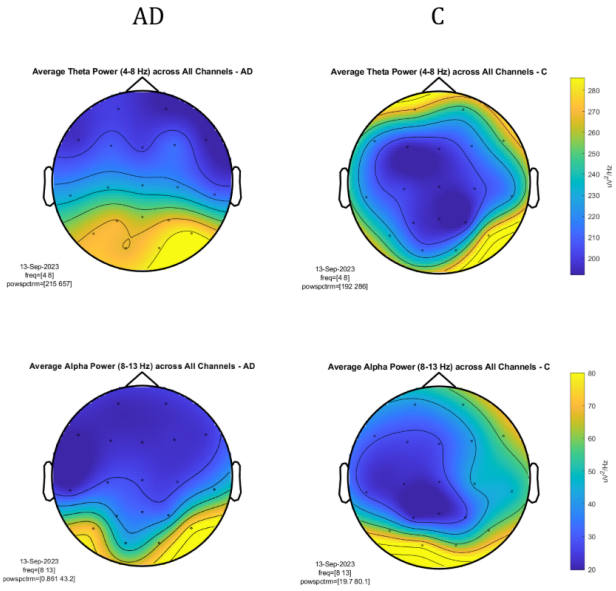
Frequency band power & connectivity patterns - a basis for Alzheimer's disease diagnosis in EEG data?

A hypothesis driven analysis of given resting state EEG data from Alzheimer's disease patients and a healthy control group



Lisa Golla
lgolla@uni-osnabrueck.de
983700

UNIVERSITÄT OSNABRÜCK

A Term paper for the course
Designing and conducting an EEG study
Prof. Dr. med. Peter König,
Debora Nolte, M. Sc. ,
Prof. Dr. Gordon Pipa
SS 2023

Frequency band power & connectivity patterns - a basis for Alzheimer's disease diagnosis in EEG data?

—Based on EEG data published in the paper [1], the data from 3 AD patients and 3 healthy controls were analyzed hypothesis driven. It was investigated whether the alpha frequency band power is reduced and the theta frequency band power is increased in the AD patient group. The hypothesis effects could be observed, also a correlation with a cognitive decline for the alpha frequency band power reduction, however not for the theta band power. The second hypothesis could also be supported since a change in the connectivity pattern of the AD group in selected channel pairs was evident. Namely, the coherence of the channel pairs was reduced for the AD group. However, for the AD group, no coherence increase of channel pairs could be reported. It was revealed that frequency band power and connectivity pattern can serve as an indicator, however are not sufficient for a diagnosis. More factors have to be included for the individual, like more biomarkers, behavior, excluding other neurogenerative illnesses, or more neural imaging procedures. In order to make reproduction easily possible, you can find a link containing code, data files and figures here.

***Index Terms*—Electroencephalography; Routine EEG; Alzheimer's disease; resting state; Diagnose of Alzheimer**

I. Introduction

Alzheimer's disease, or AD in short, can be characterized as a progressive neurodegenerative disorder caused by damage and death of neurons leading to a disturbance of cognitive and behavioral functions. There are different forms of AD, whereas AD is the most common form accounting for around 70 % of the AD cases worldwide [2]. Until now, no cure for AD exists, meaning only palliative treatments exist that temporarily slow the worsening of symptoms. Since the disease heavily reduces the life quality of patients, caregivers as well as family members, there is the interest to establish early diagnosis in order to react as fast as possible. Due to the fact that the initial symptoms are considered as 'mild', like reduced short-term-memory, or orientation difficulties, as well as they overlap with other neurodegenerative illnesses, it is often hard to diagnose AD in its early stages. This situation is grave since the extent of AD includes cognitive decline, loss of motor skills as well as memory loss [1], [2]. Better detection technologies are thus required in order to help in the early identification of these illnesses.

Electroencephalography (EEG) turned out to be a potential method for the identification and monitoring of Alzheimer's disease in addition to clinical evaluation and imaging testing [5]. EEG measures brain electrical activity and can identify anomalies in brain waves linked to certain disorders [1]. EEG signals reflect functional changes in the cerebral cortex. Therefore, EEG-based biomarkers can be used to assess neuronal degeneration caused by AD progression, long before actual tissue loss or behavioral symptoms appear. As a result, EEG is a promising technique with the potential of serving as an alternative to existing tools, like CSF and MRI/PET, but with the advantage of being noninvasive, portable, and less expensive, and as a matter of fact affordable for the common run of mankind [1], [2]. Moreover, based on new technological development and testing of machine learning methodologies, automatic detection and categorizing of diseases based on EEG signals , is an innovative current research field with a promising outlook for the future potentially facilitating an even more affordable, fast, accurate and efficient opportunity for AD diagnosis.

