

[Open Peer Review on Qeios](#)

# 14-channel neurofeedback with Auto Train Brain improves the left lateralization of the brain in dyslexia: A pilot study

Gunet Eroglu

**Funding:** No specific funding was received for this work.

**Potential competing interests:** No potential competing interests to declare.

## Abstract

Auto Train Brain is a neurofeedback-based mobile application that increases reading comprehension and reading speed in dyslexia with EMOTIV EPOC-X which has 14 channels. The clinical trials have been completed on dyslexia beforehand. The left hemisphere-related deficits are known in dyslexia. In this research, we have investigated the positive long-term effects of Auto Train Brain to improve the cognitive abilities of dyslexic people based on the measurements of the variance of gamma band sample entropy across neurofeedback sessions. The previous research indicates that the increase in the variance of the gamma band entropy shows the increased adaptations in the functional networks. 14-channel neurofeedback with Auto Train Brain increases the variance of gamma band entropy in the left temporal lobe (T7) over the right temporal lobe (T8) which may be translated as the adaptations of the functional networks in the left temporal region are increased after 100 sessions of neurofeedback in terms of electrophysiology.

## Günet Eroğlu

*Faculty of Engineering and Natural Sciences, Bahçeşehir University, İSTANBUL*

<https://orcid.org/0000-0001-8382-8417>

[gunet.eroglu@eng.bau.edu.tr](mailto:gunet.eroglu@eng.bau.edu.tr)

0-90-532- 326 55 82

**Keywords:** Neurofeedback, sample entropy, Auto Train Brain, learning disorders, dyslexia, EEG, Brain Computer Interface, wearable device.

## I. Introduction

Dyslexia is a subcategory of Specific learning impairments according to DSM 5 criteria<sup>[1]</sup>. Some people struggle with reading, despite having IQs that are normal or above average<sup>[2]</sup>. Regarding the underlying cause of dyslexia, numerous

theories have been proposed. The genetic origin of dyslexia is the most well-known of these explanations<sup>[3]</sup>. Children who have dyslexia are more likely to have dyslexic parents<sup>[4]</sup>. The other common theories about dyslexia are that genetic predisposition, environmental conditions, maternal stress, maternal activated-autoimmune response during pregnancy, and infections during pregnancy are the root causes of this phenomenon to happen<sup>[5]</sup>. Newer theories suggest that this condition is the result of a delay in brain development. According to the "Delayed Neural Commitment (DNC)" theory, dyslexic children take longer to develop (and rebuild) the neural networks that support learning to read, in addition to having delayed acquisition of skills. The framework offers a crucial time link between the early development of language, and speech networks, and the development of executive function networks<sup>[6]</sup>. The functional network in the left hemisphere of the brain in dyslexia is not fully developed, and there is hypo-functional connectivity that seems not related to IQ.

Even if children with dyslexia receive the necessary support education and adequate nutrition, it takes a very long time to close the gap between their peers. Sometimes this difference cannot be closed during their lifetime. One or more parts of phonological processing are missing, such as the ability to consciously manipulate speech sounds (phonological awareness), to temporarily store phonological information in the verbal short-term memory, and to quickly retrieve long-term phonological representations<sup>[7]</sup>.

Another theory about dyslexia is related to maternal activated-autoimmune response during pregnancy. Neurodevelopmental pathways are altered before birth. The immune response in the child is altered postnatally<sup>[8]</sup>. Excessive neurotransmitter formation triggers an autoimmune response, and inflammation begins<sup>[9]</sup> due to the insufficiency of fatty acids. The presence of inflammation delays the development of the brain. As long as the inflammation continues after birth, brain maturation lags continue. Only the right brain has the opportunity to develop for dyslexic children. The development of the left brain and left lateralization of the brain is not yet completed in dyslexia<sup>[10]</sup>. Establishment of the left-brain dominance is important before the child reaches school age<sup>[11]</sup>. Visual and auditory cues must be processed quickly in the left posterior lobe in order for the child reaches the maturity required for the development of reading skills.

Another body of research asserted that individuals with dyslexia may exhibit a disordered interhemispheric functional asymmetry<sup>[11]</sup>. As a result, the corpus callosum of the dyslexic brain undergoes alterations that impair the flow of motor and sensory information between the two hemispheres.

It is hypothesized that there is a disconnection syndrome in the left temporal lobe of dyslexia<sup>[12]</sup>. QEEG measurements display the increased slow brain waves in the left temporal region of the dyslexic brain<sup>[13]</sup> and/or there may be general EEG slowing. Temporal lobes are important for brain maturation and functional connectivity, and this connectivity seems missing in dyslexia<sup>[14]</sup>.

There are various subtypes of dyslexia. A number of recent studies have also discovered that dyslexia has been strongly linked to various characteristics, including underlying basic auditory processing deficiency<sup>[15]</sup>, impaired visual processing<sup>[16]</sup>, attentional deficits<sup>[17]</sup>, defective eye movements<sup>[18]</sup>, and irregularities of processing<sup>[19][20]</sup> and some have defects in combining the visual and auditory input in the left angular gyrus<sup>[21]</sup>. People with dyslexia are unable to efficiently decode written letters (graphemes) into their corresponding sounds (phonemes)<sup>[22]</sup>. Apart from the cortex, some subcortical structures may also be affected<sup>[23]</sup>. According to the cerebral deficit theory<sup>[24]</sup>, deficiencies are brought

on by a lack of development of articulatory skills, which in turn comes from an ontogenetic cerebellar malfunction. The cerebral deficiency theory may provide behavioral explanations for the difficulty of people with dyslexia in time estimating, motor skills, working memory, and balancing tasks.

Dyslexia causes problems in understanding words, pronunciation, and syllables. Because of this, a child with dyslexia frequently struggles with language and verbal expression and is unable to distinguish between words based on their phonemes due to poor hearing and comprehension skills. These children are normal in other aspects or just a little smarter than average. They might be daydreamers dealing with low self-esteem, anxiety, and despair as a result of their academic struggles<sup>[25]</sup>.

It is known that gluten-free diets<sup>[26]</sup>, special education, neurofeedback<sup>[27][28]</sup>, and multi-sensory learning<sup>[27]</sup> are the only effective solutions to reduce the symptoms of dyslexia. It should be noted that these solutions do not cure the root cause of dyslexia, they only improve brain maturation.

