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Abstract

Dyslexia is one of the most common neurobiological disorders, affecting about 20% of children in Norway. According to phonological theory of dyslexia, the disorder is caused by a deficit in the representation, storage and recall of speech sounds. Different brain areas have been linked to the phonological deficit by means of different brain imaging techniques, among other qEEG. Some interesting studies conducted by Breteler and colleagues (2010) and Nazari and colleagues (2012) have tried to improve reading ability among dyslexics by means of neurofeedback training. Their results have been essential to the construction and the design of the present study.

The aim of this study was to investigate neurofeedback training to improve reading ability in Norwegian dyslexic children. The study was conducted as a pre-post intervention multiple case design with 5 participants, aged 14-15 years. The intervention consisted of 25 sessions of neurofeedback, 15 Theta/Beta training and 10 individualized training mostly at language areas. The effect of intervention was measured by means of qEEG and Logos (Norwegian dyslexia test).

The results showed improvement in reading ability and phonological skills among all participants. Furthermore, qEEG analysis showed some changes in theta, alpha and beta activity in several brain areas. The results also confirm the heterogeneity of dyslexia, and the complicity of many brain areas that are involved in dyslexia.

This study is limited by the small number of participants, and low control of other variables that may have an effect on the reading ability. The improvements in reading and phonological skills in this study suggest that further research in this area, with larger sample and several training sessions, is highly required.

Contents

Concept definitions

Brodmann area (BA)	A region of the human cortex defined on the basis of Brodmann´s organization.
Coherence	Aorrelation between EEG power in two or several separate brain regions.
Desynchronization	Auppression of the rhythmic activity / decrease in power.
Frequency	EEG rhythms range in frequency from 0-70 Hz.
Oscillation	Rhythmic activity.
Phasic	Response of a brain system to sensory stimulation detected by EEG.
Phonemic awareness	The ability to hear, identify and manipulate individual sounds/phonemes in words
Phonological decoding	Conversion of written symbols into speech sounds.
Synchronization	Spindles that are synchronized over large brain areas / increase in power.
Tonic	Background activity detected by EEG

Introduction

> All schoolteachers, regardless of talent and devotion, occasionally meet a child who is resistant to reading. His intelligence is normal, or even above average for mathematics or handicrafts. When it comes to reading, however, he suddenly becomes hopeless, stumbles on every syllable, mixes speech sounds, guesses at words without thinking, quickly gets discouraged and also discourages everyone around him. A visit to the speech therapist often confirms the dreaded diagnosis: this child suffers from dyslexia. (Dehaene, 2009)

Dyslexia

Dyslexia or reading disability is one of the most common neurobiological disorders, which according to U.S. National Institute of Child Health and Human Development affects 20% or approximately 10 million American children. The prevalence rates of dyslexia are almost the same among children in Norway (Dysleksiforbundet, 2016).

There are various terms used to explain dyslexia. These includes, among others: reading difficulties, specific reading disability/difficulty, reading disability, learning disability and specific learning difficulties/disabilities (Elliott & Grigorenko, 2014). The term that will be used in this study will be dyslexia. Alongside with all the different terms in use, there are also several definitions and attempts to explain the construct of dyslexia. A universally established definition of dyslexia is yet to be produced by the field. The definition chosen to be used in this study will be one by Lyon, Shaywitz & Shaywitz (2003), since it covers the most prevalent features of dyslexia. According to these authors dyslexia is:

> A specific learning disability that is neurological in origin. It is characterized by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. These difficulties typically result from a deficit in the phonological component of language that is often unexpected in relation to other cognitive abilities and the provision of effective classroom instruction. Secondary consequences may include problems in reading comprehension and reduced reading experience that can impede growth of vocabulary and background knowledge. (Lyon, Shaywitz, & Shaywitz, 2003)

This definition points out several important aspects of dyslexia such as its biological origin and unexpectedness, deficit in phonological processing of language, and difficulties in single word recognition, spelling and reading comprehension.

There are several theories of dyslexia and each of them emphasize different biological and cognitive causes of the disorder (Ramus et al., 2003). Neuroscientific research has examined three main theories about the underlying causes of dyslexia. The cerebellar theory (Nicolson, Fawcett, & Dean, 2001) is based on the assumption of a lesion in the cerebellum, which in turn leads to automatization deficits. The magnocellular theory (Stein & Walsh, 1997) based on the idea of a deficit in the magnocellular system of visual processing and the phonological theory that postulates a specific deficit in phonological processing of speech sounds (Breteler, Arns, Peters, Giepmans, & Verhoeven, 2010; Goswami, 2008; Habib, 2000; Ramus et al., 2003). Phonological theory will be the main theory used in the present study, primarily because of the majority of researcher and scientist in the field agrees on the deficit in phonological processing as the origin of most dyslexia (Dehaene, 2009; Elliott & Grigorenko, 2014).

The phonological theory

The phonological theory postulates that a core deficit in phonological processing lies at the origin of dyslexia (Stanovich & Siegel, 1994; Vellutino, Fletcher, Snowling, & Scanlon, 2004). According to this theory, dyslexia is mainly caused by a specific impairment in the representation, storage or retrieval of speech sounds. Imperfect representation of speech sounds causes problems in precise processing of spoken words. When the representation of speech sounds becomes degraded, it leads to problems with requiring phonological skills such as phonological coding, which is the conversion of written symbols into speech sounds. That in turn makes difficult to establish strong links between visual and verbal counterparts of printed words. This is likely to affect the ability to store high quality representation of word spellings, and as a result, rapid word recognition and reading fluency is impaired (Dehaene, 2009; Elliott & Grigorenko, 2014; Habib, 2000; Ramus et al., 2003; Ramus & Szenkovits, 2008).

The phonological deficits are usually divided into three core components, which are poor phonological awareness, poor verbal short-term memory and slow lexical retrieval (Elliott & Grigorenko, 2014; Ramus & Szenkovits, 2008).

Phonological awareness refers to an individual´s ability to recognize and manipulate the sounds of spoken language. It is an explicit process, which requires reflection on

phonological structure or the sound structure of words. The development of phonological awareness proceeds from the ability to isolate larger sound units like words, to intermediate units (rhymes/onsets) to the smallest units, namely phonemes. Traditionally used tests to measure phonological awareness are tests of evaluate rhyme awareness and phonemic awareness (Goswami, 2008; Melby-Lervåg, Lyster, & Hulme, 2012). The impairments in dyslexics are usually observed in rhyme judgments or in the segmentations of words into component phonemes. For instance may they have deficits in differentiating "won" and "one", or breaking "leg" into "l", "e", "g" (Dehaene, 2009).

Learning to read involves coding, storage and retrieval of stable associations between speech and written language (Dehaene, 2009). In relation to dyslexia, most studies have focused on two types of memory, namely verbal short-term and working memory (Swanson & Ashbaker, 2000). Verbal short-term memory involves the passive storage of information while working memory involves both storage and processing of information, and requires attentional processes as well. Regulating attentional control by executive system is important in relation to for example tasks that require reading comprehension (Carretti, Borella, Cornoldi, & De Beni, 2009). Verbal short-term and working memory are usually tested by recall of digits or word-sequence (Elliott & Grigorenko, 2014; Melby-Lervåg et al., 2012).

Lexical retrieval, which refers to the speed in which an individual can name visual stimuli that are already known to the, is evidenced in rapid automatized naming (RAN). Measures of RAN involves how fast the individual can name a sequence of known items such as letters. This ability is associated with reading fluency (Elliott & Grigorenko, 2014; Ramus, 2004).

There is now agreement among theorists and researchers in the field on the central and causal role of phonological deficits in dyslexia. Several studies including Ramus et al. (2003) show that the most significant cognitive problem of dyslexic individuals lies in phonological skills. In this study the authors used several phonological tests: rapid picture naming, rapid digit naming, spoonerisms in accuracy and production time and non-word repetition. The results of their study indicate that all the dyslexics performed significantly poorer than controls in all phonological tests. Based on the results, Ramus et al. (2003) concluded that all the dyslexics in their study suffer from a phonological deficit. In agreement with Ramus et al. (2003), Paulesu and colleagues (2001) claim that the phonological processing deficits are a universal problem for dyslexics. Their study which was conducted with Italian dyslexics, showed that the dyslexic group performed poorly in reading-related phonological tasks, relative to controls (Paulesu et al., 2001; Ramus et al., 2003).

Some other studies have focused on the presence of phonological deficits before the onset of formal reading instruction (Boets et al., 2010; Pennington & Lefly, 2001). A longitudinal study conducted by Boets et al. (2010) in Belgium assessed all three dimensions of phonological ability in Dutch speaking children in three different stages of formal reading instruction: last year of kindergarten, first grade and third grade (Boets et al., 2010). Group comparison showed that dyslexics scored significant lower in all three stages, leading Boets et al. (2010) to concluded that a general phonological deficit in dyslexics is present before onset of formal reading instruction. The same observation was made in another longitudinal study carried out by Pennington and Lefly (2001), with English speaking children. In their study, Pennington & Lefly followed children from kindergarten to second grade and measured their phonological abilities four times. High-risk dyslexic readers presented consistent phonological deficits in all three measures and in all levels of formal reading instruction (Pennington & Lefly, 2001).

