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## THÈSE

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**Slow-paced breathing and cardiac vagal activity: Influence on stress regulation, sleep, and cognitive executive performance**

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**COMETE**  
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*"Yes, they are elves," Legolas said.  
"and they say that you breathe so loud  
they could shoot you in the dark."*  
Sam hastily covered his mouth.  
**J.R.R. Tolkien – Lord of the Rings**

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### 1.3 Abbreviations Index

**AOSPAN:** Automated Operation Span

**cpm:** cycles per minute

**CVA:** Cardiac Vagal Activity

**ECG:** Electrocardiogram

**HRV:** Heart Rate Variability

**HF-HRV:** High-Frequency Heart Rate Variability

**MCST:** Modified Card Sorting Test

**min:** minute

**mL:** milliliters

**ms:** millisecond

**PS:** Perceived Stress

**RMSSD:** Root Mean Square of Successive Differences

**RSA:** Respiratory Sinus Arrhythmia

#### 1.4 Publications included in the PhD thesis

**Laborde, S.**, Allen, M.S., Gohring, N., and Dosseville, F. (2017). The effect of slow-paced breathing on stress management in adolescents with intellectual disability. *Journal of Intellectual Disability Research* 61(6), 560-567. doi: 10.1111/jir.12350. (IF = 1.941)

**Laborde, S.**, Hosang, T., Mosley, E., and Dosseville, F. (2019). Influence of a 30-day slow paced breathing intervention compared to social media use on subjective sleep quality and cardiac vagal activity. *Journal of Clinical Medicine* 8. doi: 10.3390/jcm8020193. (IF = 5.688)

**Laborde, S.**, Allen, M., Borges, U., Hosang, T., Furley, P., Mosley, E., et al. (submitted). The influence of slow-paced breathing on executive function.

**Laborde, S.**, Lentès, T., Hosang, T. J., Borges, U., Mosley, E., & Dosseville, F. (2019). Influence of slow-paced breathing on inhibition after physical exertion. *Frontiers in Psychology*, 10. doi:10.3389/fpsyg.2019.01923 (IF = 2.129)

#### 1.5 Scientific communications related to this PhD thesis

##### 1.5.1 Symposia

**Laborde, S.**, Dosseville, F. (2019). Heart rate variability as a self-regulation marker. Symposium organized at the 15th European Congress for Sport Psychology (FEPSAC), 15-20th July 2019, Münster, Germany.

**Laborde, S.**, Dosseville, F. (2018). Neuroenhancement in sport psychology. Symposium organized at the 6ème Congrès International de la Société Française de Psychologie du Sport, 13-15th June 2018, Lausanne, Switzerland.

**Laborde, S.** (2018). Self-regulation and cardiac vagal activity in sport psychology. Symposium organized at the 50. Jahrestagung der Arbeitsgemeinschaft für Sportpsychologie, 10-12th May 2018, Cologne, Germany.

**Laborde, S.**, Dosseville, F. (2017, July) Personality-trait-like individual differences and psychophysiology; Symposium presented at the 14th International Congress of Sport Psychology (ISSP), Sevilla (Spain)

##### 1.5.2 Oral talks

**Laborde, S.**, Dosseville, F. (2018). Slow paced breathing in sport psychology. Paper presented at the 6ème Congrès International de la Société Française de Psychologie du Sport, 13-15th June 2018, Lausanne, Switzerland.

**Laborde, S.** (2017, July) Vagal tank theory: a functional approach to self-regulation resources, bridging the gap between neurophysiology, cognitive psychology, and social

psychology. Paper presented at the 14th International Congress of Sport Psychology (ISSP), Sevilla (Spain)

**Laborde, S., Hoffmann, S., Englert, C., & Raab, M. (2017).** Self-control revisited: The case for a motivational neurovisceral perspective on self-control. Paper presented at the 49. Jahrestagung der Arbeitsgemeinschaft für Sportpsychologie, 25-27th May 2017, Bern, Switzerland.

#### 1.5.3 Posters

**Laborde, S., Hosang, T., Mosley, E., & Dosseville, F. (2019, September)** *Influence of a 30-day slow-paced breathing intervention compared to social media use on subjective sleep quality and cardiac vagal activity.* Poster presented at the 59th congress of the Society for Psychophysiological Research (SPR), Washington DC (USA)

#### 1.5.4 Applied workshops

**Laborde, S., Mosley, E. (2019).** Heart rate variability in sport psychology: applications of the vagal tank theory. Workshop organized at the 15th European Congress for Sport Psychology (FEPSAC), 15-20th July 2019, Münster, Germany.

**Laborde, S. (2019, May).** Herzratenvariabilität im Sport und Gesundheitsmanagement. Workshop (Weiterbildung) organized at the German Sport University Cologne (Germany), 11<sup>th</sup>-12<sup>th</sup> May 2019

**Laborde, S. (2019, May).** Herzratenvariabilität im Sport und Gesundheitsmanagement. Workshop organized at the University of Heidelberg (Germany), 2<sup>nd</sup>-3<sup>rd</sup> May 2019

**Laborde, S., Ackermann, S. (2018, November).** Herzratenvariabilität im Sport und Gesundheitsmanagement. Workshop (Weiterbildung) organized at the German Sport University Cologne (Germany), 24<sup>th</sup>-25<sup>th</sup> November 2018

**Laborde, S., Ackermann, S. (2018, June).** Herzratenvariabilität im Sport und Gesundheitsmanagement. Workshop (Weiterbildung) organized at the German Sport University Cologne (Germany), 2<sup>nd</sup>-3<sup>rd</sup> June 2018

**Laborde, S. (2018).** Heart rate variability in applied sport psychology. Workshop organized at the 50. Jahrestagung der Arbeitsgemeinschaft für Sportpsychologie, 10-12th May 2018, Cologne, Germany.

**Laborde, S. (2016)** Evaluación de la variabilidad de la frecuencia cardiaca, un indicador psicofisiológico de la autorregulación, XXIV Congreso Internacional FOD “Educación Física, Deporte y Ciencias Aplicadas”, 9-11 de Noviembre, Monterrey, Nuevo León, México

## 1.6 Awards related to this PhD thesis

- Science Slam « Ma thèse en 180s », Region Normandy 1<sup>st</sup> Prize Jury (March 2019, Le Havre); then participation to the semi-final in Paris and to the national final in Grenoble (<https://www.youtube.com/watch?v=iCHv4kTHTWI>)
- Science Slam DAAD (German Academic Exchange Service): 1<sup>st</sup> Prize Public (January 2019, German Embassy Paris) (<https://www.youtube.com/watch?v=-AS6-YFWtds>)
- Teaching Prize (2<sup>nd</sup> Prize Seminar) German Sport University Cologne (*teaching concept based on the 30 days slow-paced breathing intervention of Study 2*) <https://www.dshs-koeln.de/hochschule/studium-und-lehre/lehrpreise-foerderprogramme/lehrpreise/preistraegerinnen-2019/>



## 2 Preface

The act of breathing is so natural to us, that we usually don't think about it. But breathing can also be voluntarily controlled and optimized. As I discovered the scientific foundations underlying the voluntary control of breathing, and how much it could help people to enhance a large range of self-regulation phenomena in their lives (e.g., stress management, sleep, cognitive functioning), my curiosity got stimulated to understand more about it. Slow-paced breathing is the journey I am inviting you to embark on now within this PhD thesis.

Choosing this topic made me realize once more how the brain and the body are intrinsically connected, and how much we can learn from embracing a psychophysiological approach. Along my readings and the discoveries I made during this journey, I wished I had access to many more methods to investigate my phenomenon of interest, and it was sometimes hard to accept that I had to delay those endeavors for the future. Anyway, do see this work as a plea to systematically investigate the “interrelationships between the physiological and psychological aspects of brain and behavior”<sup>1</sup> that represents the core of psychophysiology, given this is definitely to me the most exciting (and most appropriate?) road to discover the scientific truth about human behavior (quite a bold statement to start with I admit, but oh well, this is a preface, I use the chance to plead for my scientific champion's discipline).

Very often in this PhD journey, I got to read about mind-blowing scientific discoveries that keep reminding me how much we as researchers “stand on the shoulder of giants”. If I had to name only one of those, that would be the work of Claude Bernard in the 19<sup>th</sup> century, considered as the father of neurosciences, laying the ground for the neurovisceral integration

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<sup>1</sup>Psychophysiology - <https://onlinelibrary.wiley.com/page/journal/14698986/homepage/forauthors.html>

## Preface

model and the understanding of the role of the vagus nerve in the heart-brain connection (Thayer & Lane, 2009).

As a final foreword, I would like to clarify that if I do use the “I” pronoun in this manuscript, this is only to reflect the fact that I initiated this work conducted to constitute my PhD thesis, however it is by no mean an attempt to disregard the precious and invaluable help of my colleagues sharing co-authorship with me on the related studies. Any direct or indirect help related to this PhD thesis is further acknowledged at the end of this manuscript (p.138).

*Pippin: “It’s so quiet”*  
*Gandalf: “It’s the deep breath before the plunge”*  
**J.R.R. Tolkien – Lord of the Rings**

### 3 Introduction

Since thousands of years, breathing techniques are an essential aspect of most meditative and relaxation practices (Russo, Santarelli, & O'Rourke, 2017; Zaccaro et al., 2018). Chinese (Xiuling, 2003) and Hindu (van Lysbeth & Cooper, 2007) traditions recognize breathing as strongly influencing the functioning of the brain and of the body. In Hindu philosophy for example, the Sanskrit word for breath “prana” means both breath and energy at the same time, while “prana-yama” - the stop/control but also the rising/expansion of breath – is a set of breathing techniques aiming at consciously regulating one or several parameters of respiration, such as breathing frequency, deepness, or the inspiration/expiration ratio (Zaccaro et al., 2018).

Modern psychophysicologists have been building upon these ancient techniques to address some of the main challenges of our time, such as managing stress and optimizing cognitive performance. Specific voluntary respiratory patterns have been identified to optimize the adaptation to these challenges (Lehrer & Gevirtz, 2014; Van Diest et al., 2014; Vlemincx, Van Diest, & Van den Bergh, 2016; Zaccaro et al., 2018), and I will focus in this PhD thesis on those referred to as **slow-paced breathing (SPB)** techniques. SPB (based on Lehrer & Gevirtz, 2014; Lehrer, Vaschillo, & Vaschillo, 2000) refers to the voluntary control of the duration of the inhalation and exhalation phases (“paced”). Breathing is performed at a slower pace (around 6 cycles per minute) than spontaneous breathing, which is usually between 12 and 20 cycles per minute in adults (L. Sherwood, 2006).

So far, a large stream of research has focused on the effects of SPB combined with biofeedback, where the person practicing the breathing technique is equipped with sensors connected to devices displaying the physiological effects of SPB on the body (Gevirtz, 2013; Goessl, Curtiss, & Hofmann, 2017; Kennedy & Parker, 2018; Wheat & Larkin, 2010; Yu, Funk,

Hu, Wang, & Feijs, 2018; Zaccaro et al., 2018). However, even if the effectiveness of such interventions received extensive support, the fact that their implementation requires the use of additional technology makes them not directly accessible to everyone. My goal in this PhD thesis was to investigate further the effects of SPB without the use of biofeedback, given preliminary research argues that the psychophysiological effects would be mostly similar (Wells, Outhred, Heathers, Quintana, & Kemp, 2012), and given its implementation would be accessible to a broader population. **Based on the neurovisceral integration model (Smith, Thayer, Khalsa, & Lane, 2017; Thayer, Hansen, Saus-Rose, & Johnsen, 2009) and on the resonance model (Lehrer & Gevirtz, 2014), I aim in this PhD thesis to investigate the effects of SPB without biofeedback on phenomena related to several aspects playing an important role in human adaptation, namely stress, sleep, and executive functioning.**

To this end, I will first detail the main physiological mechanisms underlying breathing, then the coupling between the respiratory and cardiorespiratory functions, the way SPB is thought to improve this coupling and in turn influence positively self-regulation processes, and the role the vagus nerve is suggested to play in this optimization. After stating the main research question driving this PhD thesis and its associated hypotheses, four experimental studies will be presented. Finally, a general discussion will integrate the findings of the four experimental studies, and delineate the challenges and avenues for future research.

## *Theoretical Background*

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### 4 Theoretical background

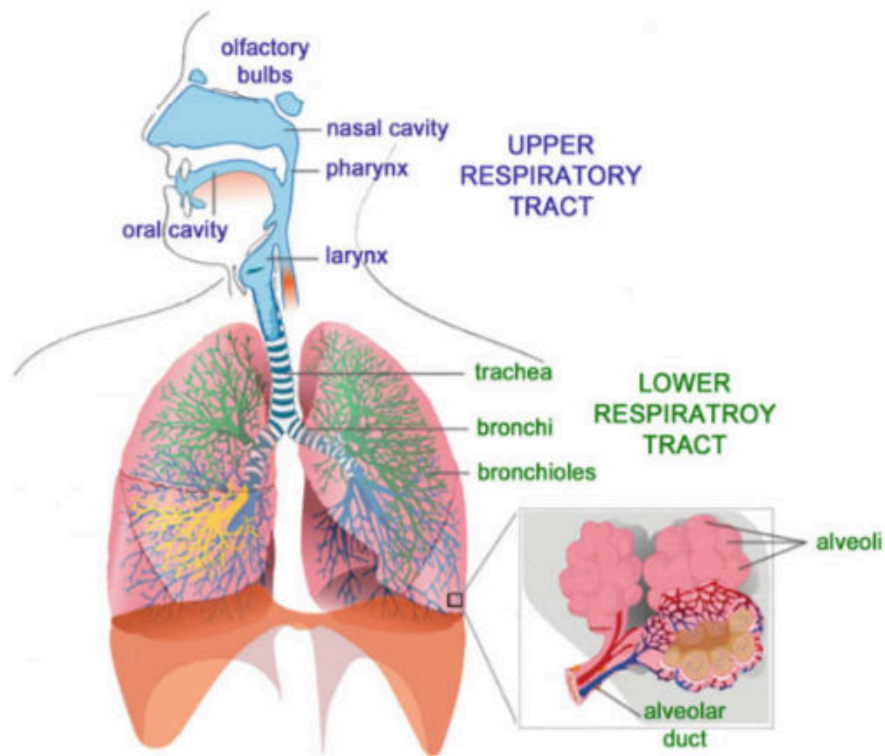
#### 4.1 Breathing

##### 4.1.1 Main role: Gas exchange between the body and the environment

Air in the atmosphere has an approximate composition of 21% oxygen, 78% nitrogen, and less than 1% of other gases (Colbert, Ankney, & Lee, 2016). In a nutshell, breathing can be characterized as the process of moving air into and out the lungs (ventilation) to enable gas exchange with the internal environment (respiration), mostly by bringing oxygen in to provide it to the body tissues, and by removing carbon dioxide (Colbert et al., 2016; Hall, 2011a). The four major functions of breathing are: 1) Pulmonary ventilation, which represents the inflow and outflow of air between the atmosphere and the lung alveoli, 2) diffusion of oxygen and carbon dioxide between the alveoli and the blood, 3) transport of oxygen and carbon dioxide in the blood and body fluids to and from the body's tissue cells, and 4) regulation of ventilation and other facets of respiration (Hall, 2011a).

##### 4.1.2 Respiratory passageways: The crucial role of the nose

The respiratory passageways (Figure 1) include the nose with the nostrils and nasal passages, paranasal sinuses, the pharynx, the larynx, trachea, bronchi and bronchioles (Colbert et al., 2016; Hall, 2011a). It is usually recommended to breath in via the nose and not via the mouth, given the nasal cavities fulfil three distinct crucial respiratory functions when the air goes through (Colbert et al., 2016; Hall, 2011a; Lorig, 2011): 1) the air is warmed by the extensive surfaces of the conchae and septum, 2) the air is humidified, 3) the air is partially filtered. Altogether, those three functions are called the air conditioning functions of the upper respiratory passageways.



*Figure 1:* Schematic representation of the respiratory system displaying the upper and lower respiratory tract regions (from Tu, Inthavong, & Ahmadi, 2013, p. 20)

#### 4.1.3 Mechanics of pulmonary ventilation

The lungs can be expanded and contracted in two ways (Colbert et al., 2016; Hall, 2011a):

- 1) by downward and upward movement of the diaphragm to lengthen or shorten the chest cavity (see Figure 2), and
- 2) elevation and depression of the ribs to increase and decrease the anteroposterior diameter of the chest cavity (see Figure 3).

## Theoretical Background

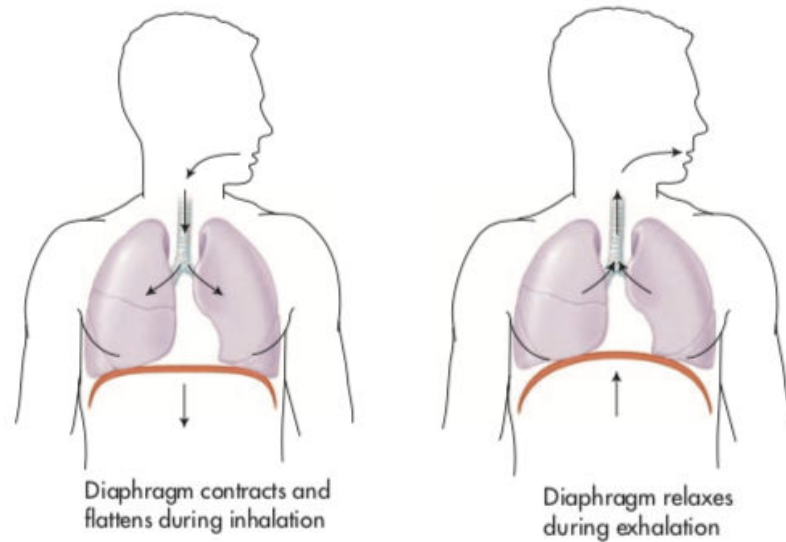


Figure 2: Diaphragm movements during inhalation and exhalation (from Colbert et al., 2016, p. 293)

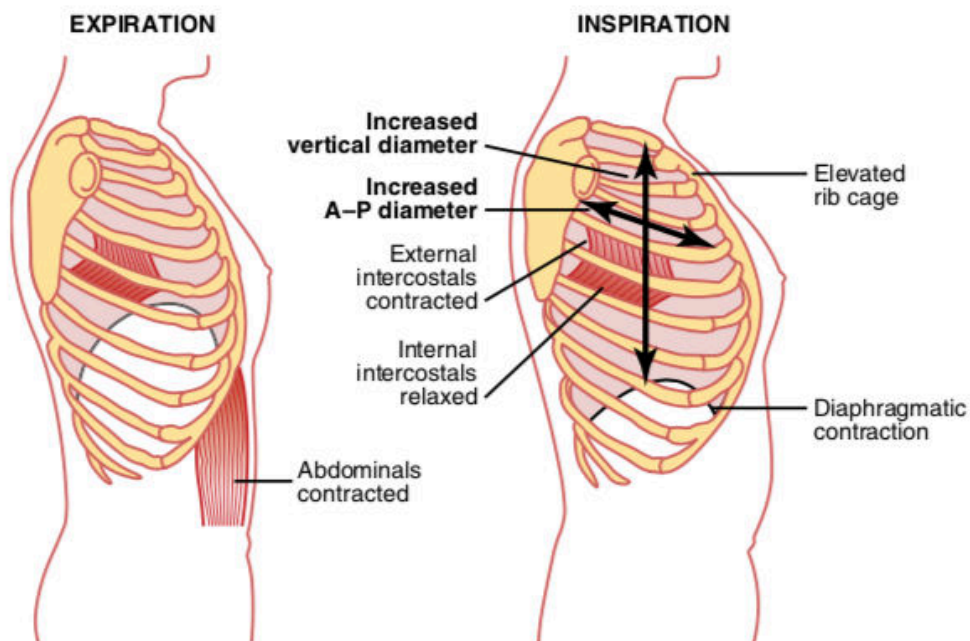


Figure 3: Illustration of the mechanics of pulmonary ventilation (from Hall, 2011a, p. 466)

Note: Figure 3 shows the contraction and expansion of the thoracic cage during expiration and inspiration, demonstrating diaphragmatic contraction, function of the intercostal muscles, and elevation and depression of the rib cage. A-P: anteroposterior

Normal spontaneous breathing is achieved almost solely with the first method, that is to say by the movement of the diaphragm (Colbert et al., 2016; Hall, 2011a). During inspiration,

contraction of the diaphragm pulls the lower surfaces of the lungs downward. Then during expiration, the diaphragm simply relaxes, and the elastic recoil (also called rebound) of the lung, chest wall, and abdominal structures compresses the lungs and expels the air.

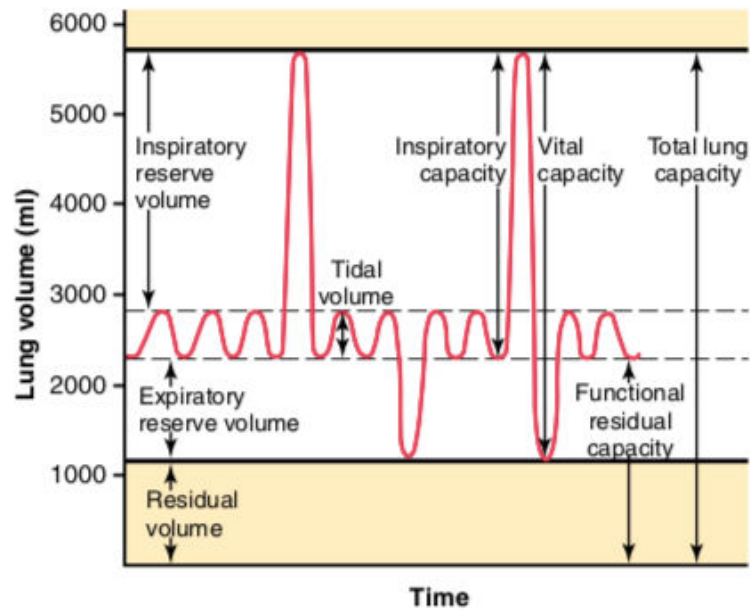
This is the breathing pattern also to be adopted with SPB – which is why it is often called diaphragmatic breathing –, to the exception that during SPB expiration would not be passive but rather active (Lehrer et al., 2000). The forced exhalation can involve abdominal muscles which, via their contraction, help to push the diaphragm back up to the thorax, thereby compressing the lungs, consequently pushing out additional air (Lorig, 2011; West, 2015; West & Luks, 2016). In SPB we would avoid to expand the lungs with the second method, that is raising the rib cage (Lehrer et al., 2000), given this would mean recruiting additional muscles, mainly the external intercostals to raise the rib cage and the abdominal recti and internal intercostals to pull the rib cage downward (Hall, 2011a; West & Luks, 2016). Recruiting additional muscles would require additional energy, which would then be counterproductive to the decrease in activation provoked by SPB (Lehrer & Gevirtz, 2014; Mather & Thayer, 2018).

#### 4.1.4 Pulmonary volumes

In order to understand the various events of pulmonary ventilation, the air in the lungs is usually subdivided in volumes and capacities, represented in Figure 4.



## Theoretical Background



*Figure 4: Respiratory patterns during normal breathing and during maximal inspiration and maximal expiration (from Hall, 2011a, p. 469)*

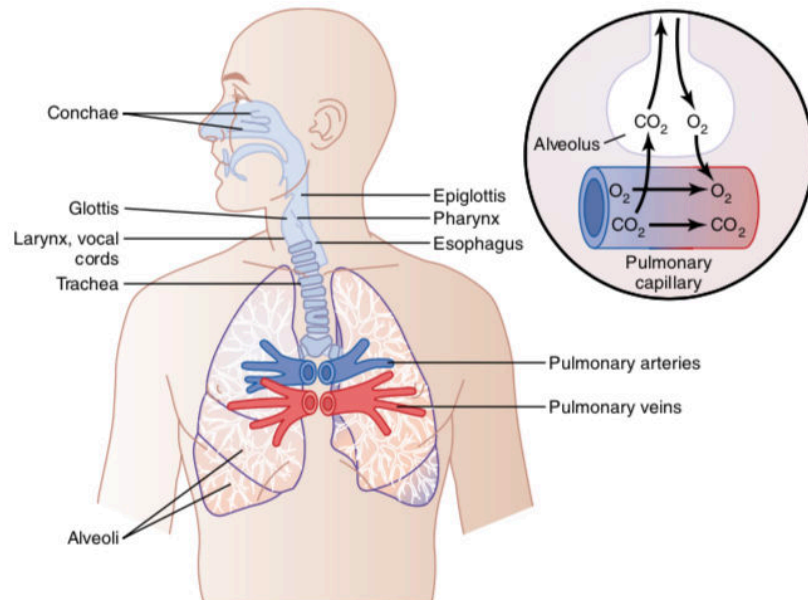
In order to better understand what happens during SPB, we focus here on the different kinds of pulmonary volumes (Hall, 2011a): The tidal volume is the volume of air inspired or expired with each normal breath (about 500mL in the adult male). The inspiratory reserve volume is the extra volume of air that can be inspired over and above the normal tidal volume when the person inspires with full force (about 3000mL). The expiratory reserve volume is the volume of air that can be expired by forceful expiration after the end of a normal tidal expiration (about 1100mL). The residual volume is the volume of air remaining in the lungs after the most forceful expiration (about 1200mL). SPB may require to breathe beyond the usual tidal volume and to take a deeper inhalation and exhalation (Lehrer & Gevirtz, 2014), which is why SPB is sometimes referred to as “deep breathing” .

### 4.1.5 Alveolar ventilation

The most important function of pulmonary ventilation is to enable to continually renew the air in the gas exchange areas of the lungs, where air is in proximity to the pulmonary blood, the alveoli (Colbert et al., 2016; Hall, 2011a; West & Luks, 2016). Other areas playing a role in this gas exchange are the alveolar sac, alveolar ducts, and respiratory bronchioles. Alveolar

## Theoretical Background

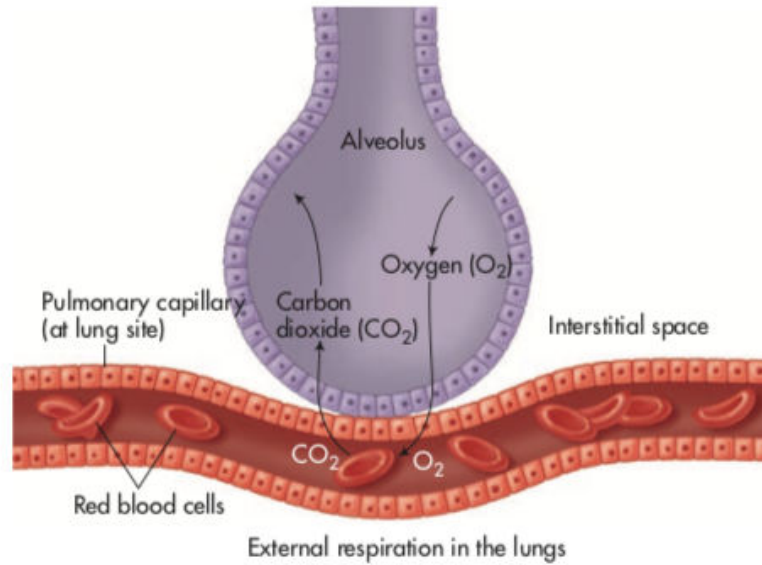
ventilation reflects the rate at which new air reaches these areas. The process of gas exchange between the alveoli and the pulmonary capillary can be seen in Figure 5. This process is suggested to be optimized by SPB, according to the resonance model (Lehrer & Gevirtz, 2014).



*Figure 5: Coupling between alveoli and pulmonary capillary (from Hall, 2011a, p. 472)*

More specifically, the process of gas exchange occurs via diffusion (Hall, 2011b), diffusion of oxygen from the alveoli into the pulmonary blood, and diffusion of carbon dioxide in the opposite direction, out of the blood (see Figure 6). The process of diffusion reflects the random motion of molecules in all directions through the respiratory membrane and adjacent fluid. Of importance will be the rate of diffusion.

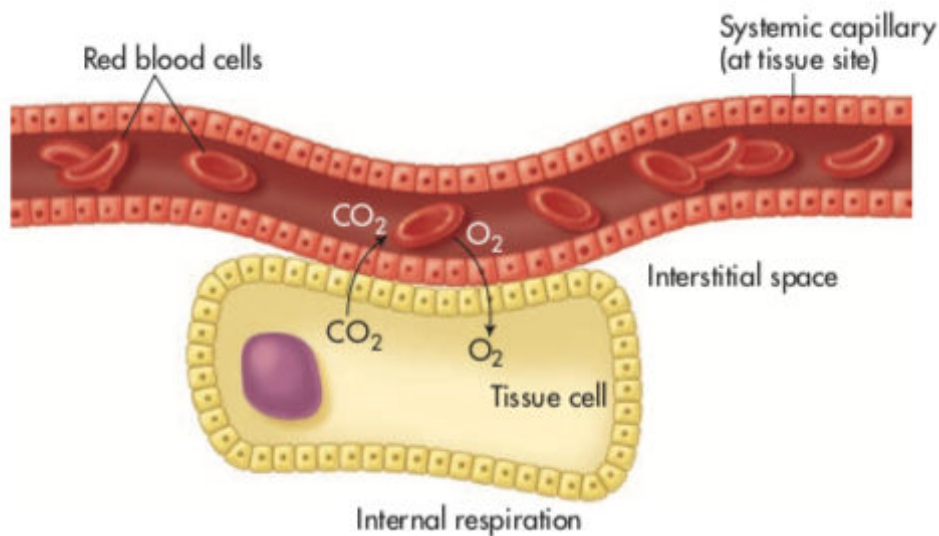
## Theoretical Background



*Figure 6:* Gas exchange at the level of the alveoli, illustration of the diffusion process (from Colbert et al., 2016, p. 293)

### 4.1.6 Transport of oxygen and carbon dioxide in blood and tissue fluids

Once oxygen has diffused from the alveoli into the pulmonary blood, it is transported to the peripheral tissue capillaries almost entirely in combination with hemoglobin (Hall, 2011c), the protein in red blood cells in charge of carrying oxygen. Hemoglobin also returns carbon dioxide from the peripheral tissues back to the lungs.



*Figure 7:* Gas exchange at the level of the tissues (from Colbert et al., 2016, p. 293)

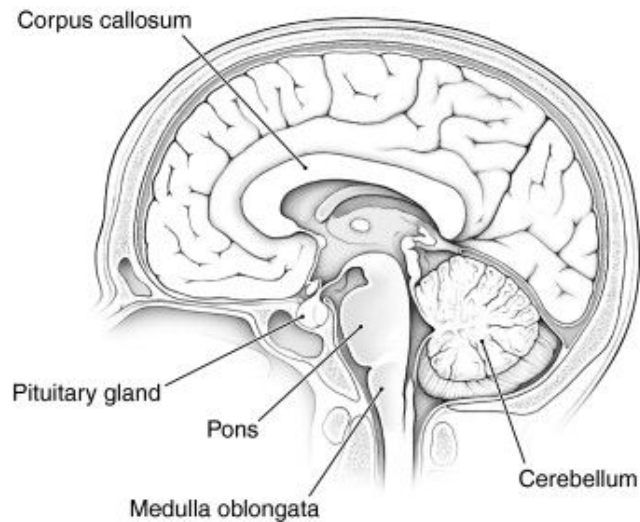
#### 4.1.7 The central regulation of respiration

The rate of alveolar ventilation is adjusted almost exactly according to the demands of the body by the nervous system. Consequently, the oxygen pressure and carbon dioxide pressure remains mostly constant in the arterial blood, even during different types of respiratory stress, such as heavy exercise (Hall, 2011d).

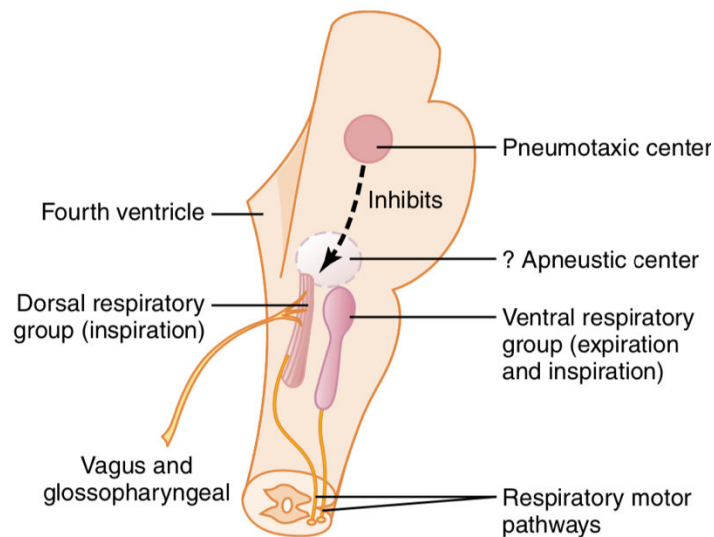
The respiratory center is composed of several groups of neurons located bilaterally in the medulla oblongata and pons of the brain stem (see Figure 8 and Figure 9). The medulla oblongata is home to all ascending and descending tracts that carry communications between the brain and the spinal cord (Waldman, 2009). In addition to the respiratory center, which fine tunes and modulates different information to regulate respiratory rate, the medulla oblongata also includes the cardiovascular center, which regulates heart rate in adjusting the strength of myocardial contractility and the dilatation and constriction of the peripheral vasculature (Waldman, 2009). Noteworthy, the sensory and motor nuclei of the **vagus nerve** are also found in the medulla oblongata. The vagus nerve is involved in many self-regulation processes (Thayer et al., 2009), as detailed later in section 4.3.1 (p.30), and will be a core focus of this PhD thesis.

The respiratory center contains three major groups of neurons: 1) the dorsal respiratory group, which is mainly responsible for inspiration, 2) the ventral respiratory group, which mainly causes expiration, and 3) the pneumotaxic center, which is mainly responsible for controlling rate and depth of breathing.

## Theoretical Background



*Figure 8: The Medulla Oblongata (from Waldman, 2009, p. 208)*



*Figure 9: Organization of the respiratory center (from Hall, 2011d, p. 506)*

The dorsal respiratory group of neurons is thought to play the most fundamental role in the control of respiration. Most of its neurons are located within the nucleus of the tractus solitarius (Zoccal, Furuya, Bassi, Colombari, & Colombari, 2014). The nucleus of the tractus solitarius is the sensory termination of two nerves: the vagus nerve and the glossopharyngeal nerves. Both nerves transmit sensory signals into the respiratory center from 1) peripheral chemoreceptors, 2) baroreceptors, and 3) other kind of receptors in the lungs such as pulmonary stretch receptors (Hall, 2011d; Zoccal et al., 2014). The vagus nerve is already particularly important to notice here, given its role in self-regulation processes (see section 4.3.1; p.30). Finally, the activity of

the respiratory center will also be modulated by chemoreceptors based on the concentrations of oxygen, carbon dioxide, and hydrogen ions in the tissues (Hall, 2011d).

The mechanisms we just described were related to the involuntary control of respiration, but interestingly for psychophysicologists, breathing can also be modified voluntarily. Some voluntary patterns will prove very useful for self-regulation (Lehrer & Gevirtz, 2014; Lorig, 2011), such as SPB, which constitutes the main focus of this PhD thesis.

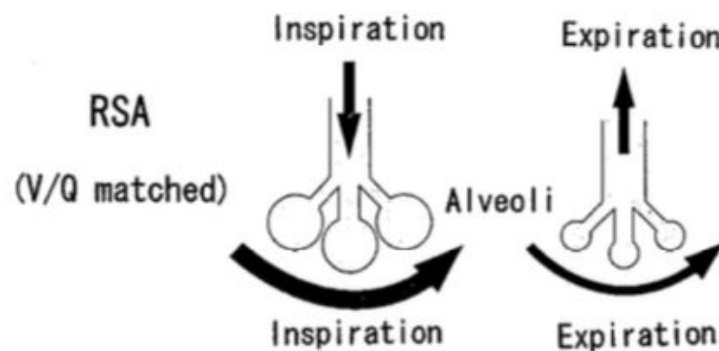
## 4.2 At the crossroads between the respiratory and the cardiovascular systems

### 4.2.1 Coupling between heart beat and respiration: Respiratory sinus arrhythmia

What makes breathing an interesting intervening variable, is that breathing is coupled to the cardiac output, in the sense that heart rate changes as a function of the respiratory cycle (Lorig, 2011). This phenomenon is called **respiratory sinus arrhythmia (RSA)**, heart rate accelerating during inhalation, and slowing down during exhalation (Angelone & Coulter, 1964; Eckberg & Eckberg, 1982). RSA is suggested to play an important role in respiration, given previous research has suggested that the efficiency of pulmonary gas exchange is improved by RSA (Giardino, Glenny, Borson, & Chan, 2003; Hayano, Yasuma, Okada, Mukai, & Fujinami, 1996; Mortola, Marghescu, & Siegrist-Johnstone, 2016, 2018; Yasuma & Hayano, 2004). The distribution of heartbeats within each respiratory cycle was suggested to improve the efficacy of respiratory gas exchange. More specifically, the matched timing of alveolar ventilation and its perfusion with RSA within each respiratory cycle was hypothesized to save energy expenditure by suppressing unnecessary heartbeats during expiration and ineffective ventilation during the ebb of perfusion (see Figure 10). In other words, heart rate tends to be higher when the air in the lungs is the richest in oxygen, and expiration occurs when carbon dioxide is at its highest level. However, this hypothesis have been questioned by further research, showing no relationship between RSA and improvement of gas exchanges (Buchheit, 2010; Sin et al., 2010; Tzeng, Sin, & Galletly, 2009). Another hypothesis, that still requires

empirical examination, is that RSA helps the heart do less work while maintaining healthy levels of blood gases (Ben-Tal, Shamailov, & Paton, 2012, 2014).

Gas exchange at the alveoli level is suggested to work best when heart rate starts to increase at the beginning of inspiration, and starts to decrease at the beginning of expiration, with would reflect a  $0^\circ$  phase relationship (Hayano et al., 1996), and which is coined cardioventilatory coupling (Elstad, O'Callaghan, Smith, Ben-Tal, & Ramchandra, 2018). However, in normal situations the relationship between heart rate and breathing is not completely in phase, which could be explained by allowing a greater degree of flexibility of the organism, so that greater efficiency can be achieved during phases of greater metabolic need, and less during decreased need (Lehrer & Gevirtz, 2014). SPB at 6 cycles per minute (cpm) is suggested to trigger a heart rate oscillating with breathing at a  $0^\circ$  phase relationship, with consequently the most efficient gas exchange (Lehrer & Gevirtz, 2014), however some posterior work strongly questioned the validity of this hypothesis (Sin, Webber, Galletly, & Tzeng, 2012). To sum up, the mechanisms by which RSA is an adaptive process still need to be understood.



*Figure 10:* Illustration of the suggested conceptual effect of the respiratory sinus arrhythmia on the relationship between alveolar gas volume and capillary blood flow during inspiration and expiration (from Yasuma & Hayano, 2004, p. 684)

*Note:* Figure 10 shows the conceptual effects of respiratory sinus arrhythmia, the curved horizontal arrow and vertical arrows indicate the volume of blood flow circulating in the pulmonary capillaries and the direction of alveolar gas interfacing with the pulmonary capillaries.

RSA is suggested to be mainly vagally-driven (Berntson, Cacioppo, & Quigley, 1993; Grossman, van Beek, & Wientjes, 1990; Yasuma & Hayano, 2004), by both tonic and phasic



vagal tone processes, which have different origins, dynamics, and functional consequences (Daly, 1985; Grossman, Karemaker, & Wieling, 1991; Grossman et al., 1990; Richter & Spyer, 1990). In particular, the vagus nerve is suggested to contribute to the efficiency of pulmonary gas exchange (Ito et al., 2006), which is to be linked to its role in self-regulation processes (see section 4.3.1, p.30). RSA is usually assessed via the peak-valley method (Grossman et al., 1990; Stange, Hamilton, Fresco, & Alloy, 2017), where the maximum heart rate during the expiration window of respiration is subtracted from the minimum heart rate during the inspiration window of respiration.

According to the resonance model (Lehrer & Gevirtz, 2014), RSA constitutes one of the core mechanisms targeted by the SPB technique to improve self-regulation processes.

#### 4.2.2 Slow-paced breathing

As mentioned earlier, SPB is a breathing technique where the inhalation and exhalation durations are controlled (“paced”), and where breathing is performed at a slower pace (around 6 cycles per minute) than spontaneous breathing, which is usually comprised between 12 and 20 cycles per minute in adults (L. Sherwood, 2006). In this PhD thesis I use the term SPB instead of the other ones found in the literature, such as “deep breathing” (e.g., Tharion, Samuel, Rajalakshmi, Gnanasenthil, & Subramanian, 2012), “abdominal breathing” (e.g., Wang et al., 2010) or “diaphragmatic” breathing (e.g., Russell, Scott, Boggero, & Carlson, 2017), given it reflects the fact that a slow, paced breathing is the necessary trigger to influence positively self-regulation processes, as we now detail. The characteristics “deep”, “abdominal”, and “diaphragmatic” are not completely inaccurate and may reflect some aspects of the breathing technique, however they are not considered as the main triggers of the effects of SPB: 1) “deep” is supposed to reflect the fact that participants are required to slow down their breathing frequency, which can result – but does not have to – in increased tidal volume; however if participants get dizzy, they are immediately recommended to stop/adopt a more shallow



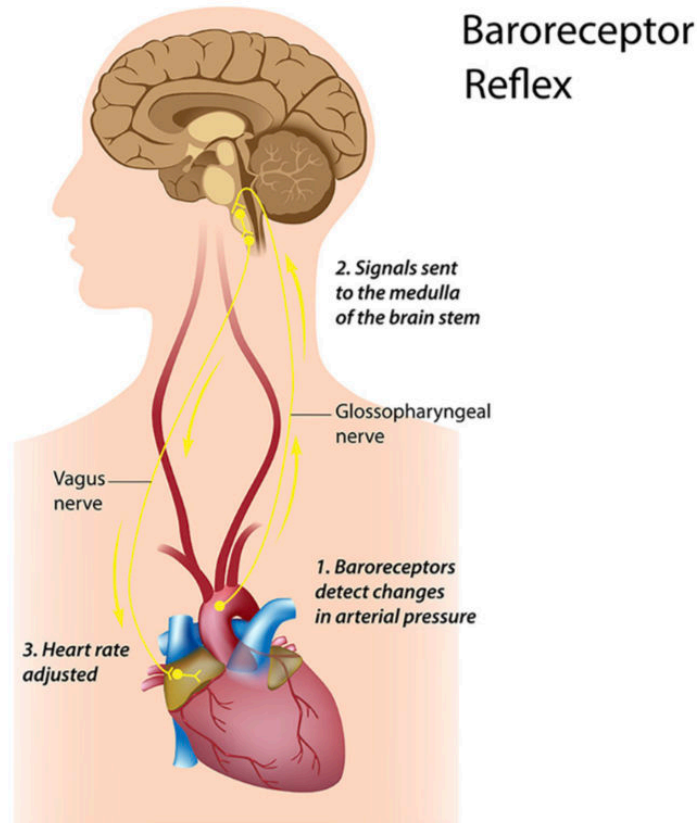
breathing (Lehrer et al., 2000); 2) “abdominal” or “diaphragmatic” reflects the characteristics of the most effective breathing pattern in resting conditions (for further details, see section 4.1.3, p.16).

