Title: Differential Effects of Nasal Versus Oral Breathing on Cognitive Performance of Individuals in the Numerical Stroop Test Following One Night of Partial Sleep Deprivation

Running Title: Nasal Breathing Ameliorates Sleep Deprivation Effects

Authors: Hanieh Riazi[1], Amir Shojaei[2,*]

1. *Department of Physiology, Faculty of Medical Sciences, Tarbiat Modares University, Tehran, Iran.*
2. *Institute for Brain and Cognition, Faculty of Medical Sciences, Tarbiat Modares University, Tehran, Iran.*

***Corresponding Author**: Amir Shojaei, Institute for Brain and Cognition, Faculty of Medical Sciences, Tarbiat Modares University, Tehran, Iran. Email: am.shojaei@modares.ac.ir

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Abstract

Objective(s): Impaired cognitive performance is a common consequence of acute partial sleep deprivation (PSD). Nasal respiration modulates neuronal activity across different brain areas and enhances the power of higher-frequency brain oscillations. The current study investigated the impact of nasal respiration on cortical brain oscillations and the cognitive performance of subjects with acute PSD in the numerical Stroop test (NST).

Materials and Methods: A total of 26 male and female subjects underwent the NST and electroencephalography (EEG) on the initial day, which established their baseline performance. Over the next 24 hours, participants were instructed to sleep only between 3 a.m. and 7 a.m. and remain awake for the rest of the period. The NST was repeated the following day. Prior to the second NST, participants in the oral respiration group performed mouth breathing, followed by resting EEG recording, while those in the nasal respiration group practiced nasal breathing for 3 minutes before resting EEG recording.

Results: Taking nasal breathing recovered the increased error rate ($p<0.01$) and shortened reaction time ($p<0.01$) observed in the oral respiration group. In addition, nasal respiration reversed the increases in delta and theta power, as well as the decreases in beta and gamma power associated with PSD, when compared to the oral respiration group.

Conclusion: This study suggests that mechanical stimulation of the olfactory epithelium through nasal respiration may improve cognitive performance in individuals with acute sleep deprivation.

Keywords: oral breathing, nasal breathing, acute sleep deprivation, electroencephalography, numerical Stroop test

Introduction

Sleep is a physiological state essential for preserving overall health and wellness (Ferrara y Gennaro, 2001). Sleep is identified by closed eyes and a significant reduction in responsiveness to sensory stimuli (Sejnowski y Destexhe, 2000). Adequate sleep at appropriate times is essential for maintaining brain function (Eugene y Masiak, 2015). It supports neuronal repair, synapse development, emotional regulation, and cognitive processes such as learning, memory, decision-making, and attention.

Sleep requirements vary between individuals and across different age groups (Watson et al., 2015). On average, healthy adults need 7 to 9 hours of sleep each night (Watson et al., 2015). Even slight reductions in sleep duration or quality can result in sleep deprivation (Eugene y Masiak, 2015). Over 30% of the worldwide population experiences acute sleep deprivation (Landolt et al., 2014) which involves either a complete lack of sleep or a reduction in its duration or quality, typically lasting one to two days (Landolt et al., 2014). Lack of sleep hinders cognitive abilities like reasoning, focus, and levels of alertness (Orzeł-Gryglewska, 2010). Electroencephalography (EEG) studies have shown that acute sleep deprivation alters brain oscillations (Corsi-Cabrera et al., 1992). It increases delta activity in the prefrontal and frontal regions and enhances theta oscillations in the central, parietal, and occipital areas. In contrast, it reduces alpha, beta, and gamma rhythms in several regions, including the frontal and parietal lobes.

Given the extensive consequences of sleep deprivation, it is essential to develop effective strategies to mitigate its effects on brain activity and cognition (Alhola y Polo-Kantola, 2007). Experimental data suggest that neuromodulation remains a promising tool in rescuing the cognitive functions in individuals with sleep deprivation (Grimaldi et al., 2020). Neuromodulation refers to the alteration of neuronal activity through targeted delivery of a stimulus or intervention, which could involve electrical, chemical, or magnetic methods (Johnson et al., 2013). Recently, stimulating the olfactory system is considered a method for neuromodulation of brain activity (Vance et al., 2024). The olfactory epithelium, which is located on the roof of the nasal cavity, detects odorant molecules that enter the nasal cavity. It sends the olfactory sensory information to the olfactory bulb (Biskamp et al., 2017).

It has been demonstrated that olfactory sensory neurons of mammals, including humans, in addition to being sensitive to chemical odor stimuli, can be mechanically stimulated by intranasal airflow (Grosmaitre et al., 2007). It has been proposed that the mechanical stimulation of the olfactory bulb during nasal breathing induces slow oscillations that are synchronized with the breath, referred to as respiration-entrained brain rhythm, across extensive regions of the cortex and subcortex, including the amygdala, hippocampus, and prefrontal cortex (Biskamp et al., 2017; Fontanini et al., 2003; Grosmaitre et al., 2007; Liu et al., 2017; Lockmann et al., 2018; Piarulli et al., 2018; Yanovsky et al., 2014; Zhong et al., 2017). These oscillations cause the coordination of different brain networks and establish connectivity between different brain regions (Heck et al., 2016). Furthermore, inhaling through the nose enhances the strength of various frequency patterns in the hippocampus, amygdala, and piriform cortex regions of the human brain. In contrast, breathing through the mouth results in a reduction of these frequency patterns in the same areas (Zelano et al., 2016). Furthermore, it has been shown that the memory and perception of fear in the condition of nasal breathing are significantly better than oral breathing (Zelano et al., 2016).

Ghazvineh et al. (2021) reported that rhythmic air-puffs delivered into the nasal passages of mechanically ventilated animals enhanced oscillatory activity in brain regions involved in cognition and memory. These regions included the ventral hippocampus and medial prefrontal cortex (Ghazvineh et al., 2021). As a result of this effect, the working memory of these animals showed improvement (Ghazvineh et al., 2021). In a different study, Salimi et al. (2022) noted that intra-nasal air-puffing enhanced the strength of gamma oscillations within the default mode network in the brains of comatose patients. It also led to improved synchronization and coherence between regions of the default mode network (Salimi et al., 2022). In another study, Qun Li et al. (2023) indicated that closed-loop electrical stimulation of the olfactory bulb decreased behaviors resembling depression and facilitated the conveyance of gamma activity from the olfactory bulb to limbic regions (Li et al., 2023). Moreover, this stimulation also influenced gamma oscillations in cortical areas (Deng et al., 2024).