Certainly, these findings raise questions: What biomarkers indicate AD? How could they be biologically explained? During the course of this paper 2 hypotheses will be elaborated that concern potential EEG signal indicators for AD. Namely, differences in frequency band powers and in connectivity patterns. A small subset of public EEG data from AD patients and a healthy control group published in [1] will be preprocessed, and analyzed hypothesis driven. For the analysis Power spectral density and coherence are employed. During the course of the paper, the results are interpreted and there will be a discussion and critical reflection about the results and the central question, whether Frequency band power and connectivity patterns could be a basis for Alzheimer's disease diagnosis. This paper is meant to employ a subset of a larger dataset and point out if the hypotheses can be proven, or in other words if the expected results can be reproduced. Generally speaking, the paper serves as a grading fundament of a course and therefore the focus lays more on analyzing data and interpreting, discussing results rather than creating expressive, representative research findings.

The main contributions are

- Preprocessing of raw EEG data from 6 different subjects
- Postulation and biological explanation of 2 hypotheses regarding AD detection via EEG signals
- Hypothesis driven analysis and interpretation of results
- Critically reflecting and discussing results

II. Material

A. Data description

Where does it come from?

The data which will be analyzed in this paper is a subset of the eeg dataset published in the paper 'A Dataset of Scalp EEG Recordings of Alzheimer's Disease, Frontotemporal Dementia and Healthy Subjects from Routine'. The dataset is a resting state EEG dataset of individuals with Alzheimer's disease (AD), frontotemporal dementia (FTD), and healthy controls (C). In total the dataset contains 36 Alzheimer's patients, 23 frontotemporal dementia patients, and 29 healthy age-matched subjects. The cognitive and neuropsychological state is provided by the international Mini-Mental State Examination (MMSE) [5]. The MMSE score ranges from 0 to 30, with a lower MMSE indicating more cognitive decline. The sampling rate is 500 Hz and the resolution is 10 uV/mm. Each recording lasts approximately 13.5 min for the AD group, 12 min for the FTD group, and 13.8 min for the CN group. These recordings took place in a clinical routine setting. Recordings were acquired from the 2nd Department of Neurology of AHEPA General Hospital of Thessaloniki by an experienced team of neurologists. A clinical EEG device (Nihon Kohden 2100), with 19 scalp electrodes (Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, and O2) and 2 electrodes (A1 and A2) was employed. The electrodes were placed according to the 10–20 international system.

What was it originally collected for?

The recordings of this dataset were collected to investigate functional differences in the EEG activity of AD compared to CN, FTD compared to CN and AD compared to FTD. The dataset contains information about the gender, the age, the MMSE score, the participant ID and the group type of the subject (AD, FTD, healthy control C). In order to compare the different conditions better, a variety of MMSE scores, gender and age are provided. On the one hand, the public dataset was meant to compare EEG characteristics between different types of dementia, which could provide insights into the underlying mechanisms of these conditions. On the other hand, the paper also indicated that the dataset has significant reuse potential since Alzheimer's EEG Machine Learning studies are increasing in popularity and there is a lack of publicly available EEG datasets. Therefore, the given dataset is also designed for further ML approaches, feature extraction, or classification tasks. For more details have a look into the original paper [1].

B. Preprocessing steps

A subset of six EEG data files was used for the analysis. It was tried to reach a representative subset, this is why for the AD patients 3 different MMSE scores were chosen. Additionally, in order to make a comparison possible also 3 healthy controls were selected. The gender and age is mixed across the AD and C condition. An overview can be seen in II-B.

Participant ID	Gender	Age	Group	MMSE
Sub-001	F	57	A	16
Sub-005	M	70	A	22
Sub-020	M	71	A	4
Sub-040	M	61	C	30
Sub-041	F	77	C	30
Sub-042	M	74	C	30

Fig. 1. Information about the participants the data contains

The data was downsampled to 512 hz and passed through a high and low pass filter with the values 0.5 and 45. Concerning the channels, There have been no channels removed since no empty or pure noise channels could be observed. Noisy segments have been rejected manually since ICA is capable of handling eye movement and blinking, but not of EMG muscle noise [7]. Accordingly, the noisy EEG data segments have been rejected as conservative as possible, to prevent deletion of valuable information. Afterwards, a baseline correction and ICA have been applied to the data and respectively rejecting components. The focus here laid mainly on receiving information of the brain and rejecting muscle, or eye components. It was decided to not epoch the given data, because the analysis at hand focuses on assessing brain activity during a continuous, uninterrupted resting state. Therefore, spontaneous, ongoing neural activity is of interest. The idea is to capture the temporal dynamics and connectivity patterns of the brain. Epoching may disrupt the natural temporal flow of brain activity, making it challenging to study continuous oscillations and connectivity over longer time periods [2], [8]. Moreover, epoching can introduce artifacts at the beginning and end of each epoch due to abrupt changes in the EEG signal. In resting-state analysis, where subtle changes in brain connectivity are of interest, these artifacts can be problematic [8]. Analyzing continuous data can help mitigate these issues.

