Contemplations into respiration: effects of breathing and meditative movement on body and mind

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Chapter 5

General Discussion

Summary

In this book we venture into the world of contemplative practices, or meditative exercises and the effects these have on cognition and stress. The main focus lies on a subset of meditative techniques, namely breathing exercises, or how respiratory patterns change body and mind. The general introduction in **Chapter 1** covers the historical distinction between classic cognitive science and the embodied cognitive perspective, adopted here. It also introduces the contemplative practices and the concept of mindfulness. It also provides a short primer on Bayesian statistics, as used throughout this book. Because the data collection in **Chapter 3** predates the theoretical work in **Chapter 2**, breathing is not a central topic in that empirical study.

Chapter 2 contains a peer-reviewed theoretical paper (Gerritsen & Band, 2018). It starts with a selected review of studies on contemplative traditions. In the review it was shown that the majority of reported beneficial effects on physical health, mental health and cognitive/emotional functioning, were within the stress-related domain. We have proposed that these benefits could be explained through a decrease in individual (chronic) stress levels and that breathing exercises, by deliberately or passively lowering respiration rates, play a critical role in achieving these effects. Because of the prevalence of breathing exercises in contemplative traditions, common findings across interventions could be attributed to this factor. We have further maintained that also exercises that just prescribe focused attention on breathing change respiratory patterns and slow its rate. The main route by which respiratory patterns affect stress-related conditions is by their effects on the autonomous nervous system. This nervous system is both responsible for arousal and relaxation, for fight-or-flight and rest-and-digest modes of operation, by its sympathetic and parasympathetic branch, respectively.

Furthermore, in **Chapter 2** we have proposed a neurophysiological model: the respiratory vagal nerve stimulation (rVNS) model of contemplative activity, that tries to explain the aforementioned emotional and cognitive effects and capture several possible mechanisms of effect. The three main predictions of the rVNS model are: 1) that respiration rate; 2) the relative duration (ratio) of inhalation versus exhalation; and 3) the relative locus — thoracic versus abdominal — of respiratory motor activity influence both acute and chronic stress levels, and thus cognition and (mental) health. Finally, we suggested a number of mechanisms focusing on the role of the vagus nerve as possible biofeedback mechanisms in producing these effects, acting also as the mediator between respiration and cognition.

The primary proposition of **Chapter 2** is that by lowering respiration rates, extending exhalation and shifting the locus to the abdomen, relaxation increases and stress goes down. Subsequently, a more flexible psychological state is reached whereby complicated tasks characterized by high mental workload — tasks that tax *executive functions* — can be performed more efficiently. In other words: breathing exercises indirectly enhance cognitive control. In **Chapter 4**, two of the main predictions (on respiration rate and locus) are empirically tested.

Chapter 3 describes an empirical study on aging, published in a peer-reviewed journal (Gerritsen et al., 2020). Here, we conducted a randomized controlled trial that examines the effects of Tai Chi Chuan (TCC)— a mind-body exercise characterized by slow movement and body awareness— on motoric and executive functioning in an aging population sample. Though the TCC intervention did include some instructions aimed at breathing, none of the predictions of the rVNS model were directly tested, because the study was conducted well before the publication of the model described in **Chapter**

2. A total of 55 participants, aged between 53 and 85, were initially assigned either to a TCC group or a control group. The final analysis included data from 43 participants. The TCC group participated in an online 10-week, 20-lesson video program of increasing difficulty level, while the control group watched educational videos of similar length and frequency. Two measures of motor functioning: motor speed, as measured by the finger tapping test, and functional balance, as measured by the timed up and go test; and three measures of executive functioning: *shifting*, as measured by the task switching test, *updating*, as measured by the 2-back task, and *inhibition*, as measured by the stop-signal reaction task, were used to evaluate the effects of TCC. We found that there were no differences in executive functioning on any of the measures between the TCC group and the control group pre-to-posttest. However, we found extreme evidence for TCC benefits on functional balance and moderate evidence for increased motor speed. Thus, we concluded that while TCC may be beneficial for improving motoric functioning in older adults, it may not have a significant impact on executive functioning.