Nasal respiration plays an important role in regulating brain oscillations, including increasing gamma power, which is critical for cognitive and sensory processing. However, no studies have distinguished the effects of nasal and oral breathing on cognitive abilities after sleep deprivation. Consequently, this study explored the cognitive performance of individuals in the numerical Stroop test (NST) and recovery of cortical oscillations after sleep deprivation, focusing on two conditions: nasal breathing and oral breathing.

Materials and Methods

Participants

This research involved 26 volunteers, comprising 14 males and 12 females, aged between 22 and 36 years. The privacy rights of human subjects have been observed, and informed consent was obtained. All participants were right-handed, in good health, and did not have any chronic or acute medical conditions. The health of the participants was confirmed based on a self-report questionnaire, and they were not subjected to medical examination. All participants either had normal vision or used corrective lenses, reported no history of substance abuse, including drug or alcohol addiction, and were only taking dietary supplements. Five participants were removed from the study for not adhering to the experimental protocols. Table 1 presents further information regarding the demographic characteristics of the participants. The Ethics Committee of Tarbiat Modares University evaluated and granted approval for the ethical aspects of this study (Date: June, 2022; reference code: IR.MODARES.REC.1401.085).

Experimental procedure

The experiments were conducted over a span of two days, totaling approximately 25 h. On the initial day (before sleep deprivation), participants arrived at the laboratory at approximately 9 a.m. Before starting the experiments, individuals signed informed consent and completed a few forms, consisting of a demographic information form, the Pittsburgh sleep quality questionnaire, and a cognitive disability questionnaire. The participants were then randomly allocated to either the oral or nasal respiration groups. Initially, participants underwent an EEG recording, which was followed by a 10-minute NST based on the method described later. (see Figure 1A). At the conclusion of the experiment on the initial day, participants were advised to refrain from ingesting central nervous system stimulants, including coffee, energy drinks, tea, or any caffeinated products, for the next 24 h. Participants were also instructed to avoid napping or participating in intense physical activity and only sleep from 3 to 7 a.m. The participants returned to the laboratory at 9 a.m. the following morning (on the next day). The experimenter provided no direct oversight; participants confirmed their adherence to the experimental protocols by completing a brief questionnaire on the second day. The questionnaire comprised eight items that asked participants about their bedtime and wake-up time, as well as whether they had consumed caffeinated beverages (e.g., tea or coffee) or alcohol or engaged in vigorous exercise within the past 24 hours. All participants reported full compliance (100%) with the study guidelines.

After completing the questionnaire, participants in the nasal respiration group were instructed to keep their mouths closed and breathe exclusively through their noses for 3 min (Riazi et al., 2024). Resting EEG was then recorded for 3 minutes, followed by the NST (see Figure 1B). For the oral respiration group, nasal tampons were inserted into subjects' nostrils, and they were directed to breathe through their mouths for 3 min and again a 3-min resting state EEG recording before undergoing the NST (see Figure 1C).

The baseline data collected from both groups on the first day, before sleep deprivation, were classified as the non-sleep-deprived state ("no SD"), and results obtained after partial sleep deprivation (PSD) on the second day were designated as the sleep-deprived condition ("SD"). Accordingly, the oral respiration measurements following PSD on the second day were labeled "SD + Oral resp," while the nasal breathing results after PSD were categorized as "SD + Nasal resp.

Pittsburgh sleep quality questionnaire

A team of researchers at the University of Pittsburgh created the Pittsburgh sleep quality index in 1989 (Buysse et al., 1989). This questionnaire evaluates the quality of sleep by examining seven aspects of the individual's sleep over the last month. These elements consist of time required to fall asleep, perceived sleep quality, consistent sleep efficiency, total duration of sleep, use of sleep medications, sleep disruptions, and daytime functional impairment.

Individuals responded to the inquiries using a Likert scale spanning from 0 to 3. The overall rating derived from the seven elements varies from 0 to 21, with sleep quality diminishing as the score rises. This questionnaire demonstrates a reliability score of 0.83 and has exhibited strong proven validity across numerous studies. Adapting the Persian version of the Pittsburgh sleep quality questionnaire has demonstrated satisfactory reliability and validity within individuals in Iran (Farrahi Moghaddam et al., 2012).

Cognitive disability questionnaire

In 2013, Nejati developed a 30-item questionnaire organized into seven domains: cognitive control and selective attention, decision-making, planning, sustained attention, social cognition, memory, and cognitive flexibility (Nejati, 2013). A five-point Likert scale ranging from 1 (never) to 5 (always) is employed to evaluate cognitive impairment, with higher scores reflecting a decline in cognitive function. The Cognitive disability questionnaire has been employed in numerous studies and demonstrates a reliability of 0.83 among the Iranian population (Fakhari et al., 2022).

Numerical Stroop Test (NST)

The NST was utilized to assess cognitive abilities and the capacity to suppress instinctive or routine responses. In each trial, participants focused on a 16-inch screen positioned approximately 80 centimeters from their eyes, which displayed two single-digit numbers. During each trial, a hashtag symbol appeared for 700 ms, followed by a blank screen for 500 ms. Following the display of a pair of numbers in white on a black background lasting 800 ms, the screen remained blank for an additional 500 ms. The next trial commenced immediately (Beldzik et al., 2015) (Figure 2A). The numbers ranged from 1 to 9 and were displayed in Arial font. Participants were instructed to use their right index finger on a keypad to select the numerically larger number, ignoring its physical size. The side with the largest number was randomly alternated. The difference between each pair of numbers ranged from 1 to 3. The number pairs were displayed under three conditions: congruent, in which the greater number appeared bigger in physical size than the other digit; neutral, where both numbers shared the same physical size but varied numerically; and incongruent, where the larger number in numerical value was smaller in physical size. There were

252 blocks in all, evenly distributed among the numerical values. The sequence of trials was arranged in a partially-randomized manner. Each NST run was split into two sessions, each consisting of 126 trials, and separated by a 5-min break to reduce mental exhaustion.

