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## Sedative effects of the jasmine tea odor and (*R*)-(-)-linalool, one of its major odor components, on autonomic nerve activity and mood states

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**Abstract** We investigated the effects of the odor of jasmine tea on autonomic nerve activity and mood states in a total of 24 healthy volunteers. We used the odor of jasmine tea at the lowest concentration that could be detected by each subject but that did not elicit any psychological effects. R–R intervals and the POMS test were measured before and after inhalation of the odors for 5 min. Both jasmine tea and lavender odors at perceived similar intensity caused significant decreases in heart rate and significant increases in spectral integrated values at high-frequency component in comparison with the control ( $P < 0.05$ ). In the POMS tests, these odors produced calm and vigorous mood states. We also examined the effects of (*R*)-(-)-linalool, one of its major odor components, at the same concentration as in the tea, and (*S*)-(+)-linalool. Only (*R*)-(-)-linalool elicited a significant decrease in heart rate ( $P < 0.05$ ) and an increase in high-frequency component in comparison with the controls, and produced calm and vigorous mood states. Thus, the low intensity of jasmine tea odor has sedative effects on both autonomic nerve activity and mood states, and (*R*)-(-)-linalool, one of its components, can mimic these effects.

**Keywords** Jasmine tea · (*R*)-(-)-linalool · Odor · Autonomic nervous activity · Mood states

### Introduction

Tea is one of the most popular beverages, and it is consumed in various ways in more than 300 forms. The pharmacological and therapeutic effects of teas have been reported since the 1970s (Chen 1992). It has been observed that tea extracts have anti-carcinogenic (Wang et al. 1988), antimutagenic (Kada et al. 1985), antioxidative (Ho et al. 1992; Yoshino et al. 1994) and hypocholesterolemic effects (Yang and Koo 1997). Jasmine tea, one of the most popular forms drunk in China, has been reported to share some of these effects (Yang and Koo 1997; Zhang et al. 1997). People not only consume jasmine tea but also enjoy its characteristic odor.

Some of the odors of essential oils are used in the treatment of depression, anxiety and some types of cognitive disorders in aromatherapy (Buchbauer and Jirovetz 1994; Buchbauer 1996). Moreover, some odors produce physiological changes in parameters such as blood pressure (Nagai et al. 2000; Suzuki and Aoki 1994), muscle tension (Schwartz 1979), blink magnitude (Ehrlichman et al. 1997), skin temperature, skin blood flow, electrodermal activity, heart rate (HR; Alaoui-Ismaili et al. 1997; Brauchli et al. 1995), brain wave pattern (Lorig 1989; Torii et al. 1988; Van Toller et al. 1993) and sleep time (Tsuchiya et al. 1992). The effects of odors on autonomic functions and mood states appear to have two mechanisms. One is pharmacological via direct interactions between odor molecules and receptors or nerve endings, and the other is psychological via the subjective effects of odor perception (Heuberger et al. 2001; Jellinek 1997).

In a previous study, we investigated the effects of the odor of jasmine tea on autonomic nerve activity at high and low concentrations, rated subjectively as high and low intensity (Inoue et al. 2003). Inhalation of this odor at low

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intensity decreased HR and increased parasympathetic nerve activity, without any influence of psychological effects due to subjective preference for or prior experience of this odor. However, we observed changes in autonomic nerve activity influenced by psychological effects, due to subjective preference for the high-intensity odor. We hypothesized that the effects of the low intensity of jasmine tea odor might be pharmacological.

In the present study, we conducted two experiments. First, we investigated the possibility of sedative effects of the odor of jasmine tea at low intensity in comparison with the odor of lavender because this has been shown pharmacologically to have sedative effects in animals and humans (Buchbauer et al. 1991; Hardy et al. 1995; Lis-Balchin and Hart 1997a) and it includes a component, (*R*)-(-)-linalool, also found in jasmine tea (Ito et al. 2002). Second, we investigated whether these effects were caused directly by (*R*)-(-)-linalool, and whether its optical isomer (*S*)-(+)-linalool has the same effects because differing pharmacological effects have been reported for the optical isomers of volatile components (Jager et al. 2000, 2001; Laska et al. 1999).

## Materials and methods

### Subjects

#### Experiment 1

Data were collected from six male and six female healthy, normotensive Japanese volunteers, aged 21–36 years (mean 26.1 years). Their mean body mass index (BMI) was 21.26 kg/m<sup>2</sup>. Informed consent was obtained from all subjects according to the guidelines established by the declaration of Helsinki.