The **resonance model** (Lehrer & Gevirtz, 2014) assumes that four processes are at stake to explain how SPB positively influences self-regulation mechanisms: 1) the phase relationship between heart rate oscillations and breathing at 6 cpm; 2) the phase relationship between heart rate and blood pressure oscillations at 6 cpm; 3) the activity of the baroreflex; and 4) the resonance characteristics of the cardiovascular system. These processes strengthen homeostasis in the baroreceptor (Lehrer et al., 2006; Vaschillo, Lehrer, Rishe, & Konstantinov, 2002; Vaschillo, Vaschillo, & Lehrer, 2006), which is suggested to result in improved gas exchanges at the level of the alveoli and in increased vagal afferences (Lehrer & Gevirtz, 2014).

However, empirical evidence challenged some of the suggested underlying mechanisms. Regarding the first one related to the **phase relationship between heart rate oscillations and breathing at 6cpm** (cardioventilatory coupling, Elstad et al., 2018), it is based on the hypothesized role of RSA in optimizing gas exchanges (Yasuma & Hayano, 2004). However, if indeed heart beat tends to redistribute towards inspiration at 6cpm (Lopes, Beda, Granja, Jandre, & Giannella-Neto, 2011), the original hypothesis regarding the physiological role of RSA to match heartbeats with pulmonary blood flow (Yasuma & Hayano, 2004) has been severely questioned (Buchheit, 2010; Sin et al., 2010; Tzeng et al., 2009), and so far no clear answer appeared to explain the adaptive physiological role of RSA. Still, the point that pulmonary gas exchange efficiency may be improved by slow breathing has received some support via reduction in ventilatory equivalents for carbon dioxide and oxygen when breathing at 6cpm (Sin et al., 2010), even if this method is not considered as the gold standard to measure gas exchange efficiency (Tharion & Subramani, 2011; Tzeng, 2011).

The **baroreflex** is a reflex mediated by blood pressure sensors in the aorta and carotid artery that help modulate blood pressure fluctuations (Eckberg & Sleight, 1992). Baroreceptors

located in the walls of these arteries detect stretching of the arteries as blood pressure increases, and when this happens, the baroreflex causes immediate decreases in heart rate. On the contrary when blood pressure falls, the baroreflex causes immediate increases in heart rate (Eckberg & Sleight, 1992; Lehrer & Gevirtz, 2014). The mechanisms underlying the functioning of the baroreceptors is displayed in Figure 11. Similar to the phase relationship mentioned for heart rate and breathing, **a similar phase relationship exists for heart rate and blood pressure**, which can be depicted like this: when an external stimulation such as breathing at a specific frequency causes heart rate to rise, it also causes blood pressure to fall, consequently triggering an additional stimulus for heart rate to rise further. When the external stimulation (e.g., breathing) causes heart rate to fall, it also causes blood pressure to rise, thus causing an additional stimulus for heart rate to further decrease. Given the  $0^\circ$  phase relationship between heart rate and breathing occurs at approximately the same frequency that external stimulation causes maximal stimulation to the baroreflex, breathing appears a natural candidate as an external stimulator (Lehrer & Gevirtz, 2014). In line with this suggestion, large increases in baroreflex gain (number of beats per minute change in heart rate per 1 mm Hg change in blood pressure) have been observed during SPB with biofeedback, illustrating the fact that the baroreflex was strengthened (Lehrer et al., 2003).



*Figure 11:* Illustration of the functioning of the baroreflex (from Shaffer, McCraty, & Zerr, 2014, p. 9)

Finally, the last mechanism suggested by the resonance model (Lehrer & Gevartz, 2014) regarding how SPB influences positively self-regulation processes is via **the resonance characteristics of the cardiovascular system**. Resonance is a physical principle stating that all oscillating feedback systems with a constant delay have the characteristic of resonance (Lehrer & Gevartz, 2014). Here an example to better picture what resonance means: imagine a person being pushed on a swing (Allen & Friedman, 2012): The push must be in rhythm with the swinger's momentum. Resonance between the natural swinging motion and the applied force makes the swing go higher than before. The same happens in the cardiovascular system when breathing at a frequency close to 6cpm, where the pattern of RSA overlaps with inherent oscillations in heart rate related to blood pressure modulation (Vaschillo et al., 2006). The point at which those two signals overlap was coined the resonant frequency, given the two signals

summate and produce large variations in heart rate (Allen & Friedman, 2012; Lehrer & Gevirtz, 2014; Vaschillo et al., 2006).

To sum up, the **resonance model** (Lehrer & Gevirtz, 2014) hypothesized that SPB, via several processes described above, influences positively self-regulation. The most supported mechanism so far is the strengthened homeostasis in the baroreceptor (Lehrer et al., 2003; Vaschillo et al., 2002; Vaschillo et al., 2006). The second mechanism, the optimization of pulmonary gas exchange based on RSA characteristics at 6cpm could not be fully supported so far (Buchheit, 2010; Sin et al., 2010; Tzeng et al., 2009). The third mechanism suggested was an increase in vagal afferents, however this path was so far largely based on the speculation that positive outcomes achieved with SPB interventions with biofeedback were related to brain areas related to vagal afferents (Lehrer & Gevirtz, 2014). In this PhD thesis, I focus on the possibility that this suggested action of SPB on vagal afferents would be reflected in vagal efferents and hence influence positively self-regulation mechanisms, given the links described in the next section.

### 4.3 How slow-paced breathing may influence self-regulation: the role of the vagus nerve

#### 4.3.1 The role of the vagus nerve in self-regulation

The vagus nerve is the tenth cranial nerve (see Figure 12), and it is the most important nerve of the parasympathetic nervous system (Brodal, 2010). It is composed of 80% afferent sensory fibers (sending signals from the body to the brain) and 20% efferent motor fibers (carrying information from the brain to the body). All branches of the vagus nerve with visceral efferent fibers also contain afferent sensory fibers, which makes it a highly sensitive nerve (Howland, 2014). As its name implies (the Latin translation of vagus means *wandering*), the vagus nerve branches to widespread regions of the body (Brodal, 2010), its fibers innervating most organs in the body including the gastrointestinal and cardiovascular systems (Bonaz,

## Theoretical Background

Bazin, & Pellissier, 2018; Brodal, 2010; H. Y. Chang, Mashimo, & Goyal, 2003). Vagal fibers release acetylcholine as neurotransmitter (Brodal, 2010). In a nutshell, thanks to its extensive network the vagus nerve allows for widespread fast acting communications within the body.

Theoretical Background

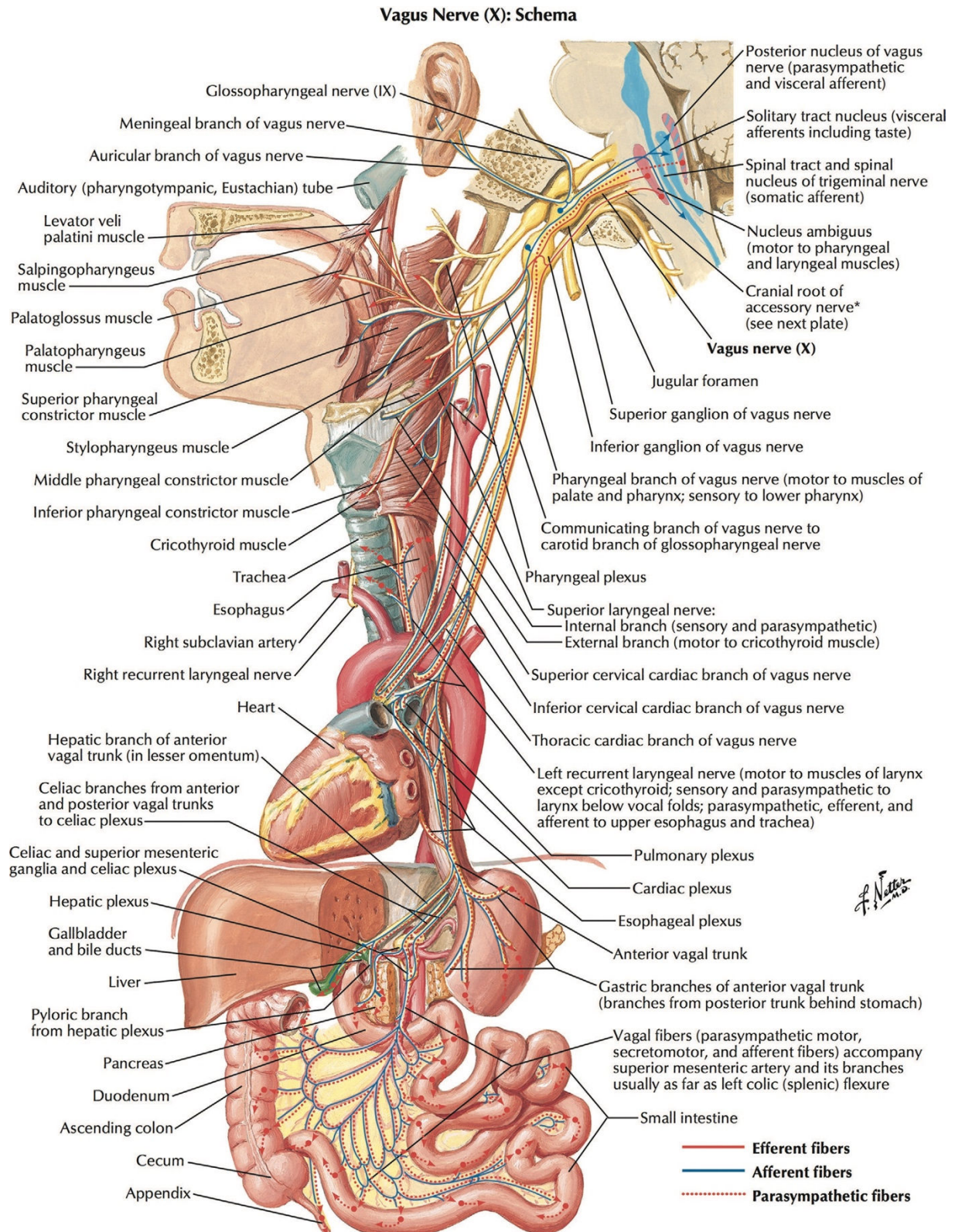


Figure 12: Anatomy of the vagus nerve including branches (from Câmara & Griessenauer, 2015, p. 386)



#### 4.3.2 Cardiac vagal activity: the output of the central autonomic network

Regarding vagal efferent fibers, those that stimulate motor action, we are particularly interested here in those innervating the heart and modulating its intrinsic activity through the sinus node, which determines heart rate. Importantly, from the two branches of the autonomic nervous system, the sympathetic and the parasympathetic, the sympathetic influence on the heart is too slow to produce beat-to-beat changes (Jose & Collison, 1970), and the heart will be mainly under parasympathetic inhibitory influence through vagal efferent fibers (Jose & Collison, 1970; Saul, 1990). This cardiac autonomic balance is a way for the organism to favor energy conservation.

Regarding vagal afferent fibers, those that are linked to sensory actions, they are largely scattered through key organs in the human body (Brodal, 2010). This gives the vagal afferent system an important adaptation role as a detector of immune-related events in the human body. This peripheral sense allows for an internal signal that can generate the appropriate autonomic, endocrine, and behavioral responses via central reflex pathways going through the nucleus of the solitary tract (Berthoud & Neuhuber, 2000). These internal inputs are then integrated to external inputs, which helps to shape the appropriate response. Importantly, SPB may play a role in triggering internal inputs, given it is suggested to stimulate vagal afferents (Lehrer & Gevirtz, 2014).

A functional network based on brain structures is suggested to facilitate the organization and regulation of vagal afferent and efferent activity (Berthoud & Neuhuber, 2000). The **central autonomic network** (Benarroch, 1993) represents a functional unit in the central nervous system supporting goal-directed behavior, adaptability, and overall self-regulation processes. **Cardiac vagal activity** (CVA), the activity of the vagus nerve regulating cardiac functioning, is suggested to be the output of this central autonomic network, and is the core of the neurovisceral integration model (Smith et al., 2017; Thayer et al., 2009).

4.3.3 The neurovisceral integration model

The **neurovisceral integration model** (Smith et al., 2017; Thayer et al., 2009) assumes a connection between the prefrontal cortex and the heart through the central autonomic network and the vagus nerve.

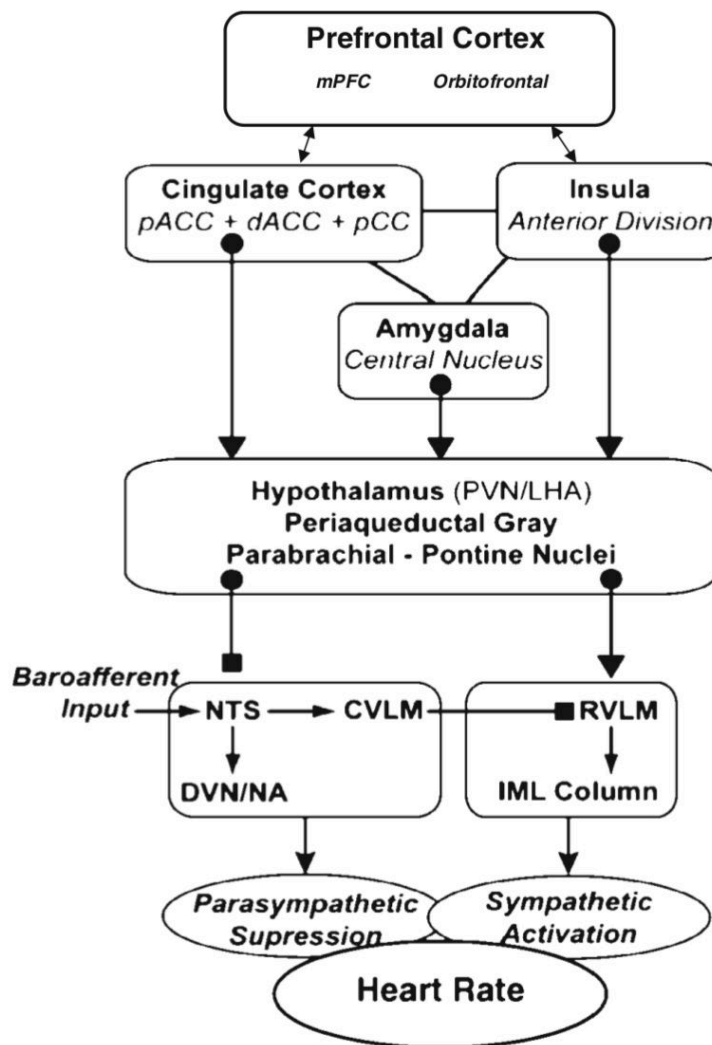


Figure 13: A composite schematic diagram showing the pathways by which the prefrontal cortex might influence the control of heart rate (from Thayer et al., 2009, p. 143)

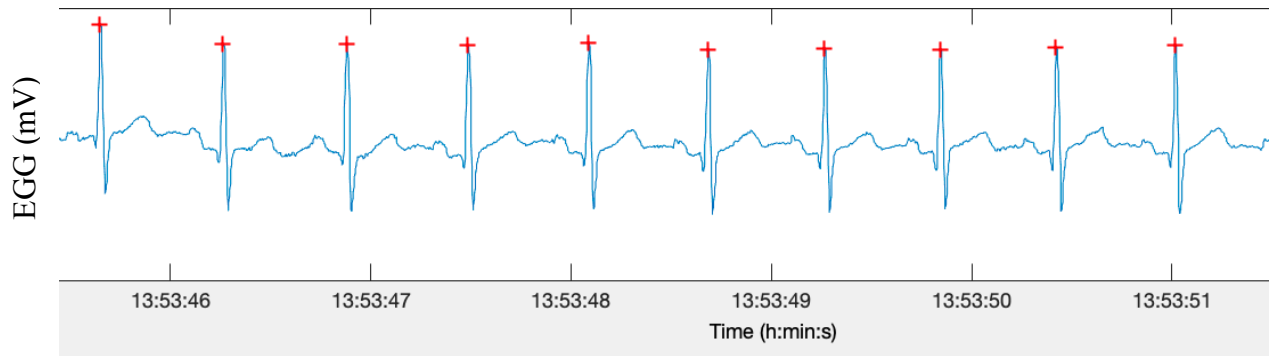
*Note.* mPFC: medial prefrontal cortex; pACC: pregenual anterior cingulate cortex; dACC: dorsal anterior cingulate cortex; pCC: posterior cingulate cortex; PVN: paraventricular nucleus of hypothalamus; LHA: lateral hypothalamus; NTS: nucleus of the solitary tract; CVLM: caudal ventrolateral medulla; DVN: dorsal vagal motor nucleus; NA; RVLM: rostral ventrolateral medulla; IML: intermediolateral nucleus



The neurovisceral integration model postulates that CVA serves as an indicator of the effectiveness of this network, and consequently serves as a basis to index phenomena underpinning self-regulation, adaptation, and health (Smith et al., 2017; Thayer et al., 2009). At rest, the medial prefrontal cortex exerts inhibitory control over the amygdala, indirectly enhancing cardiac control via the vagus nerve, which is reflected in an increase of CVA (Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). This constant inhibitory control over the amygdala would support the connection of CVA to emotion regulation. The relationship between CVA and executive functioning originates from the common structures and networks involved in cardiac and cognitive regulation (Thayer et al., 2012). The effectiveness of executive functioning in the prefrontal cortex is supported via the optimal activation of neural networks, underlined with a flow of activity along neural pathways enabling to establish adequate mappings between input, internal states, and outputs needed to perform a given task (Miller & Cohen, 2001), leading to flexible responses to changing environments (Thayer et al., 2009).

#### 4.3.4 Non-invasive assessment of cardiac vagal activity with heart rate variability

The neurovisceral integration model (Thayer et al., 2009) postulates that it is possible to index non-invasively the output of the central autonomic network, CVA, via heart rate variability (HRV, see Figure 14). HRV represents the time variation between each R peaks in the QRS complexes (Berntson et al., 1997; Laborde, Mosley, & Thayer, 2017; Malik, 1996). Two main HRV parameters reflecting CVA are the root mean square of the successive differences (RMSSD) and high-frequency HRV (Berntson et al., 1997; Laborde, Mosley, et al., 2017; Malik, 1996). SPB was found in previous research to increase vagal efferents as measured by short-term HRV (Kromenacker, Sanova, Marcus, Allen, & Lane, 2018; Lewis et al., 2015; Szulczewski & Rynkiewicz, 2018; Wells et al., 2012).



*Figure 14: Electrocardiogram - Illustration of heart rate variability calculation*

Note. Illustration of heart rate variability calculation, based on the time variation between each R peaks (marked here with a red cross); ECG: electrocardiogram; mV: millivolt

#### 4.4 The 3Rs of vagus nerve functioning

Both tonic and phasic aspects of CVA functioning are important to consider (Thayer et al., 2012). Tonic CVA has also been referred to as **resting CVA**, and is when CVA is considered at one time point. Phasic CVA shows how the system reacts, and has been coined **reactivity** when depicting the change occurring between baseline and an event, and **recovery**, representing the change between the event to the return to resting conditions (Laborde, Mosley, & Mertgen, 2018b; Laborde, Mosley, et al., 2017). These three aspects are very important to consider from an adaptation point of view (see Figure 15). Regarding resting CVA, the neurovisceral integration model (Thayer et al., 2009) would assume in general “the higher the better”, meaning that a higher resting level of CVA is associated positively to self-regulation effectiveness. Regarding reactivity, two situations have to be differentiated (Beauchaine, 2001; Laborde, Mosley, & Mertgen, 2018b; Porges, 2007b; Thayer et al., 2012; Thayer et al., 2009): if the challenge is mainly cognitive, then a lower withdrawal or even an increase of CVA would be seen as adaptive; however if metabolic demands increase, a larger decrease of CVA would be adaptive in order to provide the necessary energy to the body. Finally, regarding recovery, a faster come back to baseline is expected to reflect a better adaptation (Stanley, Peake, & Buchheit, 2013). Taking into account the 3Rs of CVA functioning will be particularly important to derive the respective hypotheses.

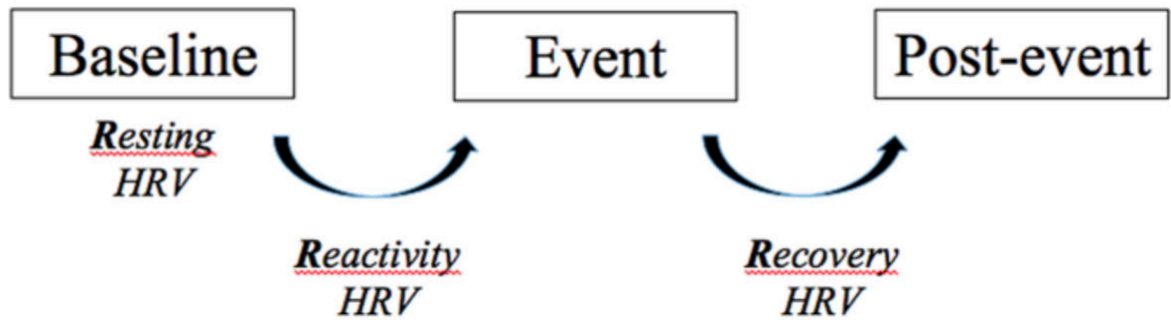


Figure 15: The 3Rs of cardiac vagal activity functioning (from Laborde, Mosley, et al., 2017, p. 6)

In summary, in the above sections we provided an overview of how SPB may be connected to self-regulation mechanisms. According to the resonance model (Lehrer & Gevirtz, 2014), if the most supported mechanism so far is the strengthening of baroreceptor homeostasis, another one being suggested is the increase in vagal afferences. This increase in vagal afferences may input into the central autonomic network (Benarroch, 1993), and according to the neurovisceral integration model (Thayer et al., 2009) consequently influence positively self-regulation phenomena. Within this PhD thesis I am going to investigate the relationship between SPB and a range of self-regulation phenomena, encompassing stress, sleep, and executive functioning.

#### 4.5 Self-regulation phenomena investigated with slow-paced breathing and cardiac vagal activity within this PhD thesis

##### 4.5.1 Stress

##### 4.5.1.1 Psychological stress

Psychological stress occurs when an individual perceives that personal or environmental demands tax or exceed his or her adaptive abilities (Lazarus & Folkman, 1984). Even if psychological stress can be considered as an idiosyncratic phenomena, given it will differ across individuals and situations, a common method of inducing stress is using tasks taxing executive functions. In addition, putting an emphasis on performing well helps to represent a situation

which differs markedly from resting states in terms of psychological demands placed on the individual. This definition of stress may be linked to CVA, in that an appraisal of threat is central to a decrease of CVA, as opposed to an appraisal of safety (Thayer et al., 2009). The influence of mental stress on CVA will be very individualized according to the appraisal process (Lazarus & Folkman, 1984), and may depend for example on the degree of threat appraisal (Thayer et al., 2009). Another kind of stress may be triggered via physical stressors.

#### *4.5.1.2 Physical stress*

In terms of CVA reactivity, physical stressors require a vagal withdrawal in order for the organism to meet the physical demands of the task (Nakamura, Yamamoto, & Muraoka, 1993; Stanley et al., 2013). This reflects the evolutionary role of CVA as a “call to arms” mechanism to nurture the fight or flight response (Porges, 2007a, 2007b; Thayer, 2009). The fight or flight response is associated with near complete CVA withdrawal (Beauchaine, Gatzke-Kopp, & Mead, 2007), in order to facilitate large increases in cardiac output by the sympathetic nervous system, which is no longer facing the opposition of vagal inhibition. The level of CVA withdrawal during the physical stressor will depend mainly on the intensity of the physical stressor rather than on its duration (Stanley et al., 2013). Finally, the initial fitness levels of the person will influence both the amplitude and kinetics of vagal recovery, individuals having a greater aerobic fitness recovering faster (Stanley et al., 2013).

#### *4.5.1.3 Stress and slow-paced breathing*

The current evidence speaks strongly for a stress reducing effect of SPB. As shown by a recent meta-analysis (Goessl et al., 2017), SPB coupled with biofeedback was found to largely reduce self-reported stress and anxiety, while no moderator (e.g., number of sessions, presence of an anxiety disorder) was found to significantly influence the results. This confirms the findings of systematic and narrative reviews on the effects of SPB coupled with biofeedback on psychophysiological stress markers (Gevirtz, 2013; Kennedy & Parker, 2018; Wheat &

Larkin, 2010; Yu et al., 2018; Zaccaro et al., 2018). Interestingly, we can notice that even if SPB has been used in many contexts to reduce stress, it is still to be investigated in the field of intellectual disabilities.

#### *4.5.1.4 Stress and intellectual disability*

Having an **intellectual disability** is associated with high levels of stress (e.g., Forte, Jahoda, & Dagnan, 2011). This association can be explained in part by additional difficulties in adaptation and finding everyday situations over demanding, as well as being socially marginalized (de Bildt, Sytema, Kraijer, Sparrow, & Minderaa, 2005). Moreover, individual with intellectual disabilities often use maladaptive coping strategies to cope with stress (Hartley & Maclean, 2008). The stress experienced by individuals with intellectual disabilities is often transferred to family members and caregivers (e.g., Hassall & Rose, 2005). In the long term, if chronic stress in persons with intellectual disabilities is not properly addressed, it can lead to serious complications including depression (Hartley & Maclean, 2009), impaired cognitive functions (Heyman & Hauser-Cram, 2015), physical health problems (Lunsky, 2008), and maladaptive coping strategies such as substance abuse (Didden, Embregts, van der Toorn, & Laarhoven, 2009). It can also lead to family members and caregivers experiencing depression (Mutkins, Brown, & Thorsteinsson, 2011) and burnout (Innstrand, Espnes, & Mykletun, 2002). Given the prevalence of stress in people with intellectual disabilities, and the absence of stress management programs implementing SPB in people having intellectual disabilities so far, the first study will investigate whether SPB can help people with individual disabilities to cope with stress (p.54).

The second self-regulation phenomena of interest in this PhD thesis will be sleep.

## *4.6 Sleep*

Issues with sleep are a pressing concern for individuals, given they directly impact life quality, and represent a risk factor at several levels (Davenne, 2009; Ferrie, Kumari, Salo,

Singh-Manoux, & Kivimaki, 2011). One of the main hypothesis regarding the cause of sleep disturbances is that they may be associated with a state of hyperarousal (Bonnet, Burton, & Arand, 2014; Riemann et al., 2010). Methods aiming to decrease a state of hyperarousal usually target an activation of the parasympathetic nervous system, and more specifically of its main nerve, the vagus nerve (Friedman, 2007; Prendiville, 2016). One way to do so is to use SPB (Gerritsen & Band, 2018; Lehrer, 2018; Lehrer & Gevirtz, 2014; Zaccaro et al., 2018).

#### 4.6.1 Sleep and cardiac vagal activity

The question whether measuring CVA during sleep ( $CVA_{\text{night}}$ ) represents an indicator of sleep quality is still debated. Some evidence points toward an association between lower  $CVA_{\text{night}}$  and sleep disorders (Stein & Pu, 2012), such as with chronic fatigue (Burton, Rahman, Kadota, Lloyd, & Vollmer-Conna, 2010) and insomnia (Yang et al., 2011). Higher  $CVA_{\text{night}}$  has also been related to higher subjective sleep quality (Brosschot, Van Dijk, & Thayer, 2007; Patel et al., 2013; Yang et al., 2011). However, some authors argue that measuring  $CVA_{\text{night}}$  across sleep stages does not provide useful information, given the variations observed in CVA during different sleep stages (Werner et al., 2015), namely CVA withdrawal during Rapid Eye Movement (REM) sleep, and CVA increase during non-REM sleep (Tobaldini et al., 2013). Further, Werner and colleagues (2015) argue that assessing CVA while sleeping is suboptimal, given CVA is supposed to reflect adaptations to environmental changes, and these do (almost) not occur during the night, so they rather recommend assessing CVA during periods where individuals are awake. In summary, even if  $CVA_{\text{night}}$  measurement cannot be considered as an index of sleep quality, it may still provide an indication of the restorative status of the body during sleep, given it indexes the activity of the parasympathetic nervous system (Laborde, Mosley, et al., 2017; Malik, 1996; Sakakibara, Hayano, Oikawa, Katsamanis, & Lehrer, 2013; L. Sherwood, 2006).

In order to address the criticisms made to  $CVA_{\text{night}}$  measurements, authors have suggested to measure CVA during wake periods (Werner et al., 2015). Particularly, a quiet awakening morning period ( $CVA_{\text{morning}}$ ) has been suggested as a good compromise, given the individual has usually not experienced heavy environmental changes beforehand (Buchheit, Simon, Piquard, Ehrhart, & Brandenberger, 2004).  $CVA_{\text{morning}}$  has already been related to subjective indices of well-being and to physical training adaptations (see for example Buchheit et al., 2006; Buchheit et al., 2004), and also more recently to subjective sleep quality measurements (Flatt, Esco, & Nakamura, 2018). Investigating  $CVA_{\text{morning}}$  together with  $CVA_{\text{night}}$  measurements seems therefore an appropriate combination to further understand the effects of SPB on CVA.

#### 4.6.2 Sleep and slow-paced breathing

To the best of our knowledge, only two previous studies investigated the effects of SPB on sleep while measuring HRV (Sakakibara et al., 2013; Tsai, Kuo, Lee, & Yang, 2015). In the first study (Sakakibara et al., 2013), SPB was delivered with the help of a biofeedback device during 20 minutes for two nights. In comparison to a control group performing autogenic training, CVA as measured with high-frequency (HF)-HRV was higher during the two nights measured, supporting the idea that it improved cardiorespiratory function. The second study (Tsai et al., 2015), focusing on self-reported insomniacs, aimed to investigate whether a 20min SPB session (6 cpm), compared to a control condition with paced breathing set at 12 cpm, would enhance objective sleep quality as assessed via polysomnography and CVA. In the SPB condition, the inspiration and expiration phases were set to 3s and 7s, while no indications were mentioned regarding the inspiration and expiration phases for the 12 cpm breathing condition. In regards to polysomnography, results showed that after a single 6 cpm session before going to sleep, sleep onset latency, number of awakenings, and awakening time during sleep were decreased, while sleep efficiency was increased, in comparison to the 12 cpm breathing

condition and to baseline. Regarding CVA, unfortunately the HRV variables mentioned in the paper (total power and R-R intervals) actually do not reflect it (Berntson et al., 1997; Laborde, Mosley, et al., 2017; Malik, 1996; Shaffer & Ginsberg, 2017; Shaffer et al., 2014), therefore it is not possible to draw any conclusions related to CVA. Moreover, HRV was not assessed during sleep, but during daytime rest. Consequently, further studies are therefore warranted to better understand the effects of SPB on subjective sleep quality and CVA, and not only on a short-term single session basis, but also on a long-term intervention basis.

The second study of this PhD thesis will target a long-term intervention (30 days) aimed to improve  $CVA_{\text{night}}$  and  $CVA_{\text{morning}}$  (p.62).

#### 4.7 Executive functioning

Another area of self-regulation that can be indexed by CVA put forward by the neurovisceral integration model is the regulation of executive functioning, based on the common networks responsible for cardiac and executive control (Thayer et al., 2012; Thayer et al., 2009). Executive functions underpin goal-directed behavior and are essential for self-control (Diamond, 2013; Kotabe & Hofmann, 2015; Miyake et al., 2000). The three core executive functions are inhibition, working memory, and cognitive flexibility (Diamond, 2013; Miyake & Friedman, 2012; Miyake et al., 2000).

##### 4.7.1 Inhibition and cardiac vagal activity

Inhibition reflects being able to control attention, behavior, thoughts, and/or emotions to override a strong impulse, and to do instead what is more appropriate according to the context (Diamond, 2013). A classical test for inhibition is the color word Stroop test (Stroop, 1935), where participants are requested to read out the color in which a word is printed while ignoring the meaning of the word. In the congruent condition the color matches the meaning of the word (e.g., the word “blue” expressed in the color blue), while in the incongruent condition the color differs from the meaning of the word (e.g., the word “blue” expressed in the color red). The



incongruent condition requires participants to inhibit the prepotent response of reading a word. Both the speed and accuracy of the responses can be measured. However, inhibition is primarily reflected as accuracy (error rate) (McDowd, Oseas-Kreger, & Filion, 1995), as it captures the ability to temporarily maintain the task goal in a retrievable state (Kane & Engle, 2003).

Previous research has found that Stroop task performance relates to CVA. A negative relationship has been observed between resting CVA and reaction times on incongruent and threat words (Johnsen et al., 2003), whereas a positive relationship has been observed between resting CVA and Stroop accuracy (i.e., Stroop interference score, Albinet, Abou-Dest, Andre, & Audiffren, 2016). These two findings are in line with the neurovisceral integration model (Thayer et al., 2009). One study observed mixed-findings between resting CVA (assessed with high-frequency HRV) and Stroop accuracy (i.e., Stroop interference score) (Subramanya & Telles, 2015). However, the experimental manipulation (meditation) occurring before the resting measurement might have introduced some confounding effects regarding the interpretation of high-frequency HRV, given that it is supposed to reflect CVA only when respiratory frequency is comprised between 9 and 24 cycles per minute (Berntson et al., 1997; Malik, 1996). As respiratory frequency was not assessed in the study, it is not possible to draw firm conclusions about CVA. The current experiment will investigate both accuracy and reaction time for the Stroop task controlling for respiratory frequency.

#### 4.7.2 Working memory and cardiac vagal activity

Working memory involves working with information no longer perceptually present (Baddeley & Hitch, 1994). Stated differently, working memory involves holding information in mind and mentally working with it (Diamond, 2013). A classical test to assess working memory capacity is the automated operation span task (AOSPAN, Unsworth, Heitz, Schrock, & Engle, 2005). The AOSPAN task requires participants to solve mathematics problems while holding a number of unrelated letters in memory.

Previous research has found a positive relationship between resting CVA and AOSPAN performance (Laborde, Furley, & Schempp, 2015), and a negative relationship between task CVA (i.e., CVA measured during the AOSPAN) and AOSPAN performance when the task is realized under high pressure (Mosley, Laborde, & Kavanagh, 2018), which might reflect the adaptation of CVA to the demands of the situation (Mosley et al., 2018). Positive relationships between resting CVA and other tasks that reflect working memory have also been reported in the literature (Hansen, Johnsen, Sollers, Stenvik, & Thayer, 2004; Hansen, Johnsen, & Thayer, 2003; Hansen, Johnsen, & Thayer, 2009; Morandi et al., 2019; Pu, Schmeichel, & Demaree, 2010; Sebastiani, Di Gruttola, Incognito, Menardo, & Santarcangelo, 2019).

#### 4.7.3 Cognitive flexibility and cardiac vagal activity

Cognitive flexibility builds on inhibition and working memory (Davidson, Amso, Anderson, & Diamond, 2006; Diamond, 2013). Cognitive flexibility involves being able to change perspective, in particular, spatially or interpersonally (Diamond, 2013). To change perspective, there is the need to inhibit a previous perspective, and “load” a new perspective into working memory. A classical way to investigate cognitive flexibility is via the Wisconsin card sorting task (WCST, Milner, 1982; Stuss et al., 2000). In this test, each card can be sorted by color, shape, or number. Participants have to deduce the correct sorting criterion on the basis of the feedback they receive, and adapt to the new sorting rule as fast as they receive feedback that the sorting rule has changed. Previous research has shown a positive relationship between resting CVA and performance on the WCST (i.e., negative relationship with decision errors; Albinet, Boucard, Bouquet, & Audiffren, 2010; Hovland et al., 2012; Mathewson, Jetha, Goldberg, & Schmidt, 2012), and this is similar to what has been found in other tasks of cognitive flexibility (Alba, Vila, Rey, Montoya, & Munoz, 2019; Colzato, Jongkees, de Wit, van der Molen, & Steenbergen, 2018).

#### 4.7.4 Executive functioning and slow-paced breathing

The influence of SPB on executive functioning has received little attention, with a focus on inhibition and working memory on the one hand (Prinsloo et al., 2011), and decision making on the other hand (De Couck et al., 2019). Decision making does not belong to the core executive functions but is considered a higher-order executive function that relies on core executive functions (Diamond, 2013). Prinsloo et al. (2011) used a modified Stroop test, where the inhibition component is combined to a working memory component, and participants were asked to remember how many white squares appeared on the screen. Participants were either allocated to a SPB condition or a control condition (where they had to breathe spontaneously), for a total of 10 minutes in each conditions. The SPB was realized with biofeedback, meaning the participants were seeing the effects of SPB on their HRV via a dedicated device. No differences were found between conditions on the Stroop inhibition component (i.e., number of errors), but the SPB group performed better than control on the working memory component. This important foundational research had some limitations including a small sample size ( $n = 18$  for a between-subject design), an assessment of inhibition and working memory that was mixed into the same modified Stroop test, and no biomarkers of CVA.

In a subsequent experiment by De Couck et al. (2019), a multiple-choice test was used to investigate decision making. The test included seven questions related to a decision-making scenario in a management context. Several possible alternative answers were provided but only one answer reflected the most efficient way a manager should act. The two conditions were a group performing SPB for two minutes and a group breathing spontaneously during the same period (control group). The SPB group was found to perform better on the decision-making task than the control group. This study also had some limitations with respect to the current research question, including no biomarkers of CVA, and the breathing was not strictly paced (participants had to mentally count how long inhalation and exhalation were lasting). Overall,

## Theoretical Background

research investigating the effects of SPB on the three core executive functions is limited (with available studies characterized by some important methodological shortcomings) and has not been grounded within a solid theoretical framework.

After reviewing the research related to SPB, CVA, and the self-regulation phenomena I will focus on in this PhD thesis (i.e., stress management, sleep, executive function), I now introduce in the next sections the main research questions and related hypotheses.

## *Research Questions & Hypotheses*

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### 5 Research Questions & Hypotheses

To sum up, if SPB interventions with biofeedback received a lot of attention so far (Gevirtz, 2013; Goessl et al., 2017; Kennedy & Parker, 2018; Wheat & Larkin, 2010; Yu et al., 2018; Zaccaro et al., 2018), this is not the case for SPB interventions without biofeedback, and further research is therefore required regarding this modality of practicing SPB. More specifically, **this PhD thesis aims to focus on understanding the underlying mechanisms of SPB regarding its action on self-regulation phenomena, and to test specifically whether SPB influences CVA**, given one of the suggested mechanisms of action of SPB is its ability to stimulate vagal afferents, as postulated by the resonance model (Lehrer & Gevirtz, 2014).

Consequently, my **main working hypothesis** is that SPB, given its suggested action on vagal afferents, would trigger an increase in CVA. Given according to the neurovisceral integration model (Smith et al., 2017; Thayer et al., 2009), CVA is assumed to reflect the effectiveness of the central autonomic network, and hence overall self-regulation effectiveness, I hypothesized that SPB influences positively self-regulation mechanisms via its action on the central autonomic network, and consequently its effects will be reflected in CVA. Within this PhD thesis, I will investigate this research question and test this main hypothesis in four contexts:

- In **Study 1**, I investigate the effects of short-term SPB in a sample of adolescents with intellectual disability. This would enable to clarify whether SPB can be applied not only in healthy samples. In this study, I hypothesize that after performing a short-term SPB session, CVA would be higher during a cognitive stress challenge, in comparison to control.

- In **Study 2**, I investigate the effects of a long-term SPB intervention (30 days) in healthy young adults on CVA measured during the night and when awakening. I hypothesize that after a 30 days SPB intervention, in comparison to baseline and in comparison to control, CVA will be higher during the night and when awakening.
- In **Study 3**, I investigate the effects of short-term SPB in healthy young adults on the three main executive functions, namely inhibition, working memory, and cognitive flexibility. I hypothesize that SPB will increase executive performance in comparison to control, and that results will be mediated by CVA.
- In **Study 4**, I investigate the effects of short-term SPB in healthy young adults on inhibition performance after performing physical exertion. I hypothesize that SPB will increase inhibition performance in comparison to control, and that results will be mediated by CVA.

In order to perform these four studies, some methodological aspects have to be considered, and the aspects related to CVA measurement and to delivering the SPB intervention will be kept mostly constant across the four studies. Those aspects are described in the next section.

## 6 General Method

### 6.1 Cardiac vagal activity measurement

An electrocardiogram (ECG) device was used to measure HRV (Faros 180°, Bittium, Kuopio, Finland). The sampling rate was 500 Hz. Two disposable ECG pre-gelled electrodes were used (Ambu L-00-S/25, Ambu GmbH, Bad Nauheim, Germany). The negative electrode was placed in the right infraclavicular fossa (just below the right clavicle) and the positive electrode was placed on the left side of the chest, below the pectoral muscle in the left anterior axillary line. The full ECG recording was inspected visually, and artefacts were corrected manually (Laborde, Mosley, et al., 2017). From ECG recording we extracted RMSSD and HF-HRV using the software Kubios (University of Eastern Finland, Kuopio, Finland). RMSSD was chosen to operationalize CVA in studies 1, 3 and 4 as it is less affected by respiration (Hill, Siebenbrock, Sollers, & Thayer, 2009), while HF-HRV was chosen in study 2 given different durations were measured during the night for each participant.