According to numerous studies, children with dyslexia have slow waves at FC5 and F7 and do not desynchronize beta-1 activity while performing a reading task in regions connected to Broca's area (FC5; speech production, articulation) and the Angular gyrus (CP5, P3), understanding semantics and mathematics<sup>[29]</sup> as well as the left parieto-occipital area (P7, O1)<sup>[30]</sup>. Right temporal and parietal (P8 and T8) areas of the brain have elevated sluggish activity in children with dyslexia<sup>[31]</sup>. According to the researchers, there is a disruption in the left temporal region<sup>[32]</sup>. Additionally, there may be a high level of frontal sluggish activity in individuals with dyslexia and ADHD. The coherence increases symmetrically. At T3 and T4, the delta and theta bands exhibit a symmetric increase in coherence, while the alpha and beta bands exhibit a distinct right-temporal central increase in coherence<sup>[31]</sup>. Bi-hemispheric hyper-coherence (between T3 and T4) appears in the delta and theta bands, however, between P7 and O1, there is hypo-coherence in the delta, theta, and alpha bands. Dyslexia is accompanied by issues with the gamma band and less functional connections<sup>[33][34]</sup>. The left and right temporal lobes are the sources of healthy functional connections. During healthy brain growth, the temporal lobes begin making new connections to other lobes, namely the frontal lobes, the parietal lobes, and the occipital lobes. Large-scale longitudinal healthy pediatric neuroimaging study results showed nonlinear changes in cortical gray matter, with a preadolescent increase followed by a post-adolescent reduction for healthy individuals, while they validated linear increases in white matter. The developmental curves for the frontal and parietal lobes peaked at around age 12, and for the temporal lobe at around age 16, but cortical gray matter increased in the occipital lobe through age 20<sup>[35]</sup>. These regional variations in cortical gray matter were present. As a child gets older, the connections between the right and left hemispheres become more stable, and the left hemisphere begins to develop more rapidly as a result of an increase in mental tasks. After becoming an adult, the brain balances its workload between the two hemispheres, allowing both to develop. It is well recognized that dyslexia suffers from poor neuronal connections, issues with functional connectivity, and a lack of grey matter production that also impairs working memory. As a result, treatments for dyslexia that lessen the disconnection syndrome, boost coherence, and raise entropy are pertinent and appropriate.

A significant connection between reading problems and auditory processing issues can be seen in the left temporal low activity region. Similar results offer neurobiological proof of underlying nervous system dysfunction in the temporo-occipital and parietal-temporal areas of the brain, among other posterior brain regions. Dyslexia may be significantly impacted by these unusual abnormalities in the left temporo-occipital region of the brain.

It is well established that neurofeedback can lessen dyslexia's consequences. The EEG data are read and displayed to the subject in real time. The subject acquires more control over their brain through operant conditioning<sup>[36]</sup>. It has been demonstrated that this phenomenon may change, and add weak connections that help the subject pay attention and learn better when the user learns to manage a particular area of the brain<sup>[37]</sup>. According to APA guidelines<sup>[38]</sup>, neurofeedback is a "possibly efficacious" technique. It is difficult to demonstrate neurofeedback's effectiveness. Typically, clinical studies have been conducted to demonstrate advancements in the psychometric tests used before and after the investigation. According to several studies, neurofeedback leads to improvements in brain structure<sup>[39]</sup>. Participants showed improved functional connectivity of the sensorimotor resting state network and increased fractional anisotropy (FA) in the corpus callosum after one hour of NFB training. The default mode network also showed increased functional connectivity<sup>[40]</sup>. fMRI is typically used in this study to display the strongly linked brain regions following neurofeedback. It is challenging to demonstrate changes in the brain following neurofeedback using QEEG. There is research that shows the causality between neurofeedback and cognitive improvement<sup>[41][42][43][44]</sup>.

Auto Train Brain is an advanced solution that includes neurofeedback from 14-channels, multimodal learning, and special education principles<sup>[27]</sup>. Machine learning algorithms are built-in features of Auto Train Brain. Previous research investigated the long-term effects of 14-channel neurofeedback with Auto Train Brain<sup>[45]</sup>. It was discovered that the variance of the gamma band entropy was increased, showing the brain's flexibility is enhanced. Using gamma band entropy variance is a good measure to understand the increased functional connectivity in certain regions across neurofeedback sessions.

In this research, we have compared the efficacy of 14-channel neurofeedback and that of 5-channel neurofeedback for dyslexia with Auto Train Brain in terms of variance in gamma band entropy changes after neurofeedback sessions.

## II. Materials & Methods

### A. Subjects & Experimental data

In this experiment, 40 dyslexic children participated providing their written consent both from themselves and from families according to the rules set by the research ethics committee. Their ages differ from 7 to 10 (34 males, 6 females). They have used Auto Train Brain (a clinically-tested mobile app for applying neurofeedback from 14 channels or 5-channels) more than 100 times to improve their reading speed and reading comprehension. The children in the experimental group were diagnosed with dyslexia by psychiatric professionals, who then recommended that their families use Auto Train Brain at home. The TILLS tests were used by psychologists and psychiatrists to examine whether the individuals met the DSM-V dyslexia criteria. The children chosen to participate in the experiment were chosen at random. The participant's primary goal in the retrospective study is to use Auto Train Brain software as a neurofeedback device at home.

The participants utilized Auto Train Brain before leaving for school in the morning. The study's inclusion requirements stipulated that participants must be of middle socioeconomic status, be drug-free, and have dyslexia as their only

comorbid condition and aged between 7-10. They lived all around Turkey in various cities. A survey of the parents of the children was done to assess their socioeconomic position. The survey asks questions about employment, education (elementary, secondary, and postsecondary), and income (low income 6,000 TL, middle income 6,000 TL to 20,000 TL, high income >20,000 TL) (staff, blue-collar workers, white-collar workers).

### *B. QEEG recording*

In the experiments, EMOTIV INSIGHT2 and EPOC-X headsets are used. The EEG data was read with 2048 per secs per channel -128 per secs per channel down sampled. EEG data were converted to the frequency band data with EMOTIV's standard procedures. The frequency band data is binned as follows: Theta (4-8 Hz), Alpha (8-12 Hz), Beta-1 (12-16 Hz), Beta-2(16-25 Hz), and Gamma (25-45 Hz). The artifacts were removed with a high pass filter (>100 Hz). EMOTIV APP is used for the calibration of the headsets, each electrode is soaked well and ensured that EEG data is read with top quality. The recorded channels were AF3, T7, P7, T8, and AF4 for EMOTIV INSIGHT2 and the recorded channels were AF3, F3, F7, FC5, T7, P7, O1, O2, P8, T8, FC6, F8, F4, and AF4 for EMOTIV EPOC-X.

