

# The Effects of Fast and Slow Yoga Breathing on Cerebral and Central Hemodynamics

## Abstract

**Background:** Yoga breathing has shown to impose significant cardiovascular and psychological health benefits. **Objective:** The mechanism (s) responsible for these health benefits remain unclear. The aim of the present study was to assess the differences in cerebral and central hemodynamic responses following fast breathing (FB) and slow breathing (SB) protocols compared to breathing awareness (BA) as a control. **Methods:** Twenty healthy participants (10 males and 10 females) volunteered to take part in the study. Participants were between ages 18–55 years (group mean:  $24 \pm 5$  years), with a height of  $168.7 \pm 9.8$  cm and a weight of  $70.16 \pm 10.9$  kg. A familiarization trial including FB and SB protocols were performed by each participant at least 24 h before the testing day. The breathing protocols were designed to achieve 6 breath/min for SB and  $\sim 120$  breaths/min for FB. **Results:** FB resulted in an increase in both right prefrontal cortex (RPFC) and left prefrontal cortex (LPFC) hemoglobin difference (Hbdiff) (brain oxygenation) compared to BA ( $P < 0.05$ ). FB resulted in an increased Hbdiff in LPFC compared to RPFC SB ( $P < 0.05$ ). FB resulted in an increased Hbdiff in LPFC compared to SB ( $P < 0.05$ ). **Conclusion:** FB may be an effective yoga breathing technique for eliciting cerebral brain oxygenation indicated by increased Hbdiff. These results may be applicable to both healthy and clinical populations.

**Keywords:** Breathing, fast breathing, hemodynamics, slow breathing

## Introduction

Yoga is well recognized for mind–body and health-related benefits by combining physical postures with meditation and breath control (pranayama). The breath control aspect of yoga has been regarded as the most important component for improving health.<sup>[1,2]</sup> Consequently, yoga breathing styles (e.g., *Sukha pranayama* and *Kapalabhati pranayama*) have been implemented into treatment practices for psychological disorders, cognitive impairment, cardiorespiratory disease, and autonomic dysfunction.<sup>[3–7]</sup>

The underlying mechanisms of how focused breathing affects these disorders are highly complex, and researchers have proposed a simplified model termed the respiratory-central nervous system (CNS)-cardiovascular interaction network.<sup>[8,9]</sup> The rate and depth of breathing (respiratory component) appears to influence autonomic (CNS) regulation, which directly affects

cardiovascular function. Increased central hemodynamics (heart rate [HR], stroke volume (SV), and blood pressure) in response to breathing are also linked to heightened oxygenation in the prefrontal cortex (PFC) regions of the brain.<sup>[10]</sup>

For example, *Kapalabhati pranayama* is a form of fast yoga breathing (FB) and has been shown to activate sympathetic outflow resulting in increased HR, systolic blood pressure, and brain oxyhemoglobin (HbO<sub>2</sub>) and total hemoglobin (totalHb) in the PFCs.<sup>[4,11]</sup> This style of breath control requires shallow, quick, rhythmic nostril breathing accomplished primarily using the abdominal muscles. It is commonly performed with two shallow breaths per second for a duration of 1 min ( $\sim 120$  breaths/min) or less followed by a recovery period typically lasting at least 1 min.<sup>[4,11,12]</sup>

*Sukha pranayama* is a form of slow breathing (SB) through the nostrils, performed by 5 s of slow inhalation followed by 5 s of slow exhalation.<sup>[12,13]</sup> This pattern of breathing is thought to increase

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parasympathetic control and lower sympathetic outflow by activating bronchi and bronchiole stretch receptors (Hering–Breuer reflex) during large inspirations.<sup>[14,15]</sup> Parasympathetic activation through SB has resulted in immediate reductions in oxygen consumption, HR, systolic blood pressure, and diastolic blood pressure.<sup>[14,16]</sup> In addition, healthy adults and those with hypertension who participated in 12 weeks of slow pranayama breathing demonstrated significant decreases in resting HR and blood pressure, which were attributed to parasympathetic reactivity.<sup>[17,18]</sup> For these reasons, SB has been attributed to improving symptoms of stress and anxiety, along with activating brain waves linked to mental quiescence, and mind–body calming, whereas FB has been shown to increase central hemodynamics and brain oxygenation and is thought to promote vigilance and focus.<sup>[19]</sup>

