be found. Although odors are well known for their ability to induce emotions (Ehrlichman and Bastone, 1993a; Miltner *et al.*, 1994), it seems that repetitive olfactory stimulation might be subject to habituation, thus inducing no emotional experience.

Change of alpha2 power differed between odors. Stimulation with valeric acid increased alpha2 power more in comparison with phenylethyl alcohol. Therefore, data of the present study suggest that stimulation with an unpleasant odor might lead to a stronger cortical deactivation than stimulation with a pleasant odor. On the other hand, it may well be that the small sample size and the resulting low statistical power are responsible for the fact that phenylethyl alcohol did not differ from the room-air blank. Data obtained in the present study go further along with previous reports that positively valenced stimuli often affect physiological variables only marginally, while the effects of negatively valenced stimuli are more distinct (Davidson *et al.*, 1990; Hubert and De Jong-Meyer, 1991; Ehrlichman and Bastone, 1992a; Miltner *et al.*, 1994).

Valeric acid showed a skin conductance response in 56% of all trials and hence a slight increase of skin conductance. When interpreting the change of skin conductance, one has to take into account that there was an increase in skin conductance during the break between two trials and therefore a decrease during any trial. These findings reveal an autonomic arousal during the presentation of valeric acid, although heart rate increased insignificantly. On the other hand, skin conductance and heart rate decreased during phenylethyl alcohol. Therefore, the third prediction, of higher autonomic arousal during presentation of valeric acid as compared with phenylethyl alcohol, is supported.

Valeric acid increased autonomic arousal but decreased cortical activation. No differential effects on alpha2 power were found when comparing the trials having a skin conductance response with those having none. Therefore, cortical deactivation and autonomic arousal in response to valeric acid seem to be dissociated processes.

One limitation that should be taken into account when interpreting the present results is that valeric acid can stimulate intranasal trigeminal structures whereas phenylethyl alcohol is not believed to do so (Doty *et al.*, 1978). Therefore, effects seen in the present study may be related to differences in sensory perception. However, as pointed out by Doty *et al.* (1978), there are few, if any, olfactory compounds which do not stimulate trigeminal structures. Therefore, this study clearly revealed that an odorous stimulus, rated as unpleasant, can increase alpha2 power.

ACKNOWLEDGEMENTS

The authors would like to thank Dr Peter M. Müller and Dr Norbert Neuner-Jehle, Givaudan-Roure Research Ltd, for delivering the olfactometer and the odorants as well as for their critique on an early version of the manuscript. Further thanks go to Mrs Adriana Tobler for her support during the investigation, and to Mrs Bonnie Strehler and Mrs Denise Surina for editorial assistance.

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Received on July 14, 1994; accepted on May 19, 1995