Given the current debate regarding whether or not to control for respiratory parameters when assessing HRV (Grossman et al., 1991; Grossman & Kollai, 1993; Laborde, Mosley, et al., 2017; Larsen, Tzeng, Sin, & Galletly, 2010; Thayer, Loerbroks, & Sternberg, 2011), respiratory frequency was also calculated in order to better understand whether potential changes in RMSSD are related to CVA or are affected by changes in respiratory frequency. Respiratory frequency was computed via the ECG derived respiration algorithm of Kubios (Tarvainen, Niskanen, Lipponen, Ranta-Aho, & Karjalainen, 2014).

## 6.2 Slow-paced breathing exercise

The most common way to realize the breathing pacing is via visual stimuli (e.g., Allen & Friedman, 2012; Laborde, Allen, Gohring, & Dosseville, 2017; Tsai et al., 2015), while auditive or kinesthetic methods (e.g., vibrations) have been used less frequently. In studies 1, 3, and 4, the SPB exercise was realized with the help of a video showing a ball moving up and down at the rate of 6cpm. The participants had to inhale continuously through the nose while the ball was going up, and exhale continuously with pursed lips when the ball was going down. This was a video capture of the software EZ-Air Plus (Biofeedback Federation of Europe<sup>2</sup>). The video displayed a 3 x 5 min SPB exercise (see Figure 16), with a 1-min break between each 5 min SPB unit, corresponding to a total of 17 min. The 1 min break between each SPB unit was introduced as some participants reported in a pilot study that 15 minutes of non-stop SPB was very demanding. Exhalation (5.5 s) lasted slightly longer than inhalation (4.5 s) as prolonged exhalation contributes to larger beat-to-beat heart fluctuations compared to a prolonged inhalation, and therefore induces a higher CVA (Komori, 2018; Matsumoto et al., 2011; Strauss-Blasche et al., 2000; Van Diest et al., 2014). In Study 2, the SPB technique was realized with the smartphone app Breath Pacer, displaying a flower slowly adding petals to indicate inhalation and exhalation phases (see Figure 17).

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<sup>2</sup> <https://bfe.org/new/try-our-breath-pacer-ez-air-plus/>



General method

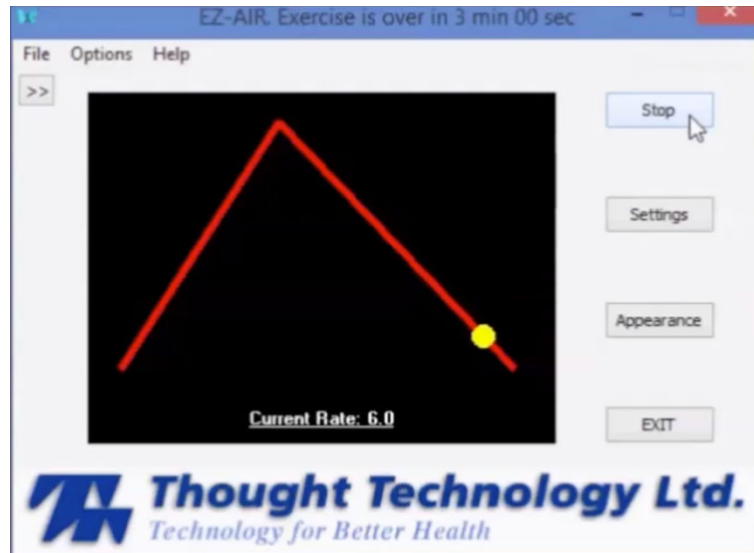


Figure 16: Video based on the software EZ-air (used in Study 1, 3, 4)



Figure 17: Smartphone App Breath Pacer (used in Study 2)

A familiarization period for SPB was created in order for participants to become familiar with the technique. Inhaling via the nose (i.e., nasal breathing) is important because the air is

more humid and clean (Lorig, 2011), and also because it provokes optimal activation of neural networks linked to stimulus processing and behavior (Zelano et al., 2016). Exhaling takes place via the mouth, which offers less ventilatory resistance than the nasal channel (Lorig, 2011). Moreover, exhalation is realized via pursed-lips, which enables greater control over the flow of air enabling participants to match it precisely to the exhalation duration. Pursed-lip breathing imposes a resistance on the expiratory flow, which leads to a positive expiratory pressure in the airways. The expiratory delay promotes homogenous emptying of the lungs, maintaining the intrabronchial pressure and favoring gas exchange and ventilation (Custodio, 1998; Nield, Soo Hoo, Roper, & Santiago, 2007; Ramos et al., 2009). Participants were then asked to put one hand on their chest and one hand on their stomach and were given the following instructions: “The hand on the chest should not move, only the hand on the belly should move: The belly should get bigger during the inhalation phase, and smaller during the exhalation phase.” This instruction reflects an optimal activation of the diaphragm. When the diaphragm contracts and goes down, it increases the volume of the thoracic cavity and creates an area of low pressure that causes air to flow into the lungs to equalize the pressure (Colbert et al., 2016; Hall, 2011a).

During spontaneous breathing exhalation is mostly passive. However, in SPB the forced exhalation can involve abdominal muscles which, via their contraction, help to push the diaphragm back up to the thorax, and consequently push out additional air (Lorig, 2011; West, 2015; West & Luks, 2016). The breathing frequency is progressively decreased with 2 min units to 10 cpm, 8 cpm, and then 6 cpm, with a 1 min break between each unit. The SPB technique requires the participant to breathe in and breathe out continuously and uniformly when the ball goes up and down respectively. When this instruction is correctly followed, the sine-waves oscillation characteristics of SPB can be observed in the R-R tachogram (Lehrer & Gevirtz, 2014). The experimenter verified whether the participant was realizing the SPB technique correctly during the familiarization before moving on to the next step of the experiment.

I now present the four studies realized within this PhD thesis:

- **Study 1** (published): The effect of slow-paced breathing on stress management in adolescents with intellectual disability (p.54)
- **Study 2** (published): Influence of a 30-day slow paced breathing intervention compared to social media use on subjective sleep quality and cardiac vagal activity (p.62)
- **Study 3** (submitted): The influence of slow-paced breathing on executive function (p.74)
- **Study 4** (published): Influence of slow-paced breathing on inhibition after physical exertion (p.90)

## 7 Study 1 (published) - The effect of slow-paced breathing on stress management in adolescents with intellectual disability



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### The effect of slow-paced breathing on stress management in adolescents with intellectual disability

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#### Abstract

**Background** Intellectual disabilities often create a state of chronic stress for both the person concerned and their significant others (family, caregivers). The development of stress management methods is therefore important for the reduction of stress in persons with intellectual disability. The aim of this experiment was to investigate the effect of slow-paced breathing on stress symptoms experienced by adolescents with intellectual disabilities during a cognitive task under time pressure.

**Method** Fourteen adolescents with intellectual disabilities ( $M_{\text{age}} = 17.39$  years, range 15–19 years) took part in two laboratory sessions – a slow-paced breathing session (experimental condition) and an audiobook session (control condition) – the order of which was counterbalanced across participants. Vagal tone was measured through heart rate variability to index stress management.

**Results** No difference in vagal tone was observed at baseline between experimental and control conditions. Compared with the control condition, vagal tone was significantly higher during the experimental condition.

**Conclusions** The slow-paced breathing task enhanced stress management to a greater extent than

did listening to an audiobook. Slow-paced breathing seems to be an easy to learn stress management technique that appears as an effective auxiliary method of lowering stress in adolescents with intellectual disabilities.

**Keywords** coping, emotion, heart rate variability, parasympathetic activity, pressure, RMSSD

#### Introduction

Having an intellectual disability (ID) is associated with high levels of stress (e.g., Forte *et al.* 2011). This association can be explained in part by additional difficulties in adaptation and finding everyday situations overdemanding, as well as being socially marginalised (de Bildt *et al.* 2005). Moreover, an individual with IDs often use maladaptive coping strategies to cope with stress (Hartley & Maclean 2008). The stress experienced by individuals with IDs is often transferred to family members and caregivers (e.g., Hassall & Rose 2005). In the long term, if chronic stress in persons with IDs is not properly addressed, it can lead to serious complications including depression (Hartley & Maclean 2009), impaired cognitive functions (Heyman & Hauser-Cram 2015), physical health problems (Lunsky 2008) and maladaptive coping strategies such as substance abuse (Didden *et al.* 2009). It can also lead to family members and caregivers experiencing depression

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(Mutkins *et al.* 2011) and burnout (Innstrand *et al.* 2002). This experiment aimed to test the effectiveness of slow-paced breathing in alleviating symptoms of stress in a sample of adolescents with IDs.

Most stress management programmes have targeted parents and caregivers rather than persons with IDs themselves (Ciechomski *et al.* 2001), and this might be explained by the challenging nature of teaching stress management strategies to persons with IDs. Few studies have targeted the individuals themselves, but those available suggest that programmes designed to reduce stress in the general population might not be particularly effective for individuals with IDs. For instance, an 8-week cardiovascular disease risk reduction intervention was found to be effective in alleviating stress for healthy adults but ineffective for those with IDs (Ewing *et al.* 2004). However, a combined assertiveness and problem-solving intervention was found to be effective in reducing subjective stress in adults with IDs (Nezu *et al.* 1991). To the best of our knowledge, no published research has investigated the efficacy of a targeted stress management method for alleviating the physiological symptoms of stress in persons with IDs. Slow-paced breathing has been identified as an effective stress management intervention in both adults (e.g., Wells *et al.* 2012) and children (e.g., Uratani *et al.* 2014). This intervention technique might also be suited to individuals with IDs given the low cognitive demands of the task.

Vagal tone is a biological process that reflects the activity of the parasympathetic nervous system (Malik 1996). It is not a stress marker per se, but rather, a marker of resources available to manage stress, emotion and health regulation (Appelhans & Luecken 2006; Thayer *et al.* 2009). Vagal tone can be measured through heart rate variability (HRV) – the change in time intervals between adjacent heartbeats (Malik 1996; Shaffer *et al.* 2014). When we refer to vagal tone, we are referring more specifically to cardiac efferent vagal tone that is measured through HRV. Under stressful conditions, vagal activity drops to enable the system to regulate metabolic output in response to environmental demands (Porges 2007). However, this response can be counterproductive if it occurs in situations that require a higher level of vagal activity (e.g., during cognitive tasks). In this instance, a lower vagal withdrawal is associated with better stress management and better cognitive performance

under stress (Laborde & Raab 2013; Laborde *et al.* 2014; Laborde *et al.*, 2015a,b).

According to the resonance frequency model, slow-paced breathing can increase vagal tone (Lehrer 2013). The mechanisms are based on the coupling between respiration at a specific pace – on average 6 cycles per minute (cpm) – and blood pressure, which triggers the resonance properties of the cardiovascular system given its action on the baroreflex. This phase coupling results in improved gas exchange and oxygen saturation, as well as an increase in vagal afferences (Lehrer 2013; Lehrer & Gevirtz 2014). Most people breathe between 12 and 20 cpm (Tortora & Derrickson 2014; Sherwood 2006), and this is why structured breathing of 6 cpm is referred to as ‘slow’-paced breathing. The term ‘paced’ means that participants have to follow a pacer regulating their inhalation and exhalation phases, with exhalation lasting slightly longer than inhalation. This is because a prolonged exhalation contributes to larger beat-to-beat heart fluctuations compared with prolonged inhalation, and hence inducing a higher vagal tone (Strauss-Blasche *et al.* 2000).

To summarise, slow-paced breathing increases vagal tone (Lehrer 2013; Lehrer & Gevirtz 2014) and has been found to have a positive (alleviating) effect on both the subjective and physiological aspects of stress in non-intellectually disabled adults (Wells *et al.* 2012) and children (Uratani *et al.* 2014). However, this technique has not been tested in persons with IDs. The aim of this experiment was to investigate the effects of a single session of slow-paced breathing on the physiological reaction to cognitive stress through vagal tone. We test this method against a standard stress management control condition (i.e., listening to an audiobook). The audiobook was chosen as it often has a calming effect on children and adolescents in comparison to a silence condition (Sunitha Suresh *et al.* 2015). Based on findings for slow-paced breathing on vagal tone (Wells *et al.* 2012; Lehrer 2013; Lehrer & Gevirtz 2014), we hypothesised that vagal tone would be higher during the cognitive stress task after the slow-paced breathing condition compared with after the audiobook condition. A within-subject design was used as advised for HRV research to reduce inter-individual differences (Quintana & Heathers 2014). To summarise, we use a within-subject experimental design in which participants attend two testing sessions (slow paced

breathing vs. audiobook), with two measurement times for vagal tone (baseline vs. during a cognitive stress task).

## Methods

### Participants

Sixteen adolescents (14 boys, 2 girls) took part in the experiment (mean age = 17.39 years, range = 15–19 years). Participants had been diagnosed with a diverse range of developmental disabilities (e.g., Down syndrome, Dravet syndrome). No participants were suffering from blood pressure problems, and no participants were taking medicine affecting the cardiovascular system. On the day of the experiment, participants were asked to follow their normal sleep routine, not to eat or drink in the 2 h before the experiment, and not to perform any physical activity the previous 24 h (parents received the instructions to ensure that their children would respond to them). Ethics approval was obtained from a university research ethics committee. All participants were attending the same specialised school. The school gave its agreement to have the study performed at the school, and written informed consent was obtained from the parents of all participants, with participants providing verbal assent.

### Cognitive stress task: Kaufman-Assessment Battery of Children

The Kaufman-Assessment Battery of Children was used as a cognitive stress task. The Kaufman-Assessment Battery of Children is a standardised test to evaluate cognitive abilities in children (Kaufman & Kaufman 1983; Kaufman *et al.* 1987). The full version contains 16 subtests and is suited for an age range between 2.6 and 12.5 years. This test can also be used with people that have an intellectual handicap (Kaufman *et al.* 1987; Maluck & Melchers 1998). Because the purpose of the experiment was to induce a stressful situation, we chose three tests that solicited cognitive abilities of participants in an ascending order of difficulty (1, Number Recall; 2, Reading/Decoding; 3, Riddles). A 5-min time limit was also given to complete the task in order to induce additional time pressure (stress). In order for the adolescents to visualise the time limit, a 5-min

hourglass was placed in front of participants for the duration of the task.

The subtest *Number Recall* required the child to repeat, in sequence, a series of numbers presented orally by the examiner. A child's performance on the number recall subtest is influenced by anxiety, attention span, and distractibility (Kaufman *et al.* 1987). The subtest *Reading/Decoding* required the child to identify letters and to read and pronounce words. As an example, the examiner presents a stimulus page with three words, pointed to each word, and asked 'what is the word?'. A child's performance on this subtest may be influenced by his or her alertness to the environment (Kaufman *et al.* 1987). The final subtest *Riddles* required the child to infer the name of a concrete or abstract concept, after having being given its attributes, functions, and other general characteristics. The child's performance on this subtest is influenced by his or her alertness to the environment, attention span, distractibility, concentration, and impulsivity (Kaufman *et al.* 1987).

### Slow-paced breathing exercise

The slow-paced breathing exercise was realised with a video showing a little ball moving up and down at the rate of 6 cpm, the participants having to inhale continuously through the nose while the ball was going up, and exhale continuously with pursed lips when the ball was going down. For the purpose of the experiment, a video was realised using the software EZ-AIR PLUS (Tought Technology Ltd.<sup>1</sup> Montreal, Canada), displaying a 3 × 5 min slow-paced breathing exercise, at a respiratory rate of 6 cpm, with a 1-min break between each unit, corresponding to a total of 17 min. Exhalation (5.5 s) was slightly longer than inhalation (4.5 s), because a prolonged exhalation contributes to larger beat-to-beat heart fluctuations compared with relatively prolonged inhalation, and therefore induces a higher vagal tone (Strauss-Blasche *et al.* 2000).

### Audiobook

As an audiobook, we used 'Die Drei Fragezeichen und der Phantomsee – Folge 2' (The 3 Interrogation Marks and the Phantom of the Lake – Episode 2). The first three tracks of the audiobook were used.

<sup>1</sup> <http://bfe.org/new/try-our-breath-pacer-ez-air-plus/>

This audiobook is based on a German children book generally recommend from 8 years on, and provides participants with a story about adventure and friendship.

#### Heart rate variability recording

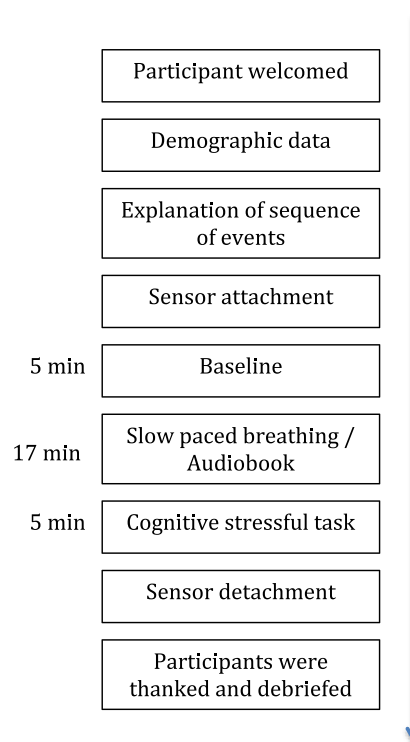
HRV was measured using an eMotion HRV device (Mega Electronics, Kuopio, Finland), with a sampling rate of 1000 Hz. We used two disposable ECG pre-gelled electrodes (Ambu L-00-S/25, Ambu GmbH, Bad Nauheim, Germany). The negative electrode was placed in the right infraclavicular fossa (just below the right clavicle) while the positive electrode was placed on the left side of the chest, below the pectoral muscle in the left anterior axillary line. From heart rate recordings, we extracted HRV using interbeat interval data that was exported to Kubios software (University of Eastern Finland, Kuopio, Finland). Artefacts were removed using the automatic low filter provided by the Kubios software. We calculated time domain parameters and used the root mean square of the successive differences (RMSSD) as an indicator of vagal tone (Malik 1996). We decided not to adjust statistically vagal tone for respiratory frequency in our analyses, given this could mask true variations in vagal tone (Denver *et al.* 2007; Thayer *et al.* 2011).

#### Procedure

Testing took place at a specialised school for adolescents with IDs in a room they were familiar with. Only the experimenter was present during testing. The same experimenter (third author) performed all testing. The children were familiar with the experimenter who was interning at the school during this time. Participants first received a group instruction session lasting 40 min, explaining the two experimental sessions, in small groups ( $n = 3-6$ ). Given the diverse cognitive abilities of the participants, a specific introduction was designed with picture cards, to explain the sequential course of events, as well as the slow-paced breathing technique itself, using pictures and very easy sentences. The picture cards were hanged in the chronological order of the sequence of events. The explanations under the pictures were read together with the participants, and open questions were answered, until all participants understood what they had to do during the

experiment. In addition, the slow-paced breathing technique was explained together with an introductory video and was practiced until the participants were able to perform it, in order to ensure they were familiar with the protocols before the experiment.

Three weeks after this introductory session, participants completed the first condition. The delay between the introductory session and the first condition was because of organisational issues at the specialised school, where the experimenter had to adapt to the school schedule and availability of facilities. The order of experimental and control conditions was counterbalanced across participants, with a 1-week delay between each condition. The full experimental protocol is depicted in Fig. 1. Upon arrival, participants were welcomed and were once more provided a description of the sequence of events



**Figure 1** Experimental protocol. [Colour figure can be viewed at [wileyonlinelibrary.com](http://wileyonlinelibrary.com)]



before electrodes were attached. For the baseline, participants were required to sit with their legs at a 90° angle, their hands on their thighs, and their eyes closed. Participants then either listened to the audiobook or completed the slow-paced breathing exercise, each of which lasted a total of 17 min. Participants then undertook the cognitive stress task in front of the 5-min hourglass. The change between the three subtests happened either after the successful completion of the final task of the previous subset or after five unsuccessful repetitions of a task. Immediately after the stress task, electrodes were detached. HRV was measured throughout testing. At each stage of testing, to visualise the sequence of events, participants were allowed to remove from the wall the card representing the event after having completed it. In total, testing lasted approximately 45 min.

#### Data preparation

We first checked our data for normality and outliers. The RMSSD data did not have a normal distribution. RMSSD data were transformed using log transform as is common in HRV research (e.g., Houtveen *et al.* 2002). After checking the slow-paced breathing data for normality, we checked visually with Kubios (HRV analysis software) whether participants correctly performed the 6-cpm breathing technique, and more specifically, we ensured that all participants followed the 4.5-s inhalation/5.5-s exhalation ratio. We found that two participants did not display the sinusoidal usually observed (Lehrer & Gevirtz 2014, Fig. 1) in the slow-paced breathing condition, meaning that they did not manage to perform the slow-paced breathing exercise correctly. These participants were therefore excluded from further analyses, and the sample size used for our analysis was  $n = 14$ .

#### Data analysis

To test our main hypothesis, where vagal activity during the cognitive stress task will be higher in the slow-paced breathing condition compared with the audiobook condition, we ran a 2 (condition: slow-paced breathing vs. audiobook)  $\times$  2 (time: baseline vs. cognitive stress task) repeated-measures ANOVA, with log<sub>10</sub> RMSSD set as the dependent variable.

## Results

Means and standard deviations for measured variables across experimental and control conditions are reported in Table 1.

For vagal tone, a repeated-measures ANOVA indicated no main effect of condition,  $F(1, 13) = 1.08$ ,  $p = .319$ ,  $\eta^2 = .08$ , Wilks'  $\lambda = 0.92$ . A main effect of time was found,  $F(1, 13) = 10.97$ ,  $p = .006$ ,  $\eta^2 = .46$ , Wilks'  $\lambda = 0.54$ ; log<sub>10</sub> RMSSD being higher at Time 2 ( $M = 1.61$ ,  $SD = 0.06$ ) in comparison with Time 1 ( $M = 1.50$ ,  $SD = 0.07$ ). A main interaction effect of condition  $\times$  time was found,  $F(1, 13) = 5.91$ ,  $p = .030$ ,  $\eta^2 = .31$ , Wilks'  $\lambda = 0.69$ . Follow-up  $t$ -tests showed that there was no difference at baseline,  $t(13) = 0.95$ ,  $p = .360$ ,  $d = 0.12$ , but there was a significant difference during the cognitive stress task,  $t(13) = 2.62$ ,  $p = .021$ ,  $d = 0.35$ . During the cognitive stress task, log<sub>10</sub> RMSSD of the slow-paced breathing condition being higher ( $M = 1.65$ ,  $SD = 0.24$ ) than log<sub>10</sub> RMSSD of the audiobook condition ( $M = 1.57$ ,  $SD = 0.23$ ).

## Discussion

The aim of this experiment was to investigate whether a single session of slow-paced breathing, in comparison with a passive relaxation condition (listening to an audiobook), would be reflected in a higher vagal activity during a cognitive stress task. Our main hypothesis was supported. Vagal tone did not differ at baseline between the two conditions, but during cognitive stress, vagal tone was higher in the slow-paced breathing condition compared with the audiobook condition.

Our findings suggest that slow-paced breathing can enhance vagal tone during stressful conditions in a

Table 1 Descriptive statistics

|                  | Slow-paced breathing |       | Audiobook |       |
|------------------|----------------------|-------|-----------|-------|
|                  | M                    | SD    | M         | SD    |
| RMSSD – Baseline | 36.66                | 22.50 | 39.48     | 22.92 |
| RMSSD – Task     | 50.33                | 22.20 | 42.79     | 24.75 |

RMSSD, root mean square of the successive differences (in milliseconds).



sample of adolescents with IDs. A higher vagal tone reflects better stress management during high-stress conditions (Laborde & Raab 2013; Laborde *et al.* 2014; Laborde *et al.* 2015a, 2015b). Our findings are in line with previous research that has found slow-paced breathing to be effective in managing stress in child (Uratani *et al.* 2014) and adult samples (Wells *et al.* 2012). As far as we are aware, this is the first study to explore slow-paced breathing as a method of reducing physiological stress symptoms in a sample of persons with IDs. Assuming these findings can be replicated through further independent research trials, there might be considerable practical value attached to our findings. The slow-paced breathing exercise can be learned and applied with relative ease, and is therefore suited to a variety of environments (home and school). Future research might explore whether there are additional benefits (such as the reduction of stress in caregivers and parents) and whether slow-paced breathing might be incorporated into multimodal interventions targeting stress reduction through other means.

The findings of this investigation are encouraging. However, there are a number of potential shortcomings that researchers must keep in mind when interpreting study findings. First, the most obvious limitation is the small sample size, meaning statistical power for the study is low and therefore we cannot discount the possibility that significant findings reflect a Type I error. The small sample size also means that more complex analytical techniques that have better error variance control (e.g., growth modelling) could not be performed on the data. Second, the sample was predominantly adolescent boys (plus two girls), and findings cannot be generalised beyond this population. Third, two participants were also unable to correctly complete the slow-paced breathing exercise during the experimental session. Further research is required to identify the optimal number of sessions needed to learn the breathing technique in adolescents with IDs. In other populations, it has been found that three sessions are often required to become familiar with the exercise (Vaschillo *et al.* 2006). The use of a biofeedback system might also facilitate the learning process, as the person can directly visualise the effects of slow-paced breathing on heart rate variability (Lehrer & Gevirtz 2014).

A fourth limitation is that we did not record any performance markers for the stress task (e.g., the number of attempts, and/or the time taken to complete each section), meaning we are unable to determine individual differences in the relative difficulty of the task between participants. Fifth, we did not use any subjective measure of stress, given it is usually hard for participants with an ID to complete self-report measures. This is compensated in our study by the objective assessment of stress regulation through vagal activity. However, what we assessed in this study reflects the physiological resources available to manage stress and not the subjective state of being stressed. Sixth, because of organisational issues at the specialised school, there was a 3-week delay between the introductory session on slow-paced breathing and the first testing session, and this might have contributed to some participants having difficulty remembering how to perform the breathing task. Nevertheless, at the beginning of the slow-paced breathing condition, the participants were reminded how to perform it, and all participants followed the 4.5-s inhalation/5.5-s exhalation ratio. A final limitation of this experiment is that we targeted only short-term effects of slow-paced breathing. Research in non-intellectually disabled adults has demonstrated important long-term effects through regular practice (Hassett *et al.* 2007; Karavidas *et al.* 2007), and further research is required to identify potential long-term adaptation effects in persons with IDs.

## Conclusion

This experiment aimed to compare the effects of slow-paced breathing and audiobook listening on stress management (measured through vagal tone) during a cognitive stress task in adolescents with IDs. We found that a single session of slow-paced breathing (preceded by an introduction session to slow paced breathing) was able to enhance vagal tone under cognitive stress to a greater degree than listening to an audiobook. This initial finding is promising, and the technique has potential applied value for a variety of settings (e.g., in school, at home). The main advantage of slow-paced breathing is that it is a brief intervention that can be implemented with relative ease and little training by

caregiving professionals or family members. We encourage further research into the potential short-term and long-term benefits accompanying slow-paced breathing exercises in persons with intellectual disabilities.

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## 8 Study 2 (published) - Influence of a 30-day slow paced breathing intervention compared to social media use on subjective sleep quality and cardiac vagal activity



Article

### Influence of a 30-Day Slow-Paced Breathing Intervention Compared to Social Media Use on Subjective Sleep Quality and Cardiac Vagal Activity

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**Abstract:** Breathing techniques are part of traditional relaxation methods; however, their influence on psychophysiological variables related to sleep is still unclear. Consequently, the aim of this paper was to investigate the influence of a 30-day slow-paced breathing intervention compared to social media use on subjective sleep quality and cardiac vagal activity (CVA, operationalized via high-frequency heart rate variability). Healthy participants ( $n = 64$ , 33 male, 31 female,  $M = 22.11$ ,  $SD = 3.12$ ) were randomly allocated to an experimental or control group. In the experimental group, they had to perform slow-paced breathing for 15 min each evening across a 30-day period. This was administered through a smartphone application. The control group used social media (e.g., Facebook, Instagram, Whatsapp) for the same duration. The night before and after the intervention, their CVA was assessed via a light portable Electrocardiogram (ECG) device, and they had to fill out the Pittsburgh Sleep Quality Index questionnaire. Results showed that in comparison to the use of social media, the slow-paced breathing technique improved subjective sleep quality and increased overnight CVA, while a tendency was observed for morning awakening CVA. Slow-paced breathing appears a promising cost-effective technique to improve subjective sleep quality and cardiovascular function during sleep in young healthy individuals.

**Keywords:** parasympathetic nervous system; cardiac vagal tone; high-frequency heart rate variability (HF-HRV); deep breathing; slow breathing; cardiac coherence; vagus nerve; respiratory sinus arrhythmia; vagal tank theory; neurovisceral integration model

#### 1. Introduction

Issues with sleep are a pressing concern for individuals, given they directly impact life quality and represent a risk factor at several levels [1]. Breathing techniques are part of traditional methods used to improve sleep [2]; however, their influence on psychophysiological variables related to sleep is still unclear. This paper is aimed to investigate the influence of a slow-paced breathing intervention on subjective sleep quality and on a psychophysiological variable linked to relaxation states, cardiac vagal activity (CVA), which reflects the activity of the vagus nerve regulating cardiac functioning [3–5].

One of the main hypothesis regarding the cause of sleep disturbances is that they may be associated with a state of hyperarousal [6,7]. Methods aiming to decrease a state of hyperarousal usually target an activation of the parasympathetic nervous system, and more specifically of its main nerve, the vagus nerve [8,9]. One way to do so is to use slow-paced breathing [10–13].

Spontaneously, most people breathe between 12 and 20 cycles per minute [14,15]. Slow-paced breathing refers to the act of voluntarily slowing down the breathing rate to a frequency close to 6 cycles per minute (cpm) [12]. The term “paced” means that participants have to follow a visual, auditory, or kinesthetics pacer regulating the duration of inhalation and exhalation phases (for example see [16,17]). Importantly, exhalation should last slightly longer than inhalation, provoking a higher increase of CVA due to the activation of the vagus nerve during exhalation [18,19]. According to the resonance frequency model [12,20], four processes help to explain the positive effects of performing slow-paced breathing at 6 cpm: (1) the phase relationship between heart rate oscillations and breathing at 6 cpm; (2) the phase relationship between heart rate and blood pressure oscillations at 6 cpm; (3) the activity of the baroreflex; and (4) the resonance characteristics of the cardiovascular system. Combined, those processes are expected to strengthen homeostasis in the baroreceptor [21–23], which results in improving gas exchanges at the level of alveoli and in increasing vagal afferences [12,20].

Slow-paced breathing is assumed to increase the activity of the afferent branch of the vagus nerve [12,20]. Although it is not possible to non-invasively measure the afferent activity of the vagus nerve, there is a way to operationalize non-invasively the efferent activity of the vagus nerve, and more specifically the activity of the vagus nerve regulating cardiac functioning (i.e., CVA) via heart rate variability [3–5,24,25]. Dozens of parameters can be extracted from heart rate variability analysis; however, only a handful have been found to reflect CVA, and in the current study we operationalize CVA via one of its most common indicators: high-frequency heart rate variability [3–5,24,25].

CVA represents the core of several theories (for a summary, see [3]), such as the neurovisceral integration model [26], the polyvagal theory [27], or the more recent vagal tank theory [28]. Taken together, those theories highlight the role of CVA in phenomena such as emotion and stress regulation, executive cognitive performance, social functioning, and health. In order to better understand CVA functioning, it is important to consider several levels of functioning: resting, reactivity, and recovery [3,28]. Further, CVA can be influenced by many factors [29,30], and slow-paced breathing is a straightforward method to increase CVA resting levels [12,16,31].

The question whether measuring CVA during sleep ( $CVA_{\text{night}}$ ) represents an indicator of sleep quality is still debated. Some evidence points toward an association between lower  $CVA_{\text{night}}$  and sleep disorders [32], such as chronic fatigue [33] and insomnia [34]. Higher  $CVA_{\text{night}}$  has also been related to higher subjective sleep quality [34–36]. However, some authors argue that measuring  $CVA_{\text{night}}$  across sleep stages does not provide useful information given the variations observed in CVA during different sleep stages [37], namely CVA withdrawal during rapid eye movement (REM) sleep, and CVA increase during non-REM sleep [38]. Further, Werner and colleagues [37] argue that assessing CVA while sleeping is suboptimal, given CVA is supposed to reflect adaptations to environmental changes, and these generally do not occur during the night. Thus, they rather recommend assessing CVA during periods where individuals are awake. In summary, even if  $CVA_{\text{night}}$  measurement cannot be considered as an index of sleep quality, it may still provide an indication of the restorative status of the body during sleep, given it indexes the activity of the parasympathetic nervous system [3,4,15].

In order to address the criticisms made to  $CVA_{\text{night}}$  measurements, authors have suggested to measure CVA during wake periods [37]. Particularly, a quiet awakening morning period ( $CVA_{\text{morning}}$ ) has been suggested as a good compromise, given the individual has usually not experienced heavy environmental changes beforehand [39].  $CVA_{\text{morning}}$  has already been related to subjective indices of well-being and to physical training adaptations (see for example [39,40]), and also more recently to subjective sleep quality measurements [41]. Investigating  $CVA_{\text{morning}}$  together with  $CVA_{\text{night}}$  measurements seems therefore an appropriate combination to further understand the effects of slow-paced breathing on CVA.

To the best of our knowledge, only one previous study investigated the effects of slow-paced breathing on sleep [42]. This study, focusing on self-reported insomniacs, aimed to investigate whether a 20-min slow-paced breathing session (6 cpm), compared to a control condition with paced breathing set at 12 cpm, would enhance objective sleep quality as assessed via polysomnography and CVA. In the slow-paced breathing condition, the inhalation and exhalation phases were set to 3 s and 7 s, while no indications were mentioned regarding the inhalation and exhalation phases for the 12 cpm breathing condition. In regards to polysomnography, results showed that after a single 6 cpm session before going to sleep, sleep onset latency, number of awakenings, and awakening time during sleep were decreased, while sleep efficiency was increased, in comparison to the 12 cpm breathing condition and to baseline. Regarding CVA, the heart rate variability variables mentioned in the paper (total power and R-R intervals) unfortunately do not reflect it [3–5,24,25]; therefore, it is not possible to draw any conclusions related to CVA. Moreover, heart rate variability was not assessed during sleep, but during daytime rest. Consequently, further studies are therefore warranted to better understand the effects of slow-paced breathing on subjective sleep quality and CVA, and not only on a short-term single session basis, but also on a long-term intervention basis.

In summary, the current study aims to address previous research gaps, investigating the effects of a 30-day slow-paced breathing intervention on subjective sleep quality and CVA<sub>night</sub> and CVA<sub>morning</sub>. Based on previous research [16,31] and on the resonance frequency model [12], we hypothesize that in comparison to a control condition involving spontaneous breathing, a 30-day slow-paced breathing intervention would increase subjective sleep quality as well as CVA<sub>night</sub> and CVA<sub>morning</sub>. Finally, due to contradictory evidence [34–37], no hypothesis was formulated regarding the relationship between subjective sleep quality with CVA<sub>night</sub> and CVA<sub>morning</sub>.

## **2. Materials and Methods**

### *2.1. Participants*

We recruited 70 participants, randomly allocated to the experimental group or to the control group. Due to technical problems ( $n = 2$ ) and inability to realize the complete intervention protocol for personal reasons ( $n = 4$ ), the data of 64 participants (33 male, 31 female,  $M = 22.11$ ,  $SD = 3.12$ , age range = 18–29 years old) were analysed. The body mass index (BMI) of participants was in the normal range, from 18.5 to  $<25 \text{ kg/m}^2$ . In order to meet the inclusion criteria for the study, participants had to be non-smokers, and not be suffering from sleep disorders (score lower than 5 on the Pittsburgh Sleep Quality Index) or from cardiovascular diseases (self-reported). All participants gave their informed consent for inclusion before they participated to the study. The study was conducted following the Declaration of Helsinki, and the protocol was approved by the Ethics committee of the German Sport University Cologne (Project identification code 42/2015).

### *2.2. Measures*

#### **2.2.1. Subjective Sleep Quality—Pittsburgh Sleep Quality Index**

In order to measure subjective sleep quality, the German version [43] of the Pittsburgh Sleep Quality Index (PSQI; [44]) was used. This self-report questionnaire assesses sleep quality for the four preceding weeks. A total of 18 items serve to generate seven component scores (which values are comprised between 0 and 3): subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. A global measure of subjective sleep quality, ranging from 0 to 21, is then calculated, with lower values indicating better sleep quality (Table 1).

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**Table 1.** Descriptive statistics for subjective variables.

|                   | Pre-Test     |           |          |           | Post-Test    |           |          |           |
|-------------------|--------------|-----------|----------|-----------|--------------|-----------|----------|-----------|
|                   | Experimental |           | Control  |           | Experimental |           | Control  |           |
|                   | <i>M</i>     | <i>SD</i> | <i>M</i> | <i>SD</i> | <i>M</i>     | <i>SD</i> | <i>M</i> | <i>SD</i> |
| Time spent in bed | 447.9        | 55.37     | 450.91   | 60.18     | 446.38       | 55.28     | 447.66   | 58.63     |
| PSQI              | 3.31         | 1.20      | 3.44     | 1.01      | 2.91         | 1.38      | 3.75     | 1.34      |

Note: PSQI = Pittsburgh Sleep Quality Index (a lower score indicates a better sleep quality).

### 2.2.2. Cardiac Vagal Activity (Operationalized via High-Frequency Heart Rate Variability)

In this study, CVA was operationalized via high-frequency heart rate variability (0.15–0.40 Hz) absolute power calculated via fast Fourier transform [3–5]. Additionally, as suggested by Laborde et al. [3], additional heart rate variability variables are presented in the descriptive statistics in Table 2, and the full data set is uploaded as Supplementary Materials. An ECG device (Faros 180°, Mega Electronics, Kuopio, Finland) was used during the experiment to assess heart rate variability, with a sampling rate of 500 Hz. We used two disposable ECG pre-gelled electrodes (Ambu L-00-S/25, Ambu GmbH, Bad Nauheim, Germany). The negative electrode was placed in the right infraclavicular fossa (just below the right clavicle) while the positive electrode was placed on the left side of the chest, below the pectoral muscle in the left anterior axillary line. From ECG recordings we extracted the heart rate variability variables with Kubios© (University of Eastern Finland, Kuopio, Finland). The full ECG recording was inspected visually, and artefacts were corrected manually [3]. Short-term morning measurements followed the five-minute duration recommendation [3,4], while overnight measurements were calculated from the time spent in bed (self-reported by the participants). As recommended by Laborde et al. [3], respiratory frequency was also assessed. In the current study respiratory frequency was computed via the ECG-derived respiration algorithm of Kubios© [45].

### 2.3. Intervention

#### 2.3.1. Experimental Group: Slow-Paced Breathing

Participants in the experimental group had to realize the slow-paced breathing technique for 15 min before going to sleep, using the smartphone app “Breath Pacer”, displaying a flower slowly adding petals to indicate inhalation (4.5 s) and exhalation (5.5 s) phases. Participants had to inhale via the nose, and exhale via pursed lips. The respiratory pattern was based on previous research investigating the influence of slow-paced breathing on psychophysiological outcomes [16,17].

#### 2.3.2. Control Group: Social Media Use

Participants in the control group had to use social media (e.g., Facebook, Instagram, Whatsapp) for 15 min before going to sleep, in order to mirror a typical smartphone use with spontaneous breathing. They were given no specific instructions related to breathing patterns.



**Table 2.** Descriptive statistics for heart rate variability parameters

|                     | Pre-Test     |         |         |         |         |         |         |         | Post-Test    |         |         |         |         |         |         |         |
|---------------------|--------------|---------|---------|---------|---------|---------|---------|---------|--------------|---------|---------|---------|---------|---------|---------|---------|
|                     | Experimental |         |         |         | Control |         |         |         | Experimental |         |         |         | Control |         |         |         |
|                     | Morning      |         | Night   |         | Morning |         | Night   |         | Morning      |         | Night   |         | Morning |         | Night   |         |
|                     | M            | SD      | M       | SD      | M       | SD      | M       | SD      | M            | SD      | M       | SD      | M       | SD      | M       | SD      |
| Interval R-R        | 1133.21      | 188.84  | 1128.98 | 151.47  | 1095.92 | 185.75  | 1098.94 | 120.37  | 1156.38      | 182.88  | 1160.48 | 177.01  | 1061.72 | 148.61  | 1072.42 | 133.96  |
| SDNN                | 123.48       | 64.87   | 147.31  | 49.99   | 129.65  | 56.88   | 160.70  | 45.68   | 128.27       | 55.05   | 152.98  | 46.83   | 111.92  | 51.47   | 152.46  | 43.09   |
| Heart rate          | 55.22        | 8.64    | 55.20   | 6.97    | 57.63   | 11.02   | 56.72   | 6.04    | 54.18        | 9.00    | 54.08   | 8.27    | 58.46   | 8.08    | 58.34   | 7.88    |
| RMSSD               | 80.93        | 34.41   | 88.14   | 41.22   | 92.84   | 42.61   | 91.46   | 37.75   | 100.38       | 42.44   | 104.13  | 45.77   | 84.18   | 43.53   | 83.30   | 32.86   |
| pnn50               | 42.49        | 19.57   | 44.52   | 21.48   | 48.14   | 20.04   | 44.98   | 16.69   | 49.79        | 23.05   | 50.19   | 20.62   | 41.07   | 18.66   | 41.62   | 15.72   |
| LF (FFT) ms2        | 2856.38      | 2712.27 | 3136.33 | 1839.50 | 3688.52 | 3036.68 | 3518.83 | 1883.52 | 2888.49      | 2014.11 | 3619.39 | 1996.06 | 3538.59 | 2993.11 | 3290.56 | 1818.72 |
| HF (FFT) ms2        | 2158.46      | 1511.91 | 2403.97 | 1751.62 | 2570.33 | 1558.90 | 2523.39 | 1491.46 | 2991.32      | 2234.31 | 3102.83 | 1932.98 | 2184.39 | 1816.48 | 2258.43 | 1448.96 |
| LF/HF (FFT)         | 1.65         | 1.28    | 1.68    | 0.87    | 1.84    | 2.02    | 1.62    | 0.81    | 1.44         | 1.25    | 1.37    | 0.72    | 1.96    | 1.40    | 1.69    | 0.80    |
| LF (AR) ms2         | 3161.06      | 2214.22 | 2955.02 | 1737.62 | 3860.79 | 3213.23 | 3241.82 | 1782.90 | 3186.44      | 2049.94 | 3433.10 | 1915.51 | 3261.93 | 2336.55 | 3042.67 | 1727.27 |
| HF (AR) ms2         | 2257.52      | 1786.46 | 2712.10 | 2361.83 | 3386.60 | 3508.39 | 3058.16 | 2791.85 | 3620.01      | 3101.24 | 3699.39 | 2959.98 | 2801.72 | 3136.46 | 2578.36 | 2409.52 |
| LF/HF (AR)          | 1.83         | 1.21    | 1.55    | 0.85    | 1.66    | 1.22    | 1.42    | 0.64    | 1.38         | 0.96    | 1.26    | 0.66    | 1.72    | 1.22    | 1.48    | 0.65    |
| Breathing frequency | 14.28        | 2.27    | 14.16   | 2.14    | 13.31   | 2.24    | 13.60   | 1.75    | 13.66        | 2.19    | 13.83   | 2.03    | 13.26   | 2.07    | 13.84   | 1.78    |

Note: SDNN = standard deviation of NN intervals, RMSSD = root mean square of successive RR interval differences, pNN50 = percentage of successive RR intervals that differ by more than 50 ms, LF = low-frequency, HF = high-frequency, FFT = fast Fourier transform, AR = autoregressive model.