#### Experiment 2

Data were collected from six male and six female nonsmoking volunteers, aged 21–36 years (mean 25.35 years). Their mean BMI was 21.37 kg/m<sup>2</sup>.

### Odorants

#### Experiment 1

Jasmine tea and essential oil of lavender were generously donated by Itoen Ltd. (Shizuoka, Japan) and Taiyo Co. (Osaka, Japan), respectively. Jasmine tea produced at Fujian province of China in September 1999 was used. It was prepared from pan-fired and basket-dried Chinese green tea (*Camellia sinensis* L.) scented with flowers of *Jasminum sambac*. After the scenting process, the flowers were removed from the tea, and dried flowers of *J. sambac* were newly added to about 1% of the weight of tea. We already reported the volatile components and

the enantiomeric ratio of linalool in the jasmine tea; 82.6% ee for the (*R*)-(-)-isomer (Ito et al. 2002). The composition of the volatile compounds and the enantiomeric purity of linalool in the lavender oil were analyzed by GC and GC–MS in the same way as the jasmine tea. Linalool (40.9%) and linalyl acetate (33.9%) were quantitatively detected as the main compounds and the enantiomeric ratio of linalool was almost optically active for the (*R*)-(-)-isomer (88.0% ee). Jasmine tea extract was prepared as for human consumption, by soaking 25 g of leaves in 1 l of boiling water for 1 min, then diluting it 20-fold using distilled water. Each subject could notice the odor at this concentration, but our previous observations showed that this was too weak to elicit significant psychological effects due to subjective preference for the odor (Inoue et al. 2003). The subjects received each odor.

To determine the concentration of lavender oil perceived by the subjects to be the same intensity as the odor of jasmine tea, a preliminary rating test was performed using lavender oil stirred sufficiently in distilled water at concentrations of 0.5, 1, 2.5, 5 and 10 µl/l. As each of the subjects rated a concentration of 1 µl/l of essential oil to be the same intensity as the odor of jasmine tea, this concentration was used in the subsequent study. The lavender oil in distilled water was used stirring sufficiently just before the experiment.

#### Experiment 2

(*R*)-(-)-Linalool, (*S*)-(+)-linalool and propylene glycol were generously donated by T. Hasegawa Co. Ltd. (Tokyo, Japan) and Wako Pure Chemical Industries Ltd. (Tokyo, Japan), respectively. Two-microliter aliquots (0.86 g/ml) of each were dissolved in propylene glycol, and then diluted with 1 l distilled water. To use the linalool at the concentration of 0.03 ppm, we used 20 µl of this diluted solution in 1 l distilled water for each subject. Analysis of odorants, by GC and GC–MS, in jasmine tea extracted from 25 g of leaves with 1 l of boiling water for 1 min showed that (*R*)-(-)-linalool was one of the main odor components present at a concentration of 530.6 ± 73.2 ppb (Ito et al. 2002). We therefore used 0.03 ppm as a comparison with 20-fold diluted jasmine tea in experiment 1. When the subjects inhaled these odors at the experiment, nobody detected the odors at this concentration by the questionnaire.

### Odor delivery

Odor was administered by inhalation via an odor delivery system developed by our group. The system consisted of a 2 l airtight bottle, an air pump, and a stainless tube (diameter 5 mm) fitted with a perforated funnel (diameter 80 mm). The bottle was filled with the 1 l diluted solution for each subject. Airflow from the bottle, produced by bubbling with the air pump, was set at 1.5 l/min and was led through the stainless tube to

near the subject's nostrils. The perforated funnel served to reduce airflow stress for the subject.

### Recording and analysis of autonomic parameters

To evaluate autonomic nervous system (ANS) activity, we used power spectral analysis on R–R intervals (the temporal durations between each heart beat). The details of this procedure have been fully described elsewhere (Moritani et al. 1993, 1995). Briefly, electrocardiographic (ECG) signals were obtained from electrodes placed in the CM5 position. The signal was digitized on-line by a 13-bit analog-to-digital converter (PS-2032GP, TEAC, Japan) at a sampling rate of 1,024 Hz. The digitized ECG signal was differentiated, and the resultant QRS spikes and the intervals of the impulses (R–R intervals) were stored sequentially on a hard disk for later analysis. The stored R–R interval data were displayed and aligned sequentially to obtain equally spaced samples with an effective sampling frequency of 2 Hz. The data were passed through a Hamming-type data window, then power spectral analysis by means of fast Fourier transform was performed on a consecutive 256 s time series of R–R interval data obtained during the test. To evaluate ANS activities in each subject in the present study, we analyzed low-frequency component (0.035–0.15 Hz, LFC) and high-frequency component (0.15–0.5 Hz, HFC) by integrating the spectrum for the respective bandwidths. In general, HFC are associated with almost entirely vagal nerve activity and LFC might be mediated by both vagal and sympathetic nerve activity.