The use of portable brain imaging technologies such as functional near-infrared spectroscopy (fNIRS) has allowed researchers to gain insight into brain oxygenation (also called brain activation) with yoga breathing practice. Cerebral blood flow is thought to be regulated by the autonomic nervous system where patients with sympathetic failure have lower brain perfusion compared to healthy control (intact sympathetic nervous system [SNS]).<sup>[20]</sup> High-frequency breathing may activate the SNS and promote cerebral blood flow. However, Telles *et al.* reported no increase in PFC oxygenation after 15 min of high-frequency breathing at a rate of 60 breaths/min among healthy male participants and stated that the frequency may need to be faster to promote activation of SNS.<sup>[21]</sup> Bhargav *et al.* demonstrated an increase in PFC oxygenation after only 1 min of high-frequency breathing at a rate of 120 breaths/min among a healthy group of individuals.<sup>[4]</sup> Therefore, it appears that short, high-frequency breathing at nearly 2 breaths/s is effective for promoting brain blood flow. It is important to highlight that PFC oxygenation and cardiovascular responses (e.g., HR or blood pressure) to breathing techniques have not been measured together in the same study.<sup>[4,21]</sup>

Therefore, the purpose of the present study was to evaluate if prefrontal cortex (PFC) brain oxygenation is influenced by breathing frequency and to further determine if central hemodynamics (HR, SV, and cardiac output [CO]) play a role in mediating the PFC oxygenation changes. We hypothesized that FB would result in heightened cerebral oxygenation and that this response would be associated with increased central hemodynamic responses compared to SB.

## Methods

### Participants

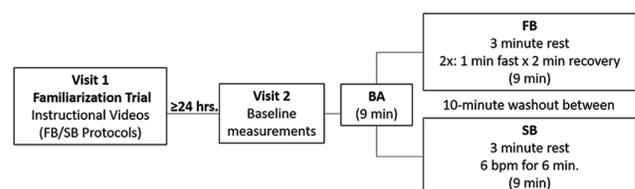
Twenty participants (10 males and 10 females) volunteered to take part in the present study. Participants were between the ages 18–55 years (mean:  $24 \pm 5$  years), with

a height of  $168.7 \pm 9.8$  cm, a weight of  $70.16 \pm 10.9$  kg, and a resting blood pressure of  $112 \pm 9.07$  mmHg. Participants had no history of yoga/meditation practice and no known cardiovascular, metabolic, kidney, or liver disease. Participants were free of any diagnoses of hypertension, diabetes, respiratory disease, autonomic dysfunction, seizure disorder, and recent abdominal surgery. Participants also reported no use of autonomic controlling medications, chronic tobacco, and marijuana use. All study procedures were performed in the Exercise Physiology Laboratory at UNM. All participants provided written consent in accordance with the Declaration of Helsinki. The study was approved by the UNM Institutional Review Board.

### Study protocol

Participants arrived at the laboratory on two occasions, at least 24 h apart [Figure 1]. Visit 1 was a familiarization trial which required participants to watch and participate in an instructional video led by a trained yoga practitioner. The video was approximately 25 min long and provided verbal and visual cues of each yoga breathing protocol: breathing awareness (BA), SB, and FB. Participants practiced each breathing technique along with the yoga instructor until the video was complete. Each participant was provided the option to watch the video as much as needed until they felt confident in their abilities to perform each breathing protocol before visit 2.

For visit 2 (study protocol), participants were instructed to arrive at the laboratory 4 h postprandial and avoid consumption of caffeine within 12 h. Baseline measurements included body weight measured on a digital scale (Holtain Limited, Crymych, Dyfed, Great Britain) to the nearest 0.1 kg and height using a stadiometer (MedWeight MS-3900, Itin Scale Company, Brooklyn, NY, USA) to the nearest 1 cm. Noninvasive impedance cardiography (PhysioFlow Enduro, PhysioFlow, Bristol, PA, USA) was used for the measurement of central hemodynamic variables (HR, SV, and CO). Electrodes were placed on the chest and back according to manufacturer guidelines. Participants were then instructed to remain seated, while the fNIRS (OctaMon, Artinis Medical Systems, Elst, The Netherlands) prefrontal cortex headband was placed on the forehead according to manufacturer guidelines. Then, a resting blood pressure was obtained using a stethoscope and an arm aneroid



**Figure 1: Study timeline.** BA = Breathing awareness, FB = Fast breathing, SB = Slow breathing, bpm = Breaths per minute

sphygmomanometer placed around the left arm (3M Littmann Select, USA).

Visit 2 began with participants completing the BA protocol followed by 10 min seated rest (in an armchair). Then, participants completed the SB and FB techniques in randomized order. Between the SB and FB techniques, a 10-min washout period required subjects to remain seated and breathe at a self-selected rate. During BA, SB, and FB techniques, central hemodynamics and PFC hemodynamics were recorded continuously after 3 min of seated rest. The protocols of each breathing technique are outlined below.