The study assessed the following variables: error rate, which represents the proportion of total trials in which a participant made an erroneous selection. Reaction time was the average duration between the presentation of numbers and the subsequent pressing of the left or right key. The missing rate was the percentage of trials where the participant failed to press any keys.

When suitable, the percentage change in these variables was computed in the following manner:

$$\text{Percentage change} = \left[\frac{\text{Value in desired state } - \text{Initial value}}{\text{Initial value on first day}}\right] \times 100$$

where "value in the desired state" refers to the NST parameter values measured on Day 2 (after PSD) following nasal respiration, and "initial value" refers to the same parameter measured on Day 1 (before PSD).

EEG recording and pre-processing

EEG was recorded in 3 min sessions as described beforehand. During the EEG recordings, participants sat comfortably in a chair with their eyes open. EEG recordings were obtained using an EEG device (V.L430 model, Liv Intelligent Technology, Iran) equipped with active electrodes following the international 10–20 system for electrode placements. Signals were recorded from 16 different channels, specifically from Fp1, Fp2, F3, F4, F7, F8, C3, C4, T7, T8, P3, P4, O1, O2, P7, and P8. The ground and reference electrodes were linked to the right and left mastoids, respectively. EEG signals were low-pass filtered at frequency range of 100 Hz and sampled at a rate of 250 Hz. Electrode impedance was maintained below 10 kΩ throughout the recordings. The collected data were then imported into MATLAB using the EEGLAB toolbox (version 2023.0;

sccn.ucsc.edu/eeglab) for preprocessing. First, baseline correction was applied to the signals, and a notch filter (48-52 Hz) was used to eliminate power line interference. The data were then subjected to band-pass filtering between 1 and 100 Hz using a zero-phase linear finite impulse response (FIR) filter. After manually inspecting and rejecting artifacts, EEG signals were re-referenced to the average electrical activity measured across all channels. Afterwards, independent component analysis (ICA) was performed using the "runica" algorithm to remove contaminating sources such as eye movements, muscle activity, eye blinks, and cardiac signals (Bell y Sejnowski, 1994). Between two and five components were discarded to enhance the quality of the EEG data. After preprocessing, a 100 sec of EEG signal was used for analyzing.

Power spectral density

Calculating the power spectral density was carried out using the pwelch function (a 4-second sliding Hamming window with 75% overlap) in MATLAB (version 2022, MathWorks Inc., California, USA). This parameter was measured within the delta (1–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta (13–30 Hz), slow gamma (30–47 Hz), medium gamma (53–70 Hz), and fast gamma (70–97 Hz) frequency ranges (Huang, 2016).

Statistical analysis

Before statistical analysis, we used the Shapiro-Wilk test to assess data normality and Levene's test to examine variance equality using OriginPro (Version 2022, OriginLab Corporation, Northampton, Massachusetts, USA). Statistical analyses were conducted using GraphPad Prism (version 8, GraphPad Software, Boston, Massachusetts, USA). NST and EEG parameters were compared between oral and nasal respiration using either a two-way repeated measures ANOVA (followed by a Sidak post-hoc test), an unpaired t-test or a Mann-Whitney U test, where applicable. Linear regression analysis was performed to assess associations between sleep quality index, cognitive disability index, and NST parameters. Pearson correlation coefficients were calculated to quantify the strength of these relationships. The data are displayed as mean ± standard error of the mean (SEM). Statistical significance was determined with a p-value below 0.05.

Cohen's d (d value) was computed in MATLAB to assess the effect size. A d value below 0.4 were categorized as weak, between 0.4 and 0.8 as medium, and values exceeding 0.8 as strong effect size (Cohen, 2013).

Results

Comparing initial characteristics of individuals in groups categorized by oral and nasal breathing

No significant difference was observed in the subjects' age and weight ($p = 0.21$ for age and $p = 0.76$ for weight; Table 1) across the nasal and oral respiration groups. Moreover, there were no observable distinctions in cognitive disability indices ($p = 0.28$) or sleep quality ($p = 0.68$) between the two groups (Table 1). Initially, there was no significant difference between the two groups in NST parameters, including error rate ($p = 0.69$), missing rate ($p = 0.94$), and reaction time ($p = 0.30$) on the initial day (Figure 2B). These results validated that individuals in both the nasal respiration and oral respiration groups exhibited similar initial conditions at the beginning of the research.

The effect of nasal respiration on the cognitive function of participants in the NST after a PSD

To investigate the impact of stimulating the olfactory epithelium via nasal breathing on cognitive performance in the NST, the results obtained after PSD (labeled 'SD') were compared with those measured before PSD on the initial day (labeled 'no SD') within the same group.

The outcomes from the oral respiration group revealed that the cognitive function of participants in NST was detrimentally affected by a PSD, leading to a notably increased error rate in the

SD+Oral resp group compared to the non-sleep-deprived state ($p < 0.01$). Moreover, PSD notably decreased the missing rate (effect size d = 0.94) and reaction times within the oral respiration group ($p < 0.01$; see Figure 3).

Interestingly, taking nasal respiration following PSD helped restore the cognitive function of individuals in the nasal respiration group so that no significant difference was observed in NST parameters between the SD+Nasal resp and no SD state (see Figure 3).

The association between sleep quality, cognitive disability and the effectiveness of nasal respiration in restoring participants' function in the NST after a night of PSD

Given the individual variances in sleep quality and cognitive abilities, our subsequent aim was to assess whether nasal respiration's impact on enhancing participants' cognitive performance after one night of PSD was influenced by their initial sleep quality or cognitive capacities. In this regard, we computed the correlation between the sleep quality index and the cognitive disability index with the percentage changes in the NST parameters. The findings demonstrated a significant correlation between the sleep quality index and the percentage of change in reaction time ($r = 0.56$, $p < 0.05$), as illustrated in Figure 4A. In other words, the effectiveness of nasal respiration in restoring the reaction time was reduced in individuals with poorer sleep quality. Furthermore, a significant negative correlation was observed between the cognitive disability index and the percentage of variations in error rate ($r = -0.57$, $p < 0.05$), as depicted in Figure 4B. It shows that the efficacy of nasal breathing in restoring cognitive function in subjects with PSD diminishes as the severity of cognitive disability increases. There were no significant correlations between the percentage change in error rate and the sleep quality index or between the percentage change in reaction time and the cognitive disability index.

