

# Acute Effects of Aroma Exposure and Breathing Patterns on Physiological Responses: A Controlled Experimental Study

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## Abstract

**Purpose:** Aromatherapy has been reported to improve mood, reduce stress, and stabilize autonomic nervous system (ANS) activity. However, it remains unclear whether these acute physiological effects are caused by aroma itself or by accompanying changes in breathing patterns. **Participants and Methods:** This study aimed to differentiate the acute effects of aroma and breathing patterns on cardiovascular and stress-related indices in healthy adults. Twenty participants completed a randomized crossover trial with four conditions: 1) aroma + normal breathing, 2) aroma + deep breathing, 3) no aroma + normal breathing, and 4) no aroma + deep breathing. Lavender essential oil (1%) was sprayed twice per minute for 5 min, while 10% ethanol served as the control. Breathing patterns were guided and monitored using a respiratory gas analyzer. Heart rate (HR), systolic (SBP) and diastolic (DBP) blood pressure, and salivary amylase (SA) were measured before and after each condition. **Results:** HR, SBP, and SA significantly decreased under deep-breathing conditions, independent of aroma exposure. No significant main effects of aroma or interaction effects were observed. DBP showed no significant changes across any condition. **Conclusion:** The acute physiological benefits observed were primarily attributed to breathing patterns rather than aroma exposure, suggesting that relaxation effects ascribed to aromatherapy may reflect vagal activation induced by deep breathing. These findings do not imply that aroma lacks effectiveness; rather, they indicate that during short-term exposure, vagal activation induced by deep breathing plays a dominant role in reducing autonomic arousal. As aroma may facilitate deeper breathing and its chronic effects have been demonstrated in previous studies, the present findings should be interpreted within the context of acute physiological responses only.

## Keywords

Aromatherapy, Breathing Pattern, Vagal Stimulation, Autonomic Nervous System, Acute Effect, Lavender

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## 1. Introduction

Aromatherapy, the controlled use of essential oils, has long been applied in complementary and alternative medicine for promoting relaxation and alleviating symptoms such as stress, anxiety, and sleep disturbance [1] [2] [3]. Evidence suggests that lavender aroma inhalation improves heart rate variability (HRV) and enhances parasympathetic (vagal) tone [1] [4]. Similarly, aromatherapy has been shown to modulate the ANS and mood in both clinical and non-clinical populations [3] [5].

On the other hand, breathing itself has a profound impact on autonomic regulation. Gerritsen and Band [6] proposed the “Respiratory Vagal Stimulation (RVS)” model, in which slow and deep breathing enhances vagal activity, thereby promoting relaxation. Prior studies demonstrated that paced breathing improves baroreflex sensitivity and reduces sympathetic arousal [7] [8].

Despite these findings, few studies have explicitly disentangled the effects of aroma from those of breathing. Inhalation of scents naturally modifies respiratory rhythm, often deepening or slowing breathing [9] [10]. Thus, reported benefits of aromatherapy may reflect a mixture of olfactory and respiratory influences rather than aroma per se. Given that olfactory stimulation has been shown to alter respiratory parameters in both human and animal studies [9] [10], it becomes critical to isolate the independent contributions of aroma versus breathing pattern to accurately interpret aromatherapy outcomes.

This study sought to clarify the acute contributions of aroma and breathing pattern to physiological responses by employing a factorial design with aroma/no-aroma and normal/deep breathing conditions. By controlling respiratory parameters with objective monitoring, we aimed to provide a clearer interpretation of aromatherapy’s mechanisms. Additionally, this study employed a manipulation check to confirm that participants could not distinguish between lavender and control conditions based on olfactory cues alone, thereby ensuring the validity of our blinding procedure.

## 2. Materials and Methods

### 2.1. Participants

A priori power analysis was conducted using G\*Power software (version 3.1). Based on previous studies examining deep breathing effects on HRV [5,6], an effect size (Cohen’s  $f$ ) of 0.4 was anticipated for the main effect of breathing. With  $\alpha = 0.05$ , power  $(1 - \beta) = 0.80$ , and a repeated measures ANOVA design with two

factors, the required sample size was calculated as  $n = 18$ . To account for potential dropouts, we recruited 20 participants.

Twenty healthy adults (13 males, 7 females; mean age  $22.5 \pm 2.1$  years) participated. Inclusion criteria were non-smokers, no history of cardiovascular, respiratory, or neurological disorders. Participants refrained from caffeine and alcohol for 12 hours prior to testing. This study was approved by Shujyukai Ethics Committee (25MA-003), and also conformed to the standard set by the Declaration of Helsinki, and written informed consent was obtained from all participants prior to the experiment.