Chapter 4 contains a first attempt at testing some predictions of the rVNS model (unpublished manuscript). Foremost, the prediction is that a phasic shift of respiratory locus from the thorax to the abdomen leads to acute relaxation, increases parasympathetic (vagal) tone, and enhances executive functioning. The chapter includes two experiments. In the first experiment, 29 people participated in three experimental sessions, where they were exposed to three different breathing interventions by audio guidance: abdominal breathing, thoracic breathing and focused breathing (semi-control condition). Participants were tested on changes in respiratory parameters, parasympathetic and sympathetic tone, as well as *response inhibition*. Although participants complied with the interventions as shown by a change

of the ratio between thoracic and abdominal circumference in accordance with the instructions (participants breathed relatively more with their stomach in the abdominal condition), expectations made by the rVNS model did not materialize. Parasympathetic and sympathetic tone, as measured by the root mean square of successive differences metric (RMSSD) of heart rate variability (HRV) and pre-ejection period, respectively, did not change pre- to post-test; and neither did executive functioning (response inhibition), as measured by the stop-signal task. However, because breathing slowed down during all three interventions, the effect of breathing locus instructions may have been mitigated. Also, response inhibition, as measured by the stop-signal task, might not be sensitive enough as an executive function to show acute enhancement — a short bout of respiratory modulation might not transfer to an increase in this functional component of cognitive control in laymen. Therefore, a second experiment was run.

In the second experiment of **Chapter 4** an attempt was made to address the two aforementioned issues present in the first experiment. Firstly, focused breathing was dropped and instructions for the other two were rewritten to prompt participants to slow their breathing in the abdominal condition — and slightly speed it in the thoracic condition. Secondly, the stop-signal task was dropped in favor of the Simon task. The form of *cognitive inhibition* mapped by the Simon task is assumed to be more sensitive to acute changes than *response inhibition*, as mapped by the stop-signal task. Thirdly, the pre-ejection period indicator of sympathetic activity was substituted for skin conductance level (SCL) for reason of feasibility of experimental set-up. Finally, a self-report on affect was added as an indication of subjective stress levels (affect grid). The data of 34 participants were analyzed. The results of the second experiment were highly similar to the first. Though participants adjusted their respiration in the instructed

direction, none of the major predictions came to pass. Cognitive inhibition was not improved from pre-test to post-test in the abdominal condition and even general speed (reaction time) did not differ between breathing conditions in the Simon task. Furthermore, autonomous tone, whether sympathetic (RMSSD) or parasympathetic (SCL), was equally unaffected. Only the self-report matched expectation (affect grid): after abdominal breathing people indicated that they felt more relaxed. Though clearly both experiments were underpowered, and this might hide the effects, there also was no numerical trend in the expected direction. Therefore, we concluded that there is no change in autonomous tone and cognitive functioning in this timeframe due to these specific breathing exercises.

Discussion

Null-results

This dissertation contains two experimental chapters, both yielding null-results on the main variables under study. The TCC intervention of **Chapter 3** did not lead to improvement in any of the executive functions under measurement — shifting, updating and (response) inhibition —as compared to control. The breathing exercises of the two experiments described in **Chapter 4** also did not affect executive functioning, as indicated by response and cognitive inhibition measures (stop-signal task and Simon task). Nor did the breathing exercises lead to the predicted state changes in autonomic functioning. A number of possible explanations for these null results have been put forth in the discussions of the previous two empirical chapters. These share a common pattern.

First, the issue might have been *dosage*. The interventions might have been too short for predicted and previously reported effects to surface. This might have been the case in the breathing studies. TCC, on the other hand, might have suffered from a lower quality and lack fundamental components such as meditative aspects. The breathing instructions in the respiration studies were auditory and did not directly manipulate respiration to a certain respiration rate, like slowing it to 6 breaths-per-minute or lower. Both breathing exercises and TCC participants might have shown failures to engage, or have other compliance problems to the instructions, for example: by difficulty level. Our breathing studies were run on young people without much *experience*, so for many participants this will have been there first dose of meditation or breathing exercise. This might have also compounded the issue of compliance. In all these cases, the dose of the effective components might have been too low to lead to a response in cognition (or autonomic functioning).