As basal ANS activities and HRs differ between individuals, the mean values for HR before inhalation were set as the baseline values and the mean values for autonomic nervous activity before inhalation were standardized to 100%, and the relative values after the inhalation of odors were compared.

### Evaluation of mood states

We performed a Japanese version of the POMS test (Yokoyama and Araki 1994) just before and after the measurement of R–R intervals. The POMS test consists of a 65-item self-rating scale (including seven dummy items) that measures present mood states. Each item is rated on a scale of 0–4, ranging from “not at all” to “extremely”. These raw scores were summed to generate six measures of emotional state: tension and anxiety (T–A), depression and dejection (D), anger and hostility (A–H), vigor (V), fatigue (F) and confusion (C). To compare the effect of each odor on mood states, we analyzed changes in the subjects' scores before and after the measurement of R–R intervals.

### Procedure

Measurements were performed in a quiet room with constant humidity (ca 50%) and temperature

( $22 \pm 0.5^\circ\text{C}$ ). Each subject was instructed to have breakfast before 0900 hours on the day of the experiment and to abstain from food, drink and exercise until finishing the experiment. To avoid the influences of circadian rhythm, each measurement was taken between 1030 and 1200 hours.

Before measurements were taken, the subjects were instructed to rest for at least 15 min in a sitting position in a quiet and relaxed manner while equipped with ECG electrodes, and then they performed the POMS test. The ECG was recorded for 18, 6 and 48 min before, during and after inhalation, respectively. After the ECG had been recorded, subjects performed the POMS test again. During ECG recording, subjects were instructed to breathe at a frequency of one breath every 4 s (0.25 Hz) in synchrony with the sound of an electric metronome because respiration greatly influences HR variability (Brown 1993; Novak et al. 1993). In each test carried out on different days, each subject was presented either jasmine tea or lavender in experiment 1 and either (R)-(-)-linalool or (S)-(+)-linalool in experiment 2 at random, with water alone as a control.

### Statistical analysis

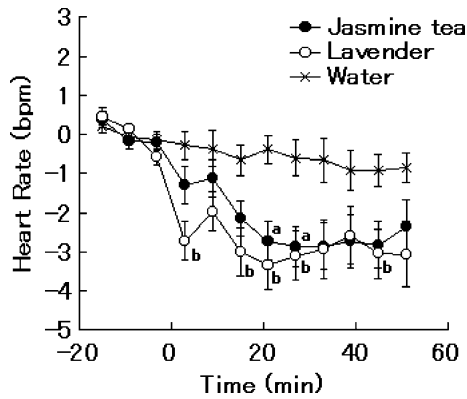
In power spectral analysis, the data were expressed as means  $\pm$  SE. The effects of time, treatment and time  $\times$  treatment were evaluated by two-way repeated measures ANOVA. Furthermore, comparisons of time, treatment and time  $\times$  treatment between the two groups were used in a contrast test. To compare each group at certain times, one-way ANOVA and post-hoc Tukey test was used. For the POMS test, the values were expressed as means  $\pm$  SE and were analyzed by one-way ANOVA followed by a post-hoc Tukey test. Statistics were calculated using the StatView software package (Windows Version J 5.0, Abacus Concepts, Berkeley, CA) and the super ANOVA software package (Macintosh version 1.11, Abacus Concepts). Probability levels of  $<0.05$  were considered as significant.

## Results

We compared males with females in experiments 1 and 2. However, the autonomic nervous response and mood states did not differ statistically between males and females, and the gender effect was not observed. The mixed result was mentioned as follows.