## 2.2. Study Design

A randomized crossover design was adopted with four conditions:

- 1) Aroma + Normal breathing;
- 2) Aroma + Deep breathing;
- 3) No aroma + Normal breathing;
- 4) No aroma + Deep breathing.

Each condition was separated by a washout period of at least 24 hours. Condition order was randomized.

## 2.3. Aroma Stimulation

Lavender essential oil (1% diluted solution) was administered as a room spray, twice per minute, for 5 minutes. For the no-aroma condition, an ethanol solution (1% diluted anhydrous ethanol) was sprayed in the same manner to control for potential olfactory and vehicle effects. Lavender was chosen for its previously reported autonomic benefits [1] [4]. To validate the blinding procedure, a manipulation check was performed: after completion of all four experimental sessions, participants were asked whether they could distinguish between the aroma and no-aroma conditions. Results indicated that 18 out of 20 participants (90%) could not reliably identify which sessions involved lavender versus ethanol, confirming effective blinding.

## 2.4. Breathing Protocol

**Normal breathing:** spontaneous breathing without modification.

**Deep breathing:** guided at six breaths per minute (inspiration 4 s, expiration 8 s). A respiratory gas analyzer monitored tidal volume, and participants received visual feedback to ensure accuracy. Nasal inhalation was required to guarantee aroma intake when present.

## 2.5. Procedure

- 1) Participants rested seated for 15 minutes. Baseline HR, SBP, DBP, and salivary amylase were measured.
- 2) Intervention (5 minutes) under the assigned condition.
- 3) Post-intervention measurements identical to baseline.

## 2.6. Outcome Measures

**Cardiovascular indices:** Heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP) (measured using an upper-arm blood pressure monitor, HCR-7104; Omron, Kyoto, Japan).

**Stress marker:** salivary amylase (SA) (measured using a salivary amylase monitor; Nipro, Osaka, Japan).

**Respiratory indices:** tidal volume and respiratory rate (verification only; measured using a respiratory gas analyzer, AE-310S; Minato Medical Science, Osaka, Japan).

## 2.7. Statistical Analysis

All statistical analyses were performed using Stat View statistical software package (Ver5.0, SAS, Cary, NC, USA). Data were expressed as mean  $\pm$  standard deviation (SD) unless otherwise indicated. Percent change from baseline was calculated. Two-way repeated measures ANOVA was applied with factors Aroma (present/absent) and Breathing (normal/deep). For significant main effects, post-hoc pairwise comparisons were conducted using Bonferroni correction. Effect sizes are reported as partial eta squared ( $\eta^2p$ ), with values of 0.01, 0.06, and 0.14 representing small, medium, and large effects, respectively. 95% confidence intervals (CI) were calculated for mean differences. Significance was set at  $p < 0.05$ .

## 3. Results

**Table 1** presents the baseline characteristics of the participants. All participants completed all four experimental conditions without adverse events.

**Table 1.** Baseline characteristics of participants (n = 20).

Characteristic	Mean $\pm$ SD	Range
Gender (Male/Female)	13/7 (65%/35%)	-
Age (years)	22.2 $\pm$ 1.0	20 - 23
Weight (kg)	60.0 $\pm$ 6.5	50.0 - 70.0
Height (m)	1.64 $\pm$ 0.08	1.46 - 1.78
BMI (kg/m <sup>2</sup> )	22.6 $\pm$ 1.0	20.0 - 24.2

Values are means  $\pm$  SD. Abbreviations: BMI, Body Mass Index.

**Heart Rate:** Deep breathing significantly reduced HR compared with normal breathing (main effect:  $F(1,19) = 28.13$ ,  $p < 0.001$ ,  $\eta^2p = 0.60$ ; mean difference =  $-6.86\%$ , 95% CI [ $-9.57\%$ ,  $-4.15\%$ ]). No main effect of aroma ( $F(1,19) = 0.32$ ,  $p = 0.58$ ) or Aroma  $\times$  Breathing interaction ( $F(1,19) = 0.15$ ,  $p = 0.70$ ) was observed.

**Blood Pressure:** SBP significantly decreased with deep breathing (main effect:  $F(1,19) = 28.48$ ,  $p < 0.001$ ,  $\eta^2p = 0.60$ ; mean difference =  $-5.04\%$ , 95% CI [ $-7.01\%$ ,  $-3.06\%$ ]). No aroma effect ( $F(1,19) = 0.41$ ,  $p = 0.53$ ) or interaction effect ( $F(1,19)$

= 0.22,  $p = 0.64$ ) was found. DBP did not change significantly across any condition (main effect of Breathing:  $F(1,19) = 0.08$ ,  $p = 0.78$ ; main effect of Aroma:  $F(1,19) = 0.03$ ,  $p = 0.86$ ; interaction:  $F(1,19) = 0.01$ ,  $p = 0.92$ ). The absence of DBP changes may reflect the relatively mild nature of the intervention and the stability of diastolic pressure in young, healthy participants (Table 2).

