Neuronal brain activity is assumed to be reflected by blood level oxygenation levels, also known as BOLD signal, measured with fMRI. A scan of the literature points to both oxygen and carbon dioxide blood levels effects on BOLD fMRI. The roles of oxygen ($O_2$; hypoxia/hypoxemia, hyperoxia) and carbon dioxide ($CO_2$; hypocapnia, hypercapnia) in neuronal activity and BOLD signal are complex and still under investigation [1,2]. Inspiration while varying levels of $O_2$ and $CO_2$ in the air have been used to investigate resting state fMRI [3].

Blood oxygenation can be influenced by breathing patterns, which is usually considered as ‘physiological noise’ that needs to be controlled for during fMRI [4,5].

Studying breathing is not straightforward because of its combination of voluntary and automatic processes involved, and factors in respiratory control versus respiratory perception (for a review on neuroimaging and cognitive/emotional processing within brain structures) [6].

Variations in arterial $CO_2$ have an effect on resting state BOLD signal [7]. Breath-holding changes blood levels of $CO_2$ which results in an increase in BOLD-signal [8] while hyperventilating can reduce BOLD signal [9].

$CO_2$ levels are stronger related to BOLD signals than volume of breathing [10]. Furthermore, there can be individual differences in the cerebrovascular effects of $CO_2$ inhalation [11]. This is important information when one wants to measure, and correct for $CO_2$ levels in BOLD fMRI - measuring respiration volume just with a belt around the abdomen would according to this evidence be insufficient for a valid measuring.

Only few researchers have stated that interpretation of respiratory ‘confounders’ in measuring brain activity could be meaningful regarding the experience of emotion [12].

Recent research shows that the experience of different emotions, anxiety and depressive state, have different effects on breathing [13]. Anxiety disorders, and especially panic disorder, are known for the effects on breathing [14,15]. From another viewpoint, there is some indirect evidence of a possible con-
necion of depression and breathing, as serotonin receptors are central respiratory chemoreceptors [16].

Giardino et al [17] conclude in their review, that anxiety can influence cerebral blood flow by its effect on CO₂. In addition, research on breathlessness could contribute to the understanding of the association between affect and breathing patterns [18].

Sighing can be seen as a way to reset breathing irregularities caused by stress or sustained attention [19] and reduces anxious physiological tension [20]. Individual differences in trait-negative affectivity reflect in differences in sighing pattern [21] and there is evidence that sighing is a way to regulate emotions [22,23].

Sighing can also have an effect on the cardiovascular system [24].

So far, breathing patterns and the effects on brain activity or fMRI BOLD signals have mainly been studied in certain medical conditions like asthma/COPD and Obstructive Sleep Apnea (OSA). There is increasing evidence for the role of OSA in cognitive decline because of the resulting intermittent hypoxia in humans [25,26]. In rats, intermittent hypoxia can cause vascular aging [27] and in mice, plaques [28].

If you look at risk factors for the development of dementia more closely, it is visible that some of them have to do with breathing one way or the other: smoking (change in blood O₂ and CO₂ levels; reduced lung capacity), exercise (lung capacity) and obesity (amongst others due to the physical limitations to breathing, for an overview of respiratory physiology in obesity) [29].

Exercise is assumed to be beneficial to prevent cognitive decline by improving vascular health [30,31].

Normal aging can increase CO₂ blood levels as a result of arterial stiffness [32] and has an effect on lung capacity [33], and for this, exercise can be helpful.

It is not clear what the exact effect is of exercise on breathing in cognitive decline. There is some evidence of this add-on effect, as training in breathing can have a positive effect on cognitive decline that exceeds that of exercise training [34].

Another link related to breathing training could come from research on mindfulness, which is known for decreasing symptoms of depression and anxiety. Mindfulness training typically incorporates attention for breathing [35] and can impact on cardiovascular reactivity to stress [36].

A potential effect could be, that by learning to observe without changing behaviour -one of the values taught in mindfulness - this can have an effect on breathing patterns. Some evidence was found for the role of perception of interoceptive cues after mindfulness training in neuronal processing [37].

A review on meditation in the elderly showed that this can be helpful in the prevention of dementia [38].

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Conclusion

Anxiety and depression are known risk-factors in the development of dementia. Breathing patterns have an effect on BOLD fMRI and can be caused by affective state or go together with certain psychopathology. Cognitive decline is reflected in decreased BOLD fMRI.

The question is, if the link between psychopathology and dementia development could be (partially) explained by the effect of breathing patterns, related to affective state and/or psychopathology.

Individual habitual patterns are expected to especially manifest during resting state. This would be important in the research on dementia prevention as the areas that are active during resting state, are the first to become impaired in the development of dementia.

Future research

Future research may want to focus on how breathing patterns are associated with psychopathological disorders and what the respective effects are on neuronal brain activity and/or connectivity, measured with BOLD fMRI; what the potential chemical/metabolic effects in the brain are, in regards to breathing patterns and blood oxygenation; and how a) breathing patterns and b) chemical or metabolic effects of breathing play a role in cognitive decline.

References