The EMOTIV EPOC-X, a commercial wearable EEG device, was used for the recordings. One of the most popular sensory EEG devices for lifestyle applications is the EMOTIV EPOC-X, which consists of 14 sensors and associated felt pads inserted in the scalp in accordance with the International 10-20 System (AF3, F3, F7, FC5, T7, P7, O1, AF4, F4, F8, FC6, T8, P8, and O2). As reference channels, two more rubber electrodes were inserted into the mastoids. The connection between the electrodes and the scalp is made using the saline liquid solution that has been administered to all of the felt pads of each sensor, and the sampling frequency is 128 Hz.

### *C. Neurofeedback treatment protocol and multi-sensory learning method*

Auto Train Brain is a mobile application that uses neurofeedback and multi-sensory learning principles. It is used with the EMOTIV EPOC+ headset. It is a non-invasive solution, that offers continuous brain performance improvement for both adults and children without any side effects. It reads QEEG from 14 channels, processes these signals, and provides real-time visual and auditory, online neurofeedback. Auto Train Brain is a patented software (patent number: PCT/TR2017/050572) specifically designed for people with dyslexia. Within this software application, a system and method for improving reading ability and cognitive functions is proposed. The system relies on a distinctive protocol of multi-sensory learning and EEG neurofeedback. The EEG neurofeedback protocol is explained below:

- Reduce theta waves at Broca area in the brain if above the threshold;
- Reduce theta waves at Wernicke area in the brain if above the threshold;
- Find the channels with the maximum absolute power of theta waves at the left hemisphere and reduce absolute theta for those channels; and
- Find the channels with the maximum absolute power of theta waves at the right hemisphere and reduce the absolute theta for those channels.

A positive reward is a green arrow on the screen, negative feedback is a red arrow and a "beep" sound. With a

positive reward, the score displayed on the screen is increased. If the slow brain waves of the subject are above the norm threshold, a red arrow is presented on the screen and the subject is asked to try to turn it to a green arrow. After the neurofeedback session, a phoneme-grapheme matching alphabet teaching system is presented. One of the significant differences between the currently available neurofeedback systems and Auto Train Brain is that it combines neurofeedback with multi-sensory learning principles.

#### *D. Study design*

All subjects used Auto Train Brain (a mobile phone application) more than 100 times, for randomly chosen 20 participants, their brain waves are read using EMOTIV INSIGHT for 5 channels, for the rest 20 participants, their brain waves are read using EMOTIV EPOC-X for 14-channels and visual and auditory neurofeedback is given for 30 minutes. After the neurofeedback session, multi-sensory alphabet learning is studied for 15 minutes.

With some assistance from their families at home, the participants completed the 30-minute neurofeedback sessions. Each participant utilized it while seated at a table at home throughout the neurofeedback session. As their parents are told to do in advance, there were 40 centimeters between the subject and the smartphone app. The participants used Auto Train Brain's arrow neurofeedback interface.

At the end of each session, session average data for each frequency band was saved to the database. During the neurofeedback session, sample entropy was calculated for each frequency band data<sup>[13]</sup>. Sample entropy is the minus of the logarithmic probability which measures the similarity of two sequences. If the two sequences of  $m$  consecutive data points, that are similar to each other (within given tolerance  $r$ ), will remain similar at the next point ( $m + 1$ ) in the dataset ( $N$ ), then the sample entropy would be higher.  $N$  is the number of samples in the session data. Normally, sample entropy is calculated based on EEG data series, however, in our calculations, we have used QEEG data as we have not reached raw data from EMOTIV INSIGHT2 or EPOC-X.

The feature set consists of 5 variables mapped from 5 channels for EMOTIV INSIGHT, and 14 variables mapped from 14 channels for EMOTIV EPOC-X. The measures are gamma band sample entropy values calculated from QEEG band power values.

#### *E. Statistical Analysis*

All analysis was performed with Excel and regression analysis has been performed.

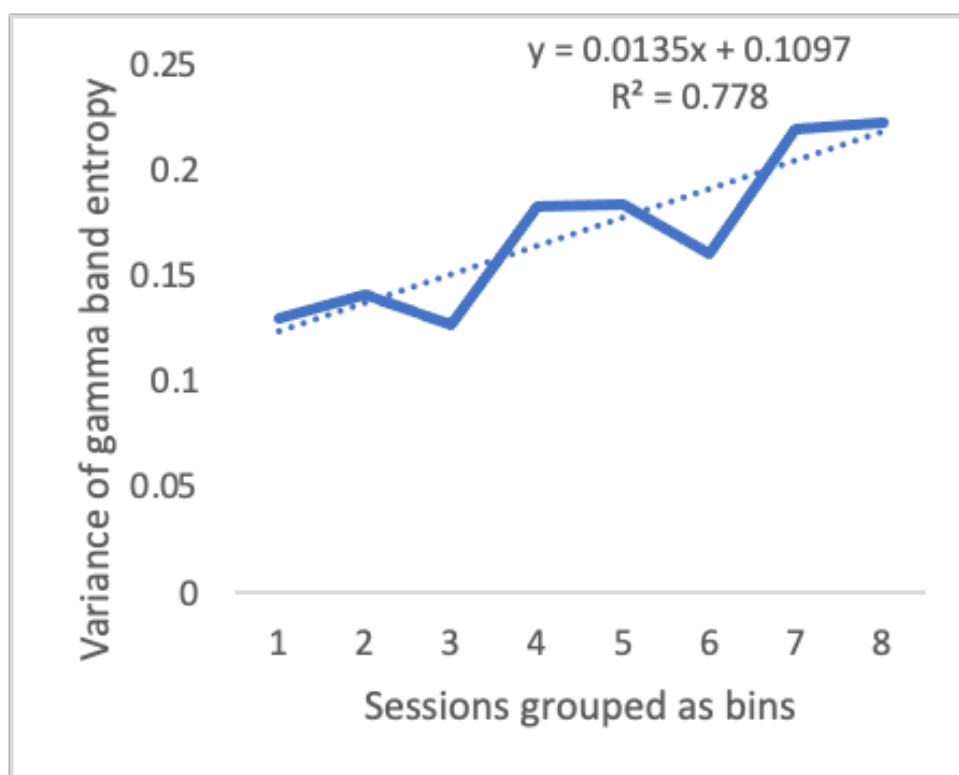
### III. Results

A regression line is drawn (the  $x$  coordinate is the session numbers and the  $y$  coordinate is the variance of gamma band sample entropy for each bin). The findings suggest that long-term neurofeedback use increased the variance of gamma band sample entropy, but we are unable to identify any long-term improvements in the gamma band sample