EEG power

To gain an understanding of how nasal breathing influences brain cortical activity, we also analyzed EEG recordings from participants. As explained before, EEG was recorded for 3 minutes in 2 sessions. The first session was performed before sleep deprivation (on day 1, known as the no SD condition), and the remaining session was performed after PSD (on day 2). It was recorded after nasal breathing (known as SD + Nasal resp) in the nasal respiration group or after oral breathing (known as SD + Oral resp) in the oral respiration group. The results of the EEG power assessment recorded from 16 electrodes are summarized in Figure 5 and Tables S1-S7. Following one night of PSD, oral respiration led to a significant increase in delta band power in the right occipital lobe ($p < 0.05$ in the O2 channel) compared to the no SD state. In contrast, nasal breathing significantly reduced delta power in the right frontal ($p < 0.05$ in Fp2 and F8), parietal ($p = 0.088$ and $d = 0.8179$ in P8), temporal ($p < 0.05$ in T8), and occipital ($p < 0.05$ in O2) cortices (Figure 5 and Table S1).

In the theta band, oral respiration significantly increased EEG power in the frontal ($p < 0.05$ in F3; $p < 0.01$ in F4), central ($p < 0.05$ in C4), parietal ($p < 0.01$ in P3, P4, P7, and P8), temporal ($p < 0.05$ in T8), and occipital ($p < 0.01$ in O2; $p < 0.001$ in O1) regions compared to no SD. In contrast, nasal breathing restored theta power to baseline levels, showing no significant differences from the no SD state (Figure 5 and Table S2).

For the alpha band, oral respiration did not significantly alter EEG power after PSD. However, nasal breathing enhanced alpha power in the frontal ($p < 0.05$ in Fp1, F4, and F8; $p < 0.01$ in Fp2), parietal ($p < 0.05$ in P8), temporal ($p < 0.05$ in T8), and occipital ($p < 0.05$ in O1 and O2) regions (Figure 5 and Table S3).

In the beta band, PSD reduced EEG power in the right frontal ($p < 0.05$ in F8) and parietal ($p < 0.05$ in P4) lobes. Nasal breathing restored beta power in these regions (F8 and P4), eliminating the PSD-induced decline. Additionally, nasal respiration significantly or substantially increased beta power in the frontal ($p < 0.01$ in F3; $p = 0.1150$ and $d = 0.8884$ in F4; $p = 0.0535$ and $d = 0.8179$ in F7) and parietal ($p = 0.1929$ and $d = 0.9168$ in P4) cortices compared to no SD (Figure 5 and Table S4).

Evaluating the EEG in the gamma band showed that 3 min of oral respiration reduced power in the frontal ($p < 0.01$ in F8), central ($p < 0.05$ in C4) and parietal ($p < 0.05$ in P7) lobes in low gamma, frontal ($p < 0.01$ in F8) and parietal ($p < 0.05$ in P4, $p < 0.01$ in P7) in medium gamma, and frontal ($p < 0.05$ in F8), parietal ($p < 0.05$ in P4, P7, and $p < 0.001$ in P8) and temporal ($p < 0.05$ in T7) cortices in fast gamma bands. Interestingly, no significant change was observed in any of the brain cortices following nasal respiration, indicating that nasal respiration has the capability of recovering the EEG in the gamma band to normal values (Figure 5 and Tables S5-S7).

Discussion

The present study showed that nasal breathing after PSD exerted a modulatory effect on brain function. Three minutes of nasal respiration following one night of PSD improved cognitive performance, restoring error rate, missing rate, and reaction time in the NST to baseline levels.

It has been demonstrated that sleep loss results in a decline in cognitive functions such as attention, concentration, working memory, episodic memory, and associative memories (Khan y Al-Jahdali, 2023; Krause et al., 2017). Additionally, sleep deprivation affects a variety of other cognitive domains, such as logical reasoning, social judgment, and decision-making (Khan y Al-Jahdali, 2023; Krause et al., 2017). In this study, oral breathing after one night of PSD led to an increase in the error rate in the NST, suggesting a decline in cognitive performance. The subjects' performance in this test depends on various facets of brain function, such as attention, mathematical reasoning, and logical thinking, to prevent automatic and habitual reactions (Sagaspe et al., 2006). Hence, it relies on the interplay among various brain regions, including the prefrontal cortex, parietal cortex, anterior cingulate cortex, and regions of the temporal lobe (Sagaspe et al., 2006). Previous studies using functional magnetic resonance imaging (fMRI) have demonstrated that functional connectivity of these regions decreases by sleep deprivation (Peng et al., 2024). It has also been indicated that following short-term sleep loss, attention becomes notably unstable and erratic, leading to fluctuating performance on tasks related to attention (Krause et al., 2017). Nevertheless, sleep deprivation diminishes overall performance in attention assessments because of heightened sleep pressure and reduced alertness (Khan y Al-Jahdali, 2023; Krause et al., 2017). Typically, the length of time spent awake during the period of sleep deprivation can forecast the extent of attentional disturbances (Khan y Al-Jahdali, 2023; Krause et al., 2017).

EEG findings in the SD + Oral group were consistent with previous studies. One night of PSD increased low-frequency power (delta and theta) and reduced high-frequency power (beta and gamma) across several cortical regions. The most pronounced changes were observed in the frontal and parietal areas. These patterns suggest increased neuronal synchrony across the cortex during oral respiration following sleep deprivation, which may impair information processing efficiency. Because frontal regions support higher-order cognition, these EEG changes may explain the cognitive deficits observed during oral respiration after sleep deprivation. EEG studies have consistently shown that sleep loss results in a compensatory increase in low-frequency oscillations, especially in the delta and theta bands, indicating an increased homeostatic drive for sleep (Brown et al., 2012). In contrast, alpha band power tends to decline following sleep restriction, suggesting that attentional control and cortical arousal are significantly impaired (Brown et al., 2012).