**Table 2.** Rate of changes in physiological indices under each condition (Mean  $\pm$  SD, %).

Outcomes	No Aroma-Normal	No Aroma-Deep	Aroma-Normal	Aroma-Deep	Main Effect (Breathing)
HR	0.41 $\pm$ 3.61	-6.35 $\pm$ 4.62*	1.06 $\pm$ 4.46	-5.90 $\pm$ 4.29#	$p < 0.001$
SBP	0.24 $\pm$ 3.17	-6.20 $\pm$ 3.50*	-1.52 $\pm$ 3.73	-5.15 $\pm$ 3.09#	$p < 0.001$
DBP	-0.97 $\pm$ 3.42	-0.73 $\pm$ 3.64	-0.66 $\pm$ 3.52	-0.76 $\pm$ 4.69	$p = 0.94$
SA	4.88 $\pm$ 10.54	-21.43 $\pm$ 14.16*	1.51 $\pm$ 11.81	-18.63 $\pm$ 9.31#	$p < 0.001$

Values are means  $\pm$  SD. \* $p < 0.05$  vs. No Aroma-Normal (post-hoc pairwise comparison). # $p < 0.05$  vs. Aroma-Normal (post-hoc pairwise comparison). Main effects were determined using two-way repeated measures ANOVA with factors Aroma (present/absent) and Breathing (normal/deep). Post-hoc comparisons used Bonferroni correction. Abbreviations: HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; SA, salivary amylase.

Salivary Amylase: Deep breathing significantly decreased salivary amylase (main effect:  $F(1,19) = 45.98$ ,  $p < 0.001$ ,  $\eta^2p = 0.71$ ; mean difference = -23.23%, 95% CI [-30.40%, -16.06%]). Aroma had no significant effect ( $F(1,19) = 0.28$ ,  $p = 0.60$ ), nor did the interaction ( $F(1,19) = 0.18$ ,  $p = 0.68$ ) (Table 3).

**Table 3.** Summary of two-way repeated measures ANOVA results.

Measure	Effect	F(1,19)	p-value	$\eta^2p$	Mean Diff	95% CI
HR	Breathing	28.13	<0.001	0.60	6.86%	[4.15%, 9.57%]
SBP	Breathing	28.48	<0.001	0.60	5.04%	[3.06%, 7.01%]
DBP	Breathing	0.01	0.94	0.00	-0.07%	[-1.91%, 1.77%]
SA	Breathing	45.98	<0.001	0.71	23.23%	[16.06%, 30.40%]

Only main effects of Breathing are shown, as main effects of Aroma and Aroma  $\times$  Breathing interactions were non-significant for all measures (all  $p > 0.05$ ).  $\eta^2p$  = partial eta squared (effect size: 0.01 = small, 0.06 = medium, 0.14 = large). Mean Diff = mean difference between Normal and Deep breathing conditions. CI = confidence interval.

Summary: Deep breathing exerted robust physiological effects across indices, while aroma alone did not induce significant changes. The large effect sizes for breathing ( $\eta^2p = 0.60 - 0.71$ ) indicate that the observed effects are both statistically significant and practically meaningful.

## 4. Discussion

This study demonstrated that acute reductions in HR, SBP, and salivary amylase

were driven by breathing patterns rather than aroma exposure. These findings align with the RVS model [6], supporting the notion that deep breathing, independent of olfactory stimulation, promotes parasympathetic activation.

#### **4.1. Reinterpretation of Aromatherapy**

Prior reports attributing relaxation to lavender aroma [1] [4] may partially reflect unmeasured breathing modifications. Our results suggest that aroma inhalation without breathing control could overestimate aroma-specific effects. Importantly, aroma may still function as a trigger for deep breathing, indirectly facilitating autonomic changes. This interpretation aligns with recent work suggesting that aromatherapy benefits may be mediated by behavioral and respiratory changes rather than direct olfactory neuromodulation [11] [12]. While our study does not diminish the potential value of aromatherapy, it highlights the need to explicitly account for respiratory mechanics when attributing outcomes to aroma exposure.