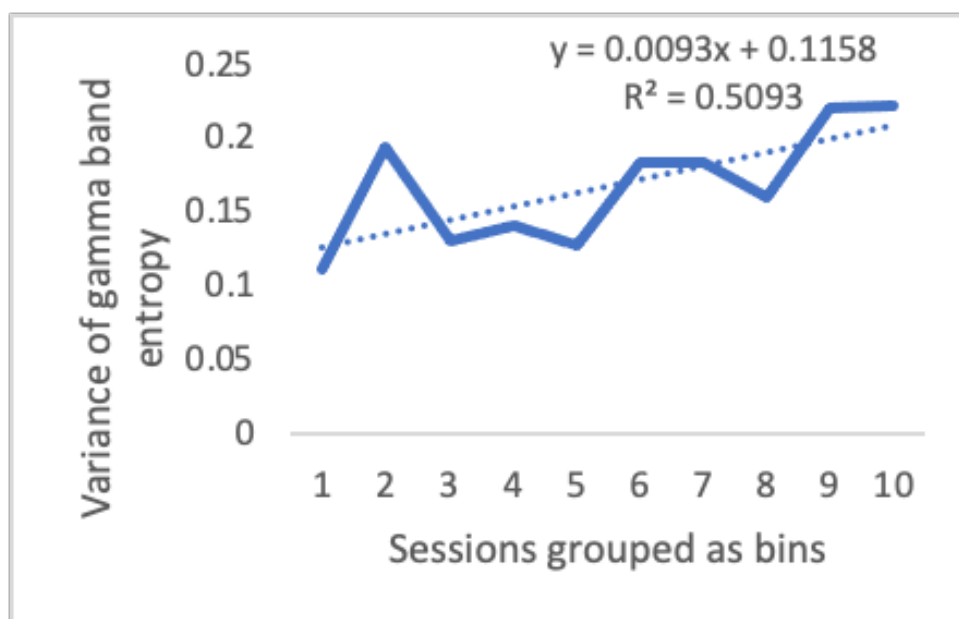
entropies across sessions.

The 100 consecutive sessions have been merged into 10 bins. Next, we determined the variance of each bin's gamma band sample entropy. Ten bins were present. We have shown the gamma band sample entropy values' bin number vs variance. In both headsets' left posterior regions, the gamma band sample entropy variance rose over time (T7).

For a 14-channel EEG headset, the regression line yields  $R^2=0.78$  when the first 30 sessions are excluded (Figure 1).  $R^2$  for the regression line is 0.50 when the first 30 sessions are considered (Figure 2). In both instances, the linear regression lines' slopes were upward.

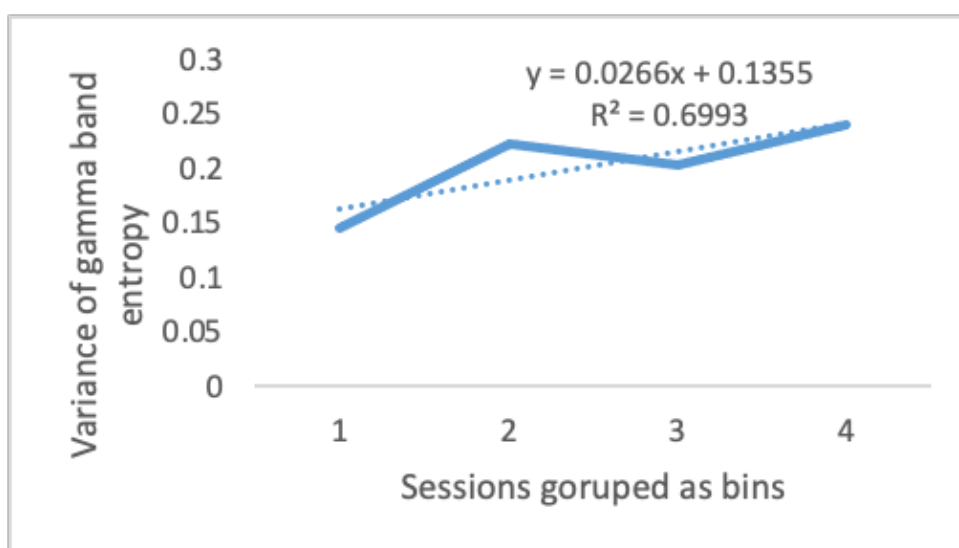


**Figure 1.** The increase in the variance of gamma band entropy (y-axis)  $y$  in the left posterior region after 30 sessions (x-axis, 1 bin=10 sessions) for a 14-channel EEG headset



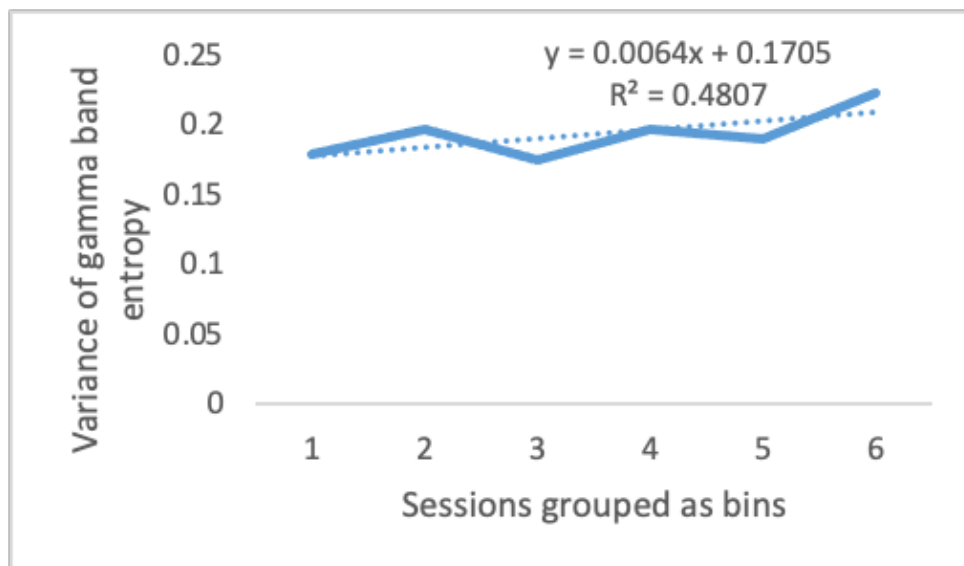
**Figure 2.** The increase in the variance of gamma band entropy (y-axis) in the left posterior region for a 14-channel EEG headset in the 100 sessions (x-axis, 1 bin= 10 sessions)

In the first 40 sessions, the regression line for a 5-channel EEG headset yields  $R^2=0.69$  (Figure 3).  $R^2$  for the regression line is 0.48 for the following 60 sessions (Figure 4). In both instances, the linear regression lines' slopes were upward.