Results of the NST also indicated that a single night of acute PSD led to a decrease in both the rate of missed responses and reaction times in the NST. These findings can be explained in the context of previous research. A study using fMRI conducted by Venkatraman and colleagues (2007) found that activity in the right nucleus accumbens heightened after participants made riskier choices during a gambling task following sleep deprivation, unlike when they were well-rested (Venkatraman et al., 2007). Additionally, following losses, there was a reduced activation observed in the orbitofrontal and insular cortices, indicating a decreased response to losses (Mullin et al., 2013). Research conducted by Olson et al. (2016) and Killgore et al. (2006) indicated that individuals deprived of sleep exhibited impulsive responses in the Iowa Gambling Task, showing a tendency for riskier decision-making and prioritizing short-term gains over long-term benefits (Killgore et al., 2006; Olson et al., 2016). In an alternative investigation, Mullin et al. (2013) discovered that following a night of sleep deprivation, participants exhibited heightened activity in the ventral striatum and decreased deactivation in the medial prefrontal cortex during a monetary reward task, compared to their performance after a complete night's sleep (Mullin et al., 2013). Taken together, these findings suggest that following sleep deprivation, neural responses do not reliably represent the significance of rewards and punishment, leading to impulsive and erroneous decision-making (Krause et al., 2017). Although oral respiration subjects exhibited a higher error rate after sleep deprivation, this could explain the reduced rate of missed responses and faster reaction times.

In this study, the taking nasal respiration led to a substantial enhancement in participants' cognitive performance following one night of PSD. This improvement was indicated by the restoration of error rate, missing rate, and reaction time to their initial baseline levels. The olfactory bulb receives input from the olfactory epithelium and transmits information to cognitive regions such as the orbitofrontal and piriform cortices (Biskamp et al., 2017). These regions process olfactory information and are associated with emotions, memory, and decision-making (Biskamp et al., 2017). Additionally, studies have shown that stimulating olfactory epithelium by odors activates the hippocampus and other regions of the brain linked to memory consolidation (Tong et al., 2014). This indicates that the olfactory system may play a significant role in memory formation and retrieval. Furthermore, olfactory cues have been connected to emotional processing in areas like the amygdala, which affects mood and behavior-related cognitive processes (Zald y Pardo, 1997). One of the important brain oscillations that has a crucial role in various cognitive functions is gamma rhythm. It has been shown that gamma activity in the primary somatosensory, motor, and visual cortices exhibits coordinated fluctuations that are synchronized with the rat's slow breathing rhythm (around 2.5 Hz) at resting state (Heck et al., 2016). Stimulating the olfactory epithelium has been found to enhance gamma oscillations in the brain (Rojas-Líbano y Kay, 2008). It also affects the emotion processing and the memory formation (Lepousez et al., 2014). Moreover, in a study, Qun Li and colleagues (2023) demonstrated that electrical stimulation of the olfactory bulb enhanced the transfer of gamma oscillations from the olfactory bulb to limbic areas, resulting in decreased depressive-like behaviors (Li et al., 2023). This stimulation also enhanced the power of gamma oscillations in cortical areas (Li et al., 2023). However, the effectiveness of nasal breathing in restoring cognitive function diminished with poorer sleep quality or lower baseline cognitive ability in individuals with PSD in our study. As the cognitive performance of subjects decreased when nasal nostrils were blocked during oral respiration, the beneficial impacts of nasal breathing on cognitive function probably stem from the stimulation of the olfactory epithelium by intranasal airflow.

EEG results showed that nasal breathing reversed PSD-induced increases in low-frequency power (especially theta) and restored high-frequency power (beta and gamma). While delta and theta oscillations are linked to certain cognitive processes, most research emphasizes the role of beta and gamma oscillations in cognition (Mai et al., 2016). Beta activity supports attention, working memory, decision-making, motor functions, sensory integration, and perception across frontal, parietal, temporal, and occipital regions (Betti et al., 2021). Gamma oscillations have been suggested to be crucial for information processing and integration, aiding sensory unification, spatial cognition, attention, perception, and memory across similar brain regions (Guan et al., 2022).

The exact mechanisms behind how olfactory epithelium stimulation impacts cognitive functions and modulates brain rhythm are still poorly understood, and further research is needed to clarify these processes. It has been suggested that mechanical stimulation of the olfactory bulb during nasal breathing generates slow oscillations that align with the breathing cycle, known as respiration-entrained brain rhythms, affecting widespread areas of both the cortex and subcortex, such as the amygdala, hippocampus, and prefrontal cortex (Biskamp et al., 2017; Fontanini et al., 2003; Grosmaitre et al., 2007; Liu et al., 2017; Lockmann et al., 2018; Piarulli et al., 2018; Yanovsky et al., 2014; Zhong et al., 2017). These oscillations help synchronize various brain networks and enhance connectivity between different brain areas (Heck et al., 2016). Furthermore, inhalation during nasal respiration amplifies the power of various frequencies in regions such as the hippocampus, amygdala, and piriform cortex in humans. In contrast, oral breathing leads to a reduction in the power of these frequencies in the same areas (Zelano et al., 2016).

In addition to the restorative effect, taking nasal breathing following PSD also reduced the power of delta and enhanced the power of high-frequency oscillations, including alpha and beta, in some cortical areas, particularly in frontal and parietal locations. These effects may have a compensatory role in the effects of PSD on the activity of these areas.

The volunteers selected for the study did not have nasal obstruction and those with a history of nasal polyps were not included in the study. However, the effects of nasal breathing were observed more in the right hemisphere. This may be due to the dominance of the left nostril or may be related to the state of arousal, cognitive level, and degree of sleep deprivation. In any case, further studies are needed to clarify this issue.