Second, there are *sample* issues. All three empirical studies were statistically underpowered: the number of participants in the samples were too low. There might also be sample characteristics that influenced the results. The aging TCC sample was characterized by high (physical) activity and might therefore have a ceiling effect on the gains that can be made from TCC's physical exercise aspect. As previously stated, the participants in the breathing studies were relatively young and inexperienced with these techniques. This might have led to non-compliance due to motivation and ability. Indeed, many participants struggled to lower their respiration rates significantly.

The aforementioned causes might hide effects in our studies that do exist. However, it might also simply be the case that the interventions just do

not lead to the predicted effects. Thus these null-results are a valid description of reality. Then we have to conclude that 1) the rVNS model is not a valid description of human neurophysiology and cognition; 2) the failure to replicate previous findings of TCC benefits on executive functioning suggests that these benefits do not exist. If we follow this latter conclusion, it remains mysterious why many studies on TCC do find positive results. Perhaps findings on the beneficial effects of contemplative practices on cognitive functioning are inflated or overstated in the literature.

Is there a *publication bias* in the field of contemplative science? According to two recent meta-analyses on the cognitive effects of mindfulness meditation there is no evidence for publication bias within the study field on these contemplative practices (Casedas et al., 2020; Gill et al., 2020). However, the findings of the two reviews do diverge strongly otherwise. Casedas and colleagues (2020) concluded that mindfulness meditation has a small to medium effect on executive functioning as a whole, though the authors acknowledge that these are preliminary findings as only 13 studies met the selection criteria and could be included. In contrast, Gill and colleagues (2020) only found a small to medium effect on higher-order functions, but no effect on executive functioning itself; and reported that overall, studies had many methodological flaws. A highly relevant difference between both studies for our discussion here, is that Gill et al. (2020) solely focused on brief mindfulness interventions (in beginners). This corresponds to our own null-findings with short interventions in laymen. But even taken together these two reviews do not provide strong confidence that contemplative practices have beneficial effects on cognition, specifically on executive functions. A randomized controlled trial published after these reviews aligns with this perspective (Baranski, 2021). This experiment investigated the acute effects of mindfulness meditation on the three

executive functioning components (shifting, updating, inhibition) and found no benefits on any of the three. Note that these are the same functional components as were under investigation in the TCC study of **Chapter 3**. The author makes the suggestion that practitioners and scientists should lower their expectations on cognitive enhancement by meditation. Another study, by Paap and colleagues (2020), reports that mindfulness meditation practice does not predict executive functioning, at least as measured by the interference scores used in multiple cognitive inhibition tasks, like the Stroop, flanker and Simon task. In this correlational study, the authors report a Bayesian statistical analysis, where extreme evidence was found, against a (positive) relationship between meditation practice and inhibitory functioning. In conclusion, aforementioned studies cast significant doubt on whether contemplative practices can enhance executive functioning, especially on the short-term; and that any potential existent effects are likely small.

This brings us to an overarching issue. Null-results and failures to replicate, even within a well-established field, where there is no indication of publication bias, are not a problem perse. Instead, they can be highly informative. Following Chalmers (1976) in his adaptation of Poppers (1959) falsificationism: a *falsification* is most useful when it provides evidence against a *dominant* hypothesis or paradigm. While *confirmation* still has value when finding evidence in favor of a bold claim (hypothesis): if it goes against the common consensus of scientific knowledge. Of course, in our studies we have not provided evidence against the dominant hypotheses (e.g. that contemplative practices benefit cognitive functioning), as our Bayesian evidence load was insufficient, due to lack of power, to make a confident claim against a model. So instead we were only unable to confirm the dominant hypotheses. Thus, I assuredly do not claim that any of the results

described in this work constitute a falsification proper. But complete rejection of the hypotheses and conclusions of previous studies into contemplative activities is not and never was the aim here; and also not in any way necessary, I believe, because there is another way to look at these results.