The dyslexic brain

Reading begins as a phonological process (Dehaene, 2009; Goswami, 2008), since learning to read an alphabetic system requires learning grapheme-phoneme correspondence. In other words, one needs to learn the association between letters and speech sounds. At the start of this process, the brain areas for the spoken language are most active. As we becomes experts in reading, an area in the visual cortex called visual word form area (VWFA), also called letterbox area, becomes increasingly active. The VWFA is active during nonsense word reading, and is thought to store orthography-phonology connections (Cohen & Dehaene, 2004; Dehaene, 2009; Goswami, 2008)

Several brain imaging studies of dyslexia find impaired brain activity in various reading-related brain areas, namely the left posterior temporal areas, left inferior frontal areas and left occipitotemporal areas (McCrory, Mechelli, Frith, & Price, 2005; Paulesu et al., 2001; B. A. Shaywitz et al., 2002; S. E. Shaywitz, Morris, & Shaywitz, 2008; S. E. Shaywitz et al., 1998; Temple et al., 2001). These areas are involved in the phonological aspects of reading and in the process of developing an orthographic lexicon (Brunswick, McCrory, Price, Frith, & Frith, 1999).

A study coordinated by Paulesu and colleagues with Italian, French and English dyslexics showed that a large area of left temporal lobe was deficiently active in dyslexics (Paulesu et al., 2001). These researcher visualized dyslexic´s brain activity using PET while the subjects were asking to read words. The same anomaly was observed for all three

nationalities at the same brain location to the same degree of impairment. Their PET study points to a universal cerebral origin for dyslexia, namely the left temporal lobe that seems consistently disorganized. A close examination of their work reveals two nearby areas in the dyslexic´s brain that are under activated, the left lateral temporal cortex and the letterbox area (VWFA). Another study conducted by McCrory, Mechelli & Price (2005) also suggest that the letterbox area may not work appropriately in dyslexics (McCrory et al., 2005). In this study, the brains of normal and dyslexic participants were scanned using PET, while written words, which they had to repeat out loud, and simple line drawing were presented to them. Dyslexics showed underactivation in the left occipitotemporal region, which is the location for letterbox area (McCrory et al., 2005; Paulesu et al., 2001).

Several neuroimaging studies of children with dyslexia show a similar pattern of activation to Paulesu et al. (2001) ´s findings. For instance, Shaywitz et al. (1998) showed that dyslexics had underactivation in posterior regions included Wernicke´s, striate cortex and angular gyrus, when compared to nonimpaired subjects. Dyslexics showed also overactivation in inferior frontal gyrus (S. E. Shaywitz et al., 1998). They concluded that the impairment in dyslexia is phonologic, and it appears as dyslexics fail to systematically increase activation in posterior cortical system, as the difficulty of mapping print to phonologic structures increase. In another study with dyslexic children, Shaywitz et al. (2002) reported underactivation in the core left temporoparietal networks among children with dyslexia, which is in agreement with their previous study with adult dyslexics (B. A. Shaywitz et al., 2002).

However, unlike adult dyslexic, dyslexic children in this study showed also underactivation in inferior frontal gyrus. Shaywitz and colleagues suggest that older dyslexic engage frontal regions to compensate for the disruption in posterior regions. In a further study, Shaywitz and colleagues found out that the brain system for reading in dyslexic readers differs when comparing to nonimpaired children (B. A. Shaywitz et al., 2007). According to the authors, dyslexics reading system are primary localized in more posterior and medial regions rather than a more anterior and lateral occipitotemporal region. Their findings indicate that there is a single reading system in the brain that develops with age. For dyslexic readers, this system is localized in posterior medial occipitotemporal region, while for nonimpaired readers it is localized in anterior lateral brain areas (B. A. Shaywitz et al., 2002; B. A. Shaywitz et al., 2007; S. E. Shaywitz et al., 1998).

As already mentioned, children with dyslexia show differences in activation of phonological brain areas such as left superior temporal and inferior frontal cortex (Georgiewa et al., 2002). Findings from a fMRI study gives support to the hypothesis that dyslexics differ

from controls in letter-sound integration. These findings demonstrate that dyslexics have an under activated superior temporal gyrus, which is directly associated with reduced auditory processing of speech sounds. This observation was in turn associated with poor performance on phonological tasks (Blau, van Atteveldt, Ekkebus, Goebel, & Blomert, 2009).

What is EEG?

Electroencephalography is an extracranial registration of the brain´s electrical activity. The rhythmic nature of the brain´s electrical activity was first discovered in human´s EEG by Berger in 1929 (Kropotov, 2010). The electric signal is a result of field potentials recorded by electrodes placed on the scalp. Each electrode reaches an area of five million pyramidal neurons. It was recognized early that the electrical activity contained bursts of sinusoidal waves occurred in a predictable way, and that they corresponded with mental states. It was also observed that the amplitude and frequency of the rhythms vary across different mental and behavioural states and across different areas in the brain. In other words, the recorded signal reflects communication processes between neurons and neuronal circuits (Destexhe & Sejnowski, 2003; Kaiser, 2007; Paul Sauseng & Klimesch, 2008).

The primary contributor to scalp recorded EEG appears to be summated postsynaptic potentials, that are synchronized IPSP´s and EPSP´s, generated particularly by cortical pyramidal cells (Kirschstein & Köhling, 2009). Changes in the resting membrane potential of the pyramidal cells that leads to action potentials, leads to the release of neurotransmitters, which in turn activate postsynaptic receptors. This activation generates IPSP´s and EPSP´s. Thus, the EEG waves are a results of summation of a large number of synchronized IPSP and EPSP (Destexhe & Sejnowski, 2003; Kirschstein & Köhling, 2009). The thalamus is considered as a critical site responsible for the rhythmic activity of cortex. The cortex, on the other hand, plays an active role in generating large-scale synchrony. It seems that it is a functional connectivity between cortex and thalamus through the generation of oscillations (Davidson, Jackson, & Larson, 2000).

Rhythms of the Brain

The brain´s rhythmic activity can vary across frequency and time. The method of spectral analysis, by means of Fourier Transformation, decomposes the EEG data into sinusoidal wave patterns (Buzsaki, 2006). This method localizes brain waves distinctly in frequency and represents the intensity and magnitude of brain waves in micro volts (absolute

power) or in percentage distribution (relative power) in the spectrogram. The frequencies of brain rhythms are usually divided into five frequency bands that are called delta (1-4 Hz), theta (4-8 Hz), alpha (8-13 Hz) Beta1 (13-21 Hz), Beta2 (21-30 Hz) and gamma for frequencies above 30 Hz (Hammond, 2007). Theta, alpha and beta oscillations can be observed in the normal EEG in different states of resting conditions, with eyes closed or eyes opened, and during different task conditions. Delta oscillations are present mainly in different states of sleep. Theta, alpha and beta oscillations in different cortical areas, and with different parameters (frequency, amplitude, coherence and phase) are thought to reflect cognitive processes and mental states (Kropotov, 2010).

Theta oscillations are the dominant oscillations in hippocampus of lower mammals, with a frequency band of 3-12 Hz, which is a much wider frequency span than in humans. Animal studies have provided evidence that theta oscillations are related to memory processes when new information is encoded, especially episodic memory. Theta activity in the rat´s hippocampus has been shown to be related to coding of locations by affecting place cells (Paul Sauseng & Klimesch, 2008). Kahana and colleagues provided evidence for dominant theta oscillations in the human hippocampus as well (Kahana, Sekuler, Caplan, Kirschen, & Madsen, 1999; Wolfgang Klimesch, 1999; Klimesch, Doppelmayr, Russegger, & Pachinger, 1996). According to Tesche and Karhu (2000) theta oscillations are not only involved in processing of input in hippocampal networks, they are also related to working memory processes (Tesche & Karhu, 2000).

Alpha oscillations of 8-13 Hz, mainly at parietooccipital areas, are the dominant rhythm of the human brain (Wolfgang Klimesch, 1999). Three main types of alpha rhythms can be recorded in different areas of the brain: posterior rhythms recorded at occipital or occipital-parietal areas, the mu rhythm recorded over sensory-motor strip and midt-temporal tau rhythm (Kropotov, 2010). Even though alpha rhythms are generated at cortical areas, it is assumed that they are driven by activity from the thalamic nuclei, especially the pulvinar nuclei (da Silva, 1991). Alpha oscillations have widespread functional relevance, they are referred to as idling rhythm of the brain and are desynchronized in the presence of sensory information. In other words, the alpha amplitude is related to the level of cortical activation. An increase in alpha activity is associated with cortical and behavioural inhibition. These oscillations are also engaged in memory processes, attentional processes and specific perceptual processes (Paul Sauseng & Klimesch, 2008; Wolfgang Klimesch, 1999; Wolfgang Klimesch, Sauseng, & Hanslmayr, 2007).