### Experiment 1

As shown in Fig. 1, significant differences in time course of changes in HR were observed between each odor, and odors of both jasmine tea and lavender significantly

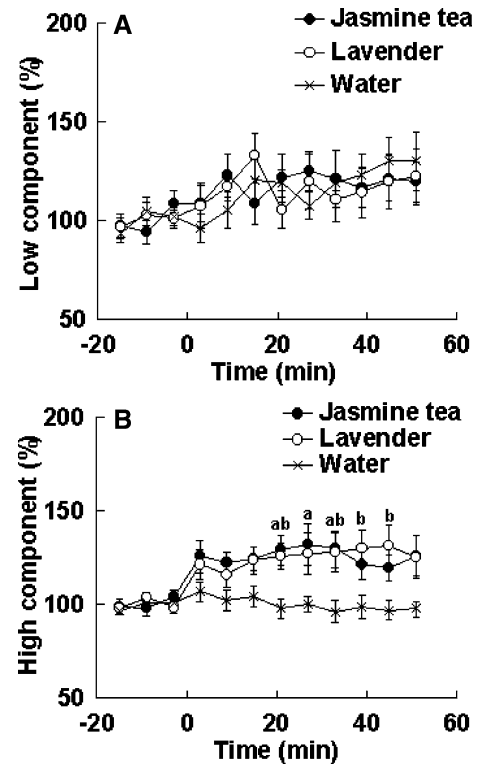


**Fig. 1** Effects of inhalation of jasmine tea odor and lavender on HR in humans. Subjects inhaled the odors at a low concentration subjectively rated as being the same intensity. The mean values of basal HR were standardized as 0. Values were expressed as mean differences from the baseline  $\pm$  SE ( $n = 10$ ). Each time plotted on the horizontal axis was defined as the midpoint of each R-R interval measurement. The decreases with time were significantly greater when subjects inhaled odors of jasmine tea or lavender than when they inhaled the control, water ( $P < 0.05$  for time  $\times$  treatment effect by two-way repeated measures ANOVA and contrast tests). A significant difference was apparent ( $P < 0.05$ : <sup>a</sup>jasmine tea vs water, <sup>b</sup>lavender vs water by ANOVA and the post-hoc Tukey test)

decreased HR for more than 40 min after inhalation in comparison with the water control [time  $\times$  treatment effect:  $F(2,39) = 2.36$ ,  $P < 0.05$ ]. The decrease of HR was significantly lower at 21 and 27 min after inhalation of jasmine tea odor compared with water [ $F(2,39) = 8.12$  and  $5.71$ , respectively,  $P < 0.05$ ] and significantly lower at 3, 15, 21, 27 and 45 min after inhalation of lavender odor compared with water [ $F(2,39) = 6.86$ ,  $5.25$ ,  $8.12$ ,  $5.71$ ,  $3.75$ , respectively,  $P < 0.05$ ].

The time course of the LFC is shown in Fig. 2a. No time effect was found for either jasmine tea or lavender. We observed significant increases in the HFC (Fig. 2b) after inhalation of odor of jasmine tea or lavender in comparison with water [time  $\times$  treatment effect:  $F(2,39) = 1.67$ ,  $P < 0.05$ ]. The increased HFC by jasmine tea odor were significantly higher at 21, 27 and 33 min than water [ $F(2,39) = 8.58$ ,  $3.63$ ,  $4.68$ , respectively,  $P < 0.05$ ]. The increased HFC by lavender odor were significantly higher at 21, 33, 39 and 45 min than water [ $F(2,39) = 8.58$ ,  $4.68$ ,  $4.14$ ,  $4.47$ , respectively,  $P < 0.05$ ].

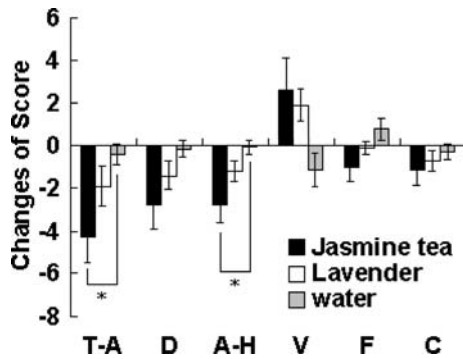
Figure 3 shows changes in scores from preinhalation to postinhalation for six subscales of the POMS test. We observed that the odors of jasmine tea significantly decreased T-A and A-H, the negative mood scores, in comparison with water [ $F(2,39) = 4.37$  and  $4.42$ , respectively,  $P < 0.05$ ]. Other negative mood score, D, was also decreased by jasmine tea odor although it was not significantly different, and F and C were unchanged. Similarly, lavender odor tended to decrease these negative mood score, T-A, D and A-H. In contrast, the odors of both jasmine tea and lavender increased the positive mood score, V, although the effect was not statistically significant.