The problem might be that we — as cognitive scientists — opt for all or nothing answers: a statement is either false or true. We would like to have a clear cut answer to the (implicit) question: does this specific effect *exist*? While the question should be: does this specific effect *occur*? The difference between these two questions is context: the particular circumstances inherent in, and perhaps unique to, a particular research setting. This means that if a study is well-designed, conscientiously conducted and the inferential statistics allow, the results stand, no matter the apparent conflict with previous findings. Current scientific background knowledge does not invalidate these results. Do note here, that when encountering an anomalous result, a scientist should always start with critically assessing their own work, such as the design and methodology of the study; this is simply the most parsimonious and likely explanation for the anomaly. But if the data has been correctly and reliably obtained, then clearly there are conditions where this specific effect does *not* manifest itself. Previous findings must have been collected in a critically different setting, albeit different in a minor matter. These critical differences in context merit discovery, analysis and further investigation. This is another reason why Bayesian statistics should be preferred over classical statistics. Classical statistics invites a dichotomous binary way of thinking: about an effect's existence or non-existence, by simply rejecting or accepting a hypothesis, while in Bayesian statistics there is a quantification of the strength of evidence, — against or for — a particular model. Just by laying findings against this grey scale, a binary

mode of inference becomes less likely. Next, I will discuss a number of circumstances that are candidates in affecting the likelihood of the *occurrence* of respiratory effects.

Contextual factors: timeframes, demographics and individual differences

As stated in the previous chapters' discussions, effects on stress systems and cognition might only show up after long-term application of the intervention under study. Specifically on the effects of breathing patterns, as of yet, there have been no longitudinal studies. There are indications in our work that some of these effects do occur, but are short-lived. For example, during the two experiments described in **Chapter 4** we observed a brief increase in parasympathetic tone (or rather vagal tone HRV) in a subset of participants during the intervention — when they were breathing slowly — but vagal tone HRV returned to baseline soon afterwards. Certainly, these effects did not last into the next experimental phase. Thus, the benefits of relaxation may only develop through continued daily practice and might only then be reflected in a more parasympathetic dominant autonomic balance and perhaps an increased cognitive flexibility. In short, acute effects of respiratory modulation on autonomic and cognitive functioning might not *occur* (or even *exist)* because the physiological effects are too short term and do not transfer to tests of cognitive performance.

Certain demographic aspects might also function as moderator variables in the chain effects of respiratory patterns, through autonomic functioning to cognition, as predicted by the rVNS model. These demographic differences — between groups of people — might affect

(psycho)physiological outcomes significantly, and thus what beneficial effects can be expected to *occur* from any intervention targeting these. Indeed, especially in the autonomous nervous system, differences between groups of people might be large: it has recently been established that autonomic functioning differs between certain populations; and thus the (supposed) mediating role of stress systems might be impacted. Some of the following population effects might have also been a factor in our experimental work.

Firstly, there seem to be sex differences in autonomic functioning. It has become increasingly more apparent over the past decade, that the female body has not been taken as the default medical model and that physiology and symptomology may vary widely between sexes. As an illustrative example, let's look at symptoms of myocardial infarction. The "atypical" symptoms, such as: stomach ache and nausea, of myocardial infarction are far more common in women and have been historically underreported and overlooked in favor of the common symptoms in men: chest pain and upper arm ache, by both science and society. To this day, knowledge that these symptoms are signs of acute infarction have been shown to be lacking in the general population (Birnbach et al., 2020). Differences in cardiac functioning between sexes do not stop at cardiac arrest. There are also strong indications that cardiac autonomic functioning differs between sexes. A meta-analysis of studies that have gathered HRV measurements (Koenig & Thayer, 2016) reports that women have higher resting state heart rate and lower HRV, within the time-domain (e.g. RMSSD). However, women show lower power in the low frequency band of HRV, but higher power in the high frequency band than do men, which is also reflected in a lower low/high frequency ratio. This suggest that cardiac activity is parasympathetically dominated (by vagal means) in women and sympathetically dominated in men. The authors

conclude that this is a likely reason for the cardiac health benefits and longevity women enjoy, as compared to men. My added conclusion is that this might also have implications for the choice of HRV metric in skewed samples of the sexes. A more recent meta-analysis has made a slightly conflicting discovery in settings of social stress (Hamidovic et al., 2020). Paradoxically, women show lower reactive HRV during a public speech task than men, as well as slightly lower HRV during anticipation and recovery. Apparently, when (social) stressors are introduced, female participants show more arousal than men, while their resting states are conversely more relaxed than men. Clearly, cardiac autonomic control differs between the sexes. Relevant here is that this implies that studies with a proportional high number of women might have difficulty finding enhancement effects due to an already healthy autonomous balance, unless a strong stressor is introduced. These studies might suffer from a ceiling effect, where HRV responsivity in females is limited by a high baseline. The two breathing experiments in **Chapter 4** have a far higher number of females than males: the sample of experiment 1 consisted of 23 females to 6 males and experiment 2 of 31 females to 3 males. It is thus not unreasonable to assume that this HRV ceiling effect might have been present in in our experimental work. Then this might have resulted in *non-occurrence* of cognitive effect as well.