Despite the promising findings, the results should be interpreted with caution. Future studies should replicate these findings in larger and more diverse samples to enhance statistical power and generalizability. In addition, the use of objective sleep measures, such as polysomnography or actigraphy, would strengthen the assessment of sleep deprivation and sleep quality beyond self-report indices. Finally, the absence of a crossover design limits the ability to fully control for inter-individual variability and potential order effects.

Conclusion

The findings of this study indicated that nasal breathing has a restorative effect on PSD-induced alteration in cortical activity and enhances the cognitive abilities of individuals experiencing acute sleep deprivation in the NST. Oral breathing bypasses the mechanical stimulation typically induced by nasal airflow, disrupting communication between the olfactory bulb and other brain regions, which may impair cognitive function.

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Authors' Contributions

Hanieh Riazi: acquisition of data, analysis and interpretation of data, Drafting the article; Amir Shojaei: The conception and design of the study, Revising the article for important intellectual content, Final approval of the version to be submitted.

Informed Consent Statement

Informed consent was obtained from all subjects involved in the study.

Conflict of Interest

The authors declare no conflicts of interest.

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Tables

Table 1. Characteristics of subjects in the nasal and oral respiration groups. Mean±SEM.

Experimental Groups	Age (years old)	Weight (kg)	Sleep quality index	Cognitive disability index
Nasal respiration (Male = 7, Female = 6)	27.31 ± 1.26	75.00 ± 7.08	6.38 ± 0.64	64.62 ± 4.54
Oral respiration (Male = 3, Female= 5)	29.88 ± 1.50	72.88 ± 10.59	5.75 ± 1.31	59.00 ± 6.71

Figures

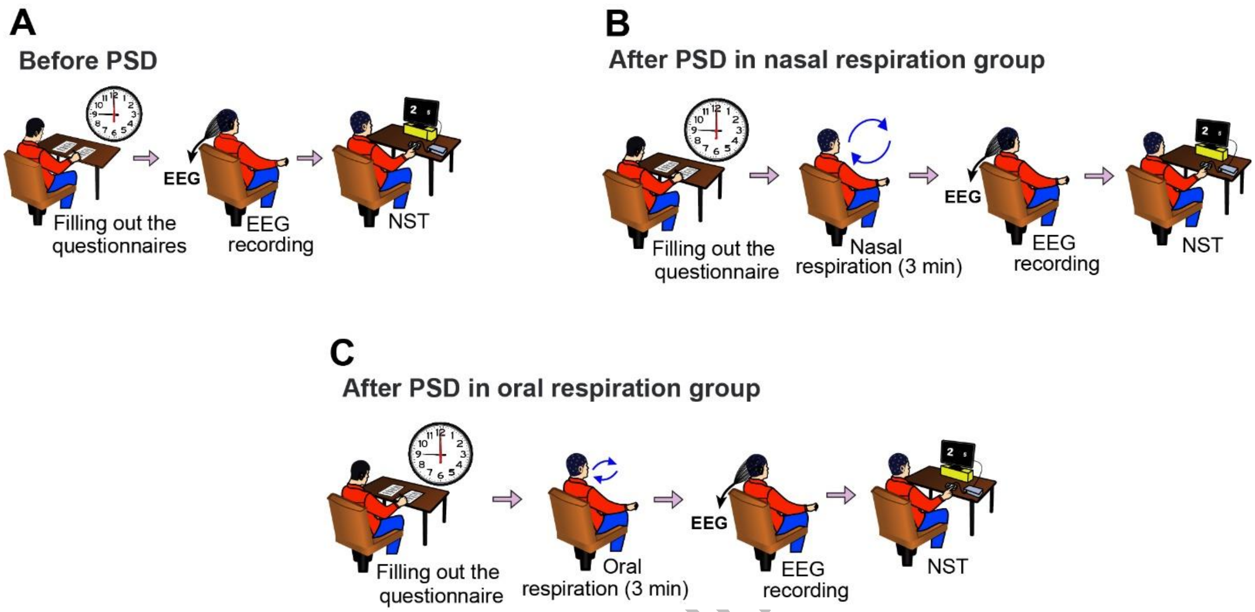

Figure 1. Design of the experiments. Part A displays the tasks that were assigned to the nasal respiration and oral respiration groups on the first day (before sleep deprivation). The tasks for the nasal respiration group and the oral respiration group on the second day (following sleep deprivation) are displayed in B and C, respectively. NST: numerical Stroop test; PSD: partial sleep deprivation.