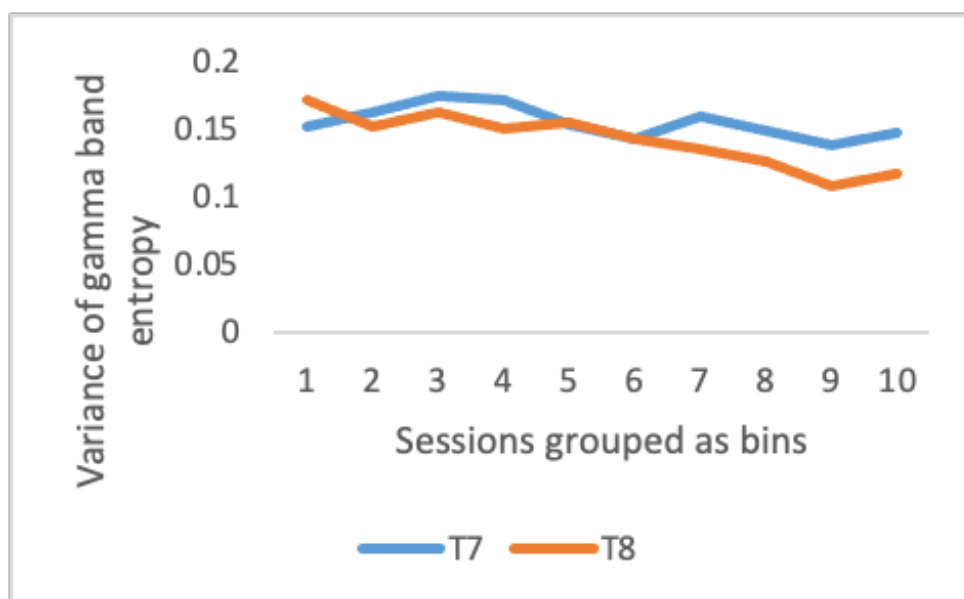
**Figure 3.** The increase in the variance of gamma band entropy (y-axis) in the left posterior region for a 5-channel EEG headset in the first 40 sessions (x-axis, 1 bin= 10 sessions)





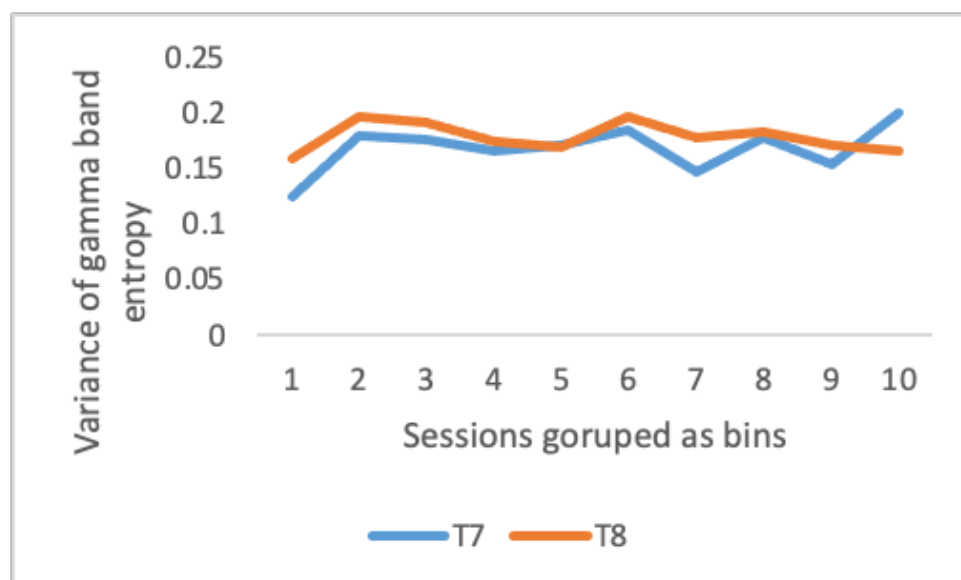
**Figure 4.** The increase in the variance of gamma band entropy (y-axis) in the left posterior region for a 5-channel EEG headset in the next 60 sessions (x-axis, 1 bin=10 sessions)

For a 14-channel headset, the variance of the gamma band entropy changes in the left temporal and the right temporal regions in the 100 sessions are plotted as follows:



**Figure 5.** The change in the variance of gamma band entropy (y-axis) in the left (T7) and right temporal (T8) regions for a 14-channel EEG headset in the next 100 sessions (x-axis, 1 bin=10 sessions)

Figure 5 shows that at around 20th sessions, the left lateralization of the brain takes place and the variance of gamma band entropy becomes permanently dominant for the left temporal region after 60 sessions.



**Figure 6.** The change in the variance of gamma band entropy (y-axis) in the left (T7) and right temporal(T8) regions for a 5-channel EEG headset in the next 100 sessions (x-axis, 1 bin=10 sessions)

Figure 6 displays the gamma band variations over sessions for 5-channel neurofeedback with Auto Train Brain in the left and right temporal lobes. Only after the 100th session, which is twice as long as with 14-channel neurofeedback, left hemispheric dominance begins to occur.

## IV. Discussion

In prior clinical research, Auto Train Brain was shown to be useful for children with dyslexia<sup>[27]</sup> with pre- and post-TILLS test comparisons. Their reading comprehension and reading speed were increased after 60 sessions and their entropy was increased. With 14-channel and 5-channel EEG headsets, we looked into the long-term use and the beneficial impacts of Auto Train Brain in this study in terms of electrophysiology.