Secondly, there are developmental differences in autonomic functioning. Lipsitz and Novak (2012) report a number of affected functions that occur in normal human aging. Overall, sympathetic nervous system activity goes up, while — paradoxically — sympathetic functioning actually decreases, due to desensitization of its receptors by spillover and lowered clearance of sympathetic neurotransmitters and hormones. In parallel, parasympathetic tone goes down. The decrease in parasympathetic functioning is also reflected in lower resting state HRV, especially in the high

frequency domain; a cardiac vagal tone indicator. Sensitivity of the baroreflex — a blood pressure regulating mechanism — and the cardiac neuroreceptors decreases. Overall blood levels of norepinephrine (noradrenalin) increase, also because of diminished clearance of its hormonal form (Pflughaupt et al., 2006). Conversely, blood levels of epinephrine (adrenaline) decrease, in response to noxious stimulation (pain), as compared to younger people. In other words: while resting state levels of stress hormones are high, the adaptive responsivity to actual stressors decreases instead (the function of these substances). A review by Hotta and Uchida (2010) therefore concludes, that autonomic reactivity decreases in normal aging and that this also results in a decreased cerebral blood flow control. So additionally, brain regions that increase in activity and demand a higher oxygen supply, are served less efficiently. From this short summary, it can be concluded that the autonomous nervous system becomes less functionally reactive overall with increasing age. This implies that expectations from interventions trying to target this system in aging populations will have to be lowered, accordingly. The TCC study in **Chapter 3** was performed in an aging population. If potential cognitive enhancement by TCC should go through an autonomic route, as suggested by the rVNS model, then this implies that especially the target population of the elderly will have a cap on its benefits by cause of a changing autonomic nervous system and would need a very large sample (power) to show these incremental effects. As stated earlier, the sample in our TCC study was small and thus autonomic non-responsivity might be a reason for the null-results. Thus, this does not rule out the *existence* of autonomic effects, but only shows that they do not *occur* in this specific sample.

Thirdly, there might be lifestyle factors, that limit the expected gain of a meditative or breathing interventions, for example: *physical conditioning*.

Similar to aging, people with a low physical fitness show a decline in autonomous functioning (Fu & Levine, 2012). People who abstain from physical exercise show a disbalance in autonomic functioning characterized by vagal withdrawal and thus suffer from an overactive arousal system (Besnier et al., 2017). Furthermore, dysfunctional autonomic balance predicts, in turn, the ability and motivation to partake in physical activity; in other words: cardiac vagal activity determines an individual's ability to do exercise (Gourine & Ackland, 2018). Concluding, physical inactivity leads to a downward spiral of autonomic dysfunction and thus most likely affects the range of benefits a respiratory modulation can be expected to produce. It is currently unknown whether progressive vagal withdrawal is reversible (by respiratory modulation). Looking back at our studies, the TCC study had a highly physically active sample, which implies two things. First, that the participants probably had a healthy stress system (and autonomic balance), that could be receptive to intervention. However, the question then remains whether there was room for improvement. Second, that the physical component of TCC is likely to have been not challenging enough for further benefits on cognition. If this is the case, then a lack of improvement of executive functions in our study might also be led back to the meditative component of TCC. If enhancement could not be reached through way of physical exercise, the expected cognitive enhancement might still be caused through meditative practice, as reported by previous studies. This leads me to conclude that: either, the meditative instructions of the intervention were of insufficient quality (or not complied to), or the meditative aspects present in TCC, such as body awareness and breathing exercises, do not enhance cognitive control factors (in isolation).