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### 2.4. Procedure

Participants were recruited via flyers at the local University. They were asked to come to the lab for a presentation of the experiment (Day 1), and they were allocated randomly to either the experimental group (slow-paced breathing) or to the control group (social media use). They were told that the study was about investigating the effects of a smartphone-based relaxation method on heart rate during sleep. Participants in the experimental group were given an introduction to the slow-paced breathing technique and were performing it for 15 min together with the experimenter, ensuring that they understood correctly how to perform it at home. Participants in the control group were given an introduction for the same duration about the relaxing effects of social media. All participants also filled out the PSQI and the demographic questionnaire related to heart rate variability from Laborde et al. [3]. Participants had to come back to the lab at Day 2 between 16:00 and 20:00 h in order to get the ECG device and electrodes attached. This day had to be a weekday, to have participants following their usual daily activities and sleeping routines. They were told that the last meal should be taken at least 2 h before going to bed, and that afterwards only drinking water was allowed. They were also not allowed to drink alcohol or have strenuous physical activity on this day or the day before.

Participants were asked to have a similar day structure for both evaluation days (pre-test and post-test), in order to provide the best comparison possible for heart rate variability measurements. No intervention was performed in the evening preceding the pre-test and post-test night measurements. Participants were told to start the device before going to bed, write down the time when they went to bed, and then turn off the light. We are aware that lying in bed does not imply that participants were sleeping, and that sleep onset latency may differ across participants, but this variable has been assessed in previous studies [42] and represents a compromise when polysomnography assessment is not available. In the morning participants had to write down waking time, and stay in bed for 5 min after awakening for the morning awakening heart rate variability measurement. During the 30-day intervention participants had to confirm every night via an online form that they either did the slow-paced breathing exercise for the experimental group or that they used social media for the control group. In case participants forgot to do so, they were gently reminded by a research assistant on the next day to continue with the procedure. The maximum of misses was set to three (10%); more than three misses was then considered as a dropout. At the end of the intervention, participants were coming back to the lab to get the ECG device for the post-test and filled out again the PSQI. The post-test night and morning measurements followed the same procedure as for the pre-test, and participants were asked to follow the same daily routine, as well as having the same times to go to bed and to wake up. The next day the participants brought the device back to the lab, and they were debriefed about the aim of the experiment. For the control group, the debriefing included an introduction to the slow-paced breathing technique.

### 2.5. Data Analysis

Data analysis was realized with JASP (Version 0.9.2, JASP Team, 2018). Data was checked for outliers and normal distribution. Outliers ( $\pm 3.27$  SD) were winsorized (3.3%). Given the heart rate variability data was not normally distributed it was log transformed ( $\log_{10}$ ), as recommended by Laborde et al. [3]. Repeated-measures ANOVAs were conducted with time (pre-test vs. post-test) as a within-subject variable, and with condition (experimental vs. control) as between-subject variable. Dependent variables were time spent in bed, PSQI,  $CVA_{\text{night}}$ , breathing frequency<sub>night</sub>,  $CVA_{\text{morning}}$ , and breathing frequency<sub>morning</sub>. When interaction effects were found, we calculated four additional Student's *t*-tests to investigate the interaction effects, and therefore for the post-hoc tests we adjusted the alpha level via Bonferroni correction to  $0.05/4 = 0.0125$ . Finally, two Pearson correlations were run between PSQI change and  $CVA_{\text{night}}$  change and between PSQI change and  $CVA_{\text{morning}}$  change, with a significant threshold set to  $0.5/2 = 0.025$ . The change was obtained subtracting the pre-test value from the post-test value. Effect sizes are indicated via partial  $\eta^2$  and Cohen's *d*.

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### 3. Results

Regarding the time spent in bed, a repeated-measure ANOVA revealed no main effect of time,  $F(1,62) = 1.808$ ,  $p = 0.184$ , partial  $\eta^2 = 0.03$ , and no time  $\times$  condition interaction effect,  $F(1,62) = 0.651$ ,  $p = 0.423$ , partial  $\eta^2 = 0.01$ .

#### 3.1. PSQI

A repeated-measure ANOVA revealed no main effect of time,  $F(1,62) = 0.070$ ,  $p = 0.685$ , partial  $\eta^2 = 0.01$ . An interaction effect time  $\times$  condition was found,  $F(1,62) = 9.744$ ,  $p = 0.003$ , partial  $\eta^2 = 0.14$ . Concerning simple main effects for condition, there was no significant difference between the conditions at pre-test,  $t(62) = 0.449$ ,  $p = 0.655$ ,  $d = 0.11$ , but there was a tendency for a difference between the conditions at post-test (i.e., PSQI score lower for the experimental group in comparison to the control group),  $t(62) = 2.481$ ,  $p = 0.016$ ,  $d = 0.62$ . Concerning simple main effects for time, there was a significant difference between pre-test and post-test for the experimental group (i.e., PSQI score decrease, indicating higher subjective sleep quality),  $t(31) = 2.881$ ,  $p = 0.007$ ,  $d = 0.51$ , but no significant difference was found for the control group,  $t(31) = 1.717$ ,  $p = 0.096$ ,  $d = 0.30$ .

#### 3.2. $CVA_{night}$

A repeated-measure ANOVA revealed a tendency for a main effect of time,  $F(1,62) = 3.967$ ,  $p = 0.051$ , partial  $\eta^2 = 0.05$ . An interaction effect time  $\times$  condition was found,  $F(1,62) = 16.449$ ,  $p < 0.001$ , partial  $\eta^2 = 0.20$ . Concerning simple main effects for condition, there was no difference between the conditions at pre-test,  $t(62) = 0.793$ ,  $p = 0.411$ ,  $d = 0.20$  nor at post-test  $t(62) = 1.383$ ,  $p = 0.172$ ,  $d = 0.35$ . Concerning simple main effects for time, there was a significant difference between pre-test and post-test for the experimental group (i.e.,  $CVA_{night}$  increase),  $t(31) = 3.868$ ,  $p < 0.001$ ,  $d = 0.68$ , but no significant difference was found for the control group,  $t(31) = 1.655$ ,  $p = 0.108$ ,  $d = 0.29$ .

#### 3.3. Breathing Frequency<sub>night</sub>

A repeated-measure ANOVA revealed no main effect of time,  $F(1,62) = 0.065$ ,  $p = 0.800$ , partial  $\eta^2 = 0$ . A significant interaction effect time  $\times$  condition was found,  $F(1,62) = 4.279$ ,  $p = 0.043$ , partial  $\eta^2 = 0.06$ . Concerning simple main effects for condition, there was no difference between the conditions at pre-test,  $t(62) = 0.296$ ,  $p = 0.296$ ,  $d = 0.26$  nor at post-test  $t(62) = 0.059$ ,  $p = 0.953$ ,  $d = 0.02$ . Concerning simple main effects for time, there was no significant difference between pre-test and post-test for the experimental group,  $t(31) = 1.500$ ,  $p = 0.004$ ,  $d = 0.27$  or for the control group,  $t(31) = 1.433$ ,  $p = 0.162$ ,  $d = 0.25$ .

#### 3.4. $CVA_{morning}$

A repeated-measure ANOVA revealed no main effect of time,  $F(1,62) = 0.360$ ,  $p = 0.551$ , partial  $\eta^2 = 0.01$ . An interaction effect time  $\times$  condition was found,  $F(1,62) = 6.533$ ,  $p = 0.013$ , partial  $\eta^2 = 0.10$ . Concerning simple main effects for condition, there was no difference between the conditions at pre-test,  $t(62) = 1.001$ ,  $p = 0.321$ ,  $d = 0.25$  nor at post-test  $t(62) = 1.163$ ,  $p = 0.249$ ,  $d = 0.29$ . Concerning simple main effects for time, there was a tendency for a difference between pre-test and post-test for the experimental group (i.e.,  $CVA_{morning}$  increase),  $t(31) = 2.372$ ,  $p = 0.024$ ,  $d = 0.42$ , but no significant difference was found for the control group,  $t(31) = 1.310$ ,  $p = 0.200$ ,  $d = 0.23$ .

#### 3.5. Breathing Frequency<sub>morning</sub>

A repeated-measure ANOVA revealed no main effect of time,  $F(1,62) = 1.481$ ,  $p = 0.228$ , partial  $\eta^2 = 0.02$ , nor any interaction effect time  $\times$  condition  $F(1,62) = 1.247$ ,  $p = 0.268$ , partial  $\eta^2 = 0.02$ .

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### 3.6. Relationships between PSQI and CVA

Finally, a significant correlation was found between PSQI change and CVA<sub>night</sub> change ( $r = -0.29$ ,  $p = 0.018$ ), while a tendency was found regarding the relationship between PSQI change and CVA<sub>morning</sub> change ( $r = -0.24$ ,  $p = 0.052$ ). The negative correlation reflects the fact that a decrease in PSQI between pre-test and post-test is related to subjective sleep quality improvement, while an increase in CVA between pre-test and post-test reflects an improvement in CVA functioning.

## 4. Discussion

This study was aimed to investigate the influence of a 30-day slow-paced breathing intervention (experimental group) in comparison to social media use (control group) on subjective sleep quality via the PSQI and on night and morning CVA, operationalized via high-frequency heart rate variability. Consistent with our hypotheses, subjective sleep quality and CVA<sub>night</sub> was increased in the experimental group but not in the control group, while there was only a tendency for CVA<sub>morning</sub> to display a pattern similar to CVA<sub>night</sub>.

Regarding subjective sleep quality, confirming our hypothesis, scores on the PSQI significantly decreased for the experimental group between pre-test and post-test, which reflects a better subjective sleep quality in the group performing slow-paced breathing. This is in line with the expected relaxing effects of slow-paced breathing [10,12,20] and also with findings demonstrating that slow-paced breathing can decrease subjective feelings of anxiety [31]. Finally, this result is in line with the effects of slow-paced breathing observed on objective sleep parameters in insomnia patients [42].

Regarding CVA<sub>night</sub>, confirming our hypothesis, there was a significant increase for the experimental group between pre-test and post-test, while no changes were found in breathing frequency, which means that CVA changes were not driven by changes in breathing frequency. This result is in line with the resonance model [12] arguing that slow-paced breathing increases vagal afferences, and this is also in line with previous research showing that slow-paced breathing increases vagal afferences, and specifically CVA [16,31]. Following Werner and colleagues [37], we do not argue that CVA<sub>night</sub> represents an indicator of sleep quality; however, we suggest that CVA<sub>night</sub> may still reflect some form of cardiovascular self-regulation and recovery processes based on the restorative function of the parasympathetic nervous system [15], given during sleep the organism is much less under the influence of external factors. This view is also supported by previous research showing a lower CVA<sub>night</sub> in individuals with sleep disorders [32–34], and also by research showing an association between higher CVA<sub>night</sub> and higher subjective sleep quality [34–36]. Finally, this is also complemented by our findings concerning the significant relationship observed between PSQI change and CVA<sub>night</sub> change (and the tendency observed with CVA<sub>morning</sub> change), reflecting an association between improvement in CVA functioning and improvement in subjective sleep quality.

Regarding CVA<sub>morning</sub>, our hypothesis is partially validated, given it displayed only marginally a similar pattern to CVA<sub>night</sub>, meaning there was a tendency ( $p = 0.024$ ) in the experimental group to display an increase in CVA<sub>morning</sub> at post-test in comparison to pre-test, the effect size being lower for CVA<sub>morning</sub> ( $d = 0.42$ ) in comparison to CVA<sub>night</sub> ( $d = 0.68$ ). While the arguments already mentioned for CVA<sub>night</sub> may also apply here, other processes may also be involved. More specifically, it has been suggested that the role of CVA during wake periods is more relevant than during sleep given a higher solicitation of self-regulation processes when the organism is awake [37].

The main strength of our study is the long-term (30 days) slow-paced breathing intervention, given most of the studies take only into account the short-term effects of slow-paced breathing on heart rate variability (e.g., [16,31,42]). Nonetheless, our study had some limitations. The main one is that the investigation of slow-paced breathing on sleep quality would require the use of the gold standard, polysomnography [46], as was done by Werner and colleagues [37]. However, it should still be mentioned that there is no established definition for objective sleep quality, and that sleep quality can refer to different variables measured with polysomnography [47]. Further, investigating separately the sleep stages seem also required, given the differential activation of CVA in REM and non-REM

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sleep [38]. Particularly, investigating CVA during slow wave sleep appears promising [39,48–50], given slow wave sleep is (mostly) free of any external confounding events, and is characterized by fewer body movements or arousals that provoke disruptions in the ECG signal, therefore ensuring higher stationarity of the ECG signal. Although some algorithms are being developed to identify sleep stages directly via heart rate variability measurements [49,50], preliminary research testing the influence of slow-paced breathing on sleep quality should definitely consider the use of polysomnography [37,46]. Another limitation is that we did not control for smartphone use before sleeping prior to the experiment. Moreover, we had no control group with paced breathing instructions, like in the study by Tsai et al. [42] where the control condition involved breathing at 12 cpm (however, the inhalation and exhalation phases were not specified). Further related to the control group, social media use has been found to decrease sleep quality in adolescents and young adults [51–53]. This was not found in our study—in our sample both PSQI scores and CVA values did not change between pre-test and post-test for the control group. This may potentially be because our participants already had habitual use of social media prior to sleeping before being recruited for the experiment. Further research may investigate alternative active control groups, as mentioned above with the 12 cpm breathing condition [42]. Finally, the present study tested young healthy individuals, and the findings cannot be generalized to other populations.

### 5. Conclusions

In summary, this study was aimed to investigate the effects of a smartphone-based slow-paced breathing intervention (6 cpm) performed for a duration of 15 min before sleeping across 30 days, compared to a control condition with participants using social media on their smartphone. Results showed that in the experimental group subjective sleep quality was improved and CVA<sub>night</sub> was increased, while a marginal increase was also found in CVA<sub>morning</sub>. Taken together, our results suggest that slow-paced breathing performed before sleeping may enhance restorative processes at the cardiovascular level during sleep. Future research should investigate the effects of slow-paced breathing on sleep via polysomnography.

**Supplementary Materials:** The following are available online at <http://www.mdpi.com/2077-0383/8/2/193/s1>, Table S1: Full dataset.

**Author Contributions:** Conceptualization, S.L., T.H., E.M., and F.D.; methodology, S.L., T.H., E.M., and F.D.; software, S.L.; validation, S.L.; formal analysis, S.L.; investigation, S.L. and T.H.; resources, S.L.; data curation, S.L.; writing—original draft preparation, S.L.; writing—review and editing, T.H., E.M., and F.D.; visualization, S.L.; supervision, S.L.; project administration, S.L.; funding acquisition, S.L.

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## *Study 3*

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### 9 Study 3 (submitted) – The influence of slow-paced breathing on executive function

#### 9.1 Introduction

Improving executive functions is a constant endeavor for humans across the lifespan (Diamond & Ling, 2016), from children (Takacs & Kassai, 2019) to older adults (Nguyen, Murphy, & Andrews, 2019). Of interest are both the short-term immediate effects, as well as long-term effects. Given there is so far a lack of studies investigating the influence of SPB on executive functioning (for exceptions, see De Couck et al., 2019; Prinsloo et al., 2011), the current study aimed to test the immediate effects of SPB on three important executive functions: inhibition, working memory, and cognitive flexibility.

Based on the predictions of the neurovisceral integration model (Thayer et al., 2009) and the resonance model (Lehrer & Gevirtz, 2014), we assumed that SPB will increase executive performance via an increase in CVA. A within-subject design was selected given the large intra-individual variability in HRV (Laborde, Mosley, et al., 2017; Quintana & Heathers, 2014). Regarding the effects of the experimental manipulation, we hypothesized that after the experimental manipulation, in comparison to the control condition (where participants will be breathing spontaneously and watching an emotionally-neutral TV documentary), at the physiological level (H<sub>1</sub>) in the SPB condition participants will display a higher CVA (operationalized via RMSSD), a lower heart rate, and a lower respiratory frequency. We further hypothesized that executive performance will be higher in the slow paced breathing condition in comparison to the control condition (H<sub>2</sub>), and that this difference will be mediated via CVA (H<sub>3</sub>).



## 9.2 Method

### 9.2.1 Participants

To determine our sample size, we explored effect sizes presented in previous research of SPB and executive functioning (De Couck et al., 2019; Prinsloo et al., 2011). We computed an a priori power analysis using the software *G\*Power 3.0* (Faul, Erdfelder, Buchner, & Lang, 2009). A medium effect size ( $f = .25$ ), for a repeated-measures MANOVA (within-factors effects), with statistical power set at .80 and an  $\alpha$  level of .05, requires a total sample size of  $N = 66$ . We recruited a larger sample of 90 participants to allow for potential dropout or technical issues with data collection. Exclusion criteria were self-reported cardiovascular conditions, and other chronic conditions that could influence breathing or heart rate patterns, such as asthma, diabetes, and neurological conditions (Laborde, Mosley, et al., 2017). Because of technical issues (excessive noise or artefacts on the ECG signal) 12 participants were excluded and the final sample was comprised of  $N = 78$  (41 men, 37 women;  $M_{\text{age}} = 23.22$  years; age range = 18-30 years; body mass index =  $22.40 \pm 2.23$ ; waist-to-hips ratio =  $0.80 \pm .05$ ). This sample size elevated statistical power to .86 with all other parameters held constant. All participants gave written informed consent before participation, and were informed that they could withdraw from the study at any time without explanation. The experiment was conducted in line with the Declaration of Helsinki, and the protocol was approved by a human research ethics committee at the German Sport University Cologne (Project identification code 42/2015).

### 9.2.2 Material and Measures

#### 9.2.2.1 *Cardiac vagal activity*

CVA was assessed in this study as described in Section 6.1 (p.49).

#### 9.2.2.2 *Slow-paced breathing technique*

SPB was realized in this study as described in Section 6.2 (p.50).

### 9.2.2.3 TV neutral documentary (control condition)

To serve as a control condition, a TV documentary (“*Abenteuer Forschung*” [Research Adventures]) about research discoveries related to space and the universe was shown to participants for the same duration as the SPB familiarization exercise. This TV documentary was found to be subjectively emotionally neutral in a pilot study prior to the experiment.

### 9.2.3 Executive Function Tasks

#### 9.2.3.1 Inhibition task

As a measure of inhibition, we used the computerized version of the colour word match Stroop task (Stroop, 1935) with verbal responding available in the Inquisit library<sup>3</sup>, and ran it with the Inquisit software (version 5; Millisecond Software, 2017). Words appeared in 28-pt Arial font in the middle of a white screen. Three stimuli were used: colored square (congruent control stimuli), colored words displayed with the color corresponding to the word (congruent stimuli, for example the word “blue” is displayed in blue color). Participants were asked to name the color in which the word was written as fast and as accurately as possible, while ignoring the written meaning of the word. A headset (Sennheiser PC 8, Wedemark, Germany) was placed on their head for stability, with a microphone directly in front of their mouth to record the answers. The familiarization included 20 trials whereas for the main assessment participants completed 84 trials: 4 colors (red, green, blue, black) x 3 color stimulus congruency (congruent, incongruent, control squares) x 7 repetitions. The stimuli remained on the screen until response and latencies were measured from onset of stimuli. The inter-trial interval was 200 ms, and the error feedback (a red cross) was 400 ms.

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<sup>3</sup> <https://www.millisecond.com/download/library/stroop/>

### 9.2.3.2 Working memory

As a measure of working memory capacity, we used the AOSPAN (Unsworth et al., 2005) which is based on the original operation span task (Turner & Engle, 1989). We used the version of the task programmed within the Inquisit database<sup>4</sup>. In this task participants are required to solve mathematics problems while trying to remember an unrelated set of letters. The task included a total of 15 trials (three trials each with 3, 4, 5, 6, and 7 letters). An example of a three item trial is  $(8 / 2) - 1 = 1?$  (correct/incorrect?) → F; is  $(6 + 1) + 2 = 8?$  (correct/incorrect?) → P; is  $(10 + 2) - 5 = 15?$  (correct/incorrect?) → Q. After completing the three questions in this example, participants were asked to select the presented letters with a mouse click from an array of 12 potential letters in the order that they were presented (in this case F, then P, then Q). A familiarization to the task is included in the Inquisit version. The primary measure of working memory capacity is the automated operation span score (Unsworth et al., 2005), calculated as the total number of letters recalled across all error-free trials. Full task details can be found in Unsworth et al. (2005).

### 9.2.3.3 Cognitive flexibility

As a measure of cognitive flexibility, we used a shorter and modified version of the Wisconsin card sorting test, the modified card sort test (Nelson, 1976). The computerized version of the Inquisit database<sup>5</sup> was used. This test consists of two decks of 24 cards (so a total of 48) and four stimulus cards. Each card includes different colors and numbers of signs. The signs are: plus sign, star, triangle, or circle. There are one, two, three, or four signs on each card. Signs can be red, green, blue, or yellow. The participant is asked to match each new card appearing on the screen with a stimulus card. Correctly matched cards are arranged in three

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<sup>4</sup> <https://www.millisecond.com/download/library/ospan/>

<sup>5</sup> <https://www.millisecond.com/download/library/cardsort/>

categories according to color, sign, and number. After the participant performs four consecutive correct matches in one category (for example, the category “color”), the computer switches without warning the rule to another category. After each choice, the participant is provided feedback about whether the response was correct or incorrect, but is not provided information regarding the correct matching category. The scoring procedures of the modified card sorting test are the same as the original Wisconsin card sorting test (Caffarra, Vezzadini, Dieci, Zonato, & Venneri, 2004; Nelson, 1976). Participants were instructed to respond as fast and accurately as possible. Perseverative errors, which reflect the number of trials with decision errors when the matching rule has changed, was selected as the main dependent variable as it is considered the closest approximation of cognitive flexibility (Miyake et al., 2000).

#### 9.2.4 Procedure

Participants were recruited via flyers on a campus of a single university and via social networks groups linked to the university. After contacting the experimenter team who first screened for potential exclusion criteria, they received an email containing the details about the experiment. Each participant was required to take part to two testing sessions (lasting around 100 minutes each; see Figure 1). The order of the experimental conditions was counterbalanced across participants. The two sessions were separated by one week, to keep learning effects to a minimum, and took place at the same time of day, given chronotype can influence HRV (van Eekelen, Houtveen, & Kerkhof, 2004) and cognitive performance (Folkard, 1990). Prior to the testing sessions, participants were instructed not to drink or eat anything but water during the 2 h before the experiment, nor to do any strenuous exercise or drink alcohol in the 24 h before the experiment (Laborde, Mosley, et al., 2017).

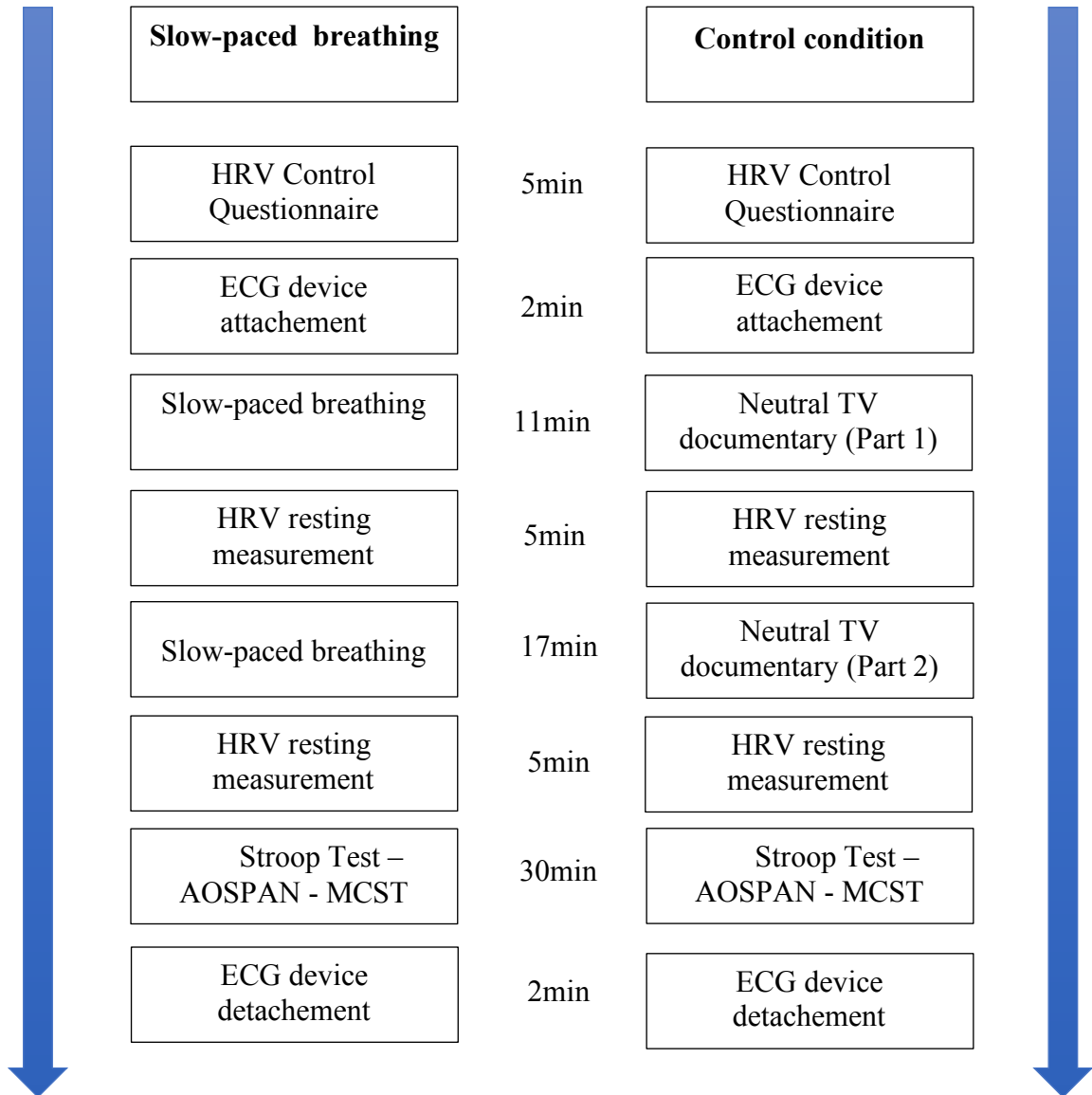
At the beginning of the experiment, participants were asked to complete an informed consent form, and at the beginning of each testing session they completed a questionnaire regarding variables potentially influencing HRV (Laborde, Mosley, et al., 2017), in order to

control whether the information sent via email has been abided. The participants were then asked to turn off their smartphone. The full experiment was protocolled by the experimenter. The course of events in both conditions was identical, except for the familiarization to SPB and the SPB exercise in the SPB condition, which were paralleled in the control condition with watching the neutral TV documentary. At the beginning of the SPB condition, participants received a short introduction video on how to perform the technique correctly, and the correct execution was checked by the experimenter. All participants managed to perform correctly the SPB technique.

The experiment then continued either with the SPB exercise, or with watching the TV documentary. A HRV resting measure was taken before and after this step. The HRV resting measure lasted 5 minutes, based on the Task Force recommendations (Malik, 1996). The participants then performed the three executive cognitive tests: the color word Stroop test, the automated operation span, and the modified card sorting test. The order of the tests was randomized across participants. However, for each participant the order was kept the same across experimental sessions. We used a 15 inch flat-screen monitor (Dell GmbH, Frankfurt, Germany; 1,280 x 960 pixels at 60 Hz) at a viewing distance of 60 cm for all tests. The HRV resting measure was realized in a sitting position with eyes closed, knees at 90°, hands on the thighs. The same body position was kept for all cognitive tests, and the participants were asked to move as little as possible during the experiment. At the end of the second testing session, participants were debriefed and thanked. The full experimental procedure is depicted in Figure 1.

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Figure 18: Study 3 - Illustration of the experimental protocol



Notes. HRV: heart rate variability; AOSPAN: Automated Operation Span; MCST: Modified Card Sorting Test; ECG: electrocardiography

### 9.2.5 Data Analysis

For the HRV data, RMSSD was extracted from the Kubios output. Respiratory frequency (the number of respiratory cycles per minute) was obtained multiplying the EDR (ECG derived respiration) value obtained via the Kubios algorithm by 60 (Tarvainen et al., 2014). All participants were breathing at 6 cpm during the SPB technique. For inhibition, accuracy was operationalized via the number of incorrect answers retrieved for the squares, congruent stimuli, and incongruent stimuli. Regarding response times, we analyzed only the correct answers. Reaction time data were subjected to two filters. In the first filter, trials with response times lower than 200ms and higher than 3000 ms were excluded in order to control for extreme results (see Putman & Berling, 2011). The second filter then screened for reaction times higher or lower than two standard deviations from the mean. These were also removed (Dresler, Meriau, Heekeren, & Van Der Meer, 2009). Working memory capacity was operationalized as the total number of letters recalled across all error-free trials. Cognitive flexibility was operationalized as the total number of perseverative errors, corresponding to a card matching error when the previous rule had changed (Nelson, 1976).

Data were checked for outliers and normality. A total of 0.90% univariate outliers cases were found and winsorized ( $\pm 2.58$ , Tabachnick & Fidell, 2012). Multivariate outliers were checked using Mahalanobis distance, with none identified. The behavioral data related to the executive functions dependent variables were normally distributed. The physiological data (RMSSD, heart rate, respiratory frequency) were not normally distributed, thus a log-transformation (log 10) was applied to achieve normal distribution, and this is consistent with previous HRV research (Laborde, Mosley, et al., 2017). For the physiological data, we ran analyses with the log-transformed values. However, for descriptive values we report the raw data for an easier interpretation.

As a manipulation check, we analyzed the difference between the resting HRV measurement before the experimental manipulation (SPB vs. TV documentary) and after the experimental manipulation, for log<sub>10</sub> heart rate, log<sub>10</sub> RMSSD, and log<sub>10</sub> respiratory frequency. For the experimental manipulation check, a repeated-measures MANOVA was conducted, with time as independent variable (pre vs. post) and with three dependent variables: log<sub>10</sub> heart rate, log<sub>10</sub> RMSSD, log<sub>10</sub> respiratory frequency. Based on our hypotheses, we focus on the condition x time interaction. For log<sub>10</sub> RMSSD, the analysis is first run without covariates and then with covariates included (age, sex, body mass, waist-to-hip ratio), in order to see whether individual difference factors affect results.

Regarding our main hypotheses, we conducted a 2 (time: pre vs. post) x 2 (group: SPB vs. control) repeated-measures MANOVA, with four dependent variables: 1) accuracy (error rate to incongruent stimuli) and reaction times (incongruent stimuli-congruent stimuli) for 2) the Stroop interference (operationalization of inhibition), 3) the automated operation span (operationalization of working memory), and 4) the number of perseverative errors (operationalization of cognitive flexibility). Regarding Stroop interference accuracy, account errors made with congruent stimuli were not considered in the calculation given there were none. Significant interactions were followed-up using independent samples *t*-tests with Bonferonni corrected significance levels. Where a significant effect for SPB was found on an executive function variable, potential mediation via log<sub>10</sub> RMSSD was tested using the PROCESS statistical software (Hayes, 2013). This custom dialog tests the total, direct, and indirect effect of an independent variable on a dependent variable through a proposed mediator and allows inferences regarding indirect effects using percentile bootstrap intervals.



### 9.3 Results

#### 9.3.1 Slow-paced Breathing and Physiological Measures

Descriptive statistics for the physiological variables concerning the manipulation check can be seen in Table 1.

*Table 1: Study 3 - Descriptive statistics for physiological variables*

|                                |                       | Resting measurement before slow-paced breathing exercise/TV documentary |       | Resting measurement after slow-paced breathing exercise/TV documentary |       |
|--------------------------------|-----------------------|---|-------|--|-------|
|                                |                       | M   | SD    | M  | SD    |
| Slow-paced breathing condition | RMSSD                 | 47.97   | 28.66 | 53.43  | 35.46 |
|                                | Heart Rate            | 71.00   | 10.93 | 69.57  | 11.02 |
|                                | Respiratory Frequency | 10.48   | 1.97  | 10.89  | 1.80  |
| Control Condition              | RMSSD                 | 52.51   | 34.14 | 52.49  | 33.85 |
|                                | Heart Rate            | 70.93   | 11.63 | 71.29  | 11.46 |
|                                | Respiratory Frequency | 13.73   | 2.58  | 13.68  | 2.32  |

*Note.* RMSSD: Root Mean Square of the Successive Differences

A repeated-measures MANOVA was conducted and showed an overall significant interaction effect for condition x time, Wilks'  $\lambda = .515$ ,  $F(3, 75) = 23.6$ ,  $p < .001$ , partial  $\eta^2 = .49$ . Univariate ANOVAs with Greenhouse-Geisser corrections were then ran for each physiological variable. A significant condition x time interaction effect was found for log10 heart rate,  $F(1, 77) = 14.9$ ,  $p < .001$ , partial  $\eta^2 = .16$ . Four follow-up post-hoc  $t$ -tests were conducted with Bonferroni correction ( $\alpha = .0125$ ). A significant difference was found in the SPB condition, with log10 heart rate at Time 2 ( $M = 1.84$ ,  $SD = 0.70$ ) being significantly lower than at Time 1 ( $M = 1.85$ ,  $SD = 0.67$ ),  $t(77) = 4.24$ ,  $p < .001$ ,  $d = 0.48$ . No other significant differences were found for the post-hoc tests with heart rate. No significant condition x time interaction effect was found for log10 RMSSD,  $F(1, 77) = 3.74$ ,  $p = .057$ , partial  $\eta^2 = .05$ .

Integrating the covariates age, sex, body mass, and waist-to-hip ratio did not change these results,  $F(1, 73) = 1.97, p = .165, \text{partial } \eta^2 = .03$ .

A significant condition x time interaction effect for log10 respiratory frequency was found  $F(1, 77) = 38.53, p < .001, \text{partial } \eta^2 = .33$ . Four follow-up post-hoc  $t$ -tests (with Bonferroni correction  $\alpha$  at .0125) showed a significant difference between both conditions at Time 1, with log10 respiratory frequency in the SPB condition ( $M = 1.01, SD = 0.71$ ) being lower than in the control condition ( $M = 1.13, SD = 0.71$ ),  $t(77) = 12.47, p < .001, d = 1.41$ . A significant difference was found between both conditions at Time 2, with log10 respiratory frequency in the SPB condition ( $M = 1.08, SD = 0.74$ ) being significantly lower than the control condition ( $M = 1.13, SD = 0.73$ ),  $t(77) = 5.39, p < .001, d = 0.61$ . A significant difference was found in the SPB condition between Time 1 and Time 2, with log10 respiratory frequency at Time 1 ( $M = 1.01, SD = 0.71$ ) being significantly lower than the log10 respiratory frequency at Time 2 ( $M = 1.08, SD = 0.74$ ),  $t(77) = 7.18, p < .001, d = 0.81$ .

### 9.3.2 Slow-paced Breathing and Behavioral Measures

Descriptive statistics for behavioral measures are reported in Table 2, and correlation matrices between all study variables are reported in the Table 3 and Table 4.

For the MANOVA, a significant overall effect for condition was found, Wilks'  $\lambda = .70, F(4, 74) = 4.69, p = .002, \text{partial } \eta^2 = .20$ . Univariate ANOVAs with Greenhouse-Geisser corrections showed a significant effect for Stroop accuracy,  $F(1, 77) = 9.21, p = .003, \text{partial } \eta^2 = .11$ . No significant effect was found for Stroop response time,  $F(1, 77) = 0.81, p = .372, \text{partial } \eta^2 = .01$ . A significant effect for working memory capacity was also found,  $F(1, 77) = 5.66, p = .020, \text{partial } \eta^2 = .07$ . A significant effect for cognitive flexibility was also found,  $F(1, 77) = 5.32, p = .024, \text{partial } \eta^2 = .07$ .

Table 2: Study 3 - Descriptive statistics for executive functions

|   | Slow-paced breathing condition |       | Control condition |       |
|---|--------------------------------|-------|-------------------|-------|
|   | M                              | SD    | M                 | SD    |
| Stroop interference reaction time               | 143.85                         | 68.02 | 138.04            | 72.84 |
| Stroop interference accuracy (number of errors) | 0.32                           | 0.47  | 0.58              | 0.73  |
| Automated operation span score                  | 42.63                          | 16.02 | 38.87             | 16.72 |
| Perseverative errors (MCST)                     | 0.40                           | 0.65  | 0.68              | 0.95  |

Note. MCST: Modified Card Sorting Test

Table 3: Study 3 - Correlation between main study variables (Control condition)

|  | 1      | 2      | 3    | 4    | 5 |
|--|--------|--------|------|------|---|
| 1. Stroop Interference Reaction Time                             |        |        |      |      |   |
| 2. Stroop Interference Accuracy (number of errors)               | .21    |        |      |      |   |
| 3. Automated operation span score                                | -.32** | -.53** |      |      |   |
| 4. Perseverative errors (MCST)                                   | .07    | .25*   | -.15 |      |   |
| 5. RMSSD (resting measurement before performing executive tasks) | -.11   | -.30** | .27* | -.20 |   |

Note: MCST: Modified Card Sorting Test; RMSSD: Root Mean Square of the Successive Differences

Table 4: Study 3 - Correlation between main study variables (Slow-paced breathing condition)

|  | 1    | 2     | 3      | 4    | 5 |
|--|------|-------|--------|------|---|
| 1. Stroop Interference Reaction Time                             |      |       |        |      |   |
| 2. Stroop Interference Accuracy (number of errors)               | .10  |       |        |      |   |
| 3. Automated operation span score                                | -.16 | -.28* |        |      |   |
| 4. Perseverative errors (MCST)                                   | .12  | .21   | -.34** |      |   |
| 5. RMSSD (resting measurement before performing executive tasks) | -.11 | -.26* | .42**  | -.22 |   |

Note: MCST: Modified Card Sorting Test; RMSSD: Root Mean Square of the Successive Differences

### 9.3.3 Mediation Analysis

To test whether the effect of SPB on executive functions was mediated by RMSSD, the experimental condition, coded as SPB (1) or control (2), was entered as the independent variable, the variables operationalizing executive functions (Stroop accuracy, automated operation span score, perseverative errors) were entered successively as dependent variables,

and RMSSD was entered as mediator variable. Using a 10,000 resampling rate, the results from the bootstrapped mediation analyses revealed no significant indirect effect for Stroop accuracy (95% CI:  $-.07, .05$ ), no significant indirect effect for automated operation span score (95% CI:  $-1.60, 2.09$ ) and no significant indirect effect for perseverative errors (95% CI:  $-.07, .05$ ).

#### 9.4 Discussion

This experiment tested the effects of SPB on immediate executive functioning (inhibition, working memory, cognitive flexibility). Based on the resonance model (Lehrer & Gevirtz, 2014), we hypothesized that CVA operationalized via RMSSD would be higher, and heart rate and respiratory frequency would be lower, after the experimental manipulation with SPB compared to control ( $H_1$ ). This hypothesis was partially supported. No difference between conditions was observed for RMSSD, while respiratory frequency and heart rate were significantly lower in the experimental group compared to control. Based on the neurovisceral integration model (Smith et al., 2017; Thayer et al., 2009), we also hypothesized that cognitive functioning would be better in the experimental group compared to control ( $H_2$ ) and that RMSSD would mediate this difference in executive functioning ( $H_3$ ). These hypotheses were also partially supported. Participants did show better executive functioning after SPB compared to control (for Stroop interference accuracy, working memory capacity, cognitive flexibility, but not Stroop interference response time), but this change in executive functioning was not mediated by RMSSD.

Regarding the physiological effects of the SPB exercise, compared to the control condition, heart rate and respiratory frequency were lower, supporting our predictions, but contrary to what we hypothesized no significant difference was found for RMSSD. Based on the resonance frequency model (Lehrer & Gevirtz, 2014), SPB at 6 cpm is thought to increase vagal afferences and the longer exhalation phase in comparison to the inhalation phase is supposed to activate the parasympathetic nervous system (Strauss-Blasche et al., 2000). This is based on the

coupling of heart beat and respiration with the RSA, where inhalation is linked to faster heart rate, and exhalation to slower heart rate (Angelone & Coulter, 1964; Yasuma & Hayano, 2004). The null finding for RMSSD might reflect the fact that an optimal SPB frequency, referred to as the resonance frequency (Vaschillo et al., 2006), is specific to each individual. As mentioned above, the value of 6 cpm was chosen to match RSA to the inherent oscillations in heart rate linked to blood pressure modulation at 0.1 Hz in order for the two signals to summate and produce larger variations in heart rate (Lehrer & Gevirtz, 2014). However it is possible that depending on physical characteristics such as height (Vaschillo et al., 2006) the resonance frequency differed across individuals, and that an individualized breathing frequency determined with an ad-hoc protocol (Lehrer et al., 2000) might have produced larger increases in CVA (Steffen, Austin, DeBarros, & Brown, 2017).