In the first 20 sessions of use, 14-channel neurofeedback in the left posterior region causes a sharp increase in the variance of the sample entropy in the gamma band. With 5-channel neurofeedback, this rise requires twice as many sessions; after 40 sessions, the variation of the gamma band entropy peaks, and the brain begins to adapt. As children adjust to and learn neurofeedback, we predict that numerous metabolic changes occur in their bodies as well as their brains and that the learning effort is particularly intense during the first month.

Given that dyslexic people are eager to employ their right hemisphere for mental tasks, the variation of the gamma band entropy increases first in the right temporal regions for dyslexia. After crossing a certain point, the burden becomes too great for the right brain, causing the left temporal region to begin to form new connections as we observe the left hemisphere's gamma band entropy variance increasing.

The hemisphere that is utilized frequently is initially more activated as we increase our mental burden, but as we do, the temporal region of the other hemisphere begins to become more active. Between the nearby temporal regions, new

functional networking occurs. Therefore, it is possible to imagine that the increase in gamma band entropy variance shows that functional connectivity is under construction to be enhanced. The variance of the gamma band entropy in the region begins to decline after the functional networking is enhanced and optimized fully, allowing the other regions to be built and developed more (Figure 5).

The variation of the sample entropy in the gamma band is reduced after the 20 sessions for 14-channel neurofeedback and after the 40 sessions for 5-channel neurofeedback with Auto Train Brain, and we assume that the functional networks prune and stabilize after some building and optimization. In the following sessions, there is an increase in the variance of the gamma band entropy. There are two further steps of adaption for both headsets in the remaining sessions.

The findings show that after using Auto Train Brain for 100 sessions with both headsets, the brain's flexibility—or its capacity to use new functional networks—increased. With 5-channel neurofeedback with Auto Train Brain, the amount of time and sessions needed to achieve brain flexibility is doubled. The brain can more quickly lateralize to the left thanks to 14-channel neurofeedback. The left hemisphere begins to dominate after the 20th session, and after some adjustments around the 60th session, the left hemisphere's dominance becomes permanent. Families who employ 14-channel neurofeedback might observe the benefits in the child's day-to-day activities much more quickly.

The frontal, parietal, and occipital lobes, as well as the temporal lobes, start developing new connections with one another as part of normal brain development. Results from a large-scale longitudinal healthy pediatric neuroimaging investigation revealed nonlinear changes in cortical gray matter, with an increase during preadolescence and a drop during adolescence for healthy individuals, whereas they confirmed linear increases in white matter. The frontal, parietal, and temporal lobes all reached their developmental peaks around the ages of 12 and 16, respectively, while the occipital lobe's cortical gray matter grew until age 20<sup>[36]</sup>. There were some regional variances in the cortical gray matter. The connections between the right and left hemispheres grow more secure as kid ages, and the left hemisphere starts to develop. After neurofeedback, we have realized that dyslexic children follow the normal growth path of the brain which increases the hypothesis of brain maturation delay.

Six dyslexic children were given neurofeedback by Nazar<sup>[46]</sup>, who did not observe any significant changes in the power bands but did observe normalization of coherence in the theta band at T4-T4, delta band at Cz-Fz, and beta band at Cz-Pz, Cz-Fz, and Cz-C4. Hypo coherence is the symptom of disconnection syndrome. He has come to the conclusion that the increases in reading ability and phonological awareness are explained by the large changes in coherence, which point to the integration of sensory and motor domains.

Coben demonstrated that coherence neurofeedback raises reading scores for those with reading problems by 1.2-grade levels<sup>[47]</sup>.

In the literature, fMRI has been used to demonstrate the increase in functional connectivity following fMRI-based functional connectivity neurofeedback<sup>[48]</sup>. To measure the improved functional connectivity<sup>[46]</sup> following coherence neurofeedback, coherence and phase lag on the EEG should be computed. It is challenging to do real-time coherence calculations using QEEG and EMOTIV headsets. Therefore, a suitable indicator of the in-session changes in functional connectivity networks is the variance of gamma band entropy across neurofeedback sessions.

It should be noted that neurofeedback does not cure the root cause of dyslexia. 14-channel neurofeedback may help

the left lateralization of the brain which is one of the most dramatic changes in the healthy brain during its projection of growth. The electrophysiological changes reflect cognition as shown by the prior research on Auto Train Brain<sup>[27]</sup>. Neurofeedback has unique advantages for affecting cognition, albeit indirectly<sup>[49]</sup>.

The unexpected finding is that the gamma band entropy is not increased constantly. It varies from one session to another and there are pruning phases after 20-30 sessions.

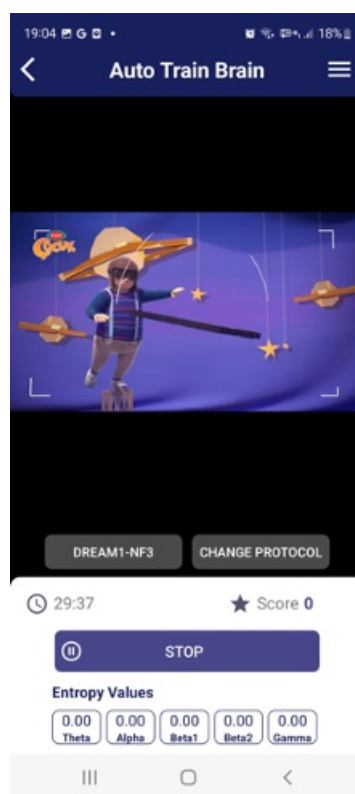
The limitation of this research is the number of participants. It would have been better if we recruited more people. The other limitations of the experiment are that there is a maturation effect and the placebo effect.

For future research, we will investigate new calculation methods of coherence and functional connectivity based on QEEG and test our hypotheses with this calculation.

This is a pilot study that should be repeated with more participants in the near future.

## Conclusion

The variance of gamma band entropy changes over neurofeedback sessions presents promising results to explain electrophysiological changes and adaptations in the brain. Auto Train Brain was proven to be effective in improving reading comprehension and reading speed beforehand. Now, with the new calculation method, we have investigated the changes in electrophysiological changes in the left temporal region after neurofeedback.



**Figure 7.** Auto Train Brain  
"youtube" interface

## Acknowledgment

We would like to thank all the children with dyslexia who have participated in our experiment and their families. Without their motivation, we couldn't have found these results.