The null-results might also be a results of differences in *individual baselines*. As described in the discussion of **Chapter 4**, the effects of

breathing interventions might very well depend on individual variations in respiratory patterns, stress states and the context they are applied in (whether ecologically valid or not). For instance, some individuals might have a high respiration rate at rest and might benefit from a manipulation that lowers their rate, decreasing stress and thereby performance on a cognitive task, as prescribed in the rVNS model. In contrast, another person might have a very relaxed state characterized by a relatively lower respiration rate and might be adversely affected by a further slowing of breathing. Instead this person might benefit from an activating manipulation, like slightly speeded breathing. A one-size-fits-all approach to modulating autonomic balance, as applied in our studies, might thus not work for everyone and this might hide the effects on those by whom it does. This would also explain null-results in our experiments. As these differences could overshadow differences between groups, or rather experimental conditions.

Fourth, human stress systems are highly individually divergent in general and in their *stress responsivity* specifically. People widely diverge in their perception of, response/adaptation to stress and susceptibility to adverse and pathological conditions, resulting from stress exposure (Ebner & Singewald, 2017; Sapolsky, 1994). The parameters of individual stress systems are already set prenatally and predispose the amplitude of adult stress responses, as was shown by the longitudinal studies of the Dutch famine that occurred during the end of World War II (Carroll et al., 2012; de Rooij, 2013). Inversely, large variation in the responsivity of individual stress systems diminishes what can be expected from manipulations of the autonomic system at the group level, for example by adjusting respiratory patterns. How far an individual is baseline stressed or reactive to stress might impact the effects of a relaxation exercise. If many non-responders – individuals that are not (much) influenced by parasympathetic activation —

are present in a sample, this also might hide the effects of the intervention, especially with a small sample. However, we do not have any indication that stress responsivity (and thus relaxation responsivity) differed or was skewed in our samples.

Lastly, the acute responsivity of individuals to respiratory interventions might very well be contingent on unknown mediating factors. This would explain why we do not find any acute effects on the stress system and cognition. However, the conclusion might then in some cases be that these acute effects do not *exist*. A prominent and necessary mediator of these effects might be *sleep*. The quantity and quality of sleep has a huge influence on all supposed affected variables of the rVNS model: cognition and (mental) health within the stress-related domain. Sleep not only plays a role in memory function, like sleep consolidation, and the development of executive functions, but a lack of it also increases the risk of developing many stress-related pathologies, such as: dementia, cardiovascular disease and immune system dysfunction. Actually, there is not much a healthy sleep pattern does not benefit (Walker, 2017). Specifically, sleep tones down the sympathetic nervous system and restores autonomic balance (Meerlo et al., 2008). Sleep is also a necessity for healthy executive functioning (Tucker et al., 2010). Thus without a good night of sleep, in quantity and quality, after an intervention aimed at these systems and before the effects are tested, there might be no benefits. Sleep consolidation is then necessary to observe the effects we are expecting to occur. Then, *acute* effects of respiration, do not *exist*. Next, I will make up the tally for the rVNS model from our current state of knowledge.

Respiration, rVNS and cognition

When we look back at the rVNS model in **Chapter 2**, we must conclude that we have not obtained any evidence for any of its predictions. For an overview and discussion of likely explanations, I refer to **Chapter 4** and the past sections. The identification of these factors leads to avenues of scientific enquiry that can either make, break or force adaptation of the rVNS model. At this point, it is too early to judge whether rVNS has any veracity and thus scientific merit. However, there are a few new studies by other authors on respiration and psychological effects that therefore deserve mention.

Grund and colleagues (2022) showed, with a tactile detection task, that when the onset of a tactile stimulus is synchronized to an individual's respiratory phase, its detection is increased. The rationale behind this occurrence is that due to the phenomenon of respiratory sinus arrythmia — the speeding of heart rate during inhalation and the slowing of heart rate during exhalation — there is an optimal timepoint in the respiratory cycle for stimulus detection. This is when heart rate is at its local peak (near the end of the inhalation phase) and thus arousal is at its highest (sympathetic tone). This is indeed what they found. Note that simply the existence of the phenomenon of respiratory sinus arrythmia indicates that respiratory modulations should have effects on arousal, stress and relaxation. Also, the uptake of oxygen — by respiratory rate and depth — is dynamically coupled with cardiac output: when breathing quickens, heart rate goes up, and vice versa (Rowell, 1993). A study by Klink and Pruessner (2023) convincingly showed that slow diaphragmatic breathing following a physiological stress induction (cold pressor test) leads to relaxation, as reflected in lowered heart rate and an increase in vagal tone HRV (RMSSD). Clearly, respiratory

patterns have an effect on autonomic functioning; on stress and relaxation. Only, this effect has not *occurred* in our experimental studies. However, the road from breathing exercises to effects on cognition remains less clear and especially the mediating role of the vagus nerve is currently causally unsubstantiated.