### **Breathing awareness, fast breathing, and slow breathing protocols**

#### *Breathing awareness protocol*

The BA protocol required normal breathing while sitting in a comfortable posture on a yoga blanket with legs crossed and an erect trunk and back (seated yoga posture). Total BA trial time was 9 min (3 min of rest followed by 6 min of normal breathing).

#### *Fast breathing protocol*

An instructional video guided the participant through the FB protocol. The video consisted of normal breathing for 3 min (baseline period) followed by FB for 1 min. The 1-min of FB was followed by 2 min of normal breathing (recovery). This protocol was repeated for two cycles (6 min total). Total FB trial time was 9 min (3 min of rest followed by 1 min of FB and then 2-min recovery [repeated two times]). The participant was instructed to follow the breathing pattern of the video instructor. Breathing frequency was monitored by a research technician through both visual and auditory inspections. Each expiration was counted as a breath. The FB protocol required fast nasal exhalation followed by recoil inhalation with a goal of reaching 120 breaths/min; however, participants in the present study achieved a range of 100–116 breaths/min during this protocol.

#### **Slow breathing protocol**

An instructional video and computerized interval breathing program guided participants through the SB protocol. The video consisted of normal breathing for 3 min (baseline period) followed by 6 min of the SB protocol. SB required participants to perform nasal inhalation for 5 s and nasal exhalation for 5 s using both thoracic and abdominal muscles equating to 6 breaths/min for a total of 6 min. The computerized interval breathing program was set at a 5-s inhalation and 5-s exhalation pace which informed participants when to start inhalation and when to start exhalation. SB rate was assessed by the ability of the participants to inhale and exhale to the pace of this program. Total SB protocol was 9 min (3 min of rest followed by 6 min of SB).

### **Cerebral and central hemodynamic measurements**

#### *Prefrontal cortex oxygenation*

fNIRS was used to measure PFC oxygenation at two wavelengths (760 nm and 850 nm) during 9 min of BA, FB, and SB trials (3 min of rest and 6 min of intervention). Differentiation of two dynamic absorbers of this device allows for the identification of the change in oxygenated hemoglobin (oxyHb) and deoxygenated hemoglobin (deoxyHb) of the brain. PFC hemodynamics and brain activation are indicative of changes in these values.<sup>[22]</sup> Four light-emitting diode optodes combined with one receiver were placed over the right and left hemispheres (4 transmitters and 1 receiver) of the PFC (8 × 2 configuration). Placement of the fNIRS was adopted by the modified international electroencephalogram 10–20 system.<sup>[23,24]</sup> The fNIRS cap was located 2 centimeters (cm) above the nasion and centering on the Fpz location.<sup>[25]</sup> The source–detector distance was 3.5 cm, which is recommended as an optimal distance to detect cortical activity among adults.<sup>[26,27]</sup> Short-separation channels were not implemented in the current study due to potential for additional error in data analyses.<sup>[28]</sup> A signal sampling rate of 10 Hz was used. Data were processed using Artinis software at a band-pass filter between 0.01 and 0.50 Hz, and concentration changes in oxyHb and deoxyHb were calculated based on a modified Beer–Lambert approach.<sup>[27]</sup> The left prefrontal cortex (LPFC) regions included Fp1 and F7 using channels 1–4. The right prefrontal cortex (RPFC) included Fp2 and F7 using readings from channels 5–8. Relative concentration changes for oxyhemoglobin (HbO<sub>2</sub>) and deoxyhemoglobin (HHb) were measured from resting baseline within each trial using the first 10 s and defined as 0 μmol. Oxyhemoglobin change is an indicator of PFC oxygenation and is used as an indirect measure of PFC activation.<sup>[21,27]</sup> In addition, oxyhemoglobin difference (Hbdiff) is considered a sensitive measure of PFC oxygenation due to the high correlation with cerebral blood flow and mean arterial pressure changes.<sup>[22,29]</sup>

#### *Central hemodynamics*

Participants were prepped for the measurement of hemodynamic function using thoracic impedance cardiography. Data were collected during 9 min of BA, FB, and SB trials (3 min of rest and 6 min of intervention). Electrodes (Skintact electrocardiography electrodes) were placed on the neck, sternum, rib cage, and erector spinae. Participants were connected to the PhysioFlow leads according to manufacturer guidelines. Calibration was performed before each breathing trial with the participant seated. Mean central hemodynamic variables (CO, SV, and HR) were obtained by averaging 5-s intervals during the breathing protocols.