Beta oscillations appear in several brain areas and are mainly associated with motor activity (Paul Sauseng & Klimesch, 2008). Increase in beta activity is associated with motor cortex activity. This rhythm is also suggested to play an important role during attentional processes and higher cognitive functions. Findings from animal studies shows that beta oscillations in neocortex is related to a state of high alertness and focused attention on a target (da Silva, 1991). Several experiments has shown that during visual attention tasks, enhanced beta activity appears in visual areas (da Silva, 1991; Neuper & Pfurtscheller, 2001). The beta rhythm in frontal areas appears also during cognitive tasks that are related to stimulus judgment and decision making (Kropotov, 2010).

EEG and dyslexia

Several studies have tried to understand the pattern of brain´s electrical activity, using EEG, in individuals with dyslexia. A study conducted by Rippon and Brunswick compared event-related EEG changes in one phonological task and one visual task, performed both by a group of dyslexic children and a control group consisting of age-matched children with normal reading ability (Rippon & Brunswick, 2000). Opposed to the control group, EEG responses from the dyslexic group showed a lack of task-related decrease in the alpha amplitude. Dyslexics showed an increase in theta amplitude while controls showed a reduction in theta activity. This increase was located frontally in the phonological task while controls showed desynchronized theta activity in the same areas. As for beta activity levels, the dyslexic´s beta amplitude increased in the parieto-occipital area while controls showed decrease in activity in this same area. Rippon and Brunswick suggest that the combination of lack of alpha desynchronization and increase in theta activity among dyslexics can indicate poor attentional and memory processes.

Several relevant studies have been coordinated by Klimesch and colleagues to investigate whether dyslexics show deficits in attentional networks and/or semantic encoding (Wolfgang Klimesch et al., 2001; W Klimesch et al., 2001). According to these researchers, EEG oscillations in alpha and theta band reflect cognitive and memory processes, if a dissociation between tonic and phasic band power changes is taken into account (Wolfgang Klimesch, 1999). In their study with dyslexics, subjects were recorded while they were reading numbers, words and pseudowords and their EEG data was analysed in theta, alpha and beta band powers. The findings showed that dyslexics fail to show increase in theta power in occipital sites during processing of pseudowords, which can reflect that dyslexics have reduced ability to encode pseudowords into the working memory system. Another interesting

finding was that, while controls show a highly selective left hemispheric processing of words, dyslexics encode words bilaterally (Klimesch et al., 2001). Findings from alpha analyses revealed that dyslexics showed significant lower desynchronization at occipital areas during word and pseudoword encoding, what can be interpreted as a reduced ability of attentional control. Additionally, dyslexics showed increased alpha desynchronization at anterior sites under all three task conditions, which according to Klimesch et al. means that dyslexics fails to show selective semantic word encoding. In respect to beta band powers, dyslexics displayed reduced selective pattern of activity compared to controls which indicate a lack of selective topographic pattern for the processing of words, pseudowords and numbers (Wolfgang Klimesch et al., 2001).

Deviant theta activity among dyslexics was demonstrated in another study by Spironelli, Penolazzi, Vio & Angrilli (Spironelli, Penolazzi, Vio, & Angrilli, 2006). Italian dyslexics were compared with controls during a phonological task consisting of whether or not two words rhyme and a semantic task comprising words that were either semantically related or not. For the phonological task, dyslexics showed greater theta amplitude over right hemisphere areas while controls showed left lateralized theta amplitude. The same pattern of theta activity was shown for the semantic task, with greater right lateralized theta activity among dyslexics. The inverted lateralization of dyslexics was mainly located in posterior brain areas. According to the authors, the right hemisphere activity is a consequence of left posterior area deficits. Spironelli and colleagues concluded that the right lateralization of theta activity in dyslexics is consistent with their linguistic deficits. Their study supports the linguistic deficit that is measured at both behavioural and electrophysiological level, especially during phonological tasks.

EEG coherence refers to functional connectivity between different brain areas. High coherence between EEG frequencies at different areas suggests increase in functional cooperation between the underlying neuronal networks (Weiss & Mueller, 2003). Some EEG studies have considered EEG coherence to be an interesting measure to demonstrate deviation in functional connectivity in individuals with dyslexia. Shiota, Koeda and Takeshita (2000) reported significantly higher coherence values both intra- and interhemispheric in dyslectic children. Another interesting finding is a study conducted by Arns, Peters, Breteler & Verhoeven (2007), which found increased coherence in frontal, central and temporal regions. The core dysfunction in dyslexia according to Arns et al. (2007) seems to consist of increased slow activity(delta and theta activity) at left frontal and right temporal regions. They also

found bilateral increased coherence in the delta and theta bands (Arns, Peters, Breteler, & Verhoeven, 2007).

Neurofeedback

Neurofeedback is a procedure in which one can train the brain directly. The method is classified as a type of EEG biofeedback that is based of one´s brain oscillatory activity. The purpose of neurofeedback is to affect the activity of the brain in a self-regulating way (Ros et al., 2009). The technique is based on principles for operant learning mechanisms, meaning that by measuring current brain activity and rewarding it, it is possible to change it into more appropriate pattern of activity. Influencing one´s brain activity is normally difficult because one is not aware of the activity´s pattern. The fact that one can see one´s own brain activity few milliseconds after it take place, gives the individual the power to control and change it. The changes are at first short-term but can progressively become more persisting. During typical trainings, a small number of electrodes are connected to the brain region of interest and one or two electrodes are placed on the earlobe as references. The electrical activity detected by the electrodes are digitized by means of specialized hardware and software and provides immediate feedback about brain´s activity (Hammond, 2007). The signal mapped into some form of feedback can be either visual, auditory or both. The changes in activity can be displayed in form of graphs or changes in colour or patterns (Yucha & Montgomery, 2008).

According to Sherlin et al. (2011) there are certain principles that must be followed for the neurofeedback training to be effective. To achieve the desired training effect, one must ensure a discrete and uncomplicated setup of the equipment, and as much as possible of noise and artefact must be avoided. The feedback and reinforcement must be fast and the signal or behaviour under training must be specific. Other reinforcements must be directly connected to the learning process and in order to ensure generalization of the neurofeedback training to real-life situations one must include transfer trials (Sherlin et al., 2011).

Neurofeedback has been applied with various purposes, to different patient groups and healthy individuals. Many clinicians are using neurofeedback in the treatment of ADHD (Arns, de Ridder, Strehl, Breteler, & Coenen, 2009; Arns, Heinrich, & Strehl, 2014) and there is an interesting meta-analysis conducted by Arns, Ridder, Strehl, Breteler & Coenen (2009) that evaluate the empirical evidence for the effect of neurofeedback on symptoms of ADHD. These researchers concluded that neurofeedback could be regarded as clinically suitable treatment in symptom reduction of ADHD. Neurofeedback is, according to Arns et al. (2009)

considered as an effective treatment of ADHD in line with medication (Arns et al., 2009). But unlike medication, there are few or none negative side effects of neurofeedback (Hammond et al., 2011; Leins et al., 2007) and the effects of the treatment are long term effects (Gani, 2009). The technique's application is not limited to ADHD, but includes among other the treatment of epilepsy with significant reduction on seizure frequency (Tan et al., 2009), treatment of addiction (Gabrielsen, 2012), children with learning disabilities (Fernández et al., 2007), and to optimize performance in healthy subjects (Gruzelier, 2014a). In order to optimize performance in healthy subjects, the focus has been on improvement of different types of attention, executive functions, reaction time, memory, spatial skills and calmness (Gruzelier, 2014a).

In accordance with the different frequency bands, and the different brain areas, which should be of interest for the neurofeedback training, there are many possible training protocols. Which protocol to use depends of the aim of the training and the symptoms of the individual. When choosing a protocol, the focus can be to increase or decrease the amplitude of one or several specific brain waves. For neurofeedback training to be effective it is important to personalize the training to the individual´s brain activity pattern. Therefore, it is essential to perform an assessment that my include the individuals current symptoms, clinical history, psychological tests and EEG recording prior to neurofeedback training (Hammond, 2007; Hammond et al., 2011). On the other hand, standardized protocols are required if the objective is to compare results across studies to evaluate the effect of neurofeedback training. Examples of standardized training protocols are among other Sensory Motor Rhythm protocol (SMR), Alpha/Theta (A/T) training protocol and Theta/Beta protocol (Arns et al., 2014; Gruzelier, 2014a)

Theta/Beta protocol has the focus on regulating the relationship between the amount of beta and theta activity and have mostly been used in studies/treatment of ADHD. The choice of Theta/Beta protocol in treatment of ADHD is based on the findings of higher levels of theta and lower levels of beta among individuals with this condition (Arns, Conners, & Kraemer, 2012; Lubar, 1991). Theta reduction by means of theta/beta protocol is an adjustment of a deviant enhanced rhythm. In ADHD children, higher theta activity is related to attentional problems (Doppelmayr & Weber, 2011), while increased theta activity among healthy individuals is related to focused sustained attention (Sauseng, Hoppe, Klimesch, Gerloff, & Hummel, 2007).However, for the neurofeedback to become a more efficient and personalized treatment, individualized protocols may be required (Yucha & Montgomery, 2008).