**Fig. 2** Effects of inhalation of jasmine tea odor and lavender on the power spectra of R-R intervals in low-frequency component (LFC, A) and high-frequency component (HFC, B) frequency regions in humans. Subjects inhaled odors at a low concentration subjectively rated as the same intensity. The mean values of basal spectral integrated values were standardized as 100%. Values were expressed as mean percentages of baseline  $\pm$  SE ( $n = 10$ ). Each time plotted on the horizontal axis was defined as the midpoint of each R-R interval measurement. For HFC, the increases with time were significantly greater when subjects inhaled the odors of jasmine tea or lavender than when they inhaled the control, water ( $P < 0.05$  for time  $\times$  treatment effect by two-way repeated measures ANOVA and contrast test). A significant difference was apparent ( $P < 0.05$ : <sup>a</sup>jasmine tea vs water, <sup>b</sup>lavender vs water by ANOVA and the post-hoc Tukey test)

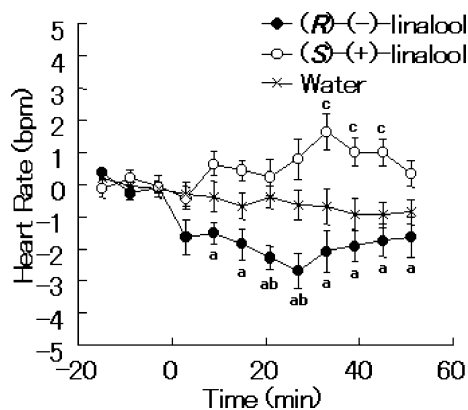
## Experiment 2

As shown in Fig. 4, (R)-(-)-linalool significantly decreased HR for more than 40 min after inhalation in comparison with water and (S)-(+)-linalool [time  $\times$  treatment effect:  $F(2,38) = 3.67$ ,  $P < 0.01$ ]. On the other hand, HR was significantly increased when subjects inhaled (S)-(+)-linalool compared with water [time  $\times$  treatment effect:  $F(2,38) = 3.67$ ,  $P < 0.05$ ]. The decrease of HR was significantly lower at 21 and 27 min after inhalation of (R)-(-)-linalool compared with (S)-(+)-linalool or water [ $F(2,38) = 11.04$  and  $11.04$ , respectively,  $P < 0.05$ ] and at 9, 15, 33, 39, 45 and 51 min compared with (S)-(+)-linalool [ $F(2,38) = 5.03$ ,  $7.37$ ,  $9.65$ ,  $8.81$ ,  $8.52$  and  $3.69$ , respectively,  $P < 0.05$ ]. The increase of HR was significantly higher at 33, 39 and 45 min after inhalation of (S)-(+)-linalool compared with water [ $F(2,38) = 9.65$ ,  $8.81$  and  $8.52$ , respectively,  $P < 0.05$ ].



**Fig. 3** Effects of inhalation of odors of jasmine tea and lavender on POMS tests performed before and after inhalations. Subjects inhaled the odors at a low concentration subjectively rated as the same intensity. Values represented the differences between the postinhalation scores and preinhalation scores for the six subscales of the POMS test: T-A (tension and anxiety), D (depression and dejection), A-H (anger and hostility), V (vigor), F (fatigue), and C (confusion). Values were expressed as means  $\pm$  SE ( $n = 10$ ). For T-A and A-H, the decrease was significantly greater when subjects inhaled odor of jasmine tea than when they inhaled the control, water (\*  $P < 0.05$  by one-way ANOVA and post-hoc Tukey test)