## References

- <sup>^</sup> American Psychiatric Association. (2013). *Anxiety disorders*. In *Diagnostic and statistical manual of mental disorders* (5th ed.). <https://doi.org/10.1176/appi.books.9780890425596.dsm05>
- <sup>^</sup> Siegel, L. S. (1988). Evidence that IQ scores are irrelevant to the definition and analysis of reading disability. *Canadian Journal of Psychology/Revue canadienne de psychologie*, 42(2), 201.
- <sup>^</sup> Francks, C., MacPhie, I. L., & Monaco, A. P. (2002). The genetic basis of dyslexia. *The Lancet Neurology*, 1(8), 483-490.
- <sup>^</sup> Van Bergen, E., De Jong, P. F., Plakas, A., Maassen, B., & van der Leij, A. (2012). Child and parental literacy levels within families with a history of dyslexia. *Journal of Child Psychology and Psychiatry*, 53(1), 28-36.
- <sup>^</sup> D'Souza, S., Backhouse-Smith, A., Thompson, J. M., Slykerman, R., Marlow, G., Wall, C., ... & Waldie, K. E. (2016). Associations between the KIAA0319 dyslexia susceptibility gene variants, antenatal maternal stress, and reading ability in a longitudinal birth cohort. *Dyslexia*, 22(4), 379-393.
- <sup>^</sup> Nicolson, R. I., & Fawcett, A. J. (2019). Development of dyslexia: The delayed neural commitment framework. *Frontiers in Behavioral Neuroscience*, 13, 112.
- <sup>^</sup> McDougall, S., Hulme, C., Ellis, A., & Monk, A. (1994). Learning to read: The role of short-term memory and phonological skills. *Journal of experimental child psychology*, 58(1), 112-133.
- <sup>^</sup> Kim, E., Paik, D., Ramirez, R. N., Biggs, D. G., Park, Y., Kwon, H. K., ... & Huh, J. R. (2022). Maternal gut bacteria drive intestinal inflammation in offspring with neurodevelopmental disorders by altering the chromatin landscape of CD4+ T cells. *Immunity*, 55(1), 145-158.
- <sup>^</sup> Richardson, A. J. (2004). Long-chain polyunsaturated fatty acids in childhood developmental and psychiatric disorders. *Lipids*, 39(12), 1215-1222.
- <sup>^</sup> YILMAZ, S., & AKYÜZ, F. The relationship between speech difficulties and brain laterality in Attention Deficit Hyperactivity Disorder and Specific Learning Disorder. *Acta Medica Alanya*, 5(3), 250-256.
- <sup>a, b</sup> Kershner, J. R. (2020). Neuroscience and education: cerebral lateralization of networks and oscillations in dyslexia. *Laterality*, 25(1), 109-125.
- <sup>^</sup> Paulesu, E., Frith, U., Snowling, M., Gallagher, A., Morton, J., Frackowiak, R. S., & Frith, C. D. (1996). Is

developmental dyslexia a disconnection syndrome? Evidence from PET scanning. *Brain*, 119(1), 143-157.

13. <sup>a, b</sup>Thornton, K. E., & Carmody, D. P. (2005). Electroencephalogram biofeedback for reading disability and traumatic brain injury. *Child and Adolescent Psychiatric Clinics*, 14(1), 137-162.
14. <sup>^</sup>Sowell, E. R., Peterson, B. S., Thompson, P. M., Welcome, S. E., Henkenius, A. L., & Toga, A. W. (2003). Mapping cortical change across the human life span. *Nature neuroscience*, 6(3), 309-315.
15. <sup>^</sup>Schulte-Körne, G., & Bruder, J. (2010). Clinical neurophysiology of visual and auditory processing in dyslexia: a review. *Clinical neurophysiology*, 121(11), 1794-1809.
16. <sup>^</sup>Lassus-Sangosse, D., N'guyen-Morel, M. A., & Valdois, S. (2008). Sequential or simultaneous visual processing deficit in developmental dyslexia?. *Vision Research*, 48(8), 979-988.
17. <sup>^</sup>Bednarek, D. B., Saldaña, D., Quintero-Gallego, E., García, I., Grabowska, A., & Gómez, C. M. (2004). Attentional deficit in dyslexia: a general or specific impairment?. *Neuroreport*, 15(11), 1787-1790.
18. <sup>^</sup>Bellocchi, S., Muneaux, M., Bastien-Toniazzo, M., & Ducrot, S. (2013). I can read it in your eyes: What eye movements tell us about visuo-attentional processes in developmental dyslexia. *Research in developmental disabilities*, 34(1), 452-460.
19. <sup>^</sup>Eden, G. F., VanMeter, J. W., Rumsey, J. M., Maisog, J. M., Woods, R. P., & Zeffiro, T. A. (1996). Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. *Nature*, 382(6586), 66-69.
20. <sup>^</sup>Tschentscher, N., Ruisinger, A., Blank, H., Díaz, B., & Von Kriegstein, K. (2019). Reduced structural connectivity between the left auditory thalamus and the motion-sensitive planum temporale in developmental dyslexia. *Journal of Neuroscience*, 39(9), 1720-1732.
21. <sup>^</sup>Horwitz, B., Rumsey, J. M., & Donohue, B. C. (1998). Functional connectivity of the angular gyrus in normal reading and dyslexia. *Proceedings of the National Academy of Sciences*, 95(15), 8939-8944.
22. <sup>^</sup>Rubinsten, O., & Henik, A. (2006). Double dissociation of functions in developmental dyslexia and dyscalculia. *Journal of Educational Psychology*, 98(4), 854.
23. <sup>^</sup>Galaburda, A. M., Sherman, G. F., Rosen, G. D., Aboitiz, F., & Geschwind, N. (1985). Developmental dyslexia: four consecutive patients with cortical anomalies. *Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society*, 18(2), 222-233.
24. <sup>^</sup>Kibby, M. Y., Fancher, J. B., Markanen, R., & Hynd, G. W. (2008). A quantitative magnetic resonance imaging analysis of the cerebellar deficit hypothesis of dyslexia. *Journal of child neurology*, 23(4), 368-380.
25. <sup>^</sup>Riddick, B., Sterling, C., Farmer, M., & Morgan, S. (1999). Self-esteem and anxiety in the educational histories of adult dyslexic students. *Dyslexia*, 5(4), 227-248.
26. <sup>^</sup>Eroglu, G. (2019). EEG, neurofeedback, GFCF diet (Gluten free/casein-free diet), ADHD, learning disability, autism. *Journal of Medical Innovation and Technology*, 1(1), 21-26.
27. <sup>a, b, c, d, e</sup>Eroğlu, G., Teber, S., Ertürk, K., Kırmızı, M., Ekici, B., Arman, F., ... & Çetin, M. (2021). A mobile app that uses neurofeedback and multi-sensory learning methods improve reading abilities in dyslexia: A pilot study. *Applied Neuropsychology: Child*, 1-11.
28. <sup>^</sup>Marins, T., Rodrigues, E. C., Bortolini, T., Melo, B., Moll, J., & Tovar-Moll, F. (2019). Structural and functional connectivity changes in response to short-term neurofeedback training with motor imagery. *Neuroimage*, 194, 283-

290.