There is a substantial amount of literature on the link between vagal tone HRV and cognitive/emotional control or flexibility. This literature has been extensively described in **Chapter 2,** with some updates in **Chapter 4**. However, most of these studies are correlational or cross-sectional in design. This has important ramifications for the conclusions and inferences allowed by these results. As the famous maxim states: correlation does not imply causation. So, for example, Spangler et al., (2018) found that human beings with higher resting states of high frequency HRV show lower response inhibition rates in a threatening situation. Though this study had an experimental design it cannot be concluded from these results that higher vagal tone HRV causes or mediates these fluctuations in executive functioning, as they do not manipulate HRV levels and it thus remains a correlational finding.

In an intervention study that did have an experimental controlled design, De Smet and colleagues (2023) performed a transcutaneous auricular vagus nerve stimulation study, where they measured effects on vagal tone HRV (RMSSD) and on perseverative cognition (cognitive inflexibility). Surprisingly enough, though they did find a decrease in perseverative cognition due to the active stimulation as contrasted with sham stimulation, they did not find an overall increase in vagal tone HRV during active versus sham conditions. However, participants who did respond to the stimulation with increases in vagal tone HRV also showed the largest increase in

cognitive flexibility. Concluding, the relationship between afferent stimulation of the vagus nerve (as is the biofeedback of slow breathing), vagal tone HRV and cognition is not linear and is modulated by individual differences in the autonomous system.

When overviewing the literature domain on the link between HRV and cognition, it is remarkable that most to all of these studies come from the same lab or are at least co-authored by scholars who have introduced the neurovisceral integration model (Thayer & Lane, 2000). This observation is not meant to suggest that these studies are in any way unsound, unreliable or invalid. Indeed, two recent meta-analyses found no evidence of publication bias in the scientific literature on the link between vagal tone HRV and executive functioning (Liu et al., 2022; Magnon et al., 2022), though it was stated that publication bias could not be ruled out either. However, what we can conclude is that hardly any other author has either studied these effects or deemed their studies fit for publication, despite the popularity of HRV research. Also, it is clear that we have not found any relationship between vagal tone HRV and cognition in our empirical work. Though it is important to note, that we did not directly test the predictions of the neurovisceral integration model (or set out to). The predictions of this model are on a different time scale, than the predictions of the rVNS model tested in this book. The neurovisceral integration model predicts that there is a positive association between tonic cardiac vagal tone (long term) and cognitive functioning (trait), whereas our studies here test the rVNS model's prediction that respiratory parameters produce phasic changes in cardiac vagal tone and this relates to acute changes in cognitive functioning (state). As stated above, there is ample evidence for this correlation trait link between vagal tone HRV and cognition. See also the aforementioned reviews (Liu et al., 2022; Magnon et al., 2022), that both conclude that there is a positive association between

vagal tone HRV and executive functioning. Though it is important to note that the correlations are small and inherently uninformative on (possible) causality and directionality.

Though the mediating role of vagal tone HRV between autonomic manipulations and cognition is at this point unsure, this does not necessarily mean that the vagus nerve is not involved. Relaxation or activation due to respiratory adjustments can be mediated in other ways. The vagus nerve is a vast complex with many afferent and efferent routes, as well as projections upwards into the central nervous system. This is not all reflected in the measurement construct of vagal tone HRV. In other words: the connection between respiratory patterns and specific adaptive cognitive parameters still seems to have promise, even within a rVNS framework. For instance, the route of transcutaneous vagus nerve stimulation might go through the locus coeruleus, suggested by studies that have shown synchronization between stimulation and neuronal firing in the locus coeruleus (Hulsey et al., 2017). This alternative route is especially promising as a rVNS pathway because respiratory activity also synchronizes with the locus coeruleus (Melnychuk et al., 2018).