Neurofeedback and dyslexia

A few research studies have been conducted with neurofeedback and dyslexia. Breteler, Arns, Peters, Giepmans & Verhoeven (2010) carried out the first randomized controlled study on neurofeedback treatment for dyslexia (Breteler et al., 2010). Their study included children aged 8-15 years, divided into an experimental and a control group, who completed 20 session of neurofeedback training. The training was based on personalized protocols in accordance with the participants qEEG outcome, and the researcher's presumed associations about deviations with dyslexia. The results showed a large improvement in spelling among the dyslexics, but no improvement was found in reading. According to Breteler and colleagues, improvement in spelling may be due to attentional processes involved in this ability. Their study was limited by lack of selection based on several subtypes of dyslexia and the fact that their training protocols were based on eyes-open qEEG only, and no reading task.

Another study conducted by Nazari, Mosanezhad, Hashemi & Jahan (2012) explored the effectiveness of neurofeedback training on reading disability and EEG coherence (Nazari, Mosanezhad, Hashemi, & Jahan, 2012). Their multiple case study involved six 8-10 years old children with reading disability receiving 20 sessions of neurofeedback training. Results from the study showed significant improvements in reading and phonological awareness skills, without any notable changes in the EEG analysis in power bands. Furthermore, there were significant changes in coherence towards normalisation in theta at T3-T4, delta at Cz-Fz and beta at Cz-Fz, Cz-Pz and Cz-C4. Nazari and colleagues suggest that normalization of coherence in dyslexics may indicate integration of sensory and motor brain areas and more cerebral maturity.

The present study

The studies conducted by Breteler et al. (2010) and Nazari et al. (2012) have been essential to the construction of the present study. The design of the present study was inspired by a combination of the two studies that has been mentioned above, with both standardized and individualized neurofeedback protocols. Choice of protocols is based on Breteler et al. (2007), which found that children with dyslexia exhibited increased slow EEG activity in frontal and temporal regions.

The main objective of this study was to investigate neurofeedback training to improve reading ability of Norwegian dyslexic children.

The following research questions are covered:

1. Does neurofeedback training improve reading abilities of dyslexic children?
2. Does neurofeedback training lead to changes in qEEG of dyslexic children?
3. Are changes in qEEG related to changes in reading ability?

Methods

Selection strategy and subjects

The sample was obtained from Stokkan secondary school in Stjørdal through educational coordinator at school. Parents of all the children with dyslexia diagnosis at Stokkan got an information paper about the study (see Appendix 1.). Interested parents and their children were invited to a meeting with project supervisor and coordinator. The only criteria for participation was dyslexia without any comorbid diagnosis.

The project was designed as a prospective pre- and post intervention study for children/adolescents with dyslexia. Five adolescents, four boys and one girl, aged 14/15 years with the dyslexia diagnosis participated in the study. All of the participants had got their diagnosis in early elementary school. No one had other diagnosis except from dyslexia, and none used medication.

Table 1

Subject data

Subject	Age	Gender
T16	15 years	Boy
V16	15 years	Boy
K16	14 years	Boy
H16	14 years	Boy
A16	15 years	Girl

EEG Recordings

EEG was recorded pre- and post treatment using 19 thin electrodes attached with conductive gel and placed on the scalp. The electrodes were distributed on the scalp according to the 10-20 system (see Figure 1.). Reference electrodes were placed on each earlobe. The signal was amplified using a Mitsar 201 multi-channel digital EEG system with a sampling rate of 250Hz. The EEG data was processed using WinEEG 2.81.25 Software.

The recording was carried out during two different conditions. It was initiated with three minutes resting state with eyes open (EO) and then three minutes resting state with eyes closed (EC). The subjects were instructed to relax and avoid muscular movement while recording.

Participants were sitting in a comfortable chair during recordings. The recording took place in a room at school, after regular school hours

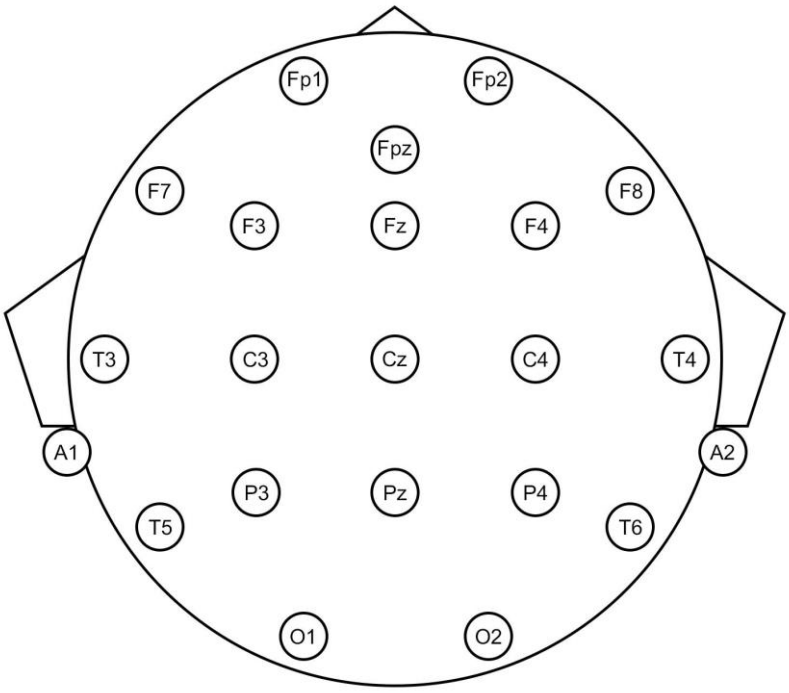


Figure 1. Positioning of the 19 electrodes according to the 10-20 international system

Eye blink artefact and muscle artefact were corrected using Independent Component Analysis (ICA) on the raw data. ICA is a separation technique that decomposes the EEG signal into better spatially and temporally components (Makeig, Bell, Jung, & Sejnowski, 1996).

Statistical analysis

Analysis of data material was performed in Mitsar WinEEG. Using Mitsar WinEEG allows estimations of statistical significance of deviations of the EEG activity, this by comparing EEG spectral variables with the database. The database used in this study is provided by Mitsar and contains data recorded from children in St Petersburg, Russia. The accepted levels of statistical significance was $p<0.05$. The EEG spectral variables for comparison were frequency (theta, alpha and beta) and region (frontal, central, parietal, temporal and occipital). Only relative power values (percentage of frequency distribution) were included in the comparison. Analysis were carried out for EO.

Source localization

In order to distinctly localize the EEG parameters, activation in different cortical areas must be decomposed by means of Independent component analysis. All significant deviations in spectral variables were localized by calculating independent component spectral analysis (ICSA), by means of WinEEG.

Low Resolution Tomography/sLORETA. EEG signals are generated by distributed sources and cortical generators. To better localize sources of the EEG signal a method named Low Resolution Tomography(LORETA) was used. sLORETA is a standardized version of LORETA. The spectral components were analysed using sLORETA to determine the standardized current density, which is distributed across the cortical gray matter.

Logos test

To evaluate the effectiveness of neurofeedback training, a reading test named Logos was also conducted pre and post-intervention. Logos was conducted at school with project coordinator present. The participant was guided through the test.

Logos is an individual, computer based test that identifies ones reading skills. The test is based on an adjusted version of Spear-Swerling and Stanovich´s reading development model named "Off-track model" (Spear-Swerling & Sternberg, 1994). The model emphasizes three processes in the acquisition of reading, namely "decoding", "reading fluency" and "reading comprehension". Logos consists of fifteen subtests that maps different skills in reading. The subtests included in Logos identifies reading fluency and reading comprehension, listening comprehension, decoding, reading-related partial skills and reaction time.

The results from Logos are displayed for each subtest in terms of percentile. Values below 15 percent are referred to as within critical level, and values between 15 and 30 percent are referred to as within concern level. Values between 30 and 100 percent are referred to as within normal level.

Neurofeedback Intervention

The intervention consisted of 25 sessions of neurofeedback, 15-20 min. each time. All the participant received the same Theta/Beta training the first 15 times, and individualized training the remaining 10 times. The intervention took place one to three times a week during the period November 2015 to June 2016.