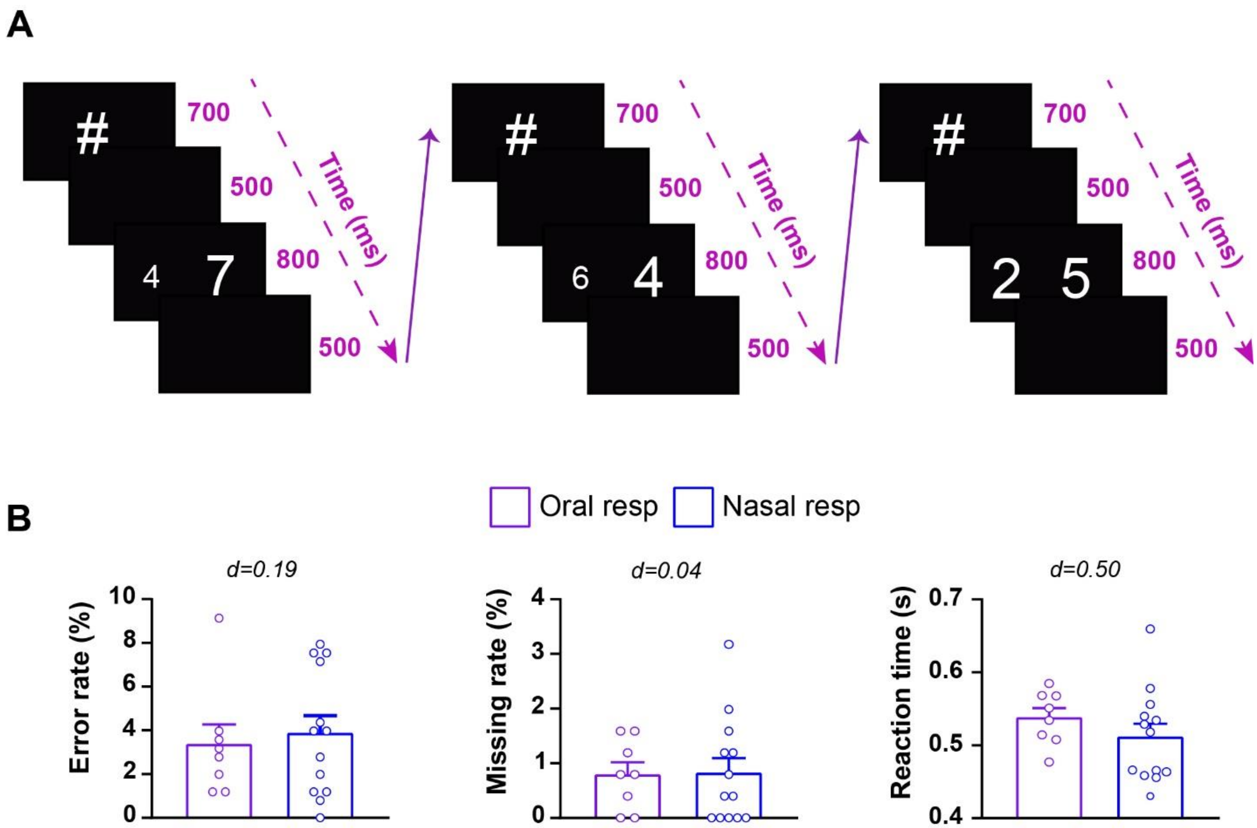

Figure 2. A) The timing procedure for showing the numbers during the NST trials. B) Comparison of NST parameters on day 1 (before sleep deprivation) between subjects in the oral respiration and nasal respiration groups. Unpaired t-test; Mean ± SEM.

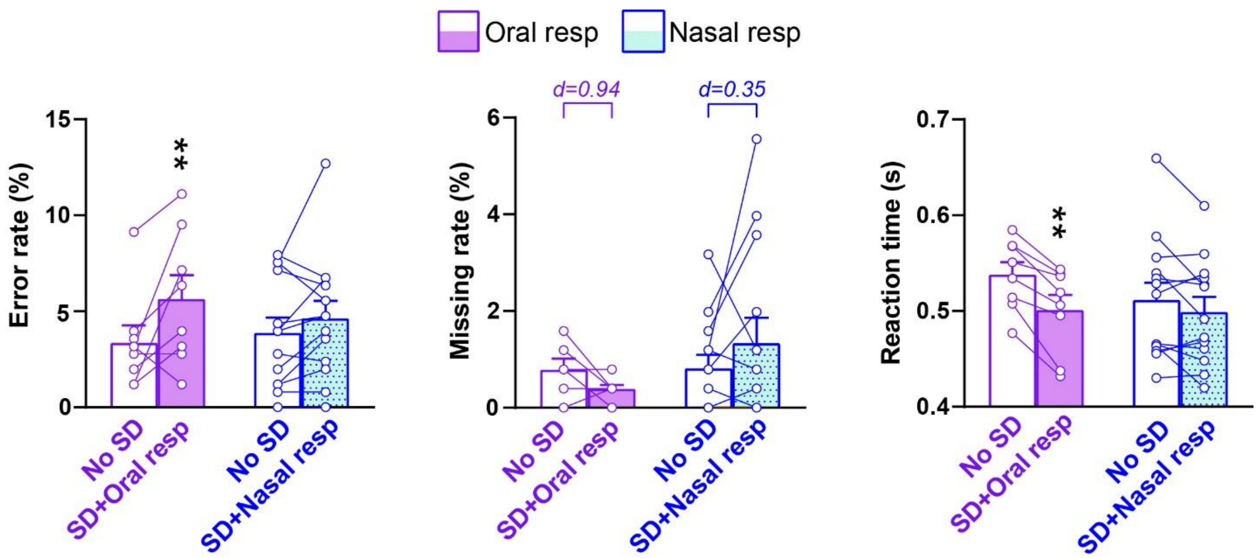

Figure 3. Impact of nasal breathing on cognitive function after PSD. ** $p < 0.01$; Two-way repeated measures ANOVA; d shows Cohen's d number; Mean ± SEM.