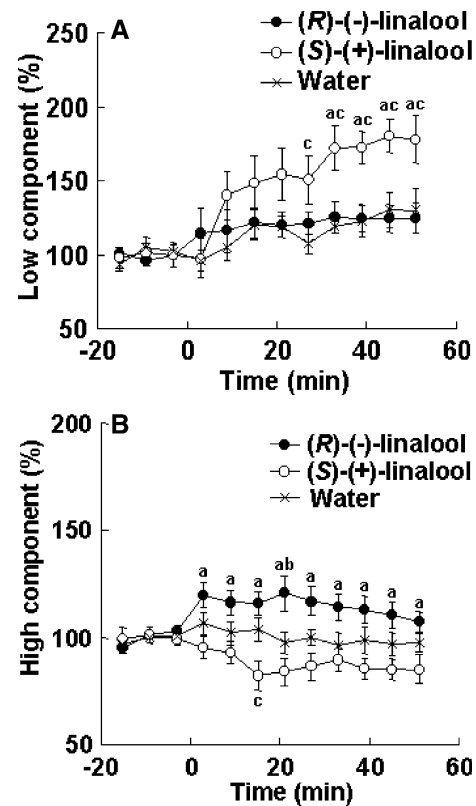
The time course of LFC is shown in Fig. 5a. No effect of (*R*)-(-)-linalool was found. On the other hand, a significant increase in LFC was observed after inhalation of (*S*)-(+)-linalool (time  $\times$  treatment effect:  $F(2,38) = 2.17$ ,  $P < 0.01$ ) in comparison with water or (*R*)-(-)-linalool. The increase of LFC by (*S*)-(+)-linalool was significantly higher at 27 min than water [ $F(2,38) = 4.44$ ,  $P < 0.05$ ] and at 33, 39, 45 and 51 min



**Fig. 4** Effects of inhalation of (*R*)-(-)-linalool and (*S*)-(+)-linalool on HR in humans. Subjects inhaled the odors at the same concentration of (*R*)-(-)-linalool found in the low intensity of jasmine tea odor. The mean values of basal HR were standardized as 0. Values were expressed as mean differences from baseline  $\pm$  SE ( $n = 7$ ). Each time plotted on the horizontal axis was defined as the midpoint of each R-R interval measurement. The decreases with time were significantly greater when subjects inhaled (*R*)-(-)-linalool than when they inhaled water ( $P < 0.05$ , time  $\times$  treatment effect, by two-way repeated measures ANOVA and a contrast test) or (*S*)-(+)-linalool ( $P < 0.01$  for time  $\times$  treatment effect by two-way repeated measures ANOVA and a contrast test). A significant difference was apparent ( $P < 0.05$ : <sup>a</sup>(*R*)-(-)-linalool vs (*S*)-(+)-linalool, <sup>b</sup>(*R*)-(-)-linalool vs water, <sup>c</sup>(*S*)-(+)-linalool vs water by ANOVA and the post-hoc Tukey test)

than water or (*R*)-(-)-linalool [ $F(2,38) = 7.21$ , 7.75, 8.35 and 4.86, respectively,  $P < 0.01$ ].

Increases and decreases in HFC were observed for more than 40 min after the inhalation of (*R*)-(-)-linalool and (*S*)-(+)-linalool, respectively, and a significant difference was observed between both effects [time  $\times$  treatment effect:  $F(2,38) = 2.21$ ,  $P < 0.01$ ; Fig. 5b]. The significant differences were observed at every time between (*R*)-(-)-linalool and (*S*)-(+)-linalool [ $F(2,38) = 4.90$ , 4.71, 8.86, 7.38, 7.11, 4.76, 5.09, 5.16 and 5.11, respectively,  $P < 0.05$ ], at 21 min between (*R*)-(-)-linalool and water [ $F(2,38) = 7.38$ ,  $P < 0.05$ ] and at 15 min between (*S*)-(+)-linalool and water [ $F(2,38) = 8.86$ ,  $P < 0.05$ ].



**Fig. 5** Effects of inhalation of (*R*)-(-)-linalool and (*S*)-(+)-linalool on the power spectra of R-R intervals in low-frequency component (LFC, A) and high-frequency component (HFC, B) frequency regions in humans. Subjects inhaled the odors at the same concentration of (*R*)-(-)-linalool included in the low-intensity odor of jasmine tea used. The mean values of basal spectral integrated values were standardized as 100%. Each time plotted on the horizontal axis was defined as the midpoint of each R-R interval measurement. Values were expressed as mean percentages of baseline  $\pm$  SE ( $n = 7$ ). **a** For the LFC, the increase with time was significantly greater when subjects inhaled (*S*)-(+)-linalool than when they inhaled water or (*R*)-(-)-linalool ( $P < 0.01$  for time  $\times$  treatment effect by two-way repeated measures ANOVA and a contrast test). **b** For the HFC, the decrease with time was greater when they inhaled (*R*)-(-)-linalool than when they inhaled (*S*)-(+)-linalool ( $P < 0.01$  for time  $\times$  treatment effect by two-way repeated measures ANOVA and a contrast test). A significant difference was apparent ( $P < 0.05$ : <sup>a</sup>(*R*)-(-)-linalool vs (*S*)-(+)-linalool, <sup>b</sup>(*R*)-(-)-linalool vs water, <sup>c</sup>(*S*)-(+)-linalool vs water by ANOVA and the post-hoc Tukey test)

Inhaling (*R*)-(-)-linalool decreased the negative mood scores T-A, D and A-H (Fig. 6), whereas (*S*)-(+)-linalool increased them and tended to decrease the positive mood score V. Furthermore, significant differences were observed between (*R*)-(-)-linalool and (*S*)-(+)-linalool in producing the mood scores T-A, D and A-H [ $F(2,38)=3.61, 4.12$  and  $5.41$ , respectively,  $P < 0.05$ ]. The other negative mood scores, F and C, were unchanged.