Theta/Beta Training Protocol. Theta/Beta protocol is meant to enhance activity in the higher frequency band and inhibit activity in lower frequency band. The training was given by means of Brain Tuner instruments (St. Petersburg, Russia), and by using one reference electrode at right earlobe and two electrodes placed at Fz and Fpz. The participant was placed in a comfortable chair about 1 meter from a 19 inch computer screen.

Each session was initiated with two minutes threshold setting, then the actual training consisting of five minutes focus-training and one minute relaxation. A horizontal line divided the screen into two halves and the participant was supposed to keep the pillar above the midline (threshold) as much as possible during training sessions. During the relaxation, the participants were instructed to relax. At the end of each session, the activity of the training and relaxation periods were displayed graphically on the computer screen. This visualization was meant as an evaluation of the session and to compare the actual training session with the participant´s own experience.

Individual protocols. T16 continued the Theta/Beta training the remaining 10 sessions. V16, A16 and H16 got the same training protocol at T4 and T6, which was supposed to inhibit activity in theta and alpha frequency bands and enhance activity in beta frequency band. K16 got the same training protocol at T3 and T5.

Ethical consideration

All of the participants and their parents were informed about the background for the study, implementation, potentially effects and side effects, and the right to resign anytime without consequences. The participants signed an informed consent form prior to the study (see Appendix 2.). The study was presented for Regional Etisk komitee (REK). According to REK, this project did not require their approval.

Results

Comparison of power spectrum

The analysis of qEEG of the individual power spectrum in pre-intervention and post-intervention measurement, compared with Mitsar reference database, showed significant higher activity for alpha (n=4), theta (n=4) and beta (n=4) frequency bands ($p<0.01$). The results of deviant spectral analysis are presented in Table 2.

Localization of deviant spectral component

The deviant spectral components revealed by comparison with the Mitsar database, which was separated by means of Independent component spectra analysis and localized by sLORETA, suggest different sources of deviation for subjects. Distributions of the deviant frequencies over cortex were mainly in occipital and language-related areas (parietal and temporal lobe).

Deviant alpha activity. Two of five subjects showed higher alpha activity than the normative database in the pre-intervention measurement, while four of the subjects showed higher alpha activity in the post-intervention measurement. The distribution of the deviant alpha activity in the pre-intervention was in occipital areas (BA 17,18,19) and temporal areas (BA 20, 21, 37). Deviant alpha activity in post-intervention was distributed mainly in occipital areas (BA 17, 18, 19), temporal areas (BA 20, 21, 22) and parietal areas (BA 40 & 42). Three subjects displayed reduction in alpha frequency after intervention, this in occipital (BA 17, 18, 19) and temporal areas (21, 37).

Deviant theta activity. Higher theta activity was displayed in four subjects in the pre-intervention, and there were still higher theta activity in three of subjects in the post-intervention. The deviant theta activity in pre-intervention was distributed across occipital cortex (BA 19), temporal areas(BA 20, 21, 37), parietal cortex (BA 40) and frontal areas (BA 40). The distribution of deviant theta activity in the post-intervention measurement was almost the same as in pre-intervention, except two subjects which showed higher theta activity in Broca´s area (BA 44 and 45) and hippocampal areas (BA 36). Reduction in theta activity was displayed in three subjects when pre-intervention and post-intervention were compared, mainly in occipital areas (BA 18,19) and temporal areas (BA 20, 21, 22) and anterior cingulate cortex. One subject had increased theta activity in the post-intervention

compared to the pre-intervention in frontal areas (BA 10, 46 and 47) which are language related areas.

Deviant beta activity. Four subjects showed higher beta activity in the pre-intervention recording. Two subjects had excessive beta activity in prefrontal and frontal areas (BA 9, 10, 44, 46 and 47), one had temporally located excessive beta activity (BA 20, 21, 22) and one showed excessive beta activity in both prefrontal and frontal areas (BA 10 and 47) and temporally areas (BA 20,21,22). In the post-intervention recording, only two subjects showed deviance in the beta-band, both with beta excess in frontal areas.

Table 2

Sources (Brodmann areas) of significant higher activity in pre- and post-intervention comparison with norm-database and pre-post intervention comparison for each subject.

	Alpha			Beta			Theta		
Subject	Pre-Norm	Post-Norm	Pre-Post	Pre-Norm	Post-Norm	Pre-Post	Pre-Norm	Post-Norm	Pre-Post
T16			19 21 37	9 44			19 40	9 18 19	9 18 19 10 46 47
V16	20 21 37	20 21 37					20 21 37	20 36 37	24
K16	17 18 19	18 19	18 19	9 10 46 47	9 10 32				
H16		17 18 19 40 21 22 42		10 47 20 21 22	9 10 47		19 40		
A16		17 18 19	17 18 19	20 21 22	20 21 22		32	32 9 44 45	20 21 22

Brodmann´s areas and the related functions localized via sLORETA of the deviant spectral activities by frequency are summarized in Table 3. The organization is according to Brodmann´s Interactive Atlas.

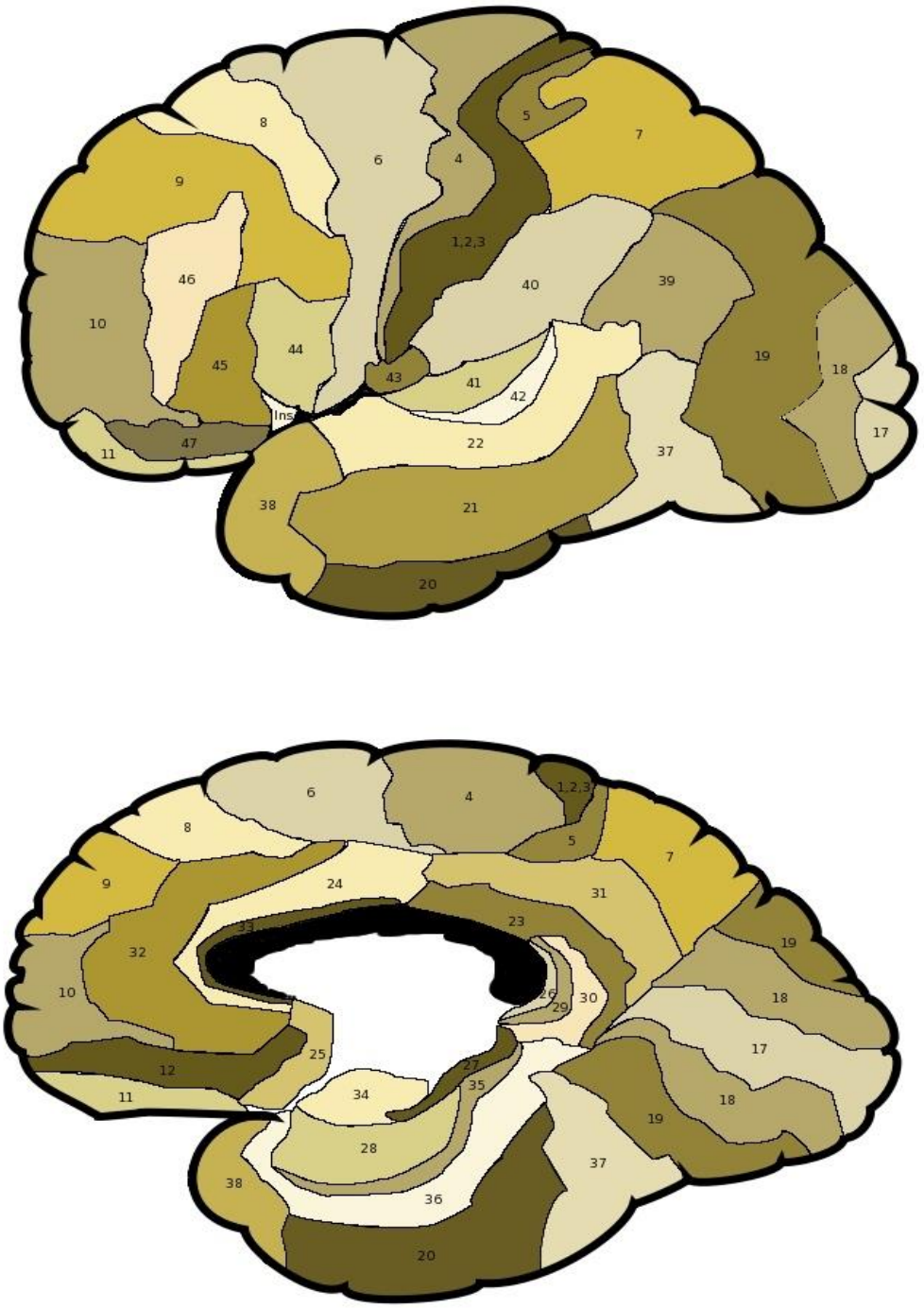


Figure 2. Brodmann´s Interactive Atlas

Table 3

The positioning of deviant spectral components, with best match in Brodmann´s area (BA),

BA	Localization	Function	Freq.
9	Prefrontal	Working memory, executive control	θ β
10	Prefrontal	Working memory, executive control	θ β
17	Occipital	Primary visual cortex	α
18	Occipital	Secondary visual cortex	α θ
19	Occipital	Secondary visual cortex	α θ
20	Temporal	Language; involved in lexico-semantic processing; language comprehention	α θ β
21	Temporal	Language; Involved in semantic processing; Word and sentence generation	α θ β
22	Temporal	Receptive language; involved in semantic processing; sentence generation; selective attention to speech; repeating words	α θ β
24 & 32	Anterior cingulate gyrus	Language; involved in semantic and phonological verbal fluency Working memory	θ β
37	Temporal	Language; involved in semantic categorization; attention to semantic relations; single letter processing	α θ
40	Parietal	Language; involved in attention to phonological relations; semantic processing	α θ
42	Temporal	Primary auditory cortex	α
44	Frontal	Broca´s area; involved in semantic and phonological fluency; phonological and syntactic processing; ghrapheme-to-phoneme conversion	θ β
45	Frontal	Broca´s area; involved in semantic-phonological processing; verbal fluency	θ β
46	Prefrontal	Language; involved in semantic processing; verbal fluency; phonological processing Working memory	θ β
47	Frontal	Language; involved in semantic encoding and processing; Phonological processing Working memory	θ β

brain localization of areas and functional description.