#### **Statistical analysis**

The sample size was determined *a priori* using mean and standard deviation values (power of 0.80 and alpha

of 0.05) for oxyhemoglobin. The criterion for selected studies was acute breathing interventions and changes in oxyhemoglobin using fNIRS technology. All results are expressed as means  $\pm$  SD and checked for homogeneity of variance and normality. Data from the leftmost side of the frontal region (fNIRS channels 1–4) and rightmost region (fNIRS channels 5–8) were combined to represent LPFC and RPFC, respectively. One-way ANOVA with repeated measures was used to analyze Hbdiff and HbO<sub>2</sub> changes in the LPFC and RPFC between trial conditions (3 conditions: BA, SB, and FB) and used to evaluate the effect of breathing techniques on regional PFC Hbdiff (2 levels: LPFC and RPFC). Similarly, a one-way ANOVA with repeated measures was used to compare average HR, SV, and CO for each trial (BA, FB, and SB).

## Results

### Cerebral oxygenation during breathing techniques

There was a significant main effect for relative change in Hbdiff,  $F(5, 395) = 26, P < 0.001$ , and HbO<sub>2</sub>,  $F(5, 392) = 22, P < 0.001$ . *Post hoc* comparisons revealed significantly higher relative changes in Hbdiff and HbO<sub>2</sub> in LPFC and RPFC regions in the FB trial compared to the same regions in the BA and SB trials [Table 1 for data]. No differences were detected between regions within any trial.

### Central hemodynamics during breathing techniques

There were no significant main effects for breathing on mean HR,  $F(2, 38) = 3.5, P = 0.06$ , or SV,  $F(2, 38) = 3.20, P = 0.06$ . A significant main effect was observed CO,  $F(2, 38) = 4.14, P = 0.03$ . However, Tukey's *post hoc* testing revealed no significant difference between breathing protocols. There was a trend between FB vs. SB on CO ( $6.12 \pm 1.75$  vs.  $5.60 \pm 1.20$  L/min,  $P = 0.07$ ).

## Discussion

This study assessed the effects of *Sukha pranayama*, SB, and *Kapalabhati pranayama*, FB, on both cerebral oxygenation and central hemodynamics among healthy adults. Key findings suggest that FB elicits a significant increase in cerebral oxygenation as observed by Hbdiff and HbO<sub>2</sub> changes in the right and left PFCs compared to BA and SB. These findings suggest that FB may be an effective

strategy for increasing brain oxygenation, which may also indicate higher PFC activation.<sup>[21,30]</sup>

In the present study, an increase in Hbdiff and HbO<sub>2</sub> was observed in both PFC regions (LPFC and RPFC) after a 100–116 breaths/min focused breathing trial compared to normal breathing and SB (6 breaths/min). Similarly, Bhargav *et al.* reported an increase in bilateral PFC oxyhemoglobin and totalHb after 1 min of FB at 120 breaths/min among healthy individuals.<sup>[4]</sup> Contrary to these findings, Telles *et al.* reported no increase in cerebral brain oxygenation following 15 min of FB at 60 breaths/min among healthy males.<sup>[21]</sup> Our findings further demonstrate that the stimulus of breathing rate may be of most importance for increasing sympathetic discharge and PFC activation among healthy adults.<sup>[4]</sup> Previous findings suggest the stimulus of breathing rate is linked to sympathetic discharge with faster breathing frequencies eliciting greater sympathetic activity among male humans.<sup>[31]</sup> This process is believed to be modulated by carotid chemoreceptors along with activation of central respiratory motor outputs.<sup>[32]</sup> During rapid breathing, the chemoreceptors increase sympathetic outflow by sensing a decrease in the partial pressure of carbon dioxide, and rapid muscle actions of the diaphragm and expiratory muscles provide afferent feedback that influences sympathetic drive.<sup>[32,33]</sup> Modulation of SNS discharge through central pattern generators has also been a suggested mechanism that explains the effect of rapid breathing on the SNS.<sup>[34]</sup> The results of our study demonstrate that FB increases PFC oxygenation among healthy adults; however, we did not measure autonomic activity to determine if the responses were mediated through sympathetic discharge.