#### **4.2. Comparison with Prior Research**

Slow breathing has been shown to enhance baroreflex sensitivity and reduce sympathetic tone [7] [8]. Similarly, deep breathing reduces pain perception and stress reactivity [9] [10]. The present findings extend this evidence by confirming that, when aroma is controlled, deep breathing remains the dominant factor in acute autonomic regulation. Although previous research has extensively documented the effects of paced breathing on autonomic function [7] [8], the present study provides novel evidence by directly comparing aroma and breathing effects within a single factorial design, thereby quantifying their relative contributions. This methodological approach clarifies that, in acute settings, breathing exerts a substantially larger effect ( $r^2p = 0.40 - 0.60$ ) than aroma exposure ( $r^2p < 0.01$ ).

#### **4.3. Clinical Implications**

Combining aroma with breathing guidance may optimize relaxation in hospitals, nursing homes, and stress management programs. For workers and students, aroma may serve as a cue to engage in paced breathing exercises. For sleep disorders, incorporating aroma with guided breathing could improve pre-sleep relaxation and sleep onset. To test the hypothesis that aroma functions as a “psychological primer” for breathing exercises, future studies could employ a conditioning paradigm: participants could be trained to associate a specific aroma with deep breathing, then tested to determine whether aroma exposure alone (without explicit breathing instructions) spontaneously elicits deeper respiratory patterns and associated autonomic changes. Such a design would directly assess aroma’s role as a conditioned cue for respiratory modulation.

#### **4.4. Additional Considerations**

The no-aroma condition used ethanol spray, which itself has a subtle olfactory property. This methodological refinement reduces the possibility that participants

distinguished conditions based on olfactory cues alone. Thus, the absence of aroma-specific effects in our results strengthens the conclusion that deep breathing was the primary driver of physiological changes.

Another important aspect concerns psychological expectancy effects. Participants may anticipate relaxation when exposed to pleasant aromas. By equating the spraying procedure with ethanol, we minimized expectancy bias. This suggests that, at least for acute effects, the physiological changes are less about olfactory hedonic qualities and more about breathing-related vagal modulation.

#### **4.5. Broader Perspective**

Our findings highlight a potential integration of Eastern and Western approaches. Eastern traditions such as yoga emphasize breathing control, while Western aromatherapy emphasizes olfactory stimulation. This study suggests a dual-pathway model, where aroma acts as a psychological primer while breathing constitutes the physiological driver.

#### **5. Limitations**

Several limitations warrant consideration. First, the sample consisted exclusively of young, healthy adults (mean age 22.5 years), which may limit generalizability. Autonomic responses to breathing and aroma may differ in older adults, individuals with chronic stress, or clinical populations (e.g., those with anxiety disorders or cardiovascular disease). Future research should examine these effects across diverse age groups and clinical conditions to determine whether the dominance of breathing over aroma generalizes beyond healthy young adults.

Second, the intervention duration was brief (5 minutes), and physiological measurements were limited to HR, blood pressure, and salivary amylase. Longer exposure periods and additional autonomic markers—such as heart rate variability (HRV), respiratory sinus arrhythmia (RSA), and cortisol levels—would provide deeper insight into autonomic modulation mechanisms and potentially reveal cumulative or delayed aroma effects.

Third, while we included objective physiological measures, we did not assess subjective psychological outcomes such as self-reported stress, relaxation, mood states, or perceived pleasantness of the aroma. Incorporating validated psychometric scales (e.g., State-Trait Anxiety Inventory, Visual Analog Scales for relaxation) in future studies would enable a more comprehensive understanding of aromatherapy's effects and clarify whether subjective and objective outcomes align.

Fourth, although the manipulation check confirmed effective blinding in 90% of participants, the use of ethanol as a control is not without limitations. Ethanol possesses a mild olfactory signature, which, while faint, may introduce subtle confounds. Future research could employ truly odorless controls or alternative pleasant non-therapeutic scents (e.g., vanilla) to further isolate aroma-specific effects.

The present results suggest that acute physiological responses are more sensitive to breathing patterns than to aroma exposure. These findings do not imply

that aroma lacks effectiveness; rather, they indicate that during short-term exposure, vagal activation induced by deep breathing plays a dominant role in reducing autonomic arousal. As aroma may facilitate deeper breathing and its chronic effects have been demonstrated in previous studies, the present findings should be interpreted within the context of acute physiological responses only.

## 6. Conclusion

The acute physiological benefits observed in this study were primarily due to breathing patterns rather than aroma exposure. Deep breathing consistently reduced HR, SBP, and salivary amylase, supporting vagal activation mechanisms. These results emphasize the importance of distinguishing between aroma-specific and breathing-related effects when interpreting aromatherapy outcomes.

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## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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