Behavioural data

The results from the logos test, which was conducted twice, both pre-intervention and post-intervention, indicate that all participants improved their scores in several subtests (see Table 4). The subtests with most improvement included phonological reading, reading fluency, working memory, rapid naming of numbers and reading comprehension. An example of Logos profile score, from one of the participants will be presented in Figure 3 and 4.

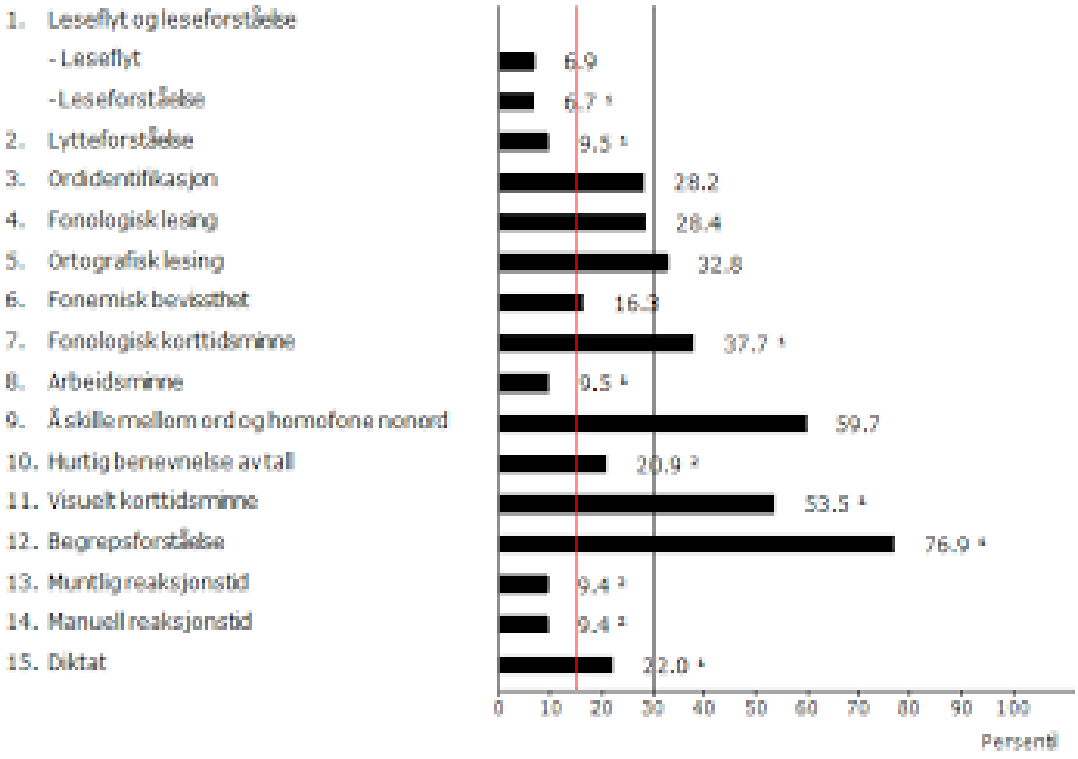
Table 4

Results from Logos improvements for each subject.

Subject	Improved subtets	From critical level to concern level	From concern level to "Normal" level	From critical to "Normal" level
T16	13 of 15	- Reading fluency - Verbal reaction time	- Word identification - Phonological reading - Phonemic awareness - Rapid naming of numbers - Dictate	- Reading comprehension - Working memory
V16	12 of 15	- Reading fluency - Dictate	- Verbal reaction time	- Distinguishing between words and homophonic nonwords
K16	12 of 15	- Orthographic reading - Distinguishing between words and homophonic nonwords		- Visual short-term memory
H16	10 of 15	- Rapid naming of numbers	- Working memory - Phonological reading - Distinguishing between words and homophonic nonwords	
A16	11 of 15	- Rapid naming of numbers	- Manual reaction time	- Phonological reading

PROFIL RAPPORT

Elev:		**Testing:**	1. testing
Trinn:	9	**Fødselsdato:**	
Skole:		**Oppgavesett:**	6 - voksne
Testleder:	Administrator, Logos	**Testnorm:**	Trinn 8

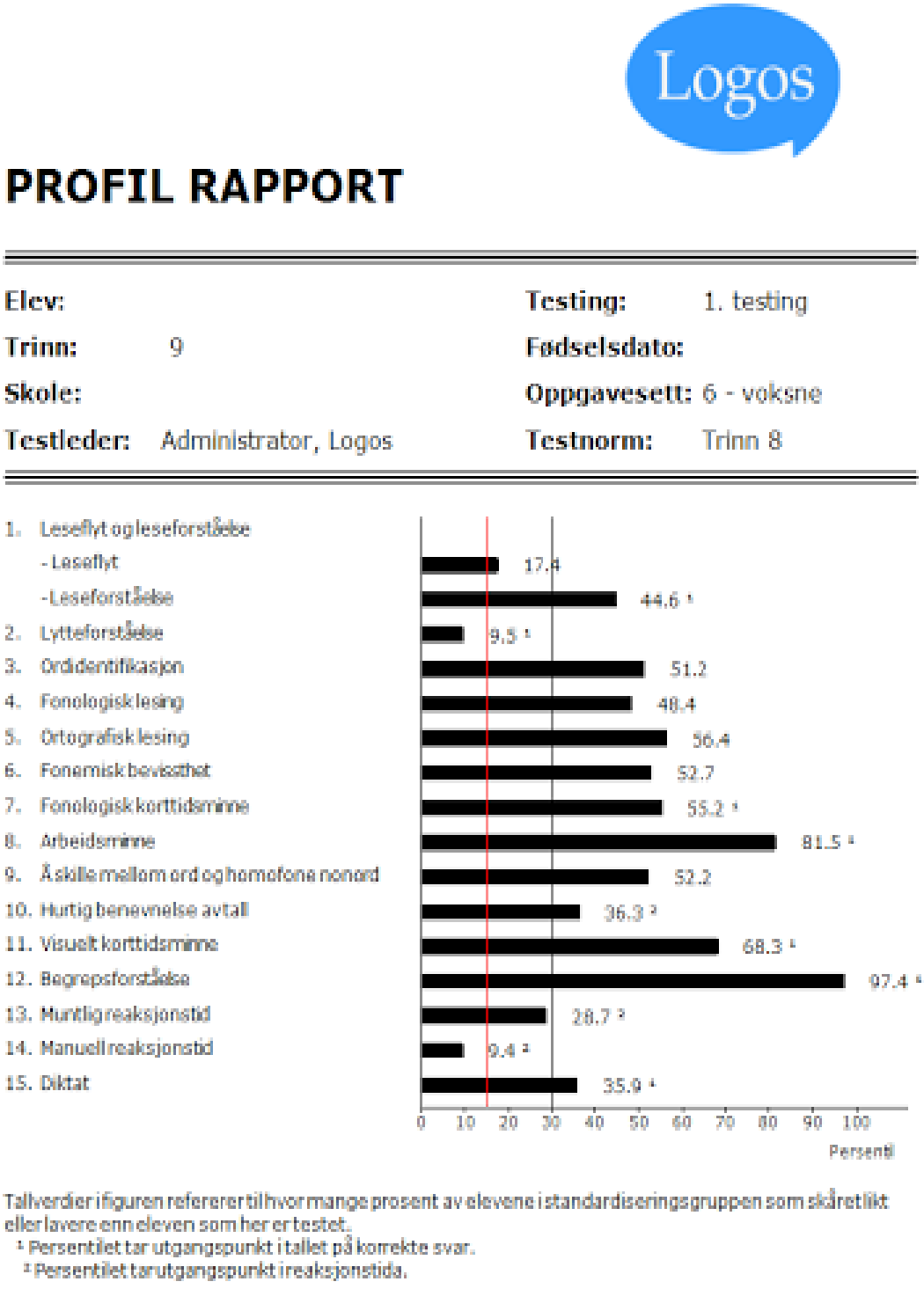


Tallverdier i figuren refererer til hvor mange prosent av elevene i standardiseringsgruppen som skåret likt eller lavere enn eleven som her er testet.