## Discussion

We investigated the sedative effects on both autonomic nerve activity and mood states of the odor of jasmine tea in comparison with lavender at a low intensity that did not elicit any psychological effects due to subjective preference for the odor in a previous study (Inoue et al. 2003). We also studied whether these effects were caused by linalool, one of the main odor components of jasmine tea, and whether these effects depended on the enantiomeric structure of linalool.

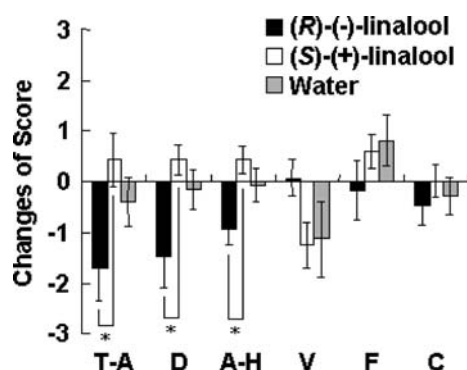
After inhalation of the odors, both jasmine tea and lavender at subjectively perceived equivalent concentrations decreased HRs and increased parasympathetic nerve activities for 40 min, but had no effect on sympathetic nerve activity. These effects reflect the sedative effects on autonomic nerve activities, and our results agree with a previous report by Saeki (2000), which showed that inhalation of odor of lavender using a foot bath increased parasympathetic nerve activities in subjects, suggesting that inhalation of its odor elicits sedative or relaxant effects. In the present study, inhalation of (*R*)-(-)-linalool, one of the main odor components of

the odor of jasmine tea, also decreased HR and increased parasympathetic nerve activity for 40 min. Moreover, these effects by the odor of (*R*)-(-)-linalool mimicked the effects by the odors of jasmine tea and lavender. In contrast, inhalation of (*S*)-(+)-linalool at the same concentration elicited significant increases in HR and sympathetic nerve activity. These suggests that the odor of jasmine tea has lavender-like sedative effects and these effects may be partly caused by (*R*)-(-)-linalool, the common main odor component of both jasmine tea and lavender because it mimics these effects on autonomic nerve activity. Furthermore, we showed that the enantiomeric structure of linalool influences these sedative effects.

Our results on mood states had the same tendency as the results of inhalation of each odor on autonomic nerve activities. Inhalation of the odors of jasmine tea or lavender decreased several negative mood scales, tension-anxiety, depression-dejection and anger-hostility, and increased the positive mood scale, vigor. Moreover, (*R*)-(-)-linalool showed similar effects. In contrast, (*S*)-(+)-linalool increased the negative mood scales and decreased the positive mood scale. Thus, the odors of jasmine tea and lavender both appear to produce calm and vigorous mood states, and (*R*)-(-)-linalool, but not (*S*)-(+)-linalool, may partly contribute to these changes.

Two possible mechanisms might contribute to the effects of odors on autonomic function and mood states. One is pharmacological, via direct interactions between odor molecules and receptors or nerve endings, and the other is psychological via the subjective experience of odors (Alaoui-Ismaili et al. 1997; Heuberger et al. 2001; Jellinek 1997). Our results suggest that the principal odor component of jasmine tea, especially, (*R*)-(-)-linalool, exerts a pharmacological sedative effect. However, a subjective psychological effect does not appear to contribute to this because the intensity of odors used in this study was too weak to induce subjective psychological effects due to subjective preference for the odor (Inoue et al. 2003). In addition, the different effects between optical isomers may support the hypothesis of a pharmacological mechanism because the concentrations of (*R*)-(-)-linalool and (*S*)-(+)-linalool used in the present study were too low to be perceived by the subjects.