Two different patterns were observed for heart rate and respiratory frequency. Heart rate was found to be lower after the SPB exercise than after watching the TV documentary, which would to some extent reflect the relaxation effect associated with SPB (Lehrer & Gevirtz, 2014). A different pattern was observed for respiratory frequency given that it was found to be lower in the SPB condition in comparison to the control condition *before* performing the SPB technique. This is most likely due to the fact that in the SPB condition, participants completed a familiarization exercise that decreased their breathing frequency (see Figure 1) prior to the first resting measurement. This reduced breathing pattern remained to some extent during the first resting measurement. The respiratory frequency then increased in the SPB condition (when comparing the measurements before and after performing the SPB exercise), that might be due to the participants becoming habituated to the reduced respiratory frequency while performing the technique, and returning faster to their normal respiratory frequency. However the fact that respiratory frequency was still lower in the SPB condition than in the control condition (after the SPB exercise and before starting the executive functioning tasks) supports the predicted effects of the SPB technique at the respiratory level (Lehrer & Gevirtz, 2014).

Regarding our second hypothesis, executive functioning improved after the SPB exercise in comparison to the control condition, for 1) inhibition with less errors for the incongruent stimuli (i.e., better Stroop interference accuracy), for 2) working memory capacity with a higher automated operation span score, and for 3) cognitive flexibility with less perseverative errors. No change was found on Stroop interference reaction time. Nevertheless, regarding hypothesis 3, the results are not in line with predictions of the neurovisceral integration model (Smith et al., 2017; Thayer et al., 2009), given the improvement in executive functioning was not mediated by RMSSD. This could suggest that other mechanisms might be contributing to the effects of SPB on executive functioning. This might include improved functional connectivity in brain networks (Mather and Thayer (2018). To explain, the slow oscillations in heart rate produced by SPB are suggested to have the potential to strengthen brain network dynamics, especially in medial prefrontal regulatory regions that are particularly sensitive to physiological oscillations (Mather & Thayer, 2018), and which are responsible for cognitive control and emotion regulation (Thayer et al., 2012). In order to uncover the mechanisms at work, that might be involved in oxygenation, blood flow or electrophysiological signals in brain areas associated with executive functions, future research should investigate the effects of SPB using brain imaging techniques such as EEG, fNIRS, or fMRI.

Our experiment has some notable limitations that need to be considered when interpreting main findings. First, our control condition involved spontaneous breathing while the participants were watching a neutral TV documentary. Therefore, we cannot rule out the possibility that participants in the control group were actively focusing on their breathing, and future studies should include a control condition that includes a similar focus on breathing at a different pace, perhaps 12 cycles per minute (Tsai et al., 2015) that would match typical lower range spontaneous respiratory frequencies (L. Sherwood, 2006). Second, respiratory frequency was obtained via a dedicated algorithm from Kubios (Tarvainen et al., 2014). However, a more precise assessment of respiratory frequency such as a respiratory belt or a pneumotachograph

(Egizio, Eddy, Robinson, & Jennings, 2011; Quintana & Heathers, 2014) and the assessment of other respiratory related variables (e.g., respiratory depth, gas exchanges) could prove helpful in explaining the effects of SPB on executive functioning (Lorig, 2011; Ritz et al., 2002). Third, some meaningful variations could be introduced to the SPB technique practiced in this study. For example, having a break introduced between inhalation and exhalation, or between exhalation and inhalation, could have different physiological consequences (Reyes del Paso, Munoz Ladron de Guevara, & Montoro, 2015; Skow, Day, Fuller, Bruce, & Steinback, 2015). In our case, future studies should investigate the use of a post-exhalation break, given that it appears to increase CVA more than no break (Russell et al., 2017).

## 9.5 Conclusion

In conclusion, this experiment tested the effects of SPB on immediate executive functioning in a sample of young adults. The SPB exercise was able to decrease heart rate and respiratory frequency, while no change was observed for RMSSD. An increase in executive functioning was observed for inhibition, working memory, and cognitive flexibility, after the SPB exercise compared to control. However, this increase in executive performance was not mediated by RMSSD. Therefore, the influence of SPB on executive functioning cannot be explained by an increase in CVA as predicted by the neurovisceral integration model (Smith et al., 2017; Thayer et al., 2009). Further research might want to test (using brain imaging) whether brain network dynamics are involved in the association between SPB and executive functioning.

## 10 Study 4 (published) - Influence of slow-paced breathing on inhibition after physical exertion



ORIGINAL RESEARCH  
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## Influence of Slow-Paced Breathing on Inhibition After Physical Exertion

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This research aims to investigate whether slow-paced breathing (SPB) improves adaptation to psychological stress, and specifically inhibition, when it is performed before or after physical exertion (PE). According to the resonance model, SPB is expected to increase cardiac vagal activity (CVA). Further, according to the neurovisceral integration model, CVA is positively linked to executive cognitive performance, and would thus play a role in the adaptation to psychological stress. We hypothesized that SPB, in comparison to a control condition, will induce a better adaptation to psychological stress, measured via better inhibitory performance. Two within-subject experiments were conducted with athletes: in the first experiment ( $N = 60$ ) SPB (or control – neutral TV documentary) was realized before PE (“relax before PE”), and in the second experiment ( $N = 60$ ) SPB (or the watching TV control) was realized after PE (“relax after PE”). PE consisted of 5 min Burpees, a physical exercise involving the whole body. In both experiments the adaptation to psychological stress was investigated with a Stroop task, a measure of inhibition, which followed PE. Perceived stress increased during PE (partial  $\eta^2 = 0.63$ ) and during the Stroop task (partial  $\eta^2 = 0.08$ ), and decreased during relaxation (partial  $\eta^2 = 0.15$ ), however, no effect of condition was found. At the physiological level PE significantly increased HR, RF, and decreased CVA [operationalized in this research via the root mean square of successive differences (RMSSD)] in both experiments. Further, the number of errors in the incongruent category (Stroop interference accuracy) was found to be lower in the SPB condition in comparison to the control condition, however, these results were not mediated by RMSSD. Additionally, the Stroop interference [reaction times (RTs)] was found to be lower overall in “relax before PE,” however, no effect was found regarding SPB and Stroop interference (RTs). Overall, our results suggest that SPB realized before or after PE has a positive effect regarding adaptation to psychological stress and specifically inhibition, however, the underlying mechanisms require further investigation.

**Keywords:** acute exercise, physical activity, executive function, cognition, heart rate variability

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## INTRODUCTION

Executive functions underpin goal-directed behavior and are essential for self-control (Miyake et al., 2000; Diamond, 2013; Kotabe and Hofmann, 2015). Executive functions may be hindered by factors such as stress (e.g., Arnsten, 2009), fatigue (Kurzban et al., 2013; Inzlicht et al., 2014; Schmit and Brisswalter, 2018), and pressure (e.g., Laborde et al., 2014). The aim of this paper is to investigate the influence of a relaxation method [slow-paced breathing (SPB)], to prevent inhibition failure during psychological stress following physical exertion (PE).

Several neurological mechanisms have been identified in the literature to explain the influence of perceived stress (PS; e.g., Archer et al., 2018), mental fatigue (e.g., Guo et al., 2018) or physical fatigue (e.g., Tanaka et al., 2013) on inhibition failure. In this research, we focus on the role of the autonomic nervous system, and more specifically of its parasympathetic branch. The neurovisceral integration model (Thayer et al., 2009; Smith et al., 2017) assumes that similar structures are involved in the regulation of executive performance and cardiac functioning. The functional organization of these structures is depicted by the central autonomic network (Benarroch, 1993), the output of this network being cardiac vagal activity (CVA), the activity of the vagus nerve which regulates cardiac functioning (Thayer et al., 2009; Laborde et al., 2017b). Based on this functional organization, the neurovisceral integration model (Thayer et al., 2009; Smith et al., 2017) assumes that the effectiveness of the central autonomic network is reflected in CVA and can be indexed via heart rate variability (HRV), the time interval between adjacent heartbeats (Malik, 1996; Berntson et al., 1997). Specifically, the neurovisceral integration model assumes that a higher resting CVA is linked to better executive functioning (Wendt et al., 2015; Albinet et al., 2016; Spangler and Friedman, 2017; Spangler et al., 2018). In this paper, we operationalize CVA with the RMSSD (Malik, 1996; Berntson et al., 1997), which has been found to be less affected by respiratory influences than other HRV variables suggested to index CVA (Hill et al., 2009). Given that CVA reflects self-regulation and higher levels of CVA promote better executive performance (Thayer et al., 2009), it serves as a valuable measure when assessing performance under both physiological and psychological stress.

Psychological stress occurs when an individual perceives that personal or environmental demands tax or exceed his or her adaptive abilities (Lazarus and Folkman, 1984). Even if psychological stress can be considered as an idiosyncratic phenomena, given it will differ across individuals and situations, a common method of inducing stress is using tasks taxing executive functions. In addition, putting an emphasis on performing well helps to represent a situation which differs markedly from resting states in terms of psychological demands placed on the individual. For example, the Color Word Stroop Test (CWST; Stroop, 1935), a classical cognitive test to investigate inhibition (Diamond, 2013), has been used to create psychological stress (e.g., Vazan et al., 2017), and was found to increase biological markers of stress (e.g., Brugnera et al., 2018). In the CWST, an inhibitory interference occurs when the processing of a stimulus feature affects the simultaneous

processing of another attribute of the same stimulus (Stroop, 1935). Many variations exist, but the basic experimental paradigm depicted in the CWST is the use of color words printed either in the same color for the congruent category (for example, "BLUE" printed in blue color) or in a different color for the incongruent category (for example, "BLUE" printed in red). The participant has then to name the color in which the word is printed. The accuracy (number of errors) and reaction times (RTs) are measured to investigate the Stroop test performance (Stroop, 1935; Scarpina and Tagini, 2017). Better performance in this test reflects better inhibitory control which directly reflects the processing of incongruent stimuli in comparison to congruent stimuli. This differential processing constitutes the basis of the so-called Stroop interference, although a high heterogeneity to calculate the Stroop interference is reported in the literature (Scarpina and Tagini, 2017). Inhibition is primarily displayed by the error rate (accuracy) (McDowd et al., 1995), which represents as well an index of the ability to maintain the task's goal temporarily in a highly retrievable state (Kane and Engle, 2003). Assessing RTs may also be useful to uncover other processes linked to inhibition (Scarpina and Tagini, 2017).

The CWST and its variations (which we refer to thereafter as "Stroop tests") have already been investigated with HRV (Hoshikawa and Yamamoto, 1997; Prinsloo et al., 2011; Satish et al., 2015; Subramanya and Telles, 2015; Vazan et al., 2017; Zeki Al Hazzouri et al., 2017), however, only limited studies exist examining Stroop performance related to CVA (Johnsen et al., 2003; Subramanya and Telles, 2015; Albinet et al., 2016). In several studies, the Stroop test has been used with the mere purpose to create psychological stress (Hoshikawa and Yamamoto, 1997; Satish et al., 2015; Vazan et al., 2017), and no link with inhibition performance has been established. In other studies, although Stroop performance has been investigated with HRV, it has not been directly related to CVA indices (Prinsloo et al., 2011; Zeki Al Hazzouri et al., 2017). Among the studies linking Stroop performance and CVA, Johnsen et al. (2003) found that patients with dental fear having a higher resting CVA had shorter RTs to the incongruent color words and to the threat words compared to patients with lower resting CVA. However, Stroop interference accuracy (number of errors) was not measured, which does not provide a holistic representation of inhibition (McDowd et al., 1995). Albinet et al. (2016) showed that CVA improvements linked to a 5-months aquaerobics training program in older adults were linked to improvements on the Stroop interference accuracy (lower number of errors). Those two studies would be in line with the assumptions of the neurovisceral integration model (Thayer et al., 2009). On the contrary, Subramanya and Telles (2015) found that Stroop performance was better (less errors in the incongruent condition) with a decrease in high-frequency HRV, which usually depicts CVA when breathing patterns are comprised between 9 and 24 (Berntson et al., 1997), and increases in low-frequency HRV. However, these results were observed following a cyclic meditation condition involving slow body movements and slow breathing patterns, so there may have been some confounding effects of the experimental manipulation, and we can also speculate that in this case low-frequency HRV may have been

mainly vagally driven. In summary, limited studies have linked Stroop performance to CVA, investigating either only RTs or accuracy (number of errors). Our research aims to address this gap and link CVA to both Stroop performance RTs and accuracy (number of errors).

When considering the effects of PE on Stroop performance, one should distinguish the timing of the Stroop test. During exercise cognitive performance is usually impaired and improves after exercise (Lambourne and Tomporowski, 2010), unless exhaustion is reached (Schmit et al., 2015). After acute exercise, RT on the Stroop interference is shorter after PE in comparison to a resting control condition (Alves et al., 2012), or in comparison to before PE (Johnson et al., 2016), regardless of the fitness level of the participants (Chang et al., 2014). Improvements on the Stroop interference accuracy (number of correct answers) after acute exercise were found (Peruyero et al., 2017), however, some mixed findings were also reported (Vincent and Hall, 2017). Improved cognitive performance after acute exercise is usually linked to an increase in physiological arousal (Chang et al., 2014). In line with the inverted-U hypothesis, moderate arousal is linked to optimal cognitive performance, while if arousal levels are either too low or too high cognitive performance is impaired (Ploughman, 2008; Kashiwara et al., 2009). Acute effects of physical activity on Stroop performance are explained in particular via increased brain-derived neurotrophic factor (Griffin et al., 2011) and brain activation in the left dorsolateral prefrontal cortex (Yanagisawa et al., 2010), but do not seem to be related to changes in cerebral blood flow (Ogoh et al., 2014). At present, we are not aware of any research linking acute physical fatigue, Stroop performance, and CVA, and our research aims to address this gap.

Physical exertion will induce a drop in CVA, due to the activation of the sympathetic nervous system and the inhibition of the parasympathetic nervous system (Goldsmith et al., 2000; Iellamo, 2001; Winsley, 2002; Aubert et al., 2003; Stanley et al., 2013; Michael et al., 2017). Stopping PE induces a parasympathetic reactivation, which speed and magnitude depends on the fitness level of the individual (Stanley et al., 2013; Romero et al., 2017). If we follow the assumptions of the neurovisceral integration model (Thayer et al., 2009), the fact cognitive performance is decreased during exercise but improved after exercise (Lambourne and Tomporowski, 2010) may be linked to the parasympathetic deactivation observed during PE and reactivation after PE. Importantly, it may be possible to influence the speed and magnitude of CVA recovery after PE via specific strategies. Indeed, many factors were found to influence CVA (Laborde et al., 2018b), and some of them will be particularly adapted for athletes (Laborde et al., 2018c). Among those, we focus in this research on SPB.

Slow-paced breathing is a breathing technique with controlled inhalation and exhalation times ("paced"), realized at a slower pace, around 6 cycles per minute (cpm) than spontaneous breathing, which is usually comprised between 12 and 20 cpm in adults (Sherwood, 2006; Tortora and Derrickson, 2014). The pacing is usually realized via a visual, audio, or kinesthetic pacer (e.g., Allen and Friedman, 2012). According to the resonance model (Lehrer and Gevirtz, 2014), four processes play a role to understand the effects of SPB at 6 cpm: (1) the phase

relationship between heart rate (HR) oscillations and breathing at 6 cpm; (2) the phase relationship between HR and blood pressure oscillations at 6 cpm; (3) the activity of the baroreflex; and (4) the resonance characteristics of the cardiovascular system. Combined, those processes are expected to strengthen homeostasis in the baroreceptor (Vaschillo et al., 2002, 2006; Lehrer et al., 2006), which results in improving gas exchanges at the level of alveoli and in increasing vagal afferences (Lehrer and Gevirtz, 2014). Evidence has already been found for both acute (Laborde et al., 2017a) and chronic (Laborde et al., 2019) increases in CVA (i.e., vagal efferent activity) following SPB interventions.

Slow-paced breathing has already been shown to improve cognitive functioning, with inhibition and working memory (Prinsloo et al., 2011). Prinsloo et al. (2011) investigated a modified Stroop test, combining the classical inhibition component (i.e., naming the ink in which a word corresponding to another color is printed) to a working memory component, asking participants to remember how many control white squares appeared on the screen. Participants were allocated either to a SPB condition with biofeedback (seeing live the effects of SPB on their HRV via a device) or to a control condition where they were breathing spontaneously, for 10 min. Results showed no differences between conditions on the Stroop test inhibition component (number of errors), however, the working memory performance of the SPB group was better when compared to the control group. Limitations of this study were a reduced sample size ( $N = 18$  in a between-subjects design), the fact that inhibition and working memory were mixed in the modified Stroop test, which did not allow for drawing clear conclusions about the specific executive functions targeted, and finally the link between Stroop performance and CVA was not investigated. To conclude, research investigating the effects of SPB on inhibition is still required.

In summary, based on the research gaps identified in the literature, the aim of this research was to investigate the influence of a short-term SPB technique (in comparison to a watching TV control condition) on adaptation to psychological stress characterized via inhibition performance, before (Experiment 1) and after (Experiment 2) PE. The overall research was conceived as a mixed-model design, both experiments were conducted as within-subject designs (i.e., meaning participants take part in both the SPB and the control conditions of the same experiment), however, participants of Experiment 1 were not participating to Experiment 2, corresponding to the between-subject part. The within-subject part is always recommended in experiments involving HRV to limit inter-individual differences (Quintana and Heathers, 2014; Laborde et al., 2017b), however, given participating in four experimental sessions may have created some habituation effect, we split our research in two experiments, so that each participant was participating to two experimental sessions. At the subjective level, we expected PE and the CWST to increase the level of PS given both activities may lead the individual to perceive that demands of the task tax or exceed current adaptive abilities, while the relaxation moment would decrease the level of PS (Lazarus and Folkman, 1984; Lehrer and Gevirtz, 2014; Conway and Rubin, 2016). Further, we expected

the SPB condition to show larger effect sizes than the control condition, based on its relaxing effects (Lehrer and Gevirtz, 2014). Further, at the physiological level, we expect PE to increase HR and RF and decrease RMSSD, reflecting cardiovascular and respiratory adaptations to acute exercise (Stanley et al., 2013). Regarding our main hypothesis, based on the neurovisceral integration model (Thayer et al., 2009) and on the resonance model (Lehrer and Gevirtz, 2014), we hypothesize that in both experiments, SPB will improve inhibition (Stroop interference) in terms of accuracy (number of errors) and RTs. The improvement in Stroop interference will be mediated by resting RMSSD before starting the Stroop test.

## MATERIALS AND METHODS

### Participants

In order to determine our sample size, we utilized previous research combining SPB and inhibition (Prinsloo et al., 2011). The study of Prinsloo et al. (2011) did not find any effect of SPB on inhibition and was likely underpowered ( $N = 18$  for a between-subject design).<sup>1</sup> Based on this previous work, we computed an *a priori* power analysis with G\*Power (Faul et al., 2007) based on a small effect size, for a repeated-measures ANOVA with a within-between interaction, with a power of 0.80 and an alpha level of 0.05, which gave us a total sample size of 120. A total of  $N = 60$  participants (35 men, 25 women, mean age = 25.57 years old, age range = 19–40, BMI:  $M = 23.62$ ,  $SD = 2.52$ ) took part in Experiment 1, where the relaxation condition (SPB vs. watching TV control) was realized before physical PE. We refer thereafter to this first experiment as “relax before PE.” Similarly, a total of  $N = 60$  participants took part in Experiment 2 (38 men, 22 women, Mean age = 24.87 years old, age range = 18–41, BMI:  $M = 22.86$ ,  $SD = 2.29$ ), where the relaxation condition (SPB vs. watching TV control) was realized after PE. We refer thereafter to this second experiment as “relax after PE.” Exclusion criteria were self-reported cardiovascular diseases, and other chronic diseases that might influence breathing or HR patterns, such as asthma, diabetes, psychiatric, and neurological diseases (Laborde et al., 2017b) or being color-blind, making them ineligible to complete the CWST. Participants were students at the German Sport University. The protocol of the study was approved by the Ethics committee of the German Sport University (N° 175/2016).

### Material and Measures

#### Cardiac Vagal Activity

Cardiac vagal activity was operationalized via HRV and more specifically with RMSSD. An electrocardiography (ECG) device (Faros 180°, Mega Electronics, Kuopio, Finland) was used during the experiment to assess HRV, with a sampling rate of 500 Hz. We used two disposable ECG pre-gelled electrodes (Ambu L-00-S/25, Ambu GmbH, Bad Nauheim, Germany). The negative electrode was placed in the right infraclavicular fossa (just below the right

clavicle) while the positive electrode was placed on the left side of the chest, below the pectoral muscle in the left anterior axillary line. The Faros device was taped to the participants' chest in order to avoid moving too much while the participant was performing PE. From ECG recordings we extracted RMSSD with Kubios® (University of Eastern Finland, Kuopio, Finland). The full ECG recording was inspected visually, and artifacts were corrected manually (Laborde et al., 2017b). As recommended by Laborde et al. (2017b), respiratory frequency (RF) was also taken into account. RF was computed via the ECG derived respiration algorithm of Kubios® (Tarvainen et al., 2014).

#### Perceived Stress

A visual analog scale (VAS) was used to measure PS (Lesage and Berjot, 2011). Participants were asked “How stressed do you feel right now?” and they responded by marking a cross on a 100 mm line with two anchors (“not at all stressed” to “very much stressed”).

#### Physical Exertion – Burpees

Physical exertion was achieved with a modified version of the Burpee test (Podstawski et al., 2013). The Burpee test was named after the American physiologist Royal H. Burpee (1940), and was originally designed to measure agility and coordination. Burpees are physical exercises involving the whole body and requires no additional equipment. The following version of the Burpee was performed,<sup>2</sup> with these instructions: (1) start from a standing position; (2) bend over and place both hands firmly on the ground in front of the feet; (3) kick (or step) both feet back into a push-up position and lower the entire body to the ground (this is not a push-up); (4) the chest and thighs need to make full contact with the ground; (5) then extend the arms, lifting the chest and jump (or step) both feet in toward the chest; and (6) stand, jump (opening the hips fully), and clap hands behind the head while in the air.

#### Slow-Paced Breathing

Similar to previous research (Laborde et al., 2017a), the SPB exercise was conducted with a video showing a little ball moving up and down at the rate of 6 cpm. Participants having to inhale continuously through the nose while the ball was going up, and exhale continuously with pursed lips when the ball was going down. The video used was the same used in Laborde et al. (2017a), displaying a 3 × 5-min SPB exercise, with a 1-min break between each unit, corresponding to a total of 17-min. Exhalation (5.5 s) was slightly longer than inhalation (4.5 s), because a prolonged exhalation contributed to larger beat-to-beat heart fluctuations compared to prolonged inhalation, and therefore induce higher CVA (Strauss-Blasche et al., 2000).

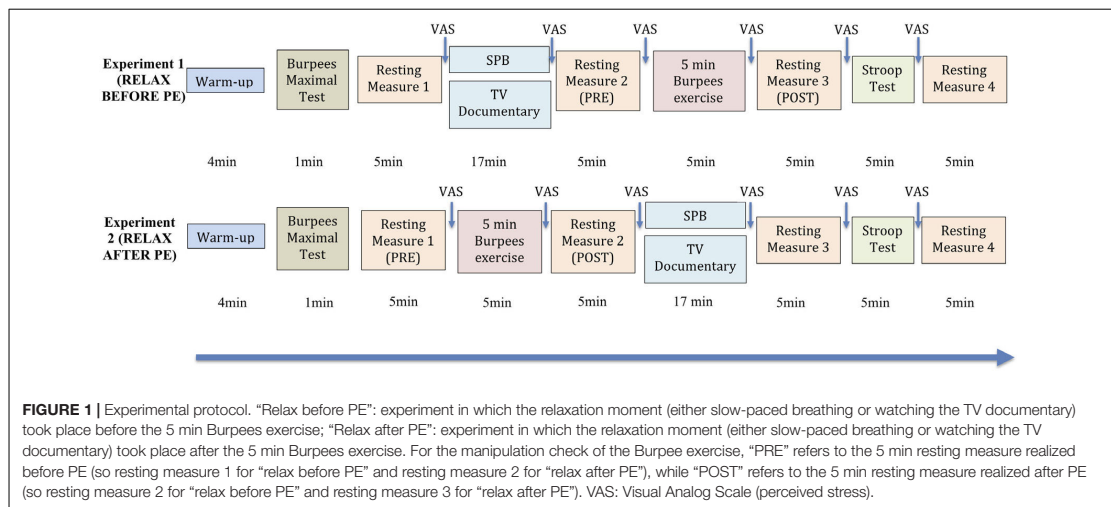
#### TV Neutral Documentary

The control condition (CON) used a TV documentary about world travel destinations, this was shown to the participants for the same duration as the SPB exercise (17 min). This TV documentary was found to be subjectively emotionally neutral in a previous pilot study.

<sup>1</sup>No precise *a posteriori* power calculation could be performed, given exact descriptive statistics related to Stroop accuracy could not be extracted from the study.

<sup>2</sup>[https://www.youtube.com/watch?v=X60BcsO\\_wE](https://www.youtube.com/watch?v=X60BcsO_wE) (retrieved on April 3, 2019).

## Study 4 - Influence of slow-paced breathing on inhibition after physical exertion



### Inhibition Performance (Measured With the Stroop Interference)

We used the computerized version of the CWST with verbal responding available in the Inquisit library,<sup>3</sup> and ran this with the Inquisit software (Inquisit 5 [Computer Software], 2016). We used a 15-in. flat-screen monitor (1,280 × 960 pixels at 60 Hz) at a viewing distance of 60 cm. Words appeared in 28-pt Arial font in the middle of a white screen. Three types of stimuli were used: colored square (congruent control stimuli), colored words displayed with the color corresponding to the word (congruent stimuli, for example the word "green" is displayed in the color green), and colored words displayed with an inconsistent color (incongruent stimuli, for example the word "green" is displayed in the color red). Participants were asked to name the color in which the word was written as fast and as accurately as possible, while ignoring the written meaning of the word. A headset mounted microphone recorded the verbal answers. A familiarization was realized with 20 trials. For the test, participants completed 84 trials (4 colors – red, green, blue, black) × 3 color stimulus congruency (congruent, incongruent, control squares) × 7 repetitions = 84 trials. Stimuli stayed on screen until response, latencies were measured from onset of stimuli. The intertrial interval was of 200 ms, and the error feedback (a red cross) of 400 ms.

### Procedure

Participants were recruited via flyers on the campus of the local University and via posts on social networks groups linked to the local University. For each experiment, there were two testing sessions involved (lasting around 90 min each, see **Figure 1** for full description). The experimental order of the sessions was counterbalanced. The two sessions were separated by 1 week, to

keep learning effects to a minimum, and took place at the same time of the day, given this parameter may influence HRV (van Eekelen et al., 2004) and performance (Folkard, 1990; Laborde et al., 2018a). Participants were either taking part in Experiment 1 or Experiment 2, they could not participate to both. They were asked to wear sports clothes to take part in the experiment. Prior to the testing sessions, participants were instructed not to drink or eat anything but water during the 2 h before the experiment, or take part in any strenuous exercise or drink alcohol for the 24 h prior testing (Laborde et al., 2017b). Both experiments were the same in their conception, the only aspect that differed was whether the relaxation moment was taking place before (Experiment 1) or after (Experiment 2) PE.

Upon arrival to the laboratory, participants were asked to fill out an informed consent form and a demographic questionnaire regarding variables potentially influencing HRV (Laborde et al., 2017b). At the beginning of the SPB condition, participants also received a short video introduction on how to perform the technique correctly, which was checked by the experimenter. Then participants were asked to perform a warm up<sup>4</sup> for 4 min involving squats, lunges, leg swings, jumping jacks, press up's, and squat thrusts. The warm up was chosen in agreement with an expert strength and conditioning coach from the German Sport University who was part of the research team. To prepare the PE (5 min Burpees exercise), participants had to perform five trials Burpees, so that the experimenter could check whether they were performed correctly. Then participants had to do a maximal Burpees test for 1 min, with the instruction to perform as many technically correct Burpees as they could. We then calculated 70% of this maximal number, and this gave the number of Burpees for the participants to realize during the 5 min Burpees exercise. For example, if the participant performed a maximal number of 20

<sup>3</sup><https://www.millisecond.com/download/library/stroop/> (retrieved on April 3, 2019).

<sup>4</sup><https://www.youtube.com/watch?v=HDfvWrGUC8> (retrieved on April 3, 2019).



Burpees,  $70\% \times 20 = 14$ , for the 5 min Burpees exercise he/she would have then to perform 14 Burpees each minute for 5 min. During the 5 min Burpees exercise, the realization of the Burpees was paced by the experimenter, giving a signal as a time marker to perform each Burpee, so the participant could better distribute his/her effort along the 5 min. The value of 70% was chosen based on a pilot study realized with 10 participants, where 70% was found to be an achievable compromise between achievability and degree of exhaustion, which had to be higher than 17 on the Borg Scale of Perceived Exertion (Borg, 1982). The 1 min Burpees maximal test was realized at the beginning of each session, given we wanted to account for potential differences in daily fitness level. The number of Burpees to be realized during the 5 min Burpees exercise was then based on the maximal number of Burpees achieved during the 1 min Burpees test at the beginning of the same session.

The relaxation task (either SPB or watching TV control) was placed either before (Experiment 1) or after (Experiment 2) PE. Finally participants had to perform the Stroop test, that lasted between 4 and 5 min. Between each block of the sessions, a HRV resting measure of 5 min was taken, based on the Task Force recommendation (Malik, 1996). HR and RF were derived from this HRV measurement. The HRV resting measure was realized in a sitting position with eyes closed, knees at 90°, hands on the thighs. At the end of the second testing session, participants were debriefed and thanked.

### Data Analysis

Due to technical problems, the ECG data of the last 13 participants of Experiment 2 were lost, therefore the sample size of Experiment 2 was reduced to  $N = 47$ . Regarding the HRV data, RMSSD was extracted from the Kubios output. RF (respiratory cycles per minute) was calculated multiplying the EDR (ECG derived respiration) value obtained via the Kubios algorithm by 60.

For the Stroop test, the number of incorrect answers was retrieved for the congruent colored squares, as well as for the congruent and incongruent stimuli. Regarding response times, we analyzed only those of the correct answers. Then we used two filters (see Lautenbach et al., 2016). In the first filter, trials with response times lower than 200 ms and higher than 3000 ms were excluded in order to account for extreme results (see Putman and Berling, 2011). The second filter then screened for RTs higher or lower than two standard deviations from the mean, which were also removed to account for outliers (see Dresler et al., 2009).

The VAS data and the Stroop performance data were normally distributed and homoscedastic. The physiological data (HRV, HR, RF) were not normally distributed, thus a log-transform (Log 10) was used to achieve normal distribution (Laborde et al., 2017b), and data were homoscedastic. Regarding the physiological data, we ran the analyses with the log-transformed values, however, we indicate as descriptive values the raw data, given they make more sense for the reader. For RMSSD, we controlled as well for the influence of covariates that have been linked to variations in CVA, such as RF, age, gender, smoking status, and BMI.

For the manipulation check related to PS, we ran three successive repeated-measures ANOVA for the relaxation technique, the 5 min Burpees exercise, and the Stroop task.

We had time (before vs. after), condition (SPB vs. CON) as within-subject independent variable, and relaxation moment ("relax before PE" or "relax after PE") as between-subject independent variable.

For the physiological manipulation check related to the 5 min Burpees exercise, "PRE" refers to the 5 min resting measure realized before PE (so resting measure 1 for "relax before PE" and resting measure 2 for "relax after PE"), while "POST" refers to the 5 min resting measure realized after PE (so resting measure 2 for "relax before PE" and resting measure 3 for "relax after PE"). As manipulation check, we wanted to ensure that PE was leading to physiological changes usually seen with acute exercise, meaning we expected a main effect of time ("PRE PE" vs. "POST PE") on HR (increase), RMSSD (decrease), and RF (increase), based on classical cardiorespiratory effects observed with PE (Stanley et al., 2013; Menz et al., 2016; Mlynczak and Krysztofiak, 2019). We investigated this manipulation check hypothesis running three repeated-measures ANOVA, with condition (SPB vs. CON), time (PRE vs. POST) as within-subject independent variables and relaxation moment ("relax before PE" or "relax after PE") as between-subject independent variables, with HR, RF, and RMSSD as dependent variables. Regarding RMSSD, in order to control for the potential effect of covariates, a linear mixed model analysis was conducted with age, gender, smoking status, BMI, and RF as covariates. The linear mixed model analysis allows to take into account time-varying covariates, in this case RF, which is not possible with the linear general model repeated-measures analysis module of SPSS. Given our hypothesis related to the manipulation check concerned only the main effect of time, we only report this result for clarity matters. Further, in order to ensure that changes are not due to a different amount of Burpees realized, the number of Burpees performed during the 5 min Burpees exercise will be checked via a repeated-measures ANOVA, with condition (SPB vs. CON) as within-subject independent variable and relaxation moment ("relax before PE" or "relax after PE") as between-subject independent variable.

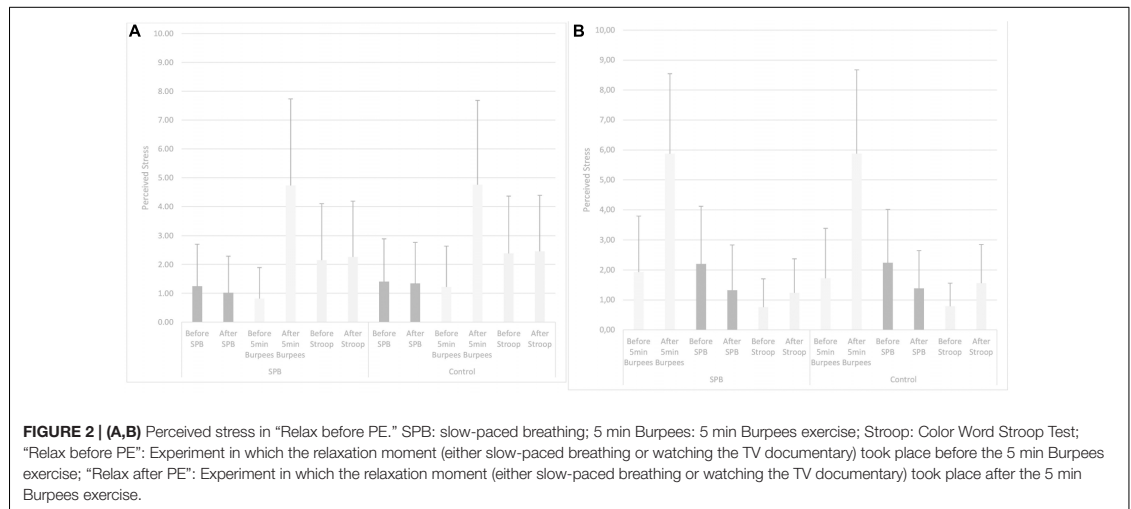
Regarding our main working hypothesis, we ran a repeated-measures ANOVA with condition (SPB vs. CON) as within-subject independent variable, and relaxation moment ("relax before PE" or "relax after PE") as between-subject independent variable. Errors (error rate to incongruent stimuli, reflecting Stroop interference accuracy) and RTs (incongruent stimuli-congruent stimuli) were used as dependent variables for the Stroop interference, and RMSSD to infer CVA. Regarding errors in the Stroop task, we decided not to take into account errors made with congruent stimuli in the calculation, given they were none. Interactions were investigated with further *t*-tests either paired or independent according to the analysis, with Bonferroni correction regarding the significance level. Regarding RMSSD, similar to the previous analysis related to PE, a linear mixed model analysis was further conducted with age, gender, smoking status, BMI, and RF as covariates.

Finally, in case a significant effect of SPB on Stroop interference (errors and/or RTs) was found, potential mediation via RMSSD was performed via the PROCESS 3.3 dialog box developed by Hayes (2013). This custom dialog tests the total, direct, and indirect effect of an independent variable on a dependent variable through a proposed mediator and allows

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Slow-Paced Breathing, Inhibition, Physical Exertion



inferences regarding indirect effects using percentile bootstrap confidence intervals.

## RESULTS

The full dataset is available in **Supplementary Material**.

### Perceived Stress Manipulation Check

See **Figures 2A,B** for full descriptive statistics about PS.

#### Relaxation (SPB vs. Watching TV)

Greenhouse-Geisser correction was applied for the tests. Regarding PS before and after the relaxation moment, no main effect of condition was found,  $F(1,105) = 1.708$ ,  $p = 0.187$ , partial  $\eta^2 = 0.02$ . No interaction condition  $\times$  relaxation moment was found,  $F(1,105) = 0.743$ ,  $p = 0.391$ , partial  $\eta^2 = 0$ . A main effect of time was found,  $F(1,105) = 17.808$ ,  $p < 0.001$ , partial  $\eta^2 = 0.15$ . PS before the relaxation was higher ( $M = 1.72$ ;  $SD = 1.57$ ) than after the relaxation ( $M = 1.45$ ;  $SD = 1.26$ ),  $t(106) = 3.111$ ,  $p = 0.002$ ; Cohen's  $d = 0.19$ . An interaction effect time  $\times$  relaxation moment was found,  $F(1,105) = 9.108$ ,  $p = 0.003$ , partial  $\eta^2 = 0.08$ . Further *post hoc t*-tests were run: they showed no significant difference for PS in "Relax before PE" being higher before ( $M = 1.32$ ;  $SD = 1.31$ ) than after ( $M = 1.18$ ;  $SD = 1.19$ ) the relaxation,  $t(59) = 1.359$ ,  $p = 0.179$ ; Cohen's  $d = 0.12$ . A significant difference was found for PS being lower before relaxation in "relax before PE" ( $M = 0.90$ ,  $SD = 0.95$ ) in comparison to before relaxation in "relax after PE" ( $M = 2.22$ ,  $SD = 1.73$ ),  $t(46) = 5.426$ ,  $p < 0.001$ ; Cohen's  $d = 0.96$ . PS was significantly lower after relaxation in "relax before PE" ( $M = 0.79$ ,  $SD = 0.77$ ) in comparison to in "relax after PE" ( $M = 1.79$ ,  $SD = 1.29$ ),  $t(46) = 6.795$ ,  $p < 0.001$ ; Cohen's  $d = 0.95$ . In "relax after PE," PS was significantly lower after ( $M = 1.79$ ,  $SD = 1.29$ ) than before ( $M = 2.22$ ,  $SD = 1.73$ ) relaxation,  $t(46) = 3.042$ ,  $p = 0.004$ ; Cohen's  $d = 0.28$ . No interaction effect condition  $\times$  time was found,  $F(1,105) = 0.302$ ,  $p = 0.584$ , partial

$\eta^2 = 0.0$ . No interaction effect condition  $\times$  time  $\times$  relaxation moment was found,  $F(1,105) = 0.178$ ,  $p = 0.674$ , partial  $\eta^2 = 0.0$ .

#### Five Minutes Burpees Exercise

Greenhouse-Geisser correction was applied for the tests. Regarding PS before and after the 5 min Burpees exercise, no main effect of condition was found,  $F(1,105) = 0.197$ ,  $p = 0.658$ , partial  $\eta^2 = 0$ . No interaction condition  $\times$  relaxation moment was found,  $F(1,105) = 1.595$ ,  $p = 0.209$ , partial  $\eta^2 = 0.02$ . A main effect of time was found,  $F(1,105) = 181.695$ ,  $p < 0.001$ , partial  $\eta^2 = 0.63$ . PS after the 5 min Burpees exercise was higher ( $M = 5.24$ ;  $SD = 2.73$ ) than before the 5 min Burpees exercise ( $M = 1.37$ ;  $SD = 1.41$ ),  $t(106) = 13.556$ ,  $p < 0.001$ ; Cohen's  $d = 1.79$ . No interaction effect time  $\times$  relaxation moment was found,  $F(1,105) = 0.310$ ,  $p = 0.579$ , partial  $\eta^2 = 0$ . No interaction effect condition  $\times$  time was found,  $F(1,105) = 0.159$ ,  $p = 0.690$ , partial  $\eta^2 = 0$ . No interaction effect condition  $\times$  time  $\times$  relaxation moment was found,  $F(1,105) = 1.995$ ,  $p = 0.161$ , partial  $\eta^2 = 0.02$ .

#### Stroop Task

Greenhouse-Geisser correction was applied for the tests. Regarding PS before and after the relaxation moment, no main effect of condition was found,  $F(1,105) = 2.102$ ,  $p = 0.150$ , partial  $\eta^2 = 0.02$ . No interaction condition  $\times$  relaxation moment was found,  $F(1,105) = 0.020$ ,  $p = 0.887$ , partial  $\eta^2 = 0$ . A main effect of time was found,  $F(1,105) = 8.976$ ,  $p = 0.003$ , partial  $\eta^2 = 0.08$ . PS was higher after Stroop ( $M = 1.94$ ;  $SD = 1.52$ ) than before Stroop ( $M = 1.61$ ;  $SD = 1.54$ ),  $t(106) = 2.696$ ,  $p < 0.001$ ; Cohen's  $d = 0.21$ . An interaction effect time  $\times$  relaxation moment was found,  $F(1,105) = 4.963$ ,  $p = 0.028$ , partial  $\eta^2 = 0.05$ . In "relax before PE," no difference in PS before ( $M = 2.27$ ,  $SD = 1.70$ ) and after Stroop ( $M = 2.36$ ,  $SD = 1.69$ ),  $t(59) = 0.500$ ,  $p = 0.619$ ; Cohen's  $d = 0.05$ . Considering the time before the Stroop task, PS was higher in "relax before PE" ( $M = 2.02$ ,  $SD = 1.69$ ) in comparison to in "relax after PE" ( $M = 0.77$ ,  $SD = 0.72$ );

$t(46) = 4.727, p < 0.001$ ; Cohen's  $d = 0.97$ . Considering the time after the Stroop task, PS was higher in "relax before PE" ( $M = 2.18, SD = 1.66$ ) in comparison to "relax after PE" ( $M = 2.02, SD = 1.69$ ),  $t(46) = 3.034, p = 0.004$ ; Cohen's  $d = 0.57$ . In "relax after PE," PS was higher after the Stroop task ( $M = 1.40, SD = 1.06$ ) than before the Stroop task ( $M = 0.77, SD = 0.72$ ),  $t(46) = 4.664, p < 0.001$ ; Cohen's  $d = 0.69$ . No interaction effect condition  $\times$  time was found,  $F(1,105) = 0.665, p = 0.417$ , partial  $\eta^2 = 0.0$ . No interaction effect condition  $\times$  time  $\times$  relaxation moment was found,  $F(1,105) = 1.099, p = 0.297$ , partial  $\eta^2 = 0.01$ .