29. <sup>^</sup>Klimesch, W., Doppelmayr, M., Yonelinas, A., Kroll, N. E., Lazzara, M., Röhm, D., & Gruber, W. (2001). Theta synchronization during episodic retrieval: neural correlates of conscious awareness. *Cognitive Brain Research*, 12(1), 33-38.
30. <sup>^</sup>Rippon, G., & Brunswick, N. (2000). Trait and state EEG indices of information processing in developmental dyslexia. *International Journal of Psychophysiology*, 36(3), 251-265.
31. <sup>a, b</sup>Arns, M., Peters, S., Breteler, R., & Verhoeven, L. (2007). Different brain activation patterns in dyslexic children: evidence from EEG power and coherence patterns for the double-deficit theory of dyslexia. *Journal of integrative neuroscience*, 6(01), 175-190.
32. <sup>^</sup>Thornton, K. E., & Carmody, D. P. (2005). Electroencephalogram biofeedback for reading disability and traumatic brain injury. *Child and Adolescent Psychiatric Clinics*, 14(1), 137-162.
33. <sup>^</sup>Fraga González, G., Smit, D. J., Van der Molen, M. J., Tijms, J., Stam, C. J., De Geus, E. J., & Van der Molen, M. W. (2018). EEG resting state functional connectivity in adult dyslexics using phase lag index and graph analysis. *Frontiers in human neuroscience*, 12, 341.
34. <sup>^</sup>Kraus, N. (2012). Atypical brain oscillations: a biological basis for dyslexia. *Trends in cognitive sciences*, 16(1), 12-13.
35. <sup>^</sup>Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F. X., Liu, H., Zijdenbos, A., ... & Rapoport, J. L. (1999). Brain development during childhood and adolescence: a longitudinal MRI study. *Nature neuroscience*, 2(10), 861-863.
36. <sup>a, b</sup>Ninaus, M., Witte, M., Kober, S.E., Friedrich, E.V., Kurzmann, J., Hartsuiker, E., Neuper, C., & Wood, G. (2015). Neurofeedback and Serious Games. In me. Management Association (Ed.), *Gamification: Concepts, Methodologies, Tools, and Applications* (pp. 83-112). Hershey, PA: IGI Global. doi:10.4018/978-1-4666-8200-9.ch005
37. <sup>^</sup>Niv, S. (2013). Clinical efficacy and potential mechanisms of neurofeedback. *Personality and Individual Differences*, 54(6), 676-686. doi:10.1016/j.paid.2012.11.037
38. <sup>^</sup>Melnikov, M. Y. (2021). The current evidence levels for biofeedback and neurofeedback interventions in treating depression: A narrative review. *Neural Plasticity*.
39. <sup>^</sup>Wing, K. (2001). Effect of neurofeedback on motor recovery of a patient with brain injury: A case study and its implications for stroke rehabilitation. *Topics in stroke rehabilitation*, 8(3), 45-53.
40. <sup>^</sup>Marins, T., Rodrigues, E. C., Bortolini, T., Melo, B., Moll, J., & Tovar-Moll, F. (2019). Structural and functional connectivity changes in response to short-term neurofeedback training with motor imagery. *Neuroimage*, 194, 283-290.
41. <sup>^</sup>Canolty RT, Edwards E, Dalal SS, Soltani M, Nagarajan SS, Kirsch HE, Berger MS, Barbaro NM, Knight RT. High gamma power is phase-locked to theta oscillations in human neocortex. *Science*. 2006 Sep 15;313(5793):1626-8. doi: 10.1126/science.1128115.
42. <sup>^</sup>Lubar, J.F., Shouse, M.N. EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR). *Biofeedback and Self-Regulation* 1, 293–306 (1976). <https://doi.org/10.1007/BF01001170>
43. <sup>^</sup>Mayoral-Rodríguez, Silvia, Pérez-Alvarez, Frederic, and Timoneda-Gallart, Carme. Brain Waves Reflect Cognition-Emotion State as a Diagnostic Tool for Intervention in Dysfunctional States: A Real-World Evidence. *Journal of Intellectual Disability Diagnosis and Treatment*, 2022, Volume 10( 4) , 154-166 <https://doi.org/10.6000/2292->



2598.2022.10.04.1

44. <sup>^</sup> Terrasa, J.L.; Barros-Loscertales, A.; Montoya, P. & Muñoz, M. A. (2020). «Self-Regulation of SMR Power Led to an Enhancement of Functional Connectivity of Somatomotor Cortices in Fibromyalgia Patients». *Frontiers in Neuroscience*. 14:236. doi: 10.3389/fnins.2020.00236
45. <sup>^</sup> EROĞLU GÜNET (2022). Auto Train Brain increases the variance of the gamma band sample entropy in the left hemisphere in dyslexia: A pilot study. *WORLD S4 2022 (Tam Metin Bildiri/Sözlü Sunum)*(Yayın No:7773551)
46. <sup>a, b</sup> Nazari, M. A., Mosanezhad, E., Hashemi, T., & Jahan, A. (2012). The effectiveness of neurofeedback training on EEG coherence and neuropsychological functions in children with reading disability. *Clinical EEG and neuroscience*, 43(4), 315-322.
47. <sup>^</sup> Coben, R., Wright, E. K., Decker, S. L., & Morgan, T. (2015). The impact of coherence neurofeedback on reading delays in learning disabled children: A randomized controlled study. *NeuroRegulation*, 2(4), 168-168.
48. <sup>^</sup> Megumi, F., Yamashita, A., Kawato, M., & Imamizu, H. (2015). Functional MRI neurofeedback training on connectivity between two regions induces long-lasting changes in intrinsic functional network. *Frontiers in human neuroscience*, 9, 160.
49. <sup>^</sup> Kvamme, T. L., Ros, T., & Overgaard, M. (2022). Can neurofeedback provide evidence of direct brain-behavior causality? *NeuroImage*, 258, 1–10. <https://doi.org/10.1016/j.neuroimage.2022.119400>