[1] Persentilet tar utgangspunkt i tallet på korrekte svar.

[2] Persentilet tar utgangspunkt i reaksjonstida.

Figur 3 Pre-intervention Logos results, subject T16.



Figur 4. Post-intervention Logos results, subject T16.

Discussion

The main aim of this study was to examine the efficiency of neurofeedback training in dyslexic children as well as to evaluate the relationship between changes in EEG data and changes in dyslexia symptoms. This was done by a pre-post measurement by means of EEG and Logos. Our results showed considerable improvements in dyslexia symptoms, and some changes in EEG data have been found. The results from Logos showed improved reading ability, which strongly supports the use of neurofeedback to reduce symptoms of dyslexia. Furthermore, individual differences in dyslexia profiles were clearly observed in the qEEG and Logos measures. Such variations make standardized intervention problematic. The fact that neurofeedback can be tailored to individual profiles makes it a suitable treatment for dyslexics.

Behavioural data changes and qEEG changes

As shown in Table 5., three subjects (T16, V16, A16) showed decreased slow activity in language areas in temporal and frontal lobe. The same subjects showed improvements in reading skills that incorporate a time component such as reading fluency, verbal reaction time and rapid naming of numbers. This can be interpreted as a general enhancement in response and reaction time, related to reading processes.

Decreased slow activity in visual areas in three subjects can also be observed. Slow activity, especially alpha activity is associated with blocking of stimulus processing. This observation may indicate improved processing of visual stimuli. Seen in connection to improved Logos results, better visual processing may be of importance to many reading abilities.

The fact that at least one participant (H16) showed improvements in Logos without significant changes in qEEG can raise questions about other variables that could have played a role during intervention phase. However, since the results by this study were encouraging, this calls for further research in this area that could control for other variables.

Table 5

Behavioural data changes and qEEG changes for each subject.

Subject	Logos Improvements	qEEG changes
T16	Reading fluency	Decreased alpha in 19, 21, 37
	Verbal reaction time	Decreased theta in 9,18,19
	Word identification	
	Phonological reading	
	Phonemic awareness	
	Rapid naming of numbers	
	Dictate	
	Reading comprehension	
	Working memory	
V16	Reading fluency	Decreased theta in 24
	Dictate	
	Verbal reaction time	
	Distinguishing between words and homophone nonwords	
K16	Orthographic reading	Decreased alpha 18, 19
	Distinguishing between words and homophone nonwords	
	Visual short term memory	
H16	Rapid naming of numbers	
	Working memory	
	Phonological reading	
	Distinguishing between words and homophone nonwords	
A16	Rapid naming of numbers	Decreased alpha in 17, 18, 19
	Manual reaction time	Decreased theta in 20, 21, 22
	Phonological reading	

Improved reading ability

Results from the behavioural test show considerable improvements in reading ability among all participants. This is clearly evident in the results from LOGOS since it shows improvements for every subject in several subtests. This effect contrasts with the study carried out by Breteler and colleagues that report improvement in spelling but no improvement in reading ability (Breteler et al., 2010). This might be, to some extent, due to the age of the subjects participating in their study, which was on average four years younger compared to the subject´s age in this study. Spelling is a more fundamental constitute of reading ability for younger readers, while more developed reading skills such as reading fluency and reading comprehension are essential for older readers. Nazari et al. (2011) on the other hand, reported improvement in both reading errors and reading time. Their results are consistent with the present study´s results, which showed both improved reading fluency and reading comprehension for several participants.

One interesting finding is that all participants had one level improvement in one or more subtests related to memory, such as working memory. As mentioned in the theory section, memory processes, especially working memory and verbal short-term memory are essential constituents of the reading process. Swanson, Zheng & Jerman (2009) conducted a meta-analysis of research that compared children with and without reading disability on measures of short-term memory and working memory (Swanson, Zheng, & Jerman, 2009). They found considerable poorer skills among children with reading disability on phoneme-related short-term memory measures and working memory measures requiring processing and storage of digits and words. According to these researchers, their results converge with other studies that suggesting that working memory system does play a critical role in the reading process. Therefore, improved memory capacity may bring advantages for dyslexic individuals. On the other hand, involvement of memory system is yet another reason why dyslexia is such a complex condition.

In addition to improved memory, the subjects improved scores with at least one level in one or several reading related subtest, such as phonological reading, orthographic reading, phonemic awareness, RAN and discriminating word and non-word. As mentioned in the theory section, skills such as phonemic awareness and phonological reading are essential contributors to reading ability. Furthermore, according to the phonological deficit theory, and data from the present study, these abilities are especially challenging for dyslexics. Therefore, improvements in these reading-related skills seem to be of significance for dyslexics.

Two subjects showed improvements in reading fluency. Increased reading fluency is important because it can enable decoding to become a more automatic process relieving cognitive resources that can then be applied to meaning related processes, or so to say, understanding the information in the text. Improved reading fluency by automatizing reading skills may lead individuals to catch more of the content of what they are reading. In addition, four of five participants improved their scores in rapid naming of numbers, which is the same as RAN. As mentioned in the theory section, RAN is related to reading fluency and as one can see in the results, four of five subjects improved their scores with one level in rapid naming of numbers.

Changes in EEG

The present study showed significant higher theta activation among the participants, compared to norm data, at language areas such as Broca´s, Wernicke´s and temporal gyrus, and in visual areas, both in pre- and post-intervention. Arns et al. (2007) have previously reported higher levels of theta activation among their dyslexic participant at Fp1, Fp2 and F7 (Arns et al., 2007). As their results are mainly based on deviant activity measured at the scalp, and does not reflect the source of the deviant activity, which makes result comparison between their results and the present study difficult. Nonetheless, when looking at coherence data, Arns et al. (2007) reported much broader deviant activity in frontal and temporal regions for both right and left hemisphere. According to Arns et al. (2007) the core dysfunction of dyslexics is increased slow activity at frontal and temporal areas. Rippon and Brunswick (2000) reported also increased theta at frontal regions during phonological tasks performed by dyslexics (Rippon & Brunswick, 2000). They further hypothesize that increased theta activity, in addition to lack of alpha synchronization may be an indicator of poor attentional and memory processes.

When comparing the pre-intervention with the post-intervention data, it can be observed that two participants showed decreasing theta activation at temporal areas (V16 and A16). This reduction may be interpreted as activity toward normalization. Another interesting finding was the increase of theta activity at prefrontal areas in two other participants (H16 and T16). As mentioned in the introduction, theta activity is involved in hippocampal and memory processes, and also in processes related to working memory (Neuper & Pfurtscheller, 2001; Tesche & Karhu, 2000). It is possible that increased theta activity in prefrontal areas can indicate enhanced memory processes. The same participant showed an increase from critical to normal level in working memory task measured in the behavioural data.

There was significant higher alpha activity in two participants in the pre-intervention condition, mainly in occipital and temporal areas. There was still significant higher alpha activity in four subjects in the post-intervention condition, at the same areas as pre-intervention. However, when pre-intervention and post-intervention registrations are compared, there was a general reduction of alpha activity, mainly in occipital areas for three subjects and occipital and temporal reduction for one of the subjects. Alpha activity is primarily associated with cortical and behavioural inhibition (Wolfgang Klimesch et al., 2007; Paul Sauseng & Klimesch, 2008). Higher alpha activity in visual areas may indicate ineffective processing of visual stimuli. It may seem that the subjects in this study face challenges when processing visual stimuli, even in the absence of direct visual stimuli. Therefore, the neurofeedback training conducted in this study may not be adequate to reduce the deviant alpha activity observed in visual areas, which was not initially the purpose of the neurofeedback training. Nevertheless, the recorded reduction at temporal regions in only one subject is both challenging and interesting, since it may show that reducing alpha activity in temporal areas, which was one of the aims of the individualized protocols, may be difficult.

Deviant beta activity was observed in four participants at temporal, parietal and frontal areas in pre-intervention condition. In post-intervention condition, there were only three subjects that still showed deviant beta activity. Two of the participant showed increased beta activity in the post-intervention condition at areas involved in working memory capacity.

Deviation in activity at visual areas measured among four subjects is an interesting finding that may provide support for the magnocellular theory. It is not surprising that several brain areas and cognitive resources are involved in the ability to read (Dehaene, 2009) and this is just another evidence confirming the complexity of dyslexia as a condition. Even though the phonological deficit theory remains the dominant theory in the field, the notion of a single theory explaining fully the complex condition of dyslexia may be hard to sustain (Snowling & Hulme, 2012).