**Five Minutes Burpees Exercise – Physiological Manipulation Check**

Descriptive statistics for all physiological variables and all measurement points can be seen in **Tables 1A,B**, while descriptive statistics specifically related to the manipulation check of the 5 min Burpees exercise can be seen in **Figures 3A–C**.

**Heart Rate**

Regarding HR, a main effect of time was found,  $F(1,105) = 344.906, p < 0.001$ , partial  $\eta^2 = 0.69$ ; with HR being higher after PE ( $M = 103.93; SD = 13.46$ ) in comparison to before PE ( $M = 80.85, SD = 12.95$ ).

**RMSSD**

Regarding RMSSD, a repeated-measures ANOVA was first ran. A main effect of time was found,  $F(1,105) = 85.517, p < 0.001$ , partial  $\eta^2 = 0.40$ , with RMSSD being lower after PE ( $M = 13.43; SD = 12.53$ ) than before PE ( $M = 24.87; SD = 24.23$ ). A linear mixed model analysis was then ran to investigate whether covariates (RF, age, gender, smoking status, BMI) were affecting the results. Relaxation moment, condition, time, and all covariates were entered as fixed effects, including intercepts, with an unstructured repeated covariance type resulting in the best model fit, with - 2 Restricted Log Likelihood = 214.341. No random effects (slopes nor intercepts) were found to improve significantly the model fit, consequently none were added to the model. Regarding the main effect of time, results remained significant within a linear mixed model analysis,  $F(1,106.956) = 158.644, p < 0.001$ , estimate of fixed effect = -0.45 ( $SE = 0.04$ ), 95% CI = -0.52 to -0.38. From the covariates two were found to have a significant effect, RF,  $F(1,263.319) = 34.114, p < 0.001$ , estimate of fixed effect = -0.70 ( $SE = 0.12$ ), 95% CI = -0.93 to -0.46; and BMI,  $F(1,101.267) = 4.074, p = 0.046$ ; estimate of fixed effect = -0.02 ( $SE = 0.01$ ), 95% CI = -0.04 to 0.

**Respiratory Frequency**

Regarding RF, a main effect of time was found,  $F(1,105) = 45.262, p < 0.001$ , partial  $\eta^2 = 0.26$ , with RF higher after PE ( $M = 18.30, SD = 3.54$ ) than before PE ( $M = 16.28, SD = 2.71$ ).

**Number of Burpees**

Regarding the number of Burpees performed during PE (descriptive statistics in **Table 2**), no main effect of condition was found  $F(1,105) = 0.161, p = 0.689$ , partial  $\eta^2 = 0$ ; and no interaction effect was found with the moment of relaxation  $F(1,105) = 0.524, p = 0.471$ , partial  $\eta^2 = 0.01$ .

TABLE 1A | Descriptive statistics physiological variables Exp. 1 "Relax before PE."

|                             | SPB                         |       |                                       |       |  |       | Control                     |       |                             |       |                                       |       |  |       |                             |       |
|-----------------------------|-----------------------------|-------|---------------------------------------|-------|--|-------|-----------------------------|-------|-----------------------------|-------|---------------------------------------|-------|--|-------|-----------------------------|-------|
|                             | Resting 1<br>(before relax) |       | Resting 2<br>(after relax/<br>PRE-PE) |       | Resting 3<br>(POST-PE/<br>before Stroop) |       | Resting 4<br>(after Stroop) |       | Resting 1<br>(before relax) |       | Resting 2<br>(after relax/<br>PRE-PE) |       | Resting 3<br>(POST-PE/<br>before Stroop) |       | Resting 4<br>(after Stroop) |       |
|                             | M                           | SD    | M                                     | SD    | M  | SD    | M                           | SD    | M                           | SD    | M                                     | SD    | M  | SD    | M                           | SD    |
| Heart rate (bpm)            | 77.16                       | 11.92 | 73.74                                 | 11.28 | 107.31                                   | 12.26 | 99.36                       | 11.90 | 76.50                       | 10.70 | 75.56                                 | 10.11 | 103.23                                   | 20.08 | 106.29                      | 43.71 |
| RMSSD (ms)                  | 43.57                       | 26.04 | 45.79                                 | 26.83 | 23.90                                    | 9.36  | 12.76                       | 8.30  | 45.90                       | 27.92 | 42.61                                 | 25.77 | 11.69                                    | 11.93 | 13.33                       | 10.52 |
| Respiratory frequency (cpm) | 14.15                       | 2.76  | 14.33                                 | 2.21  | 19.20                                    | 4.15  | 16.55                       | 3.61  | 16.52                       | 2.36  | 16.45                                 | 2.68  | 18.04                                    | 5.21  | 16.17                       | 4.09  |

TABLE 1B | Descriptive statistics physiological variables Exp. 2 "Relax after PE."

|                             | SPB                   |       |   |       |  |       | Control                     |       |                       |       |   |       |  |       |                             |       |
|-----------------------------|-----------------------|-------|---|-------|--|-------|-----------------------------|-------|-----------------------|-------|---|-------|--|-------|-----------------------------|-------|
|                             | Resting 1<br>(PRE-PE) |       | Resting 2<br>(POST-PE/<br>before relax) |       | Resting 3<br>(after relax/<br>before Stroop) |       | Resting 4<br>(after Stroop) |       | Resting 1<br>(PRE-PE) |       | Resting 2<br>(POST-PE/<br>before relax) |       | Resting 3<br>(after relax/<br>before Stroop) |       | Resting 4<br>(after Stroop) |       |
|                             | M                     | SD    | M                                       | SD    | M  | SD    | M                           | SD    | M                     | SD    | M                                       | SD    | M  | SD    | M                           | SD    |
| Heart rate (bpm)            | 89.67                 | 13.50 | 107.12                                  | 14.10 | 92.72  | 11.18 | 95.61                       | 12.20 | 87.85                 | 14.91 | 100.70                                  | 13.25 | 103.23                                       | 20.08 | 89.39                       | 10.14 |
| RMSSD (ms)                  | 25.38                 | 18.82 | 17.72                                   | 23.90 | 24.22  | 16.19 | 17.44                       | 11.39 | 27.12                 | 27.84 | 21.54                                   | 26.91 | 17.38  | 10.90 | 19.76                       | 11.23 |
| Respiratory frequency (cpm) | 17.23                 | 3.59  | 17.31                                   | 4.39  | 11.20  | 3.20  | 12.50                       | 3.02  | 17.58                 | 3.35  | 18.47                                   | 3.59  | 15.06  | 3.18  | 13.97                       | 3.05  |

bpm: beats per minute; ms: milliseconds; cpm: cycles per minute; relax: slow-paced breathing exercise or watching TV neutral documentary; PE: physical exertion (5 min Burpees exercise); SPB: slow-paced breathing; RMSSD: root mean square of successive differences. For the manipulation check of the Burpee exercise, "PRE" refers to the 5 min resting measure realized before PE (so resting measure 1 for Exp. 1 "relax before PE" and resting measure 2 for Exp. 2 "relax after PE"), while "POST" refers to the 5 min resting measure realized after PE (so resting measure 2 for Exp. 1 "relax before PE" and resting measure 3 for Exp. 2 "relax after PE").

Main Research Question – Stroop Task

Descriptive statistics related to the variables linked to our main research question with the Stroop task are displayed in Table 3. Regarding Stroop interference (RT), a repeated-measures ANOVA revealed no main effect of condition  $F(1,105) = 0.873, p = 0.352, \text{partial } \eta^2 = 0$ . No interaction effect condition  $\times$  relaxation moment was found,  $F(1,105) = 0.039, p = 0.843, \text{partial } \eta^2 = 0$ . Relaxation moment had an overall effect on the Stroop interference,  $F(1,105) = 35.031, p < 0.001, \text{partial } \eta^2 = 0.25$ , with a shorter Stroop interference found for "relax before PE" ( $M = 20,33; SD = 69.61$ ) in comparison to "relax after PE" ( $M = 103.14; SD = 64.87$ ).

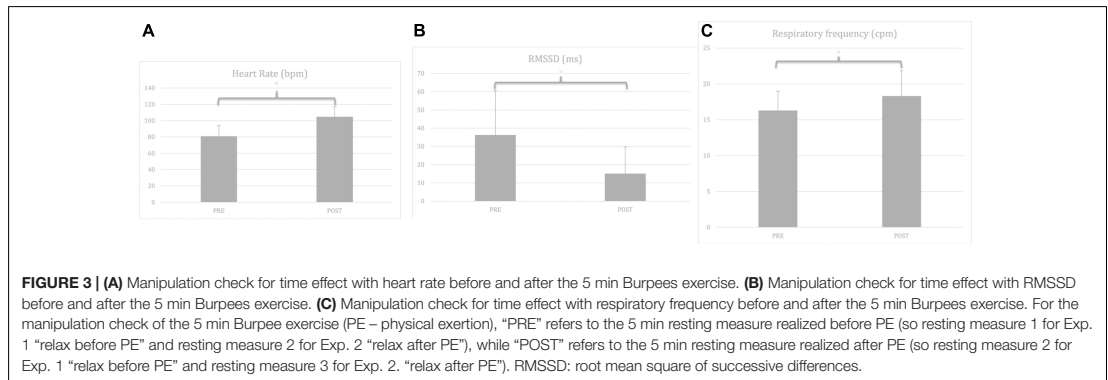
Regarding Stroop interference (errors), we found a main effect for condition,  $F(1,105) = 22.584, p < 0.001, \text{partial } \eta^2 = 0.17$ , with SPB ( $M = 0.36, SD = 0.60$ ) being lower than CON ( $M = 0.69, SD = 0.82$ ). No main relaxation moment effect was found,  $F(1,105) = 1.735, p = 0.191, \text{partial } \eta^2 = 0.02$ . An interaction effect between condition and relaxation moment was found,  $F(1,105) = 5.364, p = 0.022, \text{partial } \eta^2 = 0.04$ . Further *post hoc t*-tests were run: they showed a significant difference between SPB-"relax before PE" ( $M = 0.50; SD = 0.70$ ) and SPB-"relax after PE" ( $M = 0.17; SD = 0.38$ ), with  $t = 4.105, df = 46, p < 0.001$ , Cohen's  $d = 0.60$ ; a significant difference between SPB-"relax before PE" ( $M = 0.50, SD = 0.70$ ) and CON-"relax before PE" ( $M = 0.68, SD = 0.81$ ), with  $t = 2.647, df = 59, p = 0.010$ , Cohen's  $d = 0.34$ ; a significant difference between SPB-"relax after PE" ( $M = 0.17, SD = 0.38$ ) and CON-"relax after PE" ( $M = 0.70, SD = 0.86$ ),  $t = 3.658, df = 46, p < 0.001$ , Cohen's  $d = 0.53$ . No significant differences were found between CON-"relax before PE" and CON-"relax after PE,"  $t = 0.643, df = 46, p = 0.523$ , Cohen's  $d = 0.09$ .

Regarding RMSSD during the resting measure before starting the Stroop test: a main condition effect was found,  $F(1,105) = 5.841, p = 0.017, \text{partial } \eta^2 = 0.05$ , with SPB ( $M = 16.97, SD = 14.28$ ) being higher than CON ( $M = 14.19, SD = 11.79$ ). No interaction effect between condition and relaxation moment was found,  $F(1,105) = 3.335, p = 0.071, \text{partial } \eta^2 = 0.03$ . A main relaxation moment effect was found,  $F(1,105) = 28.870, p < 0.001, \text{partial } \eta^2 = 0.22$ , with RMSSD being higher in "relax after PE" ( $M = 20.80, SD = 14.15$ ) than in "relax before PE" ( $M = 11.49, SD = 10.69$ ).

A linear mixed model analysis was then ran to investigate whether covariates (RF, age, sex, smoking status, BMI) were affecting the results. Relaxation moment, condition, and all covariates were entered as fixed effects, including intercepts, with an unstructured repeated covariance type resulting in the best model fit, with - 2 Restricted Log Likelihood = 129.122. No random effects (slopes nor intercepts) were found to improve significantly the model fit, consequently none were added to the model. No main effect of condition was found,  $F(1,108.835) = 0.039, p = 0.844$ . A main effect of relaxation moment was found,  $F(1,126.990) = 4.890, p = 0.029$ , estimate of fixed effect = 0.27 ( $SE = 0.12$ ), 95% CI = 0.03 to 0.51. No interaction effect condition  $\times$  relaxation moment was found,  $F(1,110.459) = 0.169, p = 0.682$ . From the covariates only RF was found to have a significant effect,  $F(1,143.946) = 16.814$ ,



## Study 4 - Influence of slow-paced breathing on inhibition after physical exertion



**TABLE 2 |** Descriptive statistics for the number of Burpees realized during the 5 min Burpees exercise.

|                   | Exp. 1 – RELAX BEFORE PE |           |          |           | Exp. 2 – RELAX AFTER PE |           |          |           |
|-------------------|--------------------------|-----------|----------|-----------|-------------------------|-----------|----------|-----------|
|                   | Slow paced breathing     |           | Control  |           | Slow paced breathing    |           | Control  |           |
|                   | <i>M</i>                 | <i>SD</i> | <i>M</i> | <i>SD</i> | <i>M</i>                | <i>SD</i> | <i>M</i> | <i>SD</i> |
| Number of Burpees | 64.27                    | 11.54     | 63.00    | 11.64     | 63.60                   | 11.69     | 63.19    | 12.20     |

*PE: physical exertion (5 min Burpees exercise).*

**TABLE 3 |** Descriptive statistics for the main working hypothesis.

|  | Exp. 1 – RELAX BEFORE PE |           |          |           | Exp. 2 – RELAX AFTER PE |           |          |           |
|--|--------------------------|-----------|----------|-----------|-------------------------|-----------|----------|-----------|
|  | SPB                      |           | Control  |           | SPB                     |           | Control  |           |
|  | <i>M</i>                 | <i>SD</i> | <i>M</i> | <i>SD</i> | <i>M</i>                | <i>SD</i> | <i>M</i> | <i>SD</i> |
| Stroop interference (number of errors)   | 0.50                     | 0.70      | 0.68     | 0.81      | 0.17                    | 0.38      | 0.70     | 0.86      |
| Stroop interference (reaction times, ms) | 18.26                    | 95.70     | 25.61    | 79.45     | 104.71                  | 72.01     | 109.49   | 68.92     |
| RMSSD (ms)                               | 11.29                    | 9.36      | 11.69    | 11.93     | 24.22                   | 16.20     | 11.69    | 11.93     |
| Respiratory frequency (cpm)              | 19.20                    | 4.15      | 18.04    | 5.212     | 11.20                   | 3.201     | 15.06    | 3.18      |

*ms: milliseconds; cpm: cycles per minute; PE: physical exertion (5 min Burpees exercise); Stroop interference (accuracy) represents the number of errors in response to incongruent stimuli; Stroop interference (reaction time): reaction time to incongruent stimuli – reaction time to congruent stimuli; RMSSD, root mean square of successive differences; and respiratory frequency correspond to the resting measure 3 taken before the Stroop test.*

$p < 0.001$ , estimate of fixed effect =  $-0.47$  ( $SE = 0.11$ ), 95% CI =  $-0.70$  to  $-0.24$ .

Finally, to test whether the effect of SPB on Stroop interference (errors) was mediated by RMSSD, the experimental group (coded SPB = 1; CON = 2) was entered as independent variable, Stroop interference (errors) was entered as dependent variable, and RMSSD was entered as mediator variable. Based on a 10,000 sampling rate, the results from bootstrapping revealed no significant indirect effect, 95% CI =  $-0.15$  to  $0.18$ . Rerunning the mediation analysis taking into account the covariates (RE, age, sex, smoking status, BMI) revealed no significant indirect effect, 95% CI =  $-0.03$  to  $0.10$ .

## DISCUSSION

This research aimed to investigate the effects of SPB on the adaptation to psychological stress, and more specifically

inhibition performance, after PE. At the subjective level, in line with our hypothesis, PS was increased during the 5 min Burpees exercise and the Stroop test, and decreased during the relaxation technique, however, no effect of condition was found. At the physiological level, in line with our hypothesis, our PE manipulation was successful in increasing HR, RE, and decreasing RMSSD. Our main hypothesis was partially validated, given SPB led to better Stroop interference accuracy (lower number of errors in response to incongruent stimuli), however, no differences were found regarding Stroop interference RT.

Concerning the subjective manipulation check, confirming our hypothesis, PE and CWST increased PS, while PS was decreased with relaxation. This means that PE and the Stroop task were perceived as taxing or exceeding the adaptive resources of the individual, while relaxation contributed to decreased PS (Lazarus and Folkman, 1984). However, contrary to our hypothesis based on the relaxing effects of SPB (Lehrer and Gevirtz, 2014), the effects of SPB on PS did not differ from

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watching the TV documentary. This could be linked to the fact that many people already use TV as means of relaxation (Conway and Rubin, 2016), and therefore associate it with an activity decreasing PS. Interestingly, the increase in PS was much higher during the 5 min Burpees exercise (partial  $\eta^2 = 0.63$ ) in comparison to the Stroop task (partial  $\eta^2 = 0.08$ ). This might be due to the fact the 5 min Burpees exercise was perceived as particularly exhausting by the participants.

Concerning the physiological manipulation check of the 5 min Burpees exercise, we focused specifically on the effects involving time, given we are looking for physiological changes between the resting measures before and after the Burpees. HR and RF increased overall during the 5 min Burpees exercise, while RMSSD decreased (even after controlling for RF and other covariates). Our results cannot be directly compared to previous research, given very few studies have investigated Burpees so far, and when they did it was always as part of a more global training program involving a large range of physical exercises (McRae et al., 2012; Emberts et al., 2013; Abbas et al., 2018; Sperlich et al., 2018). However, these findings would be in line with typical cardiorespiratory adaptations to PE (Stanley et al., 2013; Menz et al., 2016; Mlynczak and Krysztofiak, 2019). The number of Burpees achieved did not differ across conditions or across relaxation moment. We should note that in the 5 min Burpees exercise, the realization of the Burpees was paced for the participants, meaning they could not go faster, even if they felt that they were able to do so. Further research may investigate whether giving the instruction to perform as many Burpees as possible during 5 min (i.e., maximal performance) may have provided different results, helping to understand whether SPB influences physical performance.

Regarding our main hypothesis linked to Stroop interference errors and RTs, Stroop interference errors decreased with SPB, while no change were observed for Stroop RTs, therefore our hypothesis was only partially validated. According to the literature, with the CWST inhibition is primarily reflected by the number of errors (accuracy) made with incongruent stimuli (McDowd et al., 1995), given it shows a typical illustration of inhibition failure. This is in line with the interpretation that Stroop interference accuracy represents as well an index of the ability to maintain the task's goal temporarily in a highly retrievable state (Kane and Engle, 2003), given making a mistake linked to incongruent stimuli means that we were temporarily not successful in the aim of the task. A follow-up analysis of the condition  $\times$  relaxation moment interaction shows that if Stroop interference errors were overall lower in the SPB condition in comparison to the CON condition, Stroop interference errors were lower when SPB was realized after PE than before PE. This may be explained by the time proximity of SPB to the realization of the Stroop test, and also by the fact that the physiological changes induced by PE may have influenced the effects of SPB realized beforehand.

Given no differences were observed with Stroop interference RTs, we have to conclude that SPB did not help to reduce the processing time of incongruent stimuli. Even if Stroop interference RTs are not the main marker of inhibition obtained with the Stroop test, RTs may still help us to understand

mechanisms related to inhibition processing (Scarpina and Tagini, 2017). This may be linked to the fact SPB induces a general decrease of general physiological arousal (Lehrer and Gevirtz, 2014), which we also observed in this research in experiment 1 ("relax before PE"), where participants had a significantly lower HR and higher RMSSD after performing SPB in comparison to CON. The fact that physiological arousal plays a role in the Stroop interference RTs is also confirmed by our data, given Stroop interference RTs are much lower right after PE (in "relax before PE"), than 17 min later after performing SPB or CON (in "relax after PE"), with a large effect size (partial  $\eta^2 = .25$ ), which is in line with previous research (Cooper et al., 2016). Cooper et al. (2016) found that RTs on the complex level (incongruent condition) of the Stroop were quicker (in comparison to RTs obtained before exercise) immediately following the sprint-based exercise, but did not differ after 45 min; while RTs on the simple level (congruent condition) of the Stroop were quicker 45 min following sprint-based exercise (Cooper et al., 2016), but did not differ from the rest condition right after exercise. It could be speculated that this difference may be due to changes in brain-derived neurotrophic factor (Griffin et al., 2011) and brain activation in the left dorsolateral prefrontal cortex (Yanagisawa et al., 2010), given these mechanisms were previously identified as playing a role in improving Stroop interference RTs after exercise, however, this should be investigated in further research. To sum up, we found that SPB was effective in improving Stroop interference accuracy (decreasing the number of errors to incongruent stimuli) but not RTs.

Root mean square of successive differences in the resting measure before the Stroop task was found to be higher in the SPB condition than in the CON condition, which would mean that the increase in vagal afferent activity assumed by the resonance model (Lehrer and Gevirtz, 2014) can also be observed in vagal efferent activity (CVA). However, this main effect of condition disappears when integrating covariates to the analyses, and in particular RF, given a main effect of RF on RMSSD was found. Given respiratory parameters (Hirsch and Bishop, 1981; Brown et al., 1993; Houtveen et al., 2002), and in particular RF (Hirsch and Bishop, 1981) were found to influence HRV, some authors recommend to correct HRV variables reflecting CVA for respiration in order to accurately capture CVA (e.g., Grossman et al., 1991; Grossman, 1992). However, another stream of research assumes a common neural basis for HRV and respiration, and regards a routine control of HRV for respiration problematic, given it would remove variability associated with neural control over the heartbeat, and therefore some of the variance playing a crucial role in HRV would be artificially removed, which would then not reflect normal physiology (Larsen et al., 2010; Thayer et al., 2011; Lewis et al., 2012; Dick et al., 2014). For further clarification whether SPB does increase CVA, future research should consider manipulating parasympathetic nervous activity via pharmacological blockade, for example with atropine (Lahiri et al., 2008), a parasympatholytic agent that inhibits the action of acetylcholine, the main neurotransmitter of the parasympathetic nervous system,

by competitively blocking muscarine receptors (Clementi and Weber-Schöndorfer, 2015). Previous research (Du Plooy and Venter, 1995) investigating deep breathing (three deep inspirations and expirations) and HRV with atropine injection showed that RMSSD was increased during deep breathing, however, this increase was canceled by atropine, which would suggest that this increase in RMSSD was vagally driven. These findings should however be replicated with the SPB exercise used in our study in order to clarify the effects of 15 min SPB on CVA.

Finally, a mediation analysis indicates that RMSSD did not mediate the effects of SPB on Stroop interference accuracy performance. This finding is not in line with the neurovisceral integration model (Thayer et al., 2009; Smith et al., 2017), which assumes that a higher CVA is linked to higher executive performance. These results are also contrary to what was found in Albinet et al. (2016), where improvements in Stroop interference (accuracy) following a 5-month aquaerobics program were correlated to increases in RMSSD, although no mediation analysis was performed. This may suggest that other mechanisms than CVA may underlie SPB effects. For example, the high amplitude oscillations in HR provoked by SPB were recently suggested to enhance the functional connectivity in brain networks associated with emotion regulation (Mather and Thayer, 2018). Future research has to clarify whether those high amplitude oscillations in HR due to SPB also provoke changes in the functional connectivity of brain networks associated with executive (inhibitory) functioning.

The strengths of this research were conducting two experiments to thoroughly investigate the effects of SPB on the adaptation to psychological stress, specifically inhibition, following PE; as well as the use of physiological measurements (HRV) to enable the investigation of potential underlying mechanisms. However, our research is not without limitation. Firstly, the participants' sporting background (e.g., fitness level, sport experience) was not assessed. Secondly, our sample was comprised of sport students, consequently future research has to investigate whether our findings would replicate to other non-athletic samples. Thirdly, no color perception test was conducted, participants only stated if they were color-blind, however, they were only allowed to continue the experiment if they were successful during the CWST familiarization phase. Fourth, the determination of the 70% of the 5 min Burpees exercise during the pilot study was based on subjective evaluation with the Borg Scale of Perceived Exertion (Borg, 1982), and further research should also measure physiological parameters during the 5 min Burpees exercise. Fifth, due to technical problems our final sample comprised 107 participants instead of the 120 originally planned, and future studies should ensure to achieve the necessary sample size to detect whether CVA mediates executive performance following SPB, to rule out the possibility that our study was underpowered to find a mediation effect. Sixth, in order to shed more light on the underlying physiological mechanisms, other parameters should be investigated, such as gaseous exchange and brain activity for example. Future research may also consider the use of biofeedback to help the participants visualize the effects of SPB on their HRV and CVA.

## CONCLUSION

The aim of this research was to investigate the role of a relaxation technique, SPB, on the adaptation to psychological stress, investigated with a measure of inhibition, following PE. Two experiments were conducted within this research. In Experiment 1, SPB (or the TV watching control condition) was realized after PE, and in Experiment 2, SPB (or the TV watching control condition) was realized before PE. Our findings showed that SPB was able to improve inhibition after PE via improving Stroop interference accuracy, meaning that participants made less errors overall after having performed SPB (either before or after PE), and the effects were stronger when SPB was performed after PE. However, SPB was not found to impact Stroop interference RTs, meaning that participants were not faster in their ability to inhibit incongruent stimuli. The applied implications of these findings are quite interesting, given athletes can consider using SPB as a quick fix to address inhibition failures. SPB may therefore help to address choking under pressure, which can be triggered by inhibition failures according to the distraction account (Roberts et al., 2017). Finally, the use of SPB could be investigated in many domains where inhibition failures would lead to unwanted behavior with serious consequences, for example police officers shooting or medical doctors operating.

## DATA AVAILABILITY

All datasets generated for this study are included in the manuscript and the **Supplementary Files**.

## ETHICS STATEMENT

This study was approved by the Ethics Committee of the German Sport University Cologne (N° 175/2016).

## AUTHOR CONTRIBUTIONS

SL, TL, and FD contributed to conceiving the design of the study. TL lead the data collection, with the help of SL. SL realized the statistical analysis with the help of TH and UB, and wrote the first draft of the manuscript. EM provided the critical comments to improve the manuscript. TH, UB, and FD suggested the final adjustments on the manuscript. All authors agreed on the final version of the manuscript.

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## SUPPLEMENTARY MATERIAL

The Supplementary Material for this article can be found online at: <https://www.frontiersin.org/articles/10.3389/fpsyg.2019.01923/full#supplementary-material>

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## *General discussion*

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### 11 General discussion

#### 11.1 Overview

The aim of this PhD thesis was to further investigate the mechanisms underlying the effects of SPB in improving self-regulation processes. The theoretical framework was based on the resonance model (Lehrer & Gevirtz, 2014) and on the neurovisceral integration model (Smith et al., 2017; Thayer et al., 2009). SPB was suggested to increase CVA, which would in turn reflect a better adaptation on a range of self-regulation phenomena including stress management, sleep, and executive functioning. This assumption was based on the fact that SPB is suggested to stimulate vagal afferences (Lehrer & Gevirtz, 2014), and that CVA is assumed to reflect the effectiveness of the central autonomic network, based on the neurovisceral integration model (Smith et al., 2017; Thayer et al., 2009).

Our working hypotheses were partially validated: first, a 15 min short-term SPB intervention (in comparison to control) triggered a higher CVA (RMSSD) in adolescents with intellectual disabilities (Study 1, p.54) performing a cognitive stressful task, indicating a better adaptation, in comparison to a condition where they were listening to an audiobook. A similar short-term SPB intervention increased resting CVA (RMSSD) in healthy young adults in Study 4 (p.90), however no changes were found in resting CVA (RMSSD) in healthy young adults in Study 3 (p.74). Regarding long-term SPB intervention (15 min per day during 30 days) in healthy young adults (Study 2, p.62),  $CVA_{\text{night}}$  (HF-HRV) was found to be improved after 30 days, while only a tendency was found with  $CVA_{\text{morning}}$  (HF-HRV). Second, executive performance was found to be improved in Study 3 (inhibition, working memory, cognitive

flexibility) and in Study 4 (inhibition), however this improvement was not mediated by CVA, contrary to our hypothesis.

Pooling our results together, we could conclude that SPB without biofeedback does influence positively self-regulation processes, similar to what is observed with SPB interventions performed with biofeedback (Gevirtz, 2013; Goessl et al., 2017; Kennedy & Parker, 2018; Wheat & Larkin, 2010; Yu et al., 2018; Zaccaro et al., 2018). This would mean that biofeedback, even if it may improve aspects related for instance to self-efficacy and motivation to perform the SPB exercise, is not a decisive criteria driving the effectiveness of SPB interventions. However, based on our findings, we cannot decisively conclude for the role of CVA to reflect the self-regulation processes influenced by SPB. If increases in CVA markers following SPB (in comparison to control) were observed in studies 1, 2, and 4, this was not the case for study 3. In addition, CVA was not found to mediate the improvement in executive functioning following SPB in studies 3 and 4. This would either mean that contrary to the assumptions of the resonance model (Lehrer & Gevirtz, 2014), SPB may not trigger vagal afferences, or that the stimulation of vagal afferences is not necessarily reflected in vagal afferences to the heart, as measured with CVA. To illustrate this speculation, a similar questioning has been reported in pain research, using invasive vagus nerve stimulation (VNS). Vagus nerve stimulation (i.e.; stimulation of vagal afferents) was suggested to send non-specific signals to the brainstem, and these signals were suggested in turn to compete with incoming pain stimuli or alternatively trigger non-specific reflexes that activate pain inhibition, for instance by releasing inhibitory neurotransmitters (Albusoda, Farmer, & Aziz, 2018). A similar mechanism may occur with SPB, but others may also be considered. As postulated by the resonance model (Lehrer & Gevirtz, 2014), SPB may strengthen baroreceptor homeostasis, or improves the gas exchange at the alveoli level. If the action on baroreceptor received support so far (Lehrer et al., 2003), additional research is required to test the hypothesis related to the effectiveness of the gas exchange, addressing the issues found in previous research focusing on



RSA (Buchheit, 2010; Giardino et al., 2003; Sin et al., 2010; Tharion & Subramani, 2011; Tzeng, 2011).

In addition to the mechanisms suggested by the resonance model (Lehrer et al., 2003), a new hypothesis related to the effects of SPB has recently been developed (Mather & Thayer, 2018). This hypothesis assumes that the oscillations induced by SPB in the heart with its action on RSA and its coupling with the baroreflex feedback loop would induce **oscillatory activity in the brain**, and hence enhance functional connectivity in brain networks associated in particular with emotion regulation. Slow oscillations in the brain are important for both local and more distant connections: more powerful and widespread slow oscillations can modulate weaker but faster local oscillations (Buzsaki & Draguhn, 2004; Canolty & Knight, 2010). Slow oscillations are therefore critical for fast information spreading within brain networks: given the limited number and speed of neuronal connections connecting distant regions, large-scale brain networks can only oscillate in tandem during slow oscillations (Buzsaki & Draguhn, 2004). Different kinds of brain imaging techniques may help to understand further the mechanisms underlying SPB effects in the brain, such as measuring blood-oxygen level-dependent (BOLD) with functional magnetic resonance imaging (*f*MRI) to investigate neural activity fluctuation in the brainstem (Pfurtscheller et al., 2019), or the influence of breathing variations on task performance (Birn, Murphy, Handwerker, & Bandettini, 2009). Measuring electroencephalography (EEG) signals would allow for example to measure how SPB correlates with slow cortical potentials (Hinterberger, Walter, Doliwa, & Loew, 2019). Finally, functional near infrared spectroscopy (*f*NIRS) would enable to investigate brain's oxygenation in relation to voluntary regulated breathing patterns (Telles, Gupta, Singh, & Balkrishna, 2016). In summary, the understanding of the underlying mechanisms of SPB would definitely benefit from the use of brain imaging techniques in the future.

I will now reflect on the main limitations of the different studies conducted within this PhD thesis, before outlining interesting perspectives that emerged from this research.

## 11.2 Limitations

This PhD thesis had several limitations that need to be considered when interpreting the findings, and that future research should consider addressing.

First, **the control conditions involved spontaneous breathing** while the participants were listening to an audiobook (Study 1), engaging with social medias (Study 2), or watching a neutral TV documentary (Study 3 and Study 4). Therefore, we cannot rule out the possibility that focusing on breathing was the key influencing factor, and future studies should include a control condition that includes a similar focus on breathing at a different pace, for instance 12 cycles per minute (Tsai et al., 2015), which would match the typical lower range of spontaneous respiratory frequencies (L. Sherwood, 2006).

Second, respiratory frequency was obtained via a dedicated algorithm from Kubios (Tarvainen et al., 2014). However, a **more precise assessment of respiratory frequency** such as with a respiratory belt or a pneumotachograph (Egizio et al., 2011; Quintana & Heathers, 2014) and the assessment of other respiratory related variables (e.g., respiratory depth, gas exchanges) could prove helpful in explaining the effects of SPB on executive functioning (Lorig, 2011; Ritz et al., 2002).

Third, **only one single breathing pattern has been used** in the four studies of this PhD thesis, based on previous research (Allen & Friedman, 2012): 6 cpm, with 4.5s inhalation and 5.5s exhalation, without breaks after the inhalation or exhalation phases. However some meaningful variations could be introduced to the SPB technique practiced in this PhD thesis. For example, having a break introduced between inhalation and exhalation, or between exhalation and inhalation, could have different physiological consequences (Reyes del Paso et al., 2015; Skow et al., 2015). Future studies may particularly want to focus on the use of a post-exhalation break, given that it appears to increase CVA more than no break (Russell et al., 2017).

### 11.3 Perspectives

Future research should consider several avenues to better understand the mechanisms underlying the effects of SPB, in addition to the use of brain imaging techniques mentioned earlier in the overview (p.105).

First, the question of an individual **resonance frequency** should be investigated. The resonance frequency is supposed to represent an optimal SPB frequency (Vaschillo et al., 2006), specific to each individual. As assumed by the resonance model (Lehrer & Gevirtz, 2014), breathing at 6 cpm allows to match RSA to the inherent oscillations in heart rate linked to blood pressure modulation at 0.1 Hz, in order for the two signals to summate and produce larger variations in heart rate. However it is possible that depending on physical characteristics such as height (Vaschillo et al., 2006), the resonance frequency differs across individuals, and an individualized breathing frequency determined with an ad-hoc protocol (Lehrer et al., 2000) may produce larger increases in CVA (Steffen et al., 2017).

Second, SPB should be tested together with additional **inspiratory threshold load** (Gholamrezaei, Van Diest, Aziz, Vlaeyen, & Van Oudenhove, 2019). To date only the study of Gholamrezaei et al. (2019) investigated this question, and the authors showed that increasing inspiratory loads tend to increase the amplitude of systolic blood pressure variation as well as RSA. The increase in CVA was mediated by stronger stimulation of the arterial baroreceptors, due to larger systolic blood pressure swings along the respiratory cycle. Inspiratory threshold load should therefore be further investigated in combination to SPB with different outcomes, in order to clarify whether it would produce stronger adaptation benefits.

Third, SPB should be investigated in a large range of **specific samples**, like it was done in Study 1 with adolescents with intellectual disability. Previous research already focused on children (Calcaterra et al., 2014; Nada, 2009) and pregnant women (van der Zwan, Huizink, Lehrer, Koot, & de Vente, 2019) for example, but SPB investigation should also be extended

to samples such as older adults, given it may help them cope with symptoms related to ageing, or to samples affected by diseases, mental or physical health conditions where SPB could potentially help relieving the symptoms, such as samples suffering from depression (Hoffmann, Ettinger, Reyes Del Paso, & Duschek, 2017) or chronic low blood pressure (Duschek, Hoffmann, Reyes Del Paso, & Ettinger, 2017).

Fourth, in relation with studying SPB with specific samples, a large range of **outcomes** related to self-regulation phenomena should also be considered. For instance SPB was already implemented with people with depression (Pinter et al., 2019), where it was found to improve treatment when associated to psychotherapy (Caldwell & Steffen, 2018), to improve the condition of patients suffering from cardiovascular diseases (Zou et al., 2017), or to improve sport performance (Jimenez Morgan & Molina Mora, 2017).

Fifth, the question of the differences of practicing **SPB with or without biofeedback** should be addressed, given some studies reported either similar (Wells et al., 2012) or divergent (Q. Chang, Liu, Li, & Shen, 2015) findings between both conditions. The influence of using biofeedback to guide the SPB practice should be investigated at different levels, for example to clarify whether it produces different psychological (e.g., self-efficacy, motivation) or physiological (e.g., HRV, blood pressure) outcomes.

Sixth, a thorough investigation of the effects of SPB on the whole autonomic nervous system would be of interest. If the parasympathetic nervous system is suggested to play the most important role in self-regulation, given the heart is under permanent inhibitory control from the vagus nerve and the parasympathetic nervous system is also the fastest to act on the heart (Jose & Collison, 1970; Saul, 1990), the role of the **sympathetic nervous system** should not be neglected. Contrary to what was thought for a long time, the sympathetic and the parasympathetic branches don't act oppositely, and it is also possible to see a co-activation, a co-inhibition, or a diverse functioning of the sympathetic nervous system in comparison to the parasympathetic one (Berntson et al., 1993). Therefore, self-regulation mechanisms may be

better explained when taking into account the sympathetic activity (Duschek et al., 2017; Michael, Graham, & Davis, 2017), which could be done using the gold standard of sympathetic influence on the heart, pre-ejection period (A. Sherwood et al., 1990).

Finally, the effects of SPB on both branches of the autonomic nervous system could also be investigated using **pharmacological blockades**. The sinus node has an intrinsic rate of depolarization, which corresponds to the intrinsic heart rate (Jose & Collison, 1970). The intrinsic heart rate is the heart rate measured in the absence of sympathetic and parasympathetic inputs to the sinus node, which can be achieved via pharmacological blockade (Lahiri, Kannankeril, & Goldberger, 2008). Different strategies can then be considered to investigate the effects of SPB on markers of the autonomic nervous system, using sympathetic blockade for example with beta-adrenergic blockers, or parasympathetic blockade with atropine (Chapleau & Sabharwal, 2011; Du Plooy & Venter, 1995; Lahiri et al., 2008). This was for example realized recently by Kromenacker et al. (2018), who found that during SPB the low-frequency HRV was mainly vagally-driven, however the breathing pattern was based on the SKY ujjayi method (four counts of inspiration, four counts of breath holding, six counts of expiration, and two counts of breath holding per respiration cycle) (Bertisch, Hamner, & Taylor, 2017), therefore further research is warranted with more classical SPB patterns, such as the one used in this PhD thesis.

To summarize, we outlined here some promising directions for future research to better understand the effects of SPB, and potentially promote its use to target a diversity of samples and outcomes.

## 12 Conclusion

This PhD thesis was aimed to investigate whether SPB without biofeedback influences CVA as well as its actions on several self-regulation processes, based on the neurovisceral integration model (Smith et al., 2017; Thayer et al., 2009) and on the resonance model (Lehrer & Gevirtz, 2014). Four studies were conducted, with adolescents with individual disabilities (Study 1) and healthy young adults (Study 2, 3, & 4). Both short-term and long-term SPB interventions have been investigated, and positive effects have been found on stress management (short-term, Study 1), sleep (long-term, Study 2), and executive functions (short-term, Studies 3 and 4), however results related to executive functions were not mediated by CVA. These results are encouraging regarding the use of SPB in acute and chronic interventions, as a “quick-fix” or daily routine, however future research has to clarify the underlying mechanisms, in particular with brain imaging methods. At the research level, this PhD thesis probably raised more additional questions than it answered previous ones, however the main applied take-home message appears encouraging: SPB is a free, non-invasive technique, with little side effects, which makes it an appropriate intervention to recommend to address a large range of self-regulation phenomena. Even if many challenges remain, I hope that this PhD thesis have helped to raise interest about this fascinating technique, and that you may be more aware of the power of voluntarily controlling your breath from now on. So let your breath be the start of your next adventure.

*“Very well”, said Bilbo.  
“It [the Ring] goes to Frodo with all the rest.”  
He drew a deep breath.  
“And now I really must be starting”*

**J.R.R. Tolkien – Opening scene of Lord of the Rings**

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## 14 Academic CV

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| 2013   | European PhD in Sport Sciences (German Sport University Cologne, Germany and University of Caen, France)   |
| 2010   | Master in Psychology (University of Caen, France)  |
| 2007   | Master in Sport Sciences (University of Caen, France)  |
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| 2014-today                                     | Lecturer and Researcher - Institute of Psychology (German Sport University Cologne)  |
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| 2012-2013                                      | Research assistant - DAAD grant – Institute of Psychology – German Sport University Cologne  |
| 2007-2012                                      | Research and teaching assistant (University of Caen, France)   |
| <b>Scientific experience</b>                   |  |
| Papers in international peer-reviewed journals | Papers in peer-reviewed international journals: 69 (35 as first author)<br><i>h</i> -index Web of Science: 18, <i>h</i> -index Google Scholar: 21<br><u>Additional activities:</u> 4 books, 13 book chapters, 6 symposia in international conferences, 32 oral presentations in international conferences, 19 posters in international conferences |
| Ad-hoc Reviewer                                | 98 international peer-reviewed scientific journals<br>Reviewer Award from Publons – Top 1% in Psychology in 2018   |
| Editorial Roles                                | <u>Associate editor for 3 peer-reviewed international journals:</u> Psychological Research; International Journal of Sport and Exercise Psychology; Sport Science and Health   |
| <b>Competitive fundings</b>                    |  |
| DFG-Costa Rica                                 | 2019 - Cortical & Autonomic Outcomes of Green Exercise, PI – 20 000 €  |
| Post-doc Grant                                 | 2019 – German Sport University, PI - 10 000€ (Slow paced breathing)  |
| BKK  | 2018 – Emotional Intelligence at school – PI – 100 000€  |

|                                 |   |
|---------------------------------|---|
| DAAD-Australia                  | 2018 – Influence of slow paced breathing on personality, PI – 5 000€  |
| French Rugby Assoc.             | 2017 – Rugby and emotional intelligence – Co-PI - 10 000€   |
| Post-doc Grant                  | 2017 – German Sport University, PI - 10 000€ (Effects of handtouch)   |
| Post-doc Grant                  | 2016 – German Sport University, PI - 10 000€ (Slow paced breathing)   |
| DAAD- Brazil                    | 2014-2015, Cultural differences intuition and creativity, Co-PI 20.000€   |
| DFG                             | 2014-2018, Joint Action (Vicon-gaze-mobile EEG), Co-PI, 334.000€  |
| Post-doc Grant                  | 2012-2013 Post-doc Grant DAAD, PI - 12 000€   |
| PhD Grant                       | 2007-2011 Region Normandy, France, PI - 100 000€  |
| <b>Awards</b>                   |   |
| Teaching Prize                  | 2019 – Seminar 2 <sup>nd</sup> prize – German Sport University Cologne  |
| Ma thèse en 180s (Science Slam) | 2019 - Region Normandie - 1st Prize Jury (+ Participation to the French semi-final and French national final)                           |
| DAAD Science Slam               | 2019 – German Embassy, Paris (France) – “Vagal tank theory: The love story between the heart and the brain” – Public Prize              |
| Young Investigator Award FEPSAC | 2011 – Young Investigator Award – (2 <sup>nd</sup> prize, oral) – European Association for Sport Psychology (FEPSAC); Madeira, Portugal |

### **Publications in international peer-reviewed journals**

Total = 69 (35 as first-author, 34 as co-author)

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### 15 A token of thanks - With or without you

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## *Synthèse de la thèse en français*

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### 16 Synthèse de la thèse en français

#### 16.1 Cadre théorique

##### 16.1.1 Introduction

Depuis des milliers d'années, les techniques de respiration représentent un aspect essentiel de la plupart des pratiques de méditation et de relaxation (Russo, Santarelli, & O'Rourke, 2017; Zaccaro et al., 2018). Les traditions chinoise (Xiuling, 2003) et hindoue (van Lysbeth & Cooper, 2007) reconnaissent que la respiration influence fortement le fonctionnement du cerveau et du corps. Dans la philosophie hindoue, par exemple, le mot sanskrit pour souffle "prana" signifie dans le même temps souffle et énergie, tandis que "pranayama", l'arrêt / le contrôle, mais aussi la montée et l'expansion du souffle, représente quand à lui un ensemble de techniques respiratoires visant à réguler consciemment un ou plusieurs paramètres de la respiration, tels que la fréquence, la profondeur ou le rapport inspiration / expiration (Zaccaro et al., 2018).