In this regard, the neurovisceral integration model is still compatible with the rVNS model. Especially, the updated version by Smith and colleagues (2017) is informative, as it provides a Bayesian brain interpretation of the model. In this hierarchical model of the central autonomous network, each level from the top level, the prefrontal cortex (or rather the executive control network), down to the bottom level — the heart — provides predictions to the level downstream. Subsequently, each level also gives prediction error feedback upstream (how far the prediction was from reality). Prediction error dynamics could be a candidate mechanism

through which biofeedback loops between respiration and executive functioning *occur*. Imagine a situation where the executive control network signals a perceived threat in a particular context. For example, a person learned that within a few moments a public speaking task must be performed. Then the prefrontal cortex (top level) instigates and readies downstream levels for a stress response (efference) and accompanies these signals with a prediction of higher peripheral stress levels (efference copy). However, downstream levels paint a different picture. Instead, cardiac vagal tone (bottom level) is high and thus speaks of low stress levels. This is because the person in question was doing slow breathing exercises, just before coming on stage. In response, the heart feeds back a high prediction error upstream (via the vagus nerve), as the prediction diverged far from the actual state of the stress system. Lastly, the executive control network reacts by turning down the perceived threat levels (i.e. adjusts its priors) and then further attenuates the stress response in its efference and efference copy downstream. In this way a clear biofeedback route has been created.

Let me offer a final suggestion how these two models might be combined. If we apply the rVNS perspective to the Bayesian neurovisceral integration model's description of the central autonomous network, the bottom level, which currently consists of the heart (vagal cardiac system) could be extended to the lungs and thus be supplanted by the more holistic cardiopulmonary system. Then, respiration rates and ratios are as informative as heart rate and variability. Thereby, the supposed mediating role of cardiac vagal tone can be circumvented as the sole possible vagal biofeedback mechanism.

Aforementioned limitations do not mean that taking vagal tone HRV measurements is without any merit. It might be useful as an indicator of

autonomic responsiveness as suggested by the results of De Smet and colleagues (2013). Another recent study (Manser et al., 2021) found vagal tone HRV reactivity to be a predictive biomarker for responsiveness in normal cognitive decline to intervention load. Individuals with higher vagal tone HRV reactivity show (larger) increases in cognitive functioning due to enhancement exercises, than do those with low reactivity.

Breathing exercises might also take other indirect ways of long-term benefits to cognition and (mental) health, for example: by increasing overall psychological well-being. Well-being is a strong predictor for many effects, such as delaying or preventing cognitive decline (Zhang et al., 2022) and all-cause mortality (Tamosiunas et al., 2019). All these aforementioned avenues deserve further study.

Conclusion

We have found no evidence for the claim that TCC enhances executive functioning, while there is strong evidence that it improves physical functioning, notably functional balance. Though the *non-occurrence* of the cognitive effects might be due to certain contextual factors, such as sample characteristics and compliance rates, we lean towards the conclusion that these cognitive effects do not *exist*.

Also, we were not able to find any evidence for the tested predictions of the rVNS model in our experiments. However, a number of studies that have been conducted by other labs have found confirmatory evidence for claims made by the rVNS model, specifically: that slow deep breathing increases vagal tone HRV. This together with the presence of contextual factors that can lead to cognitive effects to not *occu*r, that are mentioned

above, leads us to conclude that the rVNS model still holds promise and the *existence* of these predicted effects can still be shown to *occur* in other circumstances. However, we do have to conclude that *acute* enhancement effects (by respiration) of cognition have not *occurred* in our experiments and thus, we have not found evidence for their *existence*.

We started out this dissertation with a brief introduction to the history of cognitive psychology. The contrasting views on the mind between classical and embodied cognitive science were discussed in **Chapter 1**. Have we come any further in this reading? Not really. Our null-results do not give grounds to state something on this with any amount of confidence. The most I can say, is that we also did not find any cognitive effects without changes in more peripheral systems. Because we did not find any effects at all. Though we have not covered much ground on this topic, I do still maintain that cognition cannot be completely identified by computation and is therefore unsuited to be studied as such. The human mind is embodied, embedded and extended into its umwelt. But, as always, further study is required. For now, my suggestion to you, is instead of hooking up to the grid and disappearing into cyberspace, is to move into your body, experience, go outside with another human being, listen, talk.. and breathe deeply.