Dyslexia heterogeneity

There are different labels to describe the condition of dyslexia (Elliott & Grigorenko, 2014) making it difficult to compare results across studies (Vellutino et al., 2004). In addition, using various terms is one of the major reasons that complicate the creation of a universally accepted definition of dyslexia. There is still disagreement among scientists and researcher about the reasons for reading difficulty and the fundamental features of dyslexia. While most researchers agree on the phonological deficit as the primary cause for dyslexia, others still

emphasize sensorimotor deficit and see phonological deficit as a secondary cause (Ramus, 2003). Uncertainty about the manifestation of dyslexia is evidenced in the variation in prevalence rates that ranges from 5% to 17.5% (S. E. Shaywitz & Shaywitz, 2005). Furthermore, the lack of a unified definition of dyslexia enables inclusion criteria to vary in different studies. This again leads to difficulties when comparing results from different studies. Dyslexia is a neurobiological condition but is diagnosed based on behavioural data, which complicates the understanding of the condition even more.

Dyslexia can manifest itself as a wide and varied range of difficulties. Dyslexia is, therefore, not a homogeneous condition, but include different subgroups, each with its own features and properties. The results of this study also confirm the assumption of heterogeneity of dyslexia. It is important to emphasize the fact that dyslexia is not one condition, but a complex set or pattern of neurobiological and behavioural traits that may differ from one individual to another. The subjects included in the present study show how each individual may have a different pattern of difficulties when it comes to dyslexia. At the same time, one can see that all of the participants show some impairment in memory processes or executive processes, in addition to difficulties related to reading. Thus, it might be possible to find main categories of difficulties/disabilities present in all individuals suffering from dyslexia, with individual variation within each category. Creating a common understanding of difficulties related to dyslexia may lead to easier comparison across studies, while allowing variability. Additionally, clear understandings of the dyslexia construct will bring benefits for the attempts to help those who struggle to master the process of reading. Use of multi-method approach, by for instance EEG, for studying dyslexia may also contribute to a better understanding of this complex condition.

The DSM-5 framework proposed the term dyslexia to be replaced by "specific learning disorder". This according to The International Dyslexia Association will be a misleading term (Elliott & Grigorenko, 2014), since dyslexia is more than a reading difficulty, although reading difficulty is the primary marker and because the condition of dyslexia incorporates both language processing and language production, as well as other adaptive skills.

Methodological issues and limitations

The small sample size in the present study, in addition to the diversity of dyslexia symptoms based on qEEG and logos profiles leads to limitation when it comes to the generalization of the results. The small number of participants is due to restrictions in time

and resources for the present study. The few numbers of relevant participants with only dyslexia and no comorbid diagnosis is another reason why the sample size in the present study is small. On the other hand, the small numbers of participants makes it possible to have a closer look in each individual´s profile, which can be considered as strength, given the heterogen nature of dyslexia as a condition. The present study also lacked a control group.

Another limitation with the present study is the lack of coherence measures, again due to restrictions in time and resources. Several of previous studies have reported coherence data (Breteler et al., 2010; Nazari et al., 2012), which limits the possibility for comparison with the results of the present study. Conclusions based on activity in single brain areas should be done carefully since dyslexia is a complex condition that involves simultaneous activity in many brain areas.

In addition, the present study´s choose of neurofeedback protocol, in accordance with Breteler et al. (2010), was based on presumed deviant activity among dyslexics, and eyes-open qEEG only. More closely analysis of EEG data as a basis for choose of neurofeedback training, alongside with qEEG where a reading task is involved, could empower the design of the present study.

EEG recording and neurofeedback training were carried out after school when the subject were tired. This caused shorter training sessions than is recommended for best effect. Additionally, the number of training sessions were limited to 25, which according to Hammond (2007) is not enough for best learning outcomes (Hammond, 2007). However, Egner and Gruzelier (2001) reported learning effect after ten sessions (Egner & Gruzelier, 2001; Gruzelier, 2014b). It is also important to mention that the recordings and training session were conducted in a room at school with poor sound insulation.

Conclusion and implications for future research

The present study finds evidence that neurofeedback training can be an efficient treatment for symptom reduction in dyslexia. Despite the limitations of the present study, it is possible to claim considerable improvements in reading ability among the participants. Nevertheless, a clear understanding of the construct of dyslexia, in terms of definition, and inclusion criteria, is crucial for the development of the field and raise quality in research, to ultimately provide help for those who struggle to master the process of reading. Furthermore, use of assessment tools beyond behavioural assessment, such as EEG, may be beneficial in better understanding the condition of dyslexia.

Future investigations with larger samples are highly needed. It is also interesting to look at the long-lasting effect of neurofeedback training. In addition, research on individualized neurofeedback protocols is needed. Future studies should also improve control of other variables that may have an effect in addition to neurofeedback training.

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Appendices

Appendix 1

Informasjonsskriv til foreldre, foresatte og barn

Bakgrunn for studien

Mitt navn er Parissa Azadi og jeg går på masterstudiet i psykologi ved NTNU. Høsten 2015 skal jeg starte et masterprosjekt om effekten av nevrofeedback-trening på dysleksi eller dyskalkuli.

Nevrofeedback er en metode som tar utgangspunkt i individets hjernebølgeaktivitet, som på forhånd blir målt ved hjelp av EEG. I dag brukes denne metoden som alternativ behandlingsmetode for en rekke lidelser som blant annet lærevansker, eller for normalt fungerende mennesker som ønsker å forbedre sin mentale kapasitet.

Hensikten med studien er å undersøke effekten av nevrofeedback på barn med dysleksi/dyskalkuli. Det vil si å undersøke om nevrofeedback fører til forbedring på målinger av lese, skrive eller matematiske evner. Nevrofeedback er spesielt interessant da det viser få eller ingen bivirkninger. Og studier på nevrofeedback i andre land har vist potensialet for varige, positive endringer i symptombildet hos barn med lærevansker.
Metoden har inntil nylig vært ukjent i Norge, men har stor utbredelse internasjonalt. I Norge har legekontoret Barn og Unges Potensial, knyttet til Helse-Sør, praktisert nevrofeedback siden 2005 på barn og ungdom med ulike problemstillinger som skaper utfordringer i hverdagen.

Hva innebærer studien?

Deltakelse i prosjektet innebærer en pre- og postmåling av hjerneaktivitet av den enkelte ved hjelp av EEG, samt ca. 30 økter med nevrofeedback-trening.
EEG målingene foregår ved at man har på seg en hette med elektroder som måler hjerneaktiviteten til den enkelte. Det vil vare i ca. 1 time.

Deretter blir det gjennomført en tilpasset hjernetrening(nevrofeedback) i form av visuell tilbakemeldingsøvelse ved hjelp av en elektrode som festes til hodet (språkområdet) og en datamaskin. Hver hjernetreningsøkt tar ca. 30 minutter.
EEG målingen og nevrofeedback-treningen er smertefritt og uten noen form for risiko elle bivirkning.
EEG målingene vil foregå på NTNU Dragvoll. Siden nevrofeedback utstyret er mobilt, kan hjernetreningen foregå der det måtte passe for deltakeren.

Hva skjer med opptak og informasjonen om deg?

EEG-opptakene av deltakerne vil bli brukt kun som beskrevet under hensikten med studien. Alle opplysninger og EEG-opptak vil bli behandlet uten navn og fødselsnummer eller andre direkte-gjenkjente opplysninger. En kode blir knyttet til dine opplysninger og EEG-opptak gjennom en navneliste. Navnelisten oppbevares på skolen og blir ikke brukt på universitetet. Det er kun autorisert personell direkte knyttet til prosjektet og med taushetsplikt som har adgang til navnelisten. EEG-opptakene vil ikke kunne knyttes til deg som person.
Dersom du ønsker å få se resultatene fra opptaket kan du ta kontakt med prosjektansvarlig.

Frivillig deltakelse

Det er frivillig å delta i studien. Du trenger ikke å gjøre noe aktivt om du ikke ønsker å delta. Du kan når som helst og uten å oppgi noen grunn trekke ditt samtykke til å delta i studien. Dersom du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Om du nå sier ja til å delta, kan du senere trekke tilbake ditt samtykke.
Dersom du senere ønsker mer informasjon om studien, kan du kontakte:

Prosjektansvarlig og kontaktperson: Parissa Azadi
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Appendix 2

Samtykke til deltakelse i studien

Jeg ønsker å delta i denne studien.

(Signert av deltaker, dato)

Vi gir tillatelse til at vårt barn kan delta i denne studien.

(Signert av foreldre/foresatte, dato)

Jeg gir min tillatelse til at EEG-opptak av mitt barn kan benyttes i videre studier ved EEG-lab/ inngå i en EEG-database i EEG-lab JA ☐ NEI ☐