Les psychophysiolologistes modernes se sont appuyés sur ces techniques anciennes pour relever certains des principaux défis de notre époque, comme la gestion du stress et l'optimisation de la performance cognitive. Des patterns respiratoires spécifiques ont été identifiés pour optimiser l'adaptation à ces défis (Lehrer & Gevirtz, 2014; Van Diest et al., 2014; Vlemincx, Van Diest, & Van den Bergh, 2016; Zaccaro et al., 2018), et cette thèse de doctorat va se focaliser sur la respiration lente contrôlée (RLC). La RLC (basée sur Lehrer & Gevirtz, 2014; Lehrer, Vaschillo, & Vaschillo, 2000) fait référence au contrôle volontaire de la durée des phases d'inhalation et d'expiration. La respiration se fait à un rythme plus lent (environ 6 cycles par minute - cpm) que la respiration spontanée, qui se situe habituellement entre 12 et 20 cpm chez l'adulte (L. Sherwood, 2006).

Jusqu'à présent, un important courant de recherche s'est concentré sur les effets de la RLC combinés au biofeedback, où la personne pratiquant la technique respiratoire est équipée de capteurs connectés à des dispositifs montrant les effets physiologiques de la RLC en temps réel (Gevirtz, 2013; Goessl, Curtiss, & Hofmann, 2017; Kennedy & Parker, 2018; Wheat & Larkin, 2010; Yu, Funk, Hu, Wang, & Feijs, 2018; Zaccaro et al., 2018). Cependant, même si l'efficacité de ces interventions a été largement démontrée, le fait que leur mise en œuvre nécessite l'utilisation de technologies supplémentaires ne les rend pas directement accessibles

à tous. Mon but dans cette thèse de doctorat était d'étudier plus en détail les effets de la RLC sans l'utilisation d'outils biofeedback, rendant sa mise en œuvre accessible à une population élargie. En effet, les recherches préliminaires soutiennent que les effets psychophysiologiques seraient similaires avec ou sans utilisation d'outils biofeedback (Wells, Outhred, Heathers, Quintana, & Kemp, 2012). En se basant sur le modèle d'intégration neuroviscérale (Smith, Thayer, Khalsa, & Lane, 2017; Thayer, Hansen, Saus-Rose, & Johnsen, 2009) et sur le modèle de résonance (Lehrer & Gevirtz, 2014), cette thèse de doctorat vise donc à étudier les effets de la RLC sans biofeedback sur des phénomènes d'autorégulation liés à plusieurs aspects qui jouent un rôle important dans l'adaptation humaine: le stress, le sommeil et le fonctionnement exécutif.

Dans cette synthèse en français, j'éluderai la présentation des mécanismes sous-tendant la respiration que l'on retrouvera dans la version longue en anglais, pour débiter par la description du couplage entre les fonctions respiratoire et cardiorespiratoire, la façon dont la RLC est censée améliorer ce couplage et influencer positivement les processus d'autorégulation, et le rôle que le nerf vague est censé jouer dans cette optimisation. Après avoir énoncé la principale question de recherche sous-tendant cette thèse de doctorat et les hypothèses qui y sont associées, quatre études expérimentales seront présentées. Enfin, une discussion générale intégrera les résultats des quatre études expérimentales et présentera également les limites de ce travail ainsi que les pistes de recherche pour l'avenir.

### 16.1.2 À la croisée des chemins entre les systèmes respiratoire et cardiovasculaire

#### *16.1.2.1 Couplage entre le rythme cardiaque et la respiration : L'arythmie sinusale respiratoire*

Tout l'intérêt de la respiration comme variable d'intervention réside en son couplage avec le débit cardiaque, en ce sens que la fréquence cardiaque change en fonction du cycle respiratoire (Lorig, 2011). Ce phénomène est appelé arythmie sinusale respiratoire, le rythme cardiaque s'accéléral pendant l'inspiration et ralentissant pendant l'expiration (Angelone et Coulter, 1964; Eckberg et Eckberg, 1982). On suggère que l'arythmie sinusale respiratoire joue un rôle important dans la respiration, étant donné que des recherches antérieures ont suggéré que l'efficacité des échanges gazeux pulmonaires est améliorée par l'arythmie sinusale respiratoire (Giardino, Glenny, Borson et Chan, 2003; Hayano, Yasuma, Okada, Mukai et Fujinami, 1996; Mortola, Marghescu et Siegrist-Johnstone, 2016, 2018; Yasuma et Hayano, 2004). La répartition des battements cardiaques au sein des phases d'inspiration et d'expiration a été suggérée comme facteur d'amélioration de l'efficacité des échanges gazeux respiratoires. Plus précisément, on a émis l'hypothèse que la synchronisation de la ventilation alvéolaire avec

l'arythmie sinusale respiratoire dans chaque cycle respiratoire permettait d'économiser de l'énergie en supprimant les battements cardiaques inutiles pendant l'expiration et la ventilation inefficace pendant le reflux. En d'autres termes, la fréquence cardiaque tend à être plus élevée lorsque l'air dans les poumons est le plus riche en oxygène, l'expiration survenant lorsque le dioxyde de carbone est à son niveau le plus élevé. Cependant, cette hypothèse a été remise en question par d'autres recherches qui n'ont montré aucune relation entre l'arythmie sinusale respiratoire et l'amélioration des échanges gazeux (Buchheit, 2010; Sin et al., 2010; Tzeng, Sin, & Galletly, 2009). Une autre hypothèse, qui nécessite encore un examen empirique, est que l'arythmie sinusale respiratoire pourrait aider le cœur à faire moins de travail tout en maintenant des niveaux gazeux appropriés dans le sang (Ben-Tal, Shamailov, & Paton, 2012, 2014).

Il a été suggéré que les échanges gazeux au niveau des alvéoles fonctionnent mieux lorsque la fréquence cardiaque commence à augmenter au début de l'inspiration et à diminuer au début de l'expiration, avec une relation de phase de  $0^\circ$  (Hayano et al., 1996), qui est appelée le couplage cardiovasculaire (Elstad, O'Callaghan, Smith, Ben-Tal, & Ramchandra, 2018). Cependant, dans des situations normales, la relation entre la fréquence cardiaque et la respiration n'est pas complètement en phase. Il se pourrait que cela permette une plus grande flexibilité de l'organisme, de sorte qu'une plus grande efficacité peut être atteinte pendant les phases où les besoins métaboliques sont plus importants, cette efficacité n'étant pas autant nécessaire lors des phases où les besoins diminuent (Lehrer et Gevirtz, 2014). Il est possible que la RLC à 6 cpm déclenche une fréquence cardiaque oscillant avec la respiration avec une relation de phase de  $0^\circ$ , ce qui entraînerait par conséquent les échanges gazeux les plus efficaces (Lehrer & Gevirtz, 2014). Cependant certains travaux postérieurs ont fortement remis en question la validité de cette hypothèse (Sin, Webber, Galletly, & Tzeng, 2012). En résumé, les mécanismes par lesquels l'arythmie sinusale respiratoire constituerait un processus adaptatif doivent encore être compris.

L'arythmie sinusale respiratoire est principalement d'origine vagale (Berntson, Cacioppo, & Quigley, 1993; Grossman, van Beek, & Wientjes, 1990; Yasuma & Hayano, 2004). Elle serait régulée par des processus à la fois tonique et phasique du tonus vagal, qui ont des origines, des dynamiques et des conséquences fonctionnelles qui diffèrent (Daly, 1985; Grossman, Karemaker, & Wieling, 1991; Grossman et al, 1990; Richter & Spyer, 1990). En particulier, il a été suggéré que le nerf vague contribuerait à l'efficacité des échanges gazeux pulmonaires (Ito et al., 2006), ce qui est en lien avec son rôle dans les processus d'autorégulation. L'arythmie sinusale respiratoire est habituellement évaluée selon la méthode de la « valeur maximale » (Grossman et al., 1990; Stange, Hamilton, Fresco, & Alloy, 2017),

où la fréquence cardiaque maximale pendant la période d'expiration de la respiration est soustraite de la fréquence cardiaque minimale pendant la période d'inspiration de la respiration.

Selon le modèle de résonance (Lehrer & Gevirtz, 2014), l'arythmie sinusale respiratoire constitue l'un des principaux mécanismes visés par la RLC pour améliorer les processus d'autorégulation.

#### *16.1.2.2 La respiration lente contrôlée*

Comme nous l'avons déjà mentionné, la RLC est une technique respiratoire où les durées d'inhalation et d'expiration sont contrôlées et où la respiration est effectuée à un rythme plus lent (environ 6 cpm) que la respiration spontanée, qui est habituellement comprise entre 12 et 20 cpm chez les adultes (L. Sherwood, 2006). Dans ce manuscrit, le terme RLC sera préféré aux autres termes utilisés dans la littérature, comme "respiration profonde" (par ex. Tharion, Samuel, Rajalakshmi, Gnanasenthil, & Subramanian, 2012), "respiration abdominale" (par ex. Wang et al, 2010) ou "diaphragmatique" (par ex. Russell, Scott, Boggero, & Carlson, 2017), étant donné qu'elle reflète le fait qu'une respiration lente et rythmée est le déclencheur nécessaire pour influencer positivement les processus d'autorégulation, comme nous le détaillons à présent. Qualifier cette technique respiratoire de "profonde", "abdominale" et "diaphragmatique" n'est pas complètement inexact et peut refléter certains aspects de la RLC, mais ces éléments ne sont pas considérés comme les principaux déclencheurs des effets de la RLC : 1) "profonde" est supposé refléter le fait que les participants sont tenus de ralentir leur fréquence respiratoire, ce qui peut entraîner une augmentation du volume courant; cependant, si les participants commencent à avoir la tête qui tourne, ils doivent immédiatement arrêter ou adopter une respiration plus superficielle (Lehrer et al, 2000) ; 2) "abdominale" ou "diaphragmatique" reflète les caractéristiques de la respiration les plus efficaces en conditions de repos.

Le modèle de résonance (Lehrer et Gevirtz, 2014) suppose que quatre processus sont en jeu pour expliquer comment la RLC influence positivement les mécanismes d'autorégulation: 1) la relation de phase entre les oscillations de fréquence cardiaque et la respiration à 6 cpm; 2) la relation de phase entre la fréquence cardiaque et les oscillations de pression artérielle à 6 cpm; 3) l'activité du baroréflexe; et 4) les caractéristiques de résonance du système cardiovasculaire. Ces processus renforcent l'homéostasie du barorécepteur (Lehrer et al., 2006; Vaschillo, Lehrer, Rishe, & Konstantinov, 2002; Vaschillo, Vaschillo, & Lehrer, 2006), ce qui devrait entraîner une amélioration des échanges gazeux au niveau des alvéoles ainsi qu'une augmentation des afférences vagales (Lehrer & Gevirtz, 2014).

Toutefois, les données empiriques ont remis en question certains des mécanismes sous-jacents suggérés. En ce qui concerne le premier mécanisme, relatif à la relation de phase entre les oscillations du rythme cardiaque et la respiration à 6 cpm (couplage cardiovasculaire, Elstad et al., 2018), il est basé sur le rôle supposé de l'arythmie sinusale respiratoire dans l'optimisation des échanges gazeux (Yasuma & Hayano, 2004). Cependant, si le rythme cardiaque tend à se redistribuer vers l'inspiration à 6 cpm (Lopes, Beda, Granja, Jandre, & Giannella-Neto, 2011), l'hypothèse originale concernant le rôle physiologique de l'arythmie sinusale respiratoire dans l'adaptation des battements cardiaques au flux sanguin pulmonaire (Yasuma & Hayano, 2004) a été sérieusement remise en question (Buchheit, 2010 ; Sin et al., 2010 ; Tzeng et al., 2009). A ce jour, aucune réponse claire ne semble expliquer à ce stade la fonction physiologique de l'arythmie sinusale respiratoire en ce qui concerne les adaptations. Néanmoins, le fait que l'efficacité des échanges gazeux pulmonaires puisse être améliorée par la RLC a reçu un certain soutien en se basant sur la réduction des équivalents ventilatoires du dioxyde de carbone et de l'oxygène lors de la RLC à 6 cpm (Sin et al., 2010), même si la méthode des équivalents ventilatoires n'est pas considérée comme la référence en matière de mesure de l'efficacité des échanges gazeux pulmonaires (Tharion & Subramani, 2011; Tzeng, 2011).

Le baroréflexe est un réflexe médié par des capteurs de pression artérielle dans l'aorte et l'artère carotide qui aident à moduler les fluctuations de la pression artérielle (Eckberg et Sleight, 1992). Les barorécepteurs situés dans les parois de ces artères détectent l'étirement des artères à mesure que la pression artérielle augmente, et lorsque cela se produit, le baroréflexe provoque une diminution immédiate de la fréquence cardiaque. Au contraire, lorsque la pression artérielle chute, le baroréflexe provoque une augmentation immédiate de la fréquence cardiaque (Eckberg & Sleight, 1992; Lehrer & Gevirtz, 2014). Comme pour la relation de phase mentionnée pour la fréquence cardiaque et la respiration, il existe une relation de phase similaire pour la fréquence cardiaque et la pression artérielle, qui peut être représentée ainsi: lorsqu'une stimulation externe telle que la respiration à une fréquence spécifique provoque une augmentation de la fréquence cardiaque, elle provoque également une chute de la pression artérielle, ce qui déclenche un stimulus supplémentaire pour une augmentation additionnelle de la fréquence cardiaque. Lorsque la stimulation externe (par ex., la respiration) fait chuter la fréquence cardiaque, elle fait aussi monter la pression artérielle, ce qui entraîne une stimulation supplémentaire pour que la fréquence cardiaque diminue davantage. Étant donné que la relation de phase de  $0^\circ$  entre la fréquence cardiaque et la respiration se produit à peu près à la même fréquence que la stimulation externe cause une stimulation maximale du baroréflexe, la respiration apparaît alors comme un candidat naturel pour jouer le rôle de stimulateur externe

(Lehrer & Gevirtz, 2014). Conformément à cette suggestion, de fortes augmentations du gain du baroréflexe (variation en nombre de battements par minute de la fréquence cardiaque par changement de 1 mm Hg de variation de la pression artérielle) ont été observées.

Enfin, le dernier mécanisme suggéré par le modèle de résonance (Lehrer & Gevirtz, 2014) quant à la façon dont la RLC influence positivement les processus d'autorégulation se base sur les caractéristiques de résonance du système cardiovasculaire. La résonance est un principe physique selon lequel tous les systèmes de rétroaction oscillants avec un retard constant possèdent une caractéristique de résonance (Lehrer & Gevirtz, 2014). Voici un exemple pour mieux comprendre ce que signifie la résonance: imaginez une personne poussée sur une balançoire (Allen & Friedman, 2012): La poussée doit être en rythme avec l'élan de la personne assise sur la balançoire. La résonance entre le mouvement de balancement naturel et la force appliquée fait que le balancement va plus haut qu'avant. Il en va de même dans le système cardiovasculaire lorsque l'on respire à une fréquence proche de 6 cpm, où l'arythmie sinusale respiratoire se superpose aux oscillations inhérentes de la fréquence cardiaque liées à la modulation de la pression artérielle (Vaschillo et al., 2006). Le point auquel ces deux signaux se chevauchent a été appelé la fréquence de résonance, étant donné que la somme des deux signaux produit de grandes variations de la fréquence cardiaque (Allen et Friedman, 2012 ; Lehrer et Gevirtz, 2014; Vaschillo et al., 2006).

En résumé, le modèle de résonance (Lehrer & Gevirtz, 2014) a émis l'hypothèse que la RLC, par le biais de plusieurs processus décrits ci-dessus, influence positivement les mécanismes d'autorégulation. Le mécanisme le plus soutenu jusqu'à présent est l'homéostasie renforcée du barorécepteur (Lehrer et al., 2003; Vaschillo et al., 2002 ; Vaschillo et al., 2006). Le deuxième mécanisme qu'est l'optimisation des échanges gazeux pulmonaires basée sur les caractéristiques de l'arythmie sinusale respiratoire à 6 cpm n'a pu être pleinement soutenu jusqu'ici (Buchheit, 2010 ; Sin et al., 2010 ; Tzeng et al., 2009). Le troisième mécanisme suggéré était une augmentation des afférences vagales, mais cette suggestion était jusqu'à présent largement basée sur la spéculation selon laquelle les résultats positifs obtenus avec les interventions RLC avec biofeedback étaient liés aux zones du cerveau associées aux afférences vagales (Lehrer & Gevirtz, 2014). Cette thèse de doctorat se concentre sur la possibilité que cette action suggérée de la RLC sur les afférences vagales se refléterait dans les afférences vagales et influencerait donc positivement les mécanismes d'autorégulation, étant donné les liens décrits dans la section suivante.

### 16.1.3 Comment la respiration lente contrôlée peut-elle influencer l'autorégulation : le rôle du nerf vague

#### *16.1.3.1 Le rôle du nerf vague dans l'autorégulation*

Le nerf vague est le dixième nerf crânien, et c'est le nerf le plus important du système nerveux parasympathique (Brodal, 2010). Il est composé à 80% de fibres sensorielles afférentes (envoyant des signaux du corps au cerveau) et de 20% de fibres motrices efférentes (transportant l'information du cerveau au corps). Toutes les branches du nerf vague avec des fibres motrices efférentes contiennent également des fibres sensorielles afférentes, ce qui en fait un nerf très sensible (Howland, 2014). Comme son nom l'indique (la traduction latine de *vagus* signifiant « *vagabond* »), le nerf vague se ramifie vers des régions étendues du corps (Brodal, 2010), ses fibres innervant la plupart des organes du corps, y compris les systèmes gastro-intestinal et cardiovasculaire (Bonaz, Bazin et Pellissier, 2018; Brodal, 2010; H. Y. Chang, Mashimo et Goyal, 2003). Les fibres vagues libèrent de l'acétylcholine comme neurotransmetteur (Brodal, 2010). En résumé, grâce à son vaste réseau, le nerf vague permet des communications rapides et étendues à l'intérieur du corps.

#### *16.1.3.2 Activité cardiaque vagale : le produit du réseau central autonome*

En ce qui concerne les fibres efférentes vagues, celles qui stimulent l'action motrice, un intérêt particulier sera porté ici à celles qui innervent le cœur et modulent son activité intrinsèque par le nœud sinusal, qui détermine la fréquence cardiaque. Il est important de noter que le système nerveux sympathique aura une influence trop lente pour produire des changements sur les battements cardiaques (Jose & Collison, 1970), le cœur sera donc principalement sous influence inhibitrice parasympathique grâce aux fibres efférentes vagues (Jose & Collison, 1970; Saul, 1990). Cet équilibre cardiaque entre les deux branches du système nerveux autonome est un moyen pour l'organisme de favoriser la conservation de l'énergie.

Quant aux fibres afférentes vagues (liées aux actions sensorielles), elles sont largement dispersées dans les organes clés du corps humain (Brodal, 2010). Cela confère au système vagal afférent un rôle important d'adaptation en tant que détecteur d'événements liés au système immunitaire dans le corps humain. Cette sensibilité périphérique permet d'obtenir un signal interne qui peut générer les réponses autonomes, endocriniennes et comportementales appropriées par l'intermédiaire de voies réflexes centrales traversant le noyau du tractus solitaire (Berthoud & Neuhuber, 2000). Ces signaux internes sont ensuite intégrés aux signaux externes, ce qui aide à façonner la réponse appropriée. Il est important de noter que la RLC peut jouer un rôle dans le déclenchement des signaux internes, étant donné qu'elle est supposée stimuler les afférences vagues (Lehrer & Gevirtz, 2014).



Un réseau fonctionnel basé sur des structures cérébrales spécifiques est supposé faciliter l'organisation et la régulation de l'activité afférente et efférente vagale (Berthoud & Neuhuber, 2000). Le réseau autonome central (Benarroch, 1993) représente une unité fonctionnelle du système nerveux central qui soutient le comportement, l'adaptabilité et les processus d'autorégulation globaux axés sur l'atteinte de buts. L'activité vagale cardiaque, l'activité du nerf vague régulant le fonctionnement cardiaque, est supposée être le produit de ce réseau autonome central, et se trouve au cœur du modèle d'intégration neuroviscérale (Smith et al., 2017; Thayer et al., 2009).

#### *16.1.3.3 Le modèle d'intégration neuroviscérale*

Le modèle d'intégration neuroviscérale (Smith et al., 2017; Thayer et al., 2009) décrit une connexion entre le cortex préfrontal et le cœur via le réseau autonome central et le nerf vague.

Le modèle d'intégration neuroviscérale postule que l'activité vagale cardiaque sert d'indicateur de l'efficacité du réseau central autonome et, par conséquent, sert de base pour indexer les phénomènes qui sous-tendent l'autorégulation, l'adaptation et la santé (Smith et al., 2017 ; Thayer et al., 2009). Au repos, le cortex préfrontal médial exerce un contrôle inhibiteur sur l'amygdale, améliorant indirectement le contrôle cardiaque par le nerf vague, ce qui se traduit par une augmentation de l'activité vagale cardiaque (Thayer, Ahs, Fredrikson, Sollers, & Wager, 2012). Ce contrôle inhibiteur constant de l'amygdale favoriserait le lien entre l'activité vagale cardiaque et la régulation des émotions. La relation entre l'activité vagale cardiaque et le fonctionnement exécutif provient des structures et réseaux communs impliqués dans la régulation cardiaque et cognitive (Thayer et al., 2012). L'efficacité du fonctionnement exécutif dans le cortex préfrontal est soutenue par l'activation optimale des réseaux neuronaux, soulignée par un flux d'activité le long des voies neuronales permettant d'établir des correspondances adéquates entre les signaux afférents, les états internes et les signaux efférents nécessaires pour exécuter une tâche donnée (Miller & Cohen, 2001). Cela conduit à des réponses flexibles à un environnement qui évolue constamment (Thayer et al., 2009).

#### *16.1.3.4 Évaluation non invasive de l'activité vagale cardiaque avec la variabilité de la fréquence cardiaque*

Le modèle d'intégration neuroviscérale (Thayer et al., 2009) postule qu'il est possible d'indexer de manière non invasive le produit du réseau central autonome, l'activité vagale cardiaque, via la variabilité de la fréquence cardiaque. La variabilité de la fréquence cardiaque représente la variation temporelle entre chaque pic de R dans les complexes QRS (Berntson et al., 1997; Laborde, Mosley, & Thayer, 2017; Malik, 1996). Deux paramètres principaux de la

variabilité de la fréquence cardiaque reflètent l'activité vagale cardiaque: la moyenne de la racine carrée des différences successives (RMSSD) et les hautes-fréquences de la variabilité de la fréquence cardiaque (HF-HRV) (Berntson et al., 1997; Laborde, Mosley, et al., 2017; Malik, 1996). Les recherches ont montré que la RLC augmentait l'activité vagale cardiaque à court terme (Kromenacker, Sanova, Marcus, Allen, & Lane, 2018; Lewis et al, 2015; Szulczewski & Rynkiewicz, 2018; Wells et al, 2012).

#### 16.1.4 Les 3Rs du fonctionnement du nerf vague

Les aspects toniques et phasiques du fonctionnement de l'activité vagale cardiaque sont importants à considérer (Thayer et al., 2012). L'activité vagale cardiaque tonique a également été appelée activité vagale cardiaque de repos, c'est lorsque l'activité vagale cardiaque est prise en compte à un moment donné où la personne n'est pas confrontée à une tâche/événement spécifique. L'activité vagale cardiaque phasique illustre la réaction du système, elle a été qualifiée de réactivité lorsqu'il s'agit du changement survenant entre la mesure de repos et un événement, et de récupération lorsqu'il s'agit du changement entre l'événement et le retour à l'état de repos (Laborde, Mosley, & Mertgen, 2018b; Laborde, Mosley, et al., 2017). Ces trois aspects que sont les 3R (Repos, Réactivité, Récupération) sont très importants à considérer du point de vue de l'adaptation. En ce qui concerne l'activité vagale cardiaque au repos, le modèle d'intégration neuroviscérale (Thayer et al., 2009) fait l'hypothèse générale qu'un niveau plus élevé d'activité cardiaque vagale au repos est associé positivement à l'efficacité d'autorégulation. En ce qui concerne la réactivité, deux situations doivent être différenciées (Beauchaine, 2001; Laborde, Mosley, & Mertgen, 2018b; Porges, 2007b; Thayer et al., 2012; Thayer et al., 2009) : si la tâche est principalement cognitive, alors une moindre diminution, voire une augmentation de l'activité vagale cardiaque serait perçue comme favorisant l'adaptation; cependant si les demandes métaboliques augmentent, une diminution plus importante de l'activité vagale cardiaque serait alors considérée comme adaptative afin de fournir l'énergie requise au corps. Enfin, en ce qui concerne la récupération, un retour plus rapide à l'activité de repos reflète une meilleure adaptation (Stanley, Peake et Buchheit, 2013). La prise en compte des 3R pour comprendre le fonctionnement de l'activité vagale cardiaque sera particulièrement importante pour en déduire les hypothèses respectives.

En résumé, dans les sections précédentes, nous avons donné un aperçu de la façon dont la RLC peut être reliée aux mécanismes d'autorégulation. Selon le modèle de résonance (Lehrer & Gevirtz, 2014), si le mécanisme qui a reçu le plus de soutien jusqu'à présent est le renforcement de l'homéostasie des barorécepteurs, on suggère aussi que l'augmentation des afférences vagales joue un rôle. Cette augmentation des afférences vagales peut envoyer des

signaux d'entrée dans le réseau central autonome (Benarroch, 1993), et selon le modèle d'intégration neuroviscérale (Thayer et al., 2009), pourrait donc influencer positivement les phénomènes d'autorégulation. Dans le cadre de cette thèse de doctorat, je vais étudier la relation entre la RLC et une série de phénomènes d'autorégulation, incluant le stress, le sommeil et le fonctionnement exécutif.

16.1.5 Phénomènes d'autorégulation étudiés avec la respiration lente contrôlée et l'activité vagale cardiaque dans cette thèse de doctorat

16.1.5.1 *Stress*

16.1.5.1.1 *Stress psychologique*

Le stress psychologique se produit lorsqu'un individu perçoit que les demandes personnelles ou environnementales sollicitent de manière importante ses capacités d'adaptation ou bien les dépassent (Lazarus & Folkman, 1984). Même si le stress psychologique peut être considéré comme un phénomène idiosyncratique, étant donné qu'il varie selon les individus et les situations, une méthode courante pour provoquer le stress consiste à utiliser des tâches qui mettent les fonctions exécutives à l'épreuve. De plus, mettre l'accent sur la performance aide à représenter une situation qui diffère nettement d'un état de repos en ce qui concerne les exigences psychologiques imposées à l'individu. Cette définition du stress peut être liée à l'activité vagale cardiaque, en ce sens qu'une évaluation en terme de menace est essentielle à une diminution de l'activité vagale cardiaque, par opposition à une évaluation en terme de sécurité (Thayer et al., 2009). L'influence du stress mental sur l'activité vagale cardiaque sera individualisée selon le processus d'évaluation de l'individu (Lazarus & Folkman, 1984), et dépendra notamment du degré d'évaluation de la menace existante (Thayer et al., 2009). Un autre type de stress peut être déclenché par des facteurs de stress physiques.

16.1.5.1.2 *Stress physique*

Pour ce qui est de la réactivité de l'activité vagale cardiaque, les facteurs de stress physiques entraînent une diminution de l'activité vagale cardiaque afin que l'organisme puisse répondre aux exigences physiques de la tâche (Nakamura, Yamamoto, & Muraoka, 1993; Stanley et al., 2013). Cela reflète le rôle évolutif de l'activité vagale cardiaque en tant que mécanisme « d'appel aux armes » pour permettre la réponse de combat-fuite (Porges, 2007a, 2007b; Thayer, 2009). Cette réponse de combat-fuite est associée à une diminution quasi-totale de l'activité vagale cardiaque (Beauchaine, Gatzke-Kopp, & Mead, 2007), afin de faciliter une augmentation importante du débit cardiaque par le système nerveux sympathique, qui n'est alors plus confronté à l'inhibition vagale. Le niveau de diminution de l'activité vagale cardiaque quand la personne fait face à un facteur de stress physique dépendra principalement de l'intensité du facteur de stress physique plutôt que de sa durée (Stanley et al., 2013). Enfin, les

niveaux de condition physique initiaux de la personne influenceront à la fois l'amplitude et la cinétique de la récupération vagale, les individus ayant une meilleure condition physique au niveau de leur capacité aérobie récupérant plus rapidement (Stanley et al., 2013).

#### 16.1.5.1.3 Stress et respiration lente contrôlée

Les données actuelles soutiennent fortement l'effet de réduction du stress de la RLC. Comme l'a montré une méta-analyse récente (Goessl et al., 2017), il a été constaté que la RLC associée à des outils biofeedback réduisait largement le stress et l'anxiété auto-rapportés, tandis qu'aucun modérateur (par ex. le nombre de séances, la présence d'un trouble anxieux) n'a eu d'incidence significative sur les résultats. Ceci confirme les résultats d'études systématiques et narratives sur les effets de la RLC associés au biofeedback sur les marqueurs de stress au niveau psychophysiologique (Gevirtz, 2013; Kennedy & Parker, 2018; Wheat & Larkin, 2010; Yu et al, 2018; Zaccaro et al, 2018). Il est intéressant de noter que même si la RLC a été utilisée dans de nombreux contextes pour réduire le stress, il reste à l'étudier dans le domaine du handicap mental.

#### 16.1.5.1.4 Stress et handicap mental

Le fait d'avoir un handicap mental est généralement associé à des niveaux élevés de stress (Forte, Jahoda et Dagnan, 2011). Cette association s'explique en partie par des difficultés supplémentaires d'adaptation à des situations quotidiennes trop exigeantes, ainsi que par la marginalisation sociale (de Bildt, Sytema, Kraijer, Sparrow, & Minderaa, 2005). De plus, les personnes ayant un handicap mental utilisent souvent des stratégies d'adaptation inadéquates pour faire face au stress (Hartley & Maclean, 2008). Le stress vécu par les personnes ayant un handicap mental est souvent transféré aux membres de la famille et aux aidants (Hassall et Rose, 2005). A long terme, si le stress chronique chez les personnes ayant un handicap mental n'est pas traité adéquatement, il peut entraîner de graves conséquences, notamment la dépression (Hartley et Maclean, 2009), une altération des fonctions cognitives (Heyman et Hauser-Cram, 2015), des problèmes de santé physique (Lunsky, 2008) et des conduites à risque comme la toxicomanie (Didden, Embregts, van der Toorn, et Laarhoven, 2009). Ce stress chronique peut aussi mener à la dépression chez les membres de la famille et les aidants (Mutkins, Brown et Thorsteinsson, 2011), ainsi qu'à l'épuisement professionnel (Innstrand, Espnes et Mykletun, 2002). Étant donné la prévalence du stress chez les personnes ayant un handicap mental et l'absence de programmes de gestion du stress mettant en œuvre la RLC chez cette population, la première étude examinera si la RLC peut aider les personnes ayant un handicap mental à gérer leur stress.

Le deuxième phénomène d'autorégulation étudié dans ce travail sera le sommeil.

#### 16.1.5.2 Sommeil

Les problèmes de sommeil constituent une préoccupation très importante pour les individus, étant donné qu'ils ont un impact direct sur la qualité de vie et représentent un facteur de risque à plusieurs niveaux (Ferrie, Kumari, Salo, Singh-Manoux, & Kivimaki, 2011). L'une des principales hypothèses concernant les causes des troubles du sommeil est qu'ils peuvent être associés à un état d'hyper-activation (Bonnet, Burton, & Arand, 2014; Riemann et al., 2010). Les méthodes visant à diminuer un état d'hyper-activation visent généralement à mettre en jeu le système nerveux parasympathique, et plus particulièrement son nerf principal, le nerf vague (Friedman, 2007; Prendiville, 2016). Une façon de le faire est d'utiliser la RLC (Gerritsen & Band, 2018; Lehrer, 2018; Lehrer & Gevirtz, 2014 ; Zaccaro et al., 2018).

##### 16.1.5.2.1 Sommeil et activité vagale cardiaque

La question de savoir si la mesure de l'activité vagale cardiaque pendant le sommeil (activité vagale cardiaque nocturne) représente un indicateur de la qualité du sommeil est encore débattue. Certaines données indiquent une association entre une baisse de l'activité vagale cardiaque nocturne et les troubles du sommeil (Stein et Pu, 2012), comme avec la fatigue chronique (Burton, Rahman, Kadota, Lloyd, & Vollmer-Conna, 2010) et l'insomnie (Yang et al., 2011). Une activité vagale cardiaque nocturne plus élevée a également été associée à une meilleure qualité subjective du sommeil (Brosschot, Van Dijk et Thayer, 2007; Patel et coll., 2013; Yang et coll., 2011). Cependant, certains auteurs soutiennent que la mesure de l'activité vagale cardiaque nocturne globale ne fournit pas d'informations utiles, étant donné les variations observées pendant les différents stades du sommeil (Werner et al., 2015), à savoir la diminution de l'activité vagale cardiaque pendant le sommeil paradoxal et l'augmentation de l'activité vagale cardiaque pendant le sommeil lent (Tobaldini et al., 2013). De plus, Werner et ses collègues (2015) soutiennent que la signification de la mesure de l'activité vagale cardiaque est questionnable, étant donné que l'activité vagale cardiaque est censée refléter les adaptations aux changements environnementaux et que ceux-ci ne surviennent (presque) pas pendant la nuit. Ils recommandent par conséquent d'évaluer plutôt l'activité vagale cardiaque pendant les périodes de veille. En résumé, même si la mesure de l'activité vagale cardiaque pendant la nuit ne peut pas être considérée comme un indice de la qualité du sommeil, elle peut quand même fournir une indication de l'état de récupération du corps pendant le sommeil, étant donné qu'elle indexe l'activité du système nerveux parasympathique (Laborde, Mosley et al, 2017; Malik, 1996; Sakakakibara, Hayano, Oikawa, Katsamanis, & Lehrer, 2013; L. Sherwood, 2006).

Afin de répondre aux critiques formulées à l'égard des mesures nocturnes de l'activité vagale cardiaque, les auteurs ont suggéré de mesurer l'activité vagale cardiaque pendant la période d'éveil (Werner et al., 2015). En particulier, la période qui suit directement le réveil a

été suggérée comme étant un bon compromis, étant donné que l'influence de facteurs environnementaux sur l'activité vagale cardiaque peut être considérée à ce moment-là comme négligeable (Buchheit, Simon, Piquard, Ehrhart, & Brandenberger, 2004). L'activité vagale cardiaque au réveil a déjà été associée à des indices subjectifs de bien-être et à des adaptations liées à l'entraînement physique (voir par exemple Buchheit et al., 2006; Buchheit et al., 2004), ainsi qu'à des mesures subjectives de la qualité du sommeil (Flatt, Esco, & Nakamura, 2018).

L'étude de l'activité vagale cardiaque au réveil ainsi que les mesures nocturnes de l'activité vagale cardiaque apparaît donc comme une stratégie appropriée pour mieux comprendre les effets de la RLC sur l'activité vagale cardiaque.

#### 16.1.5.2.2 Sommeil et respiration lente

Deux études ont examiné les effets de la RLC sur la variabilité de la fréquence cardiaque pendant le sommeil (Sakakibara et al., 2013; Tsai, Kuo, Lee, & Yang, 2015). Dans la première étude (Sakakibara et al., 2013), la RLC a été administrée à l'aide d'un dispositif de biofeedback durant 20 minutes au cours de deux nuits consécutives. Par rapport à un groupe contrôle effectuant un entraînement basé sur le training autogène de Schulz, l'activité vagale cardiaque était plus élevée pendant les deux nuits mesurées, ce qui confirme l'idée que la RLC améliore le fonctionnement cardiovasculaire. La deuxième étude (Tsai et al., 2015), axée sur les personnes souffrant d'insomnie, visait à déterminer si une séance de RLC de 20 minutes (6 cpm), comparativement à une condition contrôle où la respiration était fixée à 12 cpm, améliorerait la qualité objective du sommeil évaluée par polysomnographie, ainsi que l'activité vagale cardiaque. Dans la condition RLC à 6 cpm, les phases d'inspiration et d'expiration étaient réglées sur 3s et 7s, alors qu'aucune indication n'était mentionnée concernant les phases d'inspiration et d'expiration pour la condition contrôle à 12 cpm. En ce qui concerne la polysomnographie, les résultats ont montré qu'après une seule séance de RLC à 6 cpm avant de s'endormir, la latence à l'endormissement, le nombre de réveils, et le temps éveillé pendant le sommeil ont diminué, tandis que l'efficacité du sommeil a augmenté, par rapport à la condition contrôle et à l'état initial. En ce qui concerne l'activité vagale cardiaque, les paramètres de variabilité de la fréquence cardiaque mentionnés dans le document (puissance totale et intervalles R-R) ne permettent pas d'obtenir d'information sur son évolution (Berntson et al., 1997; Laborde, Mosley et al., 2017; Malik, 1996; Shaffer & Ginsberg, 2017; Shaffer et al., 2014), il est donc impossible de tirer des conclusions concernant l'activité vagale cardiaque. De plus, la variabilité de la fréquence cardiaque n'a pas été évaluée pendant le sommeil, mais pendant la journée. Par conséquent, la réalisation d'autres études s'impose pour mieux comprendre les effets de la RLC sur la qualité subjective du sommeil et sur l'activité vagale

cardiaque, non seulement sur une seule séance à court terme, mais aussi sur une intervention à long terme.

La deuxième étude de cette thèse de doctorat portera sur une intervention à long terme (30 jours) visant à améliorer l'activité vagale cardiaque nocturne et au réveil.

### *16.1.5.3 Les fonctions exécutives*

Un autre domaine d'autorégulation qui peut être indexé par l'activité vagale cardiaque proposé par le modèle d'intégration neuroviscérale est la régulation du fonctionnement des fonctions exécutives, basée sur les réseaux communs responsables du contrôle cardiaque et des fonctions exécutives (Thayer et al., 2012; Thayer et al., 2009). Les fonctions exécutives soutiennent les comportements axés sur l'atteinte d'objectifs et sont essentielles au contrôle de soi (Diamond, 2013; Kotabe & Hofmann, 2015; Miyake et al., 2000). Les trois principales fonctions exécutives sont l'inhibition, la mémoire de travail et la flexibilité cognitive (Diamond, 2013; Miyake & Friedman, 2012; Miyake et al., 2000).

#### *16.1.5.3.1 Inhibition et activité vagale cardiaque*

L'inhibition reflète la capacité à contrôler l'attention, le comportement, les pensées et/ou les émotions pour contrôler une forte impulsion, afin de faire ce qui est le plus approprié en fonction du contexte (Diamond, 2013). Un test classique d'inhibition est la tâche de Stroop (Stroop, 1935), où l'on demande aux participants de lire la couleur avec laquelle un mot est présenté tout en ignorant la signification de ce mot. Pour les stimuli congruents, la couleur correspond à la signification du mot (par exemple, le mot "bleu" écrit avec la couleur bleue), tandis que pour les stimuli incongruents, la couleur diffère de la signification du mot (par exemple, le mot "bleu" écrit avec la couleur rouge). Les stimuli incongruents exigent des participants qu'ils inhibent la réponse dominante à la lecture d'un mot. La vitesse et la précision des réactions peuvent être mesurées. Cependant, l'inhibition se traduit principalement par la précision (le taux d'erreur) (McDowd, Oseas-Kreger et Fillion, 1995), puisqu'elle reflète la capacité à maintenir et accéder temporairement à l'objectif de la tâche (Kane et Engle, 2003).