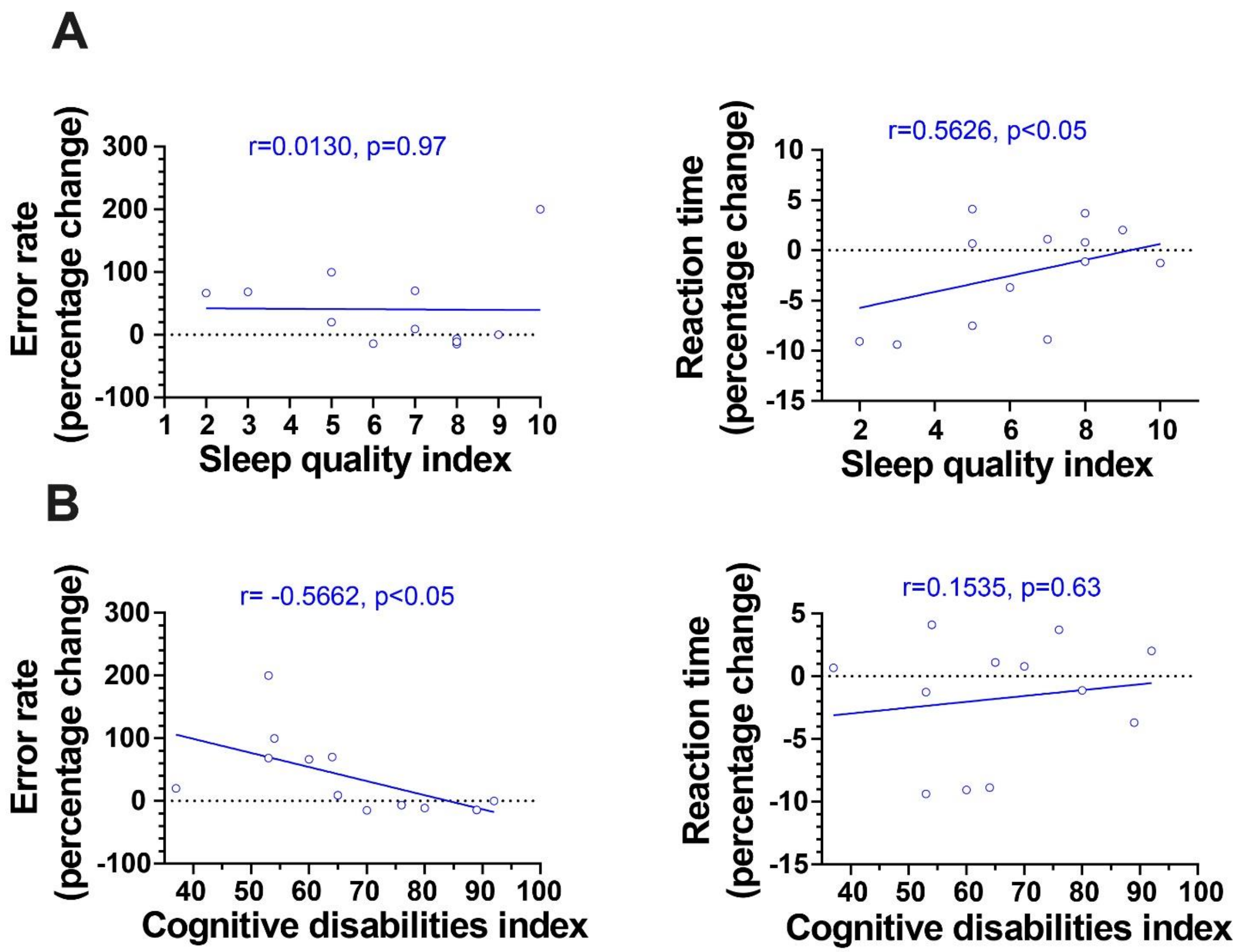

Figure 4. Correlation between nasal respiration-induced percentage changes in error rate and reaction time of participants during the NST and their baseline indices of sleep quality (A) and cognitive disability (B) after PSD. Pearson correlation; Mean ± SEM.

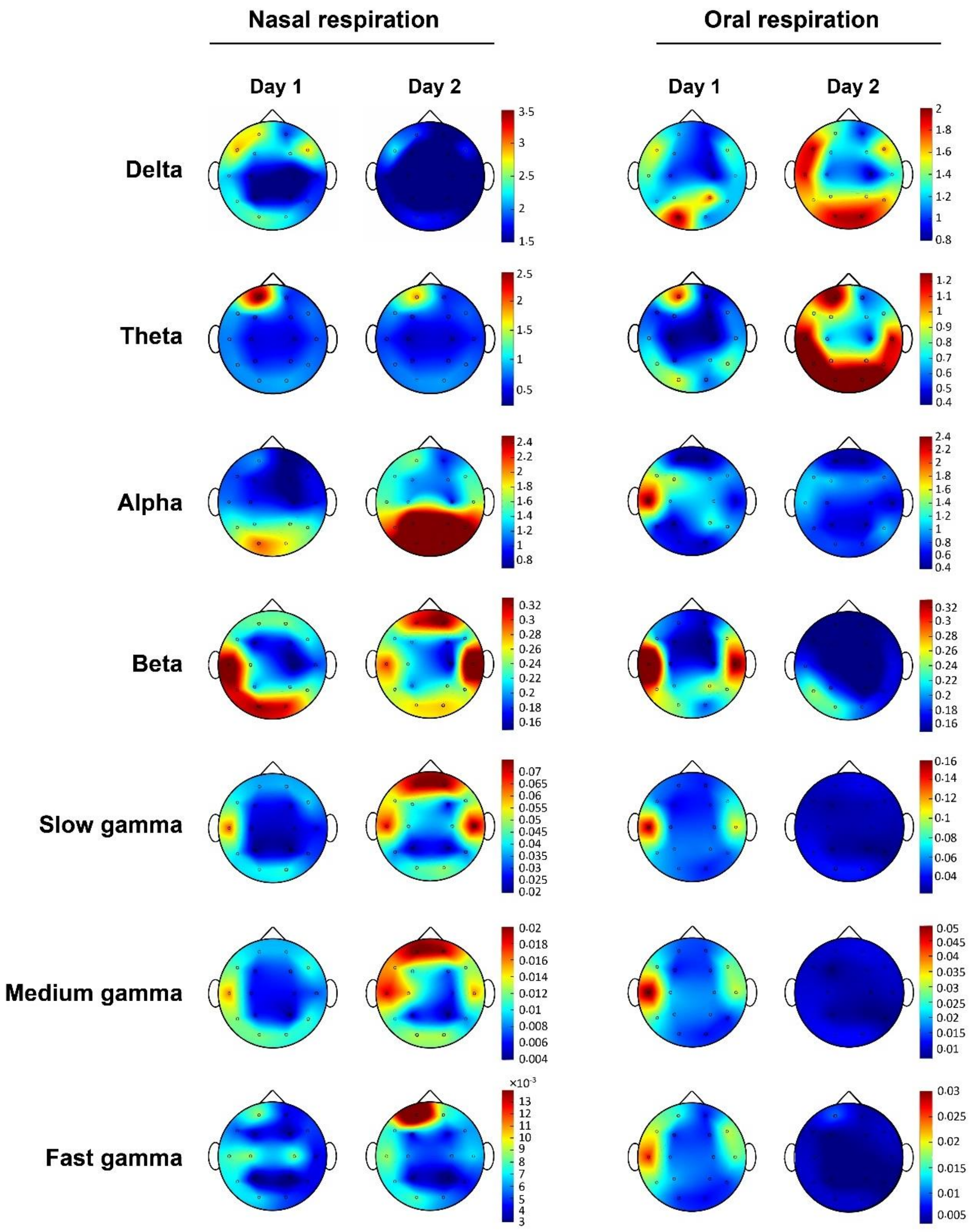

Figure 5. topographic plots representing the effect of nasal and oral breathing following one night of PSD on resting state EEG power.