Previous reports have suggested that both lavender and linalool have moderate sedative effects (Buchbauer et al. 1991, 1993). Hardy et al. (1995) showed that inhalation of the odor of lavender could replace hypnotic drugs in humans. Lis-Balchin and Hart (1997b) produced some evidence that spasmolysis or relaxation of smooth muscle of the ileum *in vitro* is correlated with this holistic relaxant effect in humans. They have also reported that the spasmolytic effect of lavender oil is most likely mediated via cAMP and not via cGMP, and they suggested that the mode of action of linalool reflects that of the whole oil (Lis-Balchin and Hart 1999). Furthermore, lavender and linalool have been reported to suppress caffeine-induced overarousal of the central



**Fig. 6** Effects of inhalation of (*R*)-(-)-linalool and (*S*)-(+)-linalool on POMS tests performed before and after inhalation. Subjects inhaled the odors at the same concentration of (*R*)-(-)-linalool included in the low-intensity odor of jasmine tea used. Values represented the differences between the postinhalation scores and preinhalation scores for the six subscales of the POMS test: T-A (tension and anxiety), D (depression and dejection), A-H (anger and hostility), V (vigor), F (fatigue) and C (confusion). Values were expressed as means  $\pm$  SE. ( $n = 7$ ). For T-A, D and A-H, the decrease was significantly greater when subjects inhaled (*R*)-(-)-linalool than when they inhaled (*S*)-(+)-linalool ( $^* P < 0.01$  by one-way ANOVA and post-hoc Tukey test)

nervous system (CNS; Buchbauer et al. 1991, 1993). Elisabetsky and Souza (1995) also showed that linalool reduces motor activities in mice and exhibits a dose-dependent binding to glutamate, a main excitatory neurotransmitter in the CNS. In addition, Buchbauer et al. (1993) showed that linalool can be detected in mice at a high concentration in blood samples from the retrobulbar venous plexus after inhalation. These reports suggest linalool might be absorbed by inhalation and consequently elicits sedative effects on the CNS. Therefore, the sedative effects on both autonomic nerve activities and mood states after inhalation of the odor of jasmine tea might be caused at least partly by these pharmacological effects of linalool.

In the ability of sensing odors, there was a gender difference. Doty et al. (2001) reported women have a better sense of smell than men and retain the ability to smell longer than men. However, when we compared male data with female data, the significant gender effect was not observed in our any data (data not shown). This might be attributable to two factors. One, the intensity of the odors used in this study might be too weak to demonstrate the gender effect. Indeed, the intensity of the odors used in this study was as weak as subjective psychological effects were not induced (Inoue et al. 2003), and both the intensity of (*R*)-(-)-linalool and (*S*)-(+)-linalool in this study were also as weak as the subjects did not detect. The other, the physiological effect of odors might be affected by the gender difference of the ability to sense odors. This might be true if the effect of the odors in this study is pharmacological, which is our hypothesis described above. In either of the cases, more research is required in order to identify the gender difference about the physiological effect of odors.

We also investigated the influence of chirality of linalool on these sedative effects. There are some reports on the influence of chirality on physiological and psychological changes. Kubota et al. (1992) showed distinct differences between different chiral isomers in their ability to influence the contingent negative variation (CNV). Heuberger et al. (2001) showed that (+)-limonene led to increases in both physiological and behavioral arousal, whereas administration of (-)-limonene only affected ANS parameters. In addition, (-)-carvone increases both physiological and behavioral arousal, whereas (+)-carvone only affects physiological arousal. They also suggested that the biological activity of odor molecules is influenced by their chirality. In the present study, we first showed that linalool caused different pharmacological effects, depending on its chirality, even at imperceptible concentrations. As (*R*)-(-)-linalool showed sedative effects and (*S*)-(+)-isomer elicited the opposite effects on both autonomic nerve activity and mood states, the sedative effects of the odors of jasmine tea and lavender were probably partly caused by (*R*)-(-)-linalool, which was present in both odors at levels of more than 90% by GC and GC-MS (Ito et al. 2002) compared with the (*S*)-(+)-isomer.

In conclusion, our findings showed that the odor of jasmine tea has lavender-like sedative effects on autonomic nerve activity and mood states at a very low intensity, and (*R*)-(-)-linalool, the main component of both jasmine tea and lavender, has the same effects. In contrast, (*S*)-(+)-linalool, an optical isomer of (*R*)-(-)-linalool, showed the opposite effects. We suggest that the sedative effects of the odor of jasmine tea are pharmacologically partly caused by (*R*)-(-)-linalool, and that the particular linalool enantiomer is important for them.

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