Les recherches ont montré que la performance sur la tâche de Stroop est liée à l'activité vagale cardiaque. Une relation négative a été observée entre l'activité vagale cardiaque au repos et les temps de réaction sur les stimuli incongruents ou bien les mots reflétant une menace (Johnsen et al., 2003), tandis qu'une relation positive a été observée entre l'activité vagale cardiaque au repos et la précision des réactions (Stroop, Albinet, Abou-Dest, Andre, & Audiffren, 2016). Ces deux résultats sont conformes au modèle d'intégration neuroviscérale (Thayer et al., 2009). Une étude a observé des résultats divergents entre l'activité vagale



cardiaque au repos (évalué avec les hautes-fréquences de la variabilité de la fréquence cardiaque) et la précision des réactions avec la tâche de Stroop (c.-à-d. le score d'interférence Stroop) (Subramanya et Telles, 2015). Cependant, la manipulation expérimentale (méditation) qui a eu lieu avant la mesure de repos pourrait avoir introduit certains effets confondants concernant l'interprétation physiologique des hautes-fréquences de la variabilité de la fréquence cardiaque, étant donné que ce paramètre est censé refléter l'activité vagale cardiaque seulement lorsque la fréquence respiratoire est comprise entre 9 et 24 cpm (Berntson et al., 1997; Malik, 1996). Comme la fréquence respiratoire n'a pas été évaluée dans cette étude, il n'est pas possible de tirer des conclusions concernant l'activité vagale cardiaque. L'étude menée dans le cadre de ce travail doctoral portera sur la vitesse et la précision des réactions à la tâche de Stroop, et l'analyse de l'activité vagale cardiaque sera accompagnée d'une mesure de la fréquence respiratoire.

#### 16.1.5.3.2 Mémoire de travail et activité vagale cardiaque

La mémoire de travail implique de travailler avec de l'information qui n'est plus directement perceptible (Baddeley & Hitch, 1994). Autrement dit, la mémoire de travail implique de garder l'information à l'esprit et de travailler mentalement avec elle (Diamond, 2013). Un test classique pour évaluer la capacité de la mémoire de travail est la tâche « Automated Operation Span » (AOSPAN, Unsworth, Heitz, Schrock, & Engle, 2005). La tâche de l'AOSPAN demande aux participants de résoudre des problèmes mathématiques tout en gardant en mémoire un certain nombre de lettres n'ayant pas de rapport entre elles.

Les recherches précédentes ont trouvé une relation positive entre l'activité vagale cardiaque au repos et la performance avec l'AOSPAN (Laborde, Furley et Schempp, 2015) et une relation négative entre l'activité vagale cardiaque durant la tâche (c.-à-d. l'activité vagale cardiaque mesurée pendant l'AOSPAN) et la performance avec l'AOSPAN lorsque la tâche est réalisée sous pression (Mosley, Laborde et Kavanagh, 2018), ce qui pourrait refléter l'adaptation de l'activité vagale cardiaque aux demandes de la tâche (Mosley et coll., 2018). Des relations positives entre l'activité vagale cardiaque au repos et d'autres tâches qui reflètent la mémoire de travail ont également été rapportées dans la littérature (Hansen, Johnsen, Sollers, Stenvik, & Thayer, 2004; Hansen, Johnsen, & Thayer, 2003; Hansen, Johnsen, & Thayer, 2009; Morandi et al, 2019; Pu, Schmeichel, & Demaree, 2010; Sebastiani, Di Gruttola, Incognito, Menardo, & Santarcangelo, 2019).

#### 16.1.5.3.3 Flexibilité cognitive et activité vagale cardiaque

La flexibilité cognitive repose sur l'inhibition et la mémoire de travail (Davidson, Amso, Anderson et Diamond, 2006; Diamond, 2013). La flexibilité cognitive implique la capacité de



changer de perspective, en particulier dans l'espace ou entre individus (Diamond, 2013). Pour changer de perspective, il faut inhiber une perspective précédente et "charger" une nouvelle perspective dans la mémoire de travail. Une façon classique d'étudier la flexibilité cognitive consiste à utiliser la tâche du « tri de cartes de Wisconsin » (WCST, Milner, 1982 ; Stuss et al., 2000). Dans ce test, chaque carte peut être triée par couleur, forme ou nombre. Les participants doivent déduire le critère de tri correct sur la base du feedback qu'ils reçoivent et s'adapter à la nouvelle règle de tri dès qu'ils reçoivent un feedback indiquant que la règle de tri a changé. Les recherches précédentes ont démontré une relation positive entre l'activité vagale cardiaque au repos et la performance sur la tâche de WCST (c.-à-d. une relation négative avec les erreurs de décision; Albinet, Boucard, Bouquet et Audiffren, 2010; Hovland et al, 2012; Mathewson, Jetha, Goldberg, & Schmidt, 2012), et ceci est similaire à ce qui a été trouvé avec d'autres tâches de flexibilité cognitive (Alba, Vila, Rey, Montoya, & Munoz, 2019; Colzato, Jongkees, de Wit, van der Molen, & Steenbergen, 2018).

#### 16.1.5.3.4 Fonctions exécutives et respiration lente contrôlée

L'influence de la RLC sur le fonctionnement exécutif n'a reçu jusqu'alors que peu d'attention de la part des chercheurs. L'accent étant mis jusqu'à présent sur l'inhibition et la mémoire de travail d'une part (Prinsloo et al., 2011), et sur la prise de décision d'autre part (De Couck et al., 2019). La prise de décision n'appartient pas aux fonctions exécutives de base, mais est considérée comme une fonction exécutive de niveau supérieur qui repose sur les fonctions exécutives de base (Diamond, 2013). Prinsloo et ses collaborateurs (2011) ont utilisé un test Stroop modifié, où la composante d'inhibition est combinée à une composante de mémoire de travail, et on a demandé aux participants de se rappeler combien de carrés blancs étaient apparus à l'écran. Les participants ont été affectés soit à une condition de RLC, soit à une condition contrôle (où ils devaient respirer spontanément), pour un total de 10 minutes dans chaque condition. La RLC a été réalisée avec biofeedback, ce qui signifie que les participants voyaient les effets de la RLC sur la variabilité de la fréquence cardiaque via un appareil dédié. Aucune différence n'a été observée entre les conditions de la composante d'inhibition Stroop (c.-à-d. le nombre d'erreurs), mais les participants de la condition RLC ont eu une meilleure performance que le groupe témoin sur la composante de la mémoire de travail. Cette étude comportait certaines limites, comme une taille réduite de l'échantillon (n = 18 pour un protocole intra-sujet), une évaluation combinée de l'inhibition et de la mémoire de travail, et une absence de prise en considération de marqueurs de l'activité vagale cardiaque.

Dans l'étude de De Couck et al (2019), un test à choix multiples a été utilisé pour étudier la prise de décision. Le test comprenait sept questions liées à un scénario décisionnel dans un

contexte de gestion. Plusieurs réponses possibles ont été fournies, mais une seule d'entre elles reflétait la façon la plus efficace avec laquelle un manager devrait agir. Les participants ont été divisés en deux conditions expérimentales: un groupe effectuant une RLC pendant deux minutes et un groupe respirant spontanément pendant la même période (groupe contrôle). Le groupe RLC s'est montré plus performant que le groupe contrôle au niveau de la prise de décision. Cette étude comportait également certaines limites, notamment l'absence de biomarqueurs de l'activité vagale cardiaque, et la respiration n'était pas strictement régulée (les participants devaient compter mentalement la durée de l'inspiration et de l'expiration). Dans l'ensemble, la recherche sur les effets de la RLC sur les trois fonctions exécutives de base est limitée, les études disponibles étant caractérisées par d'importantes lacunes méthodologiques, et elles n'ont pas été fondées sur un cadre théorique solide.

Après avoir passé en revue les recherches concernant la RLC, l'activité vagale cardiaque, et les phénomènes d'autorégulation qui seront abordés au cours de cette thèse de doctorat (gestion du stress, sommeil, fonctions exécutives), les principales questions de recherche et les hypothèses associées sont maintenant présentées.

## 16.2 Questions de recherche et hypothèses

En résumé, si les interventions de RLC avec biofeedback ont reçu beaucoup d'attention jusqu'à présent (Gevirtz, 2013; Goessl et al, 2017; Kennedy & Parker, 2018; Wheat & Larkin, 2010; Yu et al, 2018; Zaccaro et al, 2018), ce n'est pas le cas des interventions sans biofeedback, et de nouvelles recherches sont donc nécessaires concernant ce mode de pratique de la RLC. Plus spécifiquement, cette thèse de doctorat vise à comprendre les mécanismes sous-jacents de la RLC concernant son action sur les phénomènes d'autorégulation, et à tester spécifiquement si la RLC influence l'activité vagale cardiaque, étant donné que l'un des mécanismes d'action suggérés de la RLC est sa capacité à stimuler les afférences vagales, comme le propose le modèle de résonance (Lehrer et Gevirtz, 2014).

Par conséquent, la principale hypothèse de travail est que la RLC, étant donné son action suggérée sur les afférences vagales, déclencherait une augmentation de l'activité vagale cardiaque (activité vagale efférente au niveau du cœur). Selon le modèle d'intégration neuroviscérale (Smith et al., 2017 ; Thayer et al., 2009), l'activité vagale cardiaque reflète l'efficacité du réseau autonome central, et donc l'efficacité globale de l'autorégulation. Par conséquent, il est fait l'hypothèse que la RLC influence positivement les mécanismes d'autorégulation par son action sur le réseau autonome central, et que ses effets se reflètent dans l'activité vagale cardiaque. Dans le cadre de cette thèse de doctorat, je vais étudier cette question de recherche et tester cette hypothèse principale dans quatre contextes:

- Dans l'étude 1, j'examine les effets de la RLC à court terme chez un échantillon d'adolescents ayant un handicap mental. Cela permettra de clarifier si la RLC peut être mise en œuvre dans ce contexte. Pour cette étude, l'hypothèse est qu'après une séance de RLC de courte durée, l'activité vagale cardiaque serait plus élevée pendant une période de stress cognitif, comparativement à la condition contrôle.
- Dans l'étude 2, j'étudie les effets d'une intervention à long terme de RLC (30 jours) sur l'activité vagale cardiaque nocturne et au réveil de jeunes adultes sains. L'hypothèse est qu'après une intervention RLC de 30 jours, par rapport au niveau initial et par rapport au groupe contrôle, l'activité vagale cardiaque sera plus élevée pendant la nuit et au réveil.
- Dans l'étude 3, j'étudie les effets de la RLC à court terme chez de jeunes adultes sains sur les trois principales fonctions exécutives, c'est à dire l'inhibition, la mémoire de travail et la flexibilité cognitive. L'hypothèse est que la RLC augmentera la performance des fonctions exécutives par rapport au groupe contrôle et que ces effets seront médiés par l'activité vagale cardiaque.
- Dans l'étude 4, j'étudie les effets de la RLC à court terme chez de jeunes adultes sains sur la performance d'inhibition après un effort physique. Je fais l'hypothèse que la RLC augmentera la performance d'inhibition par rapport à la condition contrôle, et que les résultats seront médiés par l'activité vagale cardiaque.

Afin de réaliser ces quatre études, certains aspects méthodologiques doivent être pris en compte. Plus particulièrement, les aspects liés à la mesure de l'activité vagale cardiaque et à la réalisation de la RLC seront maintenus constants dans les quatre études. Ces aspects sont décrits dans la section suivante.

## 16.3 Méthode générale

### 16.3.1 Mesure de l'activité vagale cardiaque

La variabilité de la fréquence cardiaque a été calculée à partir d'un signal ECG mesuré avec le Faros 180° (Bittium, Kuopio, Finlande). La fréquence d'échantillonnage était de 500 Hz. Deux électrodes ECG pré-gélifiées jetables ont été utilisées (Ambu L-00-S/25, Ambu GmbH, Bad Nauheim, Allemagne). L'électrode négative a été placée dans la fosse infraclaviculaire droite (juste en-dessous de la clavicule droite) et l'électrode positive a été placée sur le côté gauche de la poitrine, sous le muscle pectoral dans la ligne axillaire antérieure gauche. L'enregistrement complet de l'ECG a été inspecté visuellement et les artefacts ont été corrigés manuellement (Laborde, Mosley, et al., 2017). A partir de l'enregistrement ECG, les variables RMSSD et HF-HRV ont été calculées en utilisant le logiciel Kubios (University of

Eastern Finland, Kuopio, Finlande). La variable RMSSD a été choisie pour opérationnaliser l'activité vagale cardiaque dans les études 1, 3 et 4 car elle est moins affectée par la respiration (Hill, Siebenbrock, Sollers, & Thayer, 2009), tandis que la variable HF-HRV a été choisie dans l'étude 2 étant donné que des durées différentes étaient mesurées pendant la nuit pour chaque participant.

Dans la mesure où il existe un débat actuel sur la question de savoir s'il faut ou non contrôler les paramètres respiratoires lors de l'évaluation de la variabilité de la fréquence cardiaque (Grossman et coll., 1991; Grossman et Kollai, 1993; Laborde, Mosley et coll., 2017; Larsen, Tzeng, Sin et Galletly, 2010; Thayer, Loerbroks et Sternberg, 2011), la fréquence respiratoire est également calculée afin de mieux comprendre si des changements possibles de la RMSSD sont attribuables à l'activité vagale cardiaque ou bien sont principalement affectés par un changement de fréquence respiratoire. La fréquence respiratoire a été calculée à l'aide de l'algorithme de respiration dérivé de l'ECG du logiciel Kubios (Tarvainen, Niskanen, Lipponen, Ranta-Aho, & Karjalainen, 2014).

### 16.3.2 La respiration lente contrôlée

La manière la plus commune de réaliser la respiration lente contrôlée se fait via l'utilisation d'un régulateur visuel (Allen et Friedman, 2012; Laborde, Allen, Gohring et Dosseville, 2017; Tsai et coll., 2015), tandis que les méthodes auditives ou kinesthésiques (par ex., vibrations) ont été utilisées moins fréquemment. Dans les études 1, 3 et 4, l'exercice de RLC a été réalisé à l'aide d'une vidéo montrant une balle se déplaçant de haut en bas à 6 cpm. Les participants devaient inspirer continuellement par le nez pendant que la balle montait, et expirer continuellement avec les lèvres pincées lorsque la balle descendait. Il s'agissait d'une capture vidéo du logiciel EZ-Air Plus (Biofeedback Federation of Europe). La vidéo montrait un exercice de RLC de 3 x 5 min, avec une pause d'une minute entre chaque unité RLC de 5 min, soit un total de 17 min. La pause d'une minute entre chaque unité de RLC a été introduite car certains participants avaient signalé dans une étude pilote que 15 minutes de RLC sans pause pouvaient se révéler fatigantes. L'expiration (5,5 s) durait plus longtemps que l'inhalation (4,5 s), car l'expiration prolongée induit une activité vagale cardiaque plus élevée (Komori, 2018; Matsumoto et al, 2011; Strauss-Blasche et al, 2000; Van Diest et al, 2014). Dans l'étude 2, la technique RLC a été réalisée avec l'application Breath Pacer, qui montre une fleur ajoutant lentement des pétales pour indiquer les phases d'inspiration et d'expiration.

Une période de familiarisation à la RLC a été créée afin de permettre aux participants de se familiariser avec la technique. L'inhalation par le nez (c'est-à-dire la respiration nasale) est importante parce que l'air est humidifié, réchauffé, et nettoyé (Lorig, 2011), mais aussi parce

qu'elle provoque une activation optimale des réseaux neuronaux liés au traitement des stimulus et au comportement (Zelano et al., 2016). L'expiration se fait par la bouche, qui offre moins de résistance ventilatoire que le canal nasal (Lorig, 2011). De plus, l'expiration est réalisée en ayant les lèvres pincées, ce qui permet un meilleur contrôle du flux d'air et permet aux participants de l'adapter précisément à la durée de l'expiration. La respiration avec les lèvres pincées impose une résistance au débit expiratoire, ce qui conduit à une pression expiratoire positive dans les voies respiratoires. Ce retard expiratoire favorise une expulsion d'air homogène des poumons, maintenant la pression intrabronchiale et favorisant l'échange gazeux et la ventilation (Custodio, 1998; Nield, Soo Hoo, Roper, & Santiago, 2007; Ramos et al., 2009). Il fut ensuite demandé aux participants de poser une main sur leur poitrine et une main sur leur ventre, alors qu'ils recevaient les instructions suivantes: « La main sur la poitrine ne doit pas bouger, seule la main sur le ventre doit bouger: Le ventre doit grossir pendant la phase d'inhalation, et rétrécir pendant la phase d'expiration. » Cette instruction reflète une activation optimale du diaphragme. Lorsque le diaphragme se contracte et descend, il augmente le volume de la cavité thoracique et crée une zone de basse pression qui fait circuler l'air dans les poumons pour égaliser la pression (Colbert et al., 2016; Hall, 2011a).

Pendant la respiration spontanée, l'expiration est surtout passive. Cependant, avec la RLC, l'expiration forcée peut impliquer des muscles abdominaux qui, par leur contraction, aident à repousser le diaphragme vers le thorax, et par conséquent à expulser l'air plus efficacement (Lorig, 2011; West, 2015; West & Luks, 2016). La fréquence respiratoire est progressivement diminuée avec des séquences de 2 minutes à 10 cpm, 8 cpm, puis 6 cpm, avec une pause de 1 minute entre chaque séquence. La technique de RLC exige que le participant inspire et expire continuellement et uniformément respectivement lorsque la balle monte et descend. Lorsque cette instruction est correctement suivie, les caractéristiques d'oscillation sinusoïdale de RLC peuvent être observées dans le tachogramme R-R (Lehrer & Gevirtz, 2014). L'expérimentateur devait vérifier si le participant réalisait correctement la technique de la RLC pendant la familiarisation avant de passer à l'étape suivante du protocole.

## 16.4 Études

### 16.4.1 Étude 1 - résumé

Contexte: Le handicap mental crée souvent un état de stress chronique à la fois pour la personne concernée et ses proches (famille, aidants). Le développement de méthodes de gestion du stress est donc important pour la réduction du stress chez les personnes ayant un handicap mental. Le but de cette étude était d'étudier l'effet de la respiration lente sur les symptômes de

stress vécus par les adolescents ayant un handicap mental au cours d'une tâche cognitive sous pression temporelle.

Méthode: Quatorze adolescents ayant un handicap mental ( $M_{\text{âge}} = 17,39$  ans, de 15 à 19 ans) ont participé à deux séances en laboratoire - une séance de RLC (condition expérimentale) et une séance d'écoute de livre audio (condition contrôle) - dont l'ordre était alterné entre les participants. L'activité vagale cardiaque a été mesurée avec la variabilité de la fréquence cardiaque pour indexer la gestion du stress.

Résultats: Aucune différence dans l'activité vagale cardiaque n'a été observée au départ entre les conditions expérimentale et contrôle. Comparativement à la condition contrôle, l'activité vagale cardiaque était significativement plus élevée pendant la condition expérimentale.

Conclusions : La RLC a amélioré la gestion du stress dans une plus grande mesure que l'écoute d'un livre audio. La RLC semble être une technique de gestion du stress facile à apprendre qui apparaît comme une méthode efficace pour aider les adolescents ayant un handicap mental à gérer leur stress.

#### 16.4.2 Étude 2 - résumé

Les techniques de respiration font partie des méthodes traditionnelles de relaxation, mais leur influence sur les variables psychophysiologiques liées au sommeil doit encore être élucidée. Par conséquent, le but de cette étude était d'investiguer l'influence d'une intervention RLC de 30 jours par rapport à l'utilisation des réseaux sociaux sur la qualité subjective du sommeil et l'activité vagale cardiaque (opérationnalisée par HF-HRV). Les participants ( $n = 64$ , 33 hommes, 31 femmes,  $M_{\text{âge}} = 22,11$ ,  $ET = 3,12$ ) ont été répartis de manière aléatoire dans un groupe expérimental ou contrôle. Dans le groupe expérimental, ils ont dû effectuer la RLC pendant 15 minutes chaque soir pendant 30 jours. La RLC a été réalisée à l'aide d'une application pour smartphone. Le groupe contrôle a utilisé les réseaux sociaux (par ex. Facebook, Instagram, Whatsapp) pour la même durée. La nuit précédant et suivant l'intervention de 30 jours, l'activité vagale cardiaque a été évaluée au moyen d'un électrocardiogramme (ECG) portatif léger, et ils ont dû remplir le questionnaire d'indice de qualité du sommeil de Pittsburgh. Les résultats ont montré que par rapport à l'utilisation des réseaux sociaux, la RLC a amélioré la qualité subjective du sommeil et augmenté l'activité vagale cardiaque nocturne, tandis qu'en ce qui concerne l'activité vagale cardiaque au réveil une augmentation non significative a été observée. En conclusion, la RLC apparaît comme une technique accessible pour améliorer la qualité subjective du sommeil ainsi que l'activité du système nerveux parasympathique pendant le sommeil chez les jeunes adultes sains.

### 16.4.3 Étude 3 - résumé

Le but de cette étude était de tester les effets immédiats de la respiration lente contrôlée sur les fonctions exécutives (inhibition, mémoire de travail et flexibilité cognitive). Deux modèles théoriques constituent les bases de cette expérience. Le modèle de résonance prédit que la RLC augmente l'activité vagale cardiaque et le modèle d'intégration neuroviscérale prédit qu'une plus grande activité vagale cardiaque entraîne un meilleur fonctionnement exécutif. Au total, 78 participants (41 hommes, 37 femmes;  $M_{\text{âge}} = 23,22$  ans, de 18 à 32 ans) ont pris part à deux conditions expérimentales: une condition RLC de 3 x 5 minutes et une condition contrôle où il s'agissait de regarder un documentaire télévisé pendant une durée équivalente. Après chaque condition, la variabilité de la fréquence cardiaque a été mesurée et les participants ont effectué trois tâches concernant les fonctions exécutives: la tâche de Stroop (inhibition), la tâche AOSPAN (mémoire de travail) et la tâche de « tri des cartes de Wisconsin » (flexibilité cognitive). Les résultats ont montré que la performance dans les tâches mobilisant les fonctions exécutives était meilleure dans la condition RLC par rapport à la condition contrôle, avec des scores plus élevés pour la précision de l'interférence Stroop, pour le score AOSPAN et moins d'erreurs persévérantes dans la tâche de tri des cartes de Wisconsin. Aucune différence n'a cependant été relevée concernant le temps de réaction de l'interférence Stroop. Cependant, cette différence de performance au niveau des fonctions exécutives entre les conditions expérimentales n'a pas été médiée par l'activité vagale cardiaque. Par conséquent, les résultats ne concordent que partiellement avec les prédictions du modèle d'intégration neuroviscérale et du modèle de résonance. La RLC semble être une technique prometteuse pour améliorer la performance immédiate des fonctions exécutives, mais de nouvelles études doivent être conduites pour comprendre les mécanismes sous-jacents possibles ainsi que les effets à long terme.

### 16.4.4 Étude 4 - résumé

Cette étude vise à déterminer si la RLC améliore l'adaptation au stress psychologique, et en particulier l'inhibition, lorsqu'elle est effectuée avant ou après un effort physique. Selon le modèle de résonance, on s'attend à ce que la RLC augmente l'activité vagale cardiaque. De plus, selon le modèle d'intégration neuroviscérale, l'activité vagale cardiaque est positivement liée à la performance cognitive des fonctions exécutives et jouerait donc un rôle dans l'adaptation au stress psychologique. L'hypothèse est que la RLC, par rapport à une condition contrôle, induira une meilleure adaptation au stress psychologique, mesurée par une meilleure performance inhibitrice. Deux expériences intra-sujet ont été réalisées avec des sportifs: dans la première expérience ( $N = 60$ ), la RLC (ou la condition contrôle - documentaire télévisé



émotionnellement neutre) a été réalisée avant l'activité physique (« relaxation avant activité physique »), et dans la deuxième expérience (N = 60), la RLC (ou la condition contrôle avec le documentaire) a été réalisée après l'activité physique (« relaxation après activité physique »). L'activité physique se composait de 5 min Burpees, un exercice physique impliquant tout le corps. Dans les deux expériences, l'adaptation au stress psychologique a été étudiée avec une tâche de Stroop, une mesure d'inhibition, qui fut exécutée après l'activité physique. Le stress perçu a augmenté pendant l'activité physique ( $\eta^2$  partiel = 0,63) et pendant la tâche de Stroop ( $\eta^2$  partiel = 0,08), et a diminué pendant la relaxation ( $\eta^2$  partiel = 0,15), mais aucun effet de condition n'a été observé. Au niveau physiologique, l'activité physique a augmenté la fréquence cardiaque et la fréquence respiratoire, et diminué l'activité vagale cardiaque [opérationnalisé avec RMSSD] dans les deux expériences. De plus, le nombre d'erreurs dans la catégorie des erreurs incongruentes (précision d'interférence Stroop) s'est avéré inférieur dans la condition RLC par rapport à la condition contrôle; toutefois, ces résultats n'ont pas été médiés par la RMSSD. De plus, le temps de réaction de l'interférence Stroop s'est avéré plus faible dans l'ensemble pour la "relaxation avant l'exercice physique", mais aucun effet n'a été observé pour la RLC et le temps de réaction de l'interférence Stroop. Dans l'ensemble, ces résultats suggèrent que la RLC réalisée avant ou après l'activité physique a un effet positif sur l'adaptation au stress psychologique et plus particulièrement par rapport à la capacité d'inhibition, mais la compréhension des mécanismes sous-jacents nécessite de faire l'objet de futures recherches.

## 16.5 Discussion générale

### 16.5.1 Synthèse des résultats

L'objectif de cette thèse de doctorat était d'étudier plus en détail les mécanismes sous-jacents concernant les effets de la RLC sur l'amélioration des processus d'autorégulation. Le cadre théorique était basé sur le modèle de résonance (Lehrer & Gevirtz, 2014) et sur le modèle d'intégration neuroviscérale (Smith et al., 2017; Thayer et al., 2009). Selon ces modèles théoriques, il a été postulé que la RLC augmenterait l'activité vagale cardiaque, ce qui provoquerait en retour un meilleur fonctionnement des phénomènes d'autorégulation, tels que la gestion du stress, le sommeil et les fonctions exécutives. Cette hypothèse était basée sur le fait que la RLC est supposée stimuler les afférences vagales (Lehrer & Gevirtz, 2014), et que l'activité vagale cardiaque est supposée refléter l'efficacité du réseau autonome central, selon le modèle d'intégration neuroviscérale (Smith et al., 2017; Thayer et al., 2009).

Nos hypothèses de travail ont été partiellement validées: tout d'abord, dans l'étude 1, une intervention RLC à court terme de 15 minutes a entraîné une activité vagale cardiaque (RMSSD) plus élevée chez des adolescents ayant un handicap mental effectuant une tâche



cognitive stressante, indiquant ainsi une meilleure adaptation, comparativement à une condition où ils écoutaient un livre audio. Une intervention similaire à court terme de RLC a provoqué une augmentation de l'activité vagale cardiaque au repos chez de jeunes adultes sains dans l'étude 4, mais aucun changement n'a été observé dans l'activité vagale cardiaque au repos chez une population similaire dans l'étude 3. En ce qui concerne l'intervention à long terme de RLC (15 min par jour pendant 30 jours) chez de jeunes adultes sains (étude 2), on a constaté une amélioration de l'activité vagale cardiaque (HF-HRV) nocturne après 30 jours, alors que seule une tendance était observée avec l'activité vagale cardiaque (HF-HRV) au réveil. Deuxièmement, la performance des fonctions exécutives a été améliorée dans l'étude 3 (inhibition, mémoire de travail, flexibilité cognitive) et dans l'étude 4 (inhibition), mais cette amélioration n'était pas médiée par l'activité vagale cardiaque, contrairement à notre hypothèse.

En associant les résultats, il est possible de conclure que la RLC sans biofeedback influence positivement les processus d'autorégulation, de façon similaire à ce qui a été observé avec les interventions de RLC réalisées avec biofeedback (Gevirtz, 2013; Goessl et al, 2017; Kennedy & Parker, 2018; Wheat & Larkin, 2010; Yu et al, 2018; Zaccaro et al, 2018). Cela signifierait que le biofeedback, même s'il peut améliorer des aspects liés par exemple à l'auto-efficacité et à la motivation à effectuer la RLC, n'est pas un critère décisif de l'efficacité des interventions de RLC. Toutefois, d'après nos résultats, nous ne pouvons conclure de façon décisive sur le rôle joué par l'activité vagale cardiaque concernant les processus d'autorégulation influencés par la RLC. Si une augmentation des marqueurs de l'activité vagale cardiaque à la suite d'une RLC (par rapport à la condition contrôle) a été observée dans les études 1, 2 et 4, ce ne fut pas le cas dans l'étude 3. De plus, les résultats ont montré que l'activité vagale cardiaque n'a pas joué le rôle de médiateur de l'amélioration des fonctions exécutives à la suite d'une RLC dans les études 3 et 4. Cela signifierait que, contrairement à l'hypothèse du modèle de résonance (Lehrer et Gevirtz, 2014), la RLC peut ne pas déclencher d'afférences vagales, ou que la stimulation des afférences vagales ne se traduit pas nécessairement par une augmentation des afférences vagales au niveau cardiaque. Pour illustrer cette spéculation, un questionnement similaire a été rapporté dans une recherche concernant la douleur, utilisant la stimulation invasive du nerf vague (VNS). La stimulation du nerf vague (c.-à-d. la stimulation des afférences vagales) est sensée envoyer des messages nerveux non spécifiques au tronc cérébral, et ces messages nerveux sont supposés concurrencer les stimuli de douleur entrants ou déclencher des réflexes non spécifiques qui activent l'inhibition de la douleur, par exemple en libérant des neurotransmetteurs inhibiteurs (Albusoda, Farmer, et Aziz, 2018). Un mécanisme similaire peut se produire avec la RLC, mais d'autres mécanismes peuvent

également être envisagés. Comme le suppose le modèle de résonance (Lehrer et Gevirtz, 2014), la RLC peut améliorer le fonctionnement du baroréflexe ou améliorer les échanges gazeux au niveau des alvéoles. Si l'action de la RLC sur les barorécepteurs a déjà été démontrée (Lehrer et al., 2003), des recherches supplémentaires sont nécessaires pour vérifier l'hypothèse relative à l'efficacité des échanges gazeux, en abordant les questions soulevées par les recherches précédentes sur l'arythmie sinusale respiratoire (Buchheit, 2010; Giardino et al., 2003; Sin et al., 2010; Tharion & Subramani, 2011; Tzeng, 2011).

#### 16.5.2 Limites

Parmi les limites de ce travail doctoral, il appartient de souligner particulièrement les suivantes:

- Le fait que les conditions contrôles ont impliqué une respiration spontanée, ce qui ne permet pas de conclure de façon certaine au rôle de la respiration lente contrôlée. De futures recherches devraient prendre en compte une condition contrôle impliquant une respiration contrôlée à un rythme proche de la fréquence spontanée de respiration, par exemple 12 cpm (Sherwood, 2006; Tsai, Kuo, Lee, & Yang, 2015).
- La fréquence respiratoire a été déterminée grâce à un algorithme du logiciel Kubios, à partir du signal ECG. Les recherches futures pourraient intégrer une ceinture de respiration afin de la mesurer directement (Lorig, 2011; Ritz et al., 2002).
- Un seul pattern de respiration a été utilisé dans les quatre études, avec 6cpm (4,5s inspiration et 5,5s expiration). De futures études pourraient se consacrer aux effets de l'utilisation de patterns de respiration différents, ou bien rechercher les effets liés à l'introduction de pauses, par exemple entre les phases expiratoire et inspiratoire (Russell, Scott, Boggero, & Carlson, 2017).

#### 16.5.3 Perspectives

Parmi les perspectives de recherche amorcées par ce travail doctoral, on peut noter les suivantes :

- La fréquence de résonance individuelle (Lehrer & Gevirtz, 2014; Vaschillo, Vaschillo, & Lehrer, 2006) devrait faire l'objet de futures recherches, car elle permettrait potentiellement d'améliorer encore plus efficacement les processus d'autorégulation.
- La RLC devrait être testée avec une résistance inspiratoire additionnelle, qui permettrait selon Gholamrezaei, Van Diest, Aziz, Vlaeyen, and Van Oudenhove (2019) d'améliorer encore plus efficacement l'effet sur la pression artérielle systolique et sur l'activité vagale cardiaque.

- Les effets de la RLC doivent encore être testés chez des populations différentes que celles étudiées lors de ce travail doctoral. Elle pourrait par exemple particulièrement bénéficier aux enfants, aux personnes âgées, et aux personnes souffrant de conditions médicales comme l'hypertension.
- En lien avec l'élargissement des recherches sur la RLC à d'autres types de populations que celles étudiées lors de ce travail doctoral, il convient également de s'intéresser aux effets de la respiration lente contrôlée sur d'autres types d'indicateurs, tels que par exemple la performance sportive (Jimenez Morgan & Molina Mora, 2017)
- La question de l'utilisation d'outils de biofeedback doit encore être abordée, pour savoir si cela permettrait d'atteindre de meilleurs résultats, ou bien si les résultats seraient similaires à ceux obtenus grâce à la pratique de la RLC sans l'utilisation additionnelle d'outils de biofeedback.
- Finalement, l'analyse de l'activité vagale cardiaque devrait se faire conjointement dans le futur avec celle de l'activité sympathique cardiaque, qui pourrait éventuellement modérer les prédictions du modèle d'intégration neuroviscérale (Duschek et al., 2017; Michael, Graham, & Davis, 2017).

## 16.6 Conclusion

Cette thèse de doctorat visait à déterminer si la RLC sans biofeedback influence l'activité vagale cardiaque, et à étudier son effet sur plusieurs processus d'autorégulation, en se basant sur le modèle d'intégration neuroviscérale (Smith et al., 2017; Thayer et al., 2009) et sur le modèle de résonance (Lehrer & Gevirtz, 2014). Quatre études ont été menées auprès d'adolescents ayant un handicap mental (étude 1) et auprès de jeunes adultes sains (études 2, 3 et 4). Des interventions à court et à long terme de RLC ont été testées et des effets positifs ont été constatés sur la gestion du stress (court terme, étude 1), le sommeil (long terme, étude 2) et les fonctions exécutives (court terme, études 3 et 4), mais les résultats liés aux fonctions exécutives n'ont pas été médiés par l'activité vagale cardiaque. Ces résultats sont encourageants en ce qui concerne l'utilisation de la RLC en tant que " solution rapide " ou routine quotidienne, mais les futures recherches doivent clarifier les mécanismes sous-jacents, en particulier avec des méthodes d'imagerie cérébrale. Cette thèse de doctorat a probablement soulevé plus de questions qu'elle n'a répondu aux interrogations déjà existantes, mais le message principal que l'on peut en tirer apparaît encourageant: La RLC semble être une technique efficace, non invasive, avec peu d'effets secondaires, ce qui en fait une intervention appropriée à recommander pour traiter un large éventail de phénomènes d'autorégulation. Même s'il reste de nombreux défis à relever, j'espère que cette thèse de doctorat a contribué à susciter l'intérêt pour

cette technique fascinante, et que vous serez peut-être plus conscient de l'intérêt du contrôle volontaire de votre respiration à partir de maintenant.

## 17 French short abstract – La respiration lente contrôlée et l'activité vagale cardiaque : Influence sur la gestion du stress, le sommeil, et les performances cognitives exécutives

Cette thèse de doctorat a pour but d'étudier l'influence de la respiration lente contrôlée sans biofeedback sur l'activité vagale cardiaque ainsi que son action sur plusieurs processus d'autorégulation, en se basant sur le modèle d'intégration neuroviscérale (Smith et al., 2017 ; Thayer et al., 2009) et sur le modèle de résonance (Lehrer & Gevirtz, 2014). Quatre études ont été menées, auprès d'adolescents ayant un handicap mental (étude 1) et de jeunes adultes sains (études 2, 3 et 4). Les effets d'interventions courtes et longues basées sur la respiration lente contrôlée ont été étudiés, et des effets positifs ont été constatés sur la gestion du stress (intervention courte, étude 1), le sommeil (intervention longue, étude 2) et les fonctions exécutives (intervention courte, études 3 et 4), mais les effets sur les fonctions exécutives n'ont pas été médiés par l'activité vagale cardiaque. Ces résultats sont encourageants en ce qui concerne l'utilisation de la respiration lente contrôlée en tant qu'intervention à court et long terme, en tant que "solution rapide" ou routine quotidienne, mais les recherches futures doivent clarifier les mécanismes sous-jacents, en particulier en utilisant des méthodes d'imagerie cérébrale. En conclusion, la respiration lente contrôlée est une technique gratuite, non invasive, simple à mettre en œuvre, et avec peu d'effets secondaires, ce qui en fait une intervention appropriée pour traiter un éventail varié de phénomènes liés à l'autorégulation.

## 18 German short abstract - Langsam kontrollierte Atmung und kardiovagale Aktivität: Einfluss auf Stressregulation, Schlaf und kognitive Exekutivfunktionen

Diese Dissertation hat zum Ziel, den Einfluss langsam kontrollierte Atmung (eng. *slow-paced breathing*; SPB) auf die kardiovagale Aktivität und verschiedene Selbstregulierungsprozesse zu untersuchen. Als theoretische Grundlage dient hierbei das neuroviszerale Integrationsmodell (Smith et al., 2017; Thayer et al., 2009) und das Resonanzmodell von Lehrer and Gevirtz (2014). Im Rahmen der vorliegenden Dissertation wurden vier verschiedene Studien durchgeführt, bei denen sowohl Jugendliche mit geistiger Behinderungen (Studie 1), als auch gesunde, junge Erwachsene (Studie 2, 3, & 4) untersucht wurden. Dabei wurden sowohl akute, sowie chronische Effekte von SPB-Interventionen untersucht. Hierbei konnten positive Auswirkungen des SPB auf das Stressmanagement (akute Effekte, Studie 1), den Schlaf (chronische Effekte, Studie 2) und die Exekutivfunktionen (akute Effekte, Studien 3 und 4) festgestellt werden. Hinsichtlich der Ergebnisse der Exekutivfunktionen konnte jedoch keine Mediation durch die kardiovagale Aktivität festgestellt werden. Die vorliegenden Ergebnisse weisen darauf hin, dass SPB in akuter und chronischer Form für die praktische Anwendung geeignet ist (z.B. in Form eines „Quick-Fix“ oder in Form von Tagesroutinen). Zukünftige Forschung sollte sich mit den zugrunde liegenden Mechanismen befassen, wobei insbesondere biopsychologische Methoden einen besonders gewinnbringenden Zugang darstellen könnten. Zusammenfassend lässt sich feststellen, dass SPB ein kostengünstiges, nicht-invasives Verfahren darstellt, welches mit geringen Nebenwirkungen assoziiert ist. Vor diesem Hintergrund und den dargestellten Ergebnissen, stellt SPB ein sinnvolles Verfahren zur positiven Beeinflussung von Selbstregulation dar.

## 20 English short abstract

This PhD thesis was aimed to investigate the influence of slow-paced breathing (SPB) without biofeedback on CVA, as well as its action on several self-regulation processes, based on the neurovisceral integration model (Smith et al., 2017; Thayer et al., 2009) and on the resonance model (Lehrer & Gevirtz, 2014). Four studies were conducted, adolescents with individual disabilities (Study 1) and healthy young adults (Study 2, 3, & 4). Both short-term and long-term SPB interventions have been investigated, and positive effects have been found on stress management (short-term, Study 1), sleep (long-term, Study 2), and executive functions (short-term, Studies 3 and 4), however results related to executive functions were not mediated by CVA. These results are encouraging regarding the use of SPB in acute and chronic interventions, as a “quick-fix” or daily routine, however future research has to clarify the underlying mechanisms, in particular with brain imaging methods. To conclude, SPB is a technique that is free, non-invasive, easy to implement, and with little side effects, which makes it an appropriate intervention to recommend to address a large range of self-regulation phenomena.

## **Slow-paced breathing and cardiac vagal activity: Influence on stress regulation, sleep, and cognitive executive performance**

This PhD thesis was aimed to investigate the influence of slow-paced breathing (SPB) without biofeedback on CVA, as well as its action on several self-regulation processes, based on the neurovisceral integration model (Smith et al., 2017; Thayer et al., 2009) and on the resonance model (Lehrer & Gevirtz, 2014). Four studies were conducted, adolescents with individual disabilities (Study 1) and healthy young adults (Study 2, 3, & 4). Both short-term and long-term SPB interventions have been investigated, and positive effects have been found on stress management (short-term, Study 1), sleep (long-term, Study 2), and executive functions (short-term, Studies 3 and 4), however results related to executive functions were not mediated by CVA. These results are encouraging regarding the use of SPB in acute and chronic interventions, as a “quick-fix” or daily routine, however future research has to clarify the underlying mechanisms, in particular with brain imaging methods. To conclude, SPB is a technique that is free, non-invasive, easy to implement, and with little side effects, which makes it an appropriate intervention to recommend to address a large range of self-regulation phenomena.

**Keywords:** slow-paced breathing, cardiac vagal activity, parasympathetic nervous system, executive functions, stress, sleep

## **La respiration lente contrôlée et l'activité vagale cardiaque : Influence sur la gestion du stress, le sommeil, et les performances cognitives exécutives**

Cette thèse de doctorat a pour but d'étudier l'influence de la respiration lente contrôlée sans biofeedback sur l'activité vagale cardiaque ainsi que son action sur plusieurs processus d'autorégulation, en se basant sur le modèle d'intégration neuroviscérale (Smith et al., 2017 ; Thayer et al., 2009) et sur le modèle de résonance (Lehrer & Gevirtz, 2014). Quatre études ont été menées, auprès d'adolescents ayant un handicap mental (étude 1) et de jeunes adultes sains (études 2, 3 et 4). Les effets d'interventions courtes et longues basées sur la respiration lente contrôlée ont été étudiés, et des effets positifs ont été constatés sur la gestion du stress (intervention courte, étude 1), le sommeil (intervention longue, étude 2) et les fonctions exécutives (intervention courte, études 3 et 4), mais les effets sur les fonctions exécutives n'ont pas été médiés par l'activité vagale cardiaque. Ces résultats sont encourageants en ce qui concerne l'utilisation de la respiration lente contrôlée en tant qu'intervention à court et long terme, en tant que "solution rapide" ou routine quotidienne, mais les recherches futures doivent clarifier les mécanismes sous-jacents, en particulier en utilisant des méthodes d'imagerie cérébrale. En conclusion, la respiration lente contrôlée est une technique gratuite, non invasive, simple à mettre en œuvre, et avec peu d'effets secondaires, ce qui en fait une intervention appropriée pour traiter un éventail varié de phénomènes liés à l'autorégulation.

**Mots-clés :** Respiration lente contrôlée, activité vagale cardiaque, système nerveux parasympathique, fonctions exécutives, stress, sommeil