Previous results regarding the effect of SB on PFC oxygenation are mixed. Singh *et al.* demonstrated that SB performed through the right nostril for 10 min at a rate of 4 breaths/m increases RPFC and decreases LPFC brain oxygenation among healthy individuals.<sup>[30]</sup> In current study, no increase in PFC oxygenation was detected in the SB trial for either PFC region (both RPFC and LPFC). The conflicting results may be due to single nostril breathing and the slower rate in the Singh *et al.* study. Evidence suggests that the right nostril can activate the SNS, whereas the left nostril leads to an increase in vagal tone.<sup>[30,35]</sup> In both nostril breathing, deep inspiration

**Table 1: Means and standard deviations of prefrontal cortex hemoglobin changes during fast breathing, slow breathing, and control trials (breathing awareness)**

Variable	PFC	Mean $\pm$ SD			P
		BA	FB	SB	
HbO <sub>2</sub> ( $\mu$ mol/L)	Left	0.68 $\pm$ 1.87	2.34 $\pm$ 2.88* <sup>^</sup>	0.22 $\pm$ 2.55	*= $<$ 0.001, <sup>^</sup> = $<$ 0.001
	Right	0.52 $\pm$ 1.68	2.12 $\pm$ 1.46* <sup>^</sup>	-0.05 $\pm$ 1.54	*= $<$ 0.018, <sup>^</sup> = $<$ 0.001
Hbdiff ( $\mu$ mol/L)	Left	0.94 $\pm$ 2.02	3.06 $\pm$ 2.89* <sup>^</sup>	1.07 $\pm$ 2.27	*= $<$ 0.001, <sup>^</sup> =0.001
	Right	0.63 $\pm$ 1.61	2.81 $\pm$ 1.42* <sup>^</sup>	0.75 $\pm$ 1.66	*=0.002, <sup>^</sup> = $<$ 0.001

\*Significantly greater than BA ( $P < 0.05$ ), <sup>^</sup>Significantly greater than slow ( $P < 0.05$ ). HbO<sub>2</sub>=Oxyhemoglobin, Hbdiff=Hemoglobin difference, BA=Breathing awareness, FB=Fast breathing, SB=Slow breathing, PFC=Prefrontal cortex, SD=Standard deviation

is thought to decrease vagal tone leading to small rise in HR, whereas slow exhalation increases vagal tone causing a small decline in HR. Therefore, these actions may cancel out autonomic control of HR during SB. Single nostril breathing or alterations in the inhalation and exhalation cycle periods may lead to an overall increase in vagal tone.

It is difficult to identify the mechanisms that explain how breathing frequency increases PFC oxygenation. Previous researchers have identified a link between respiratory activation of sympathetic drive leading to an increase in cardiac function and PFC oxygenation.<sup>[8,9]</sup> No significant changes in central hemodynamics were detected in the current study. However, a significant main effect for breathing rate was detected for CO, and trend for a higher CO in the FB trial ( $6.12 \pm 1.75$  L/min) compared to the SB trial ( $5.60 \pm 1.20$  L/min) was observed. It has been well documented that the average rate of cerebral blood flow accounts for ~ 15% of CO.<sup>[10]</sup>

Brain function is highly dependent on oxygen supply, and therefore, monitoring oxygenation has implications for clinical practice and research.<sup>[10,36,37]</sup> PFC activation in response to aerobic exercise has been linked to improved cognitive functioning among healthy and those with mental disorders.<sup>[38,39]</sup> The heightened PFC oxygenation that occurred in response to FB may also indicate increased PFC activation. Therefore, these findings may be applicable for enhancing mental stimulation in conditions involving impaired cerebral oxygenation such as ischemic stroke, coronary artery disease, type 2 diabetes, and sleep apnea.<sup>[39-42]</sup>

### Limitations

It is important to note that this study has potential limitations. First, the sample size is small. Second, the stimulus of breathing rate may be considered a primary stimulus for increasing sympathetic discharge and PFC activation. In the present study, we did not assess physiologic signals linked to increases in sympathetic discharge such respiratory CO<sub>2</sub>. In addition, we did not assess tidal volume and breathing frequency which are major components of minute ventilation.<sup>[43]</sup> Future studies investigating the effects of yoga breathing on PFC activation may benefit from assessing breathing frequency using a contact-based technique such as CO<sub>2</sub> sensing capnography.<sup>[43]</sup> Measuring these physiologic variables in addition to using fNIRS technology will lead to further understandings of mechanism driving PFC activation during the breathing component of yoga.

### Conclusion

The primary finding of this study indicates that FB (120 breaths/min) stimulates cerebral brain oxygenation in both the right and left PFCs and may be an effective approach for improving brain blood flow in healthy humans and clinical populations. Based on these data, it is unknown

if central hemodynamics mediate the increase in PFC oxygenation; however, a small trend was detected for an increase in CO in response to FB.

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### Conflicts of interest

There are no conflicts of interest.

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