III. Hypotheses

A. Alpha & Theta Power and Cognitive Decline

As elaborated in several papers [1]–[5], it is possible to identify some markers, or characteristics in EEG signals of AD patients. Particularly, it is reported that the Relative Band Power (RBP) of the five frequency bands (Delta, Theta, Alpha, Beta, Gamma) show significant differences compared to healthy controls [1], [2]. In more detail, AD patients exhibit changes in the RBP such as reduced alpha power and increased theta power. This phenomenon is thought to be linked to the neurodegenerative processes and structural brain changes associated with Alzheimer's disease [2]. Increased theta power in EEG is often associated with cognitive impairment [1]. In AD, the rise in theta power may reflect the disruption of normal neuronal networks and the loss of inhibitory control. Theta oscillations are associated with memory processes, and

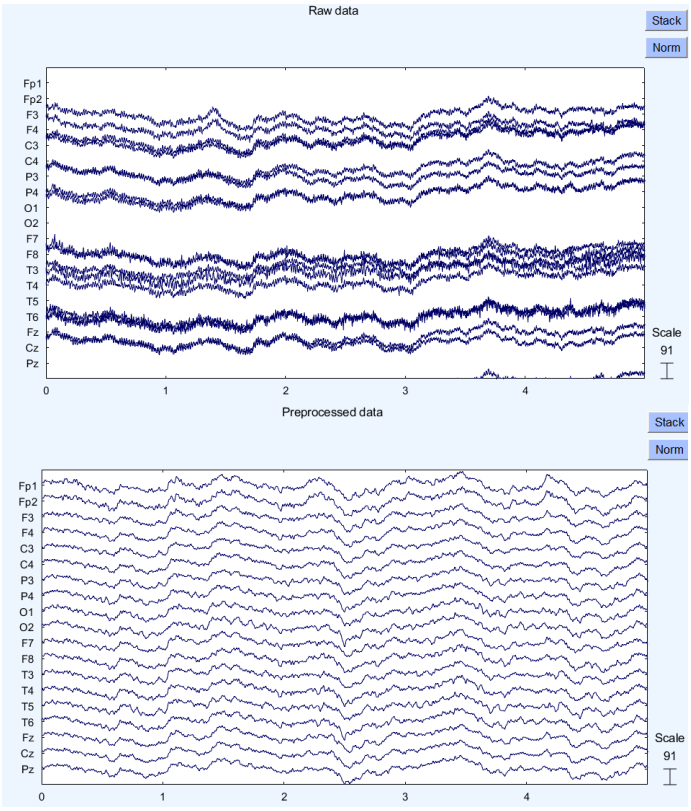


Fig. 2. A snapshot of the same signal before and after being preprocessed.

their increase may suggest compensatory mechanisms as the brain attempts to cope with memory deficits. It can also indicate decreased coherence in brain networks, reflecting difficulties in synchronizing neural activity [2]. By contrast, the reduced alpha power can be attributed to various factors. Alpha rhythms are typically associated with the brain's resting state and are prominent in awake individuals with closed eyes. First of all, the neuronal loss may play a role since neurons degenerate in AD and therefore there are fewer neurons available to generate alpha oscillations [1]. Next to that, AD disrupts synaptic transmission that may be a crucial part in generating alpha rhythms. As a result, the synaptic dysfunction might impair the alpha power. Another crucial factor to be named is the network disruption. AD disrupts functional brain networks. The changes in relative band powers may reflect altered network dynamics and connectivity, resulting from the breakdown of functional networks in the brain. In particular, the disruption can lead to a reduction of the amplitude of alpha oscillations [1]. Based on findings in [2] this effect increases in later stages with more cognitive decline. The data that will be analyzed provides information about the cognitive decline which makes it possible in theory to reproduce the finding. Nevertheless, EEG findings in AD are not uniform across all patients, and individual variations can occur. Since the first hypothesis was already tested and proven with the data that is going to be analyzed further, the idea is to report, if the results could be reproduced with the analyzed excerpt of data. A successful reproduction would therefore support the hypothesis and the findings on the paper.

AD patients will show reduced alpha power and increased theta power in posterior regions compared to healthy controls, which may correlate with cognitive decline.

In order to analyze the hypothesis, it was decided to calculate the spectral power in the alpha & theta frequency band and perform correlation analyses between alpha, theta power and cognitive test scores.

B. Connectivity Changes

Furthermore, there is another factor possibly indicating Alzheimer's disease in EEG signals. Namely, as several papers suggest, AD patients will exhibit altered functional connectivity patterns, including increased connectivity in certain regions (possibly compensatory mechanisms) and decreased connectivity in others [2], [4], [5]. This could potentially be biologically explained by the complex neurodegenerative processes and structural changes that occur in Alzheimer's disease. The neurodegeneration for example, is leading to a loss of neurons and synaptic connections. This neuronal loss can disrupt normal functional connectivity patterns in the brain. As neurons degenerate and neural networks are compromised, some regions may compensate for the loss of function by increasing their connectivity to other regions to maintain cognitive function or adapt to challenges [2]. Besides, Neuroinflammation is a common feature of AD. It involves the activation of immune cells and the release of pro-inflammatory cytokines. Neuroinflammation can disrupt neural circuits and alter functional connectivity patterns [5]. Increased connectivity may be an adaptive response to counteract the effects of inflammation. It is also reported that functional connectivity patterns may vary depending on the stage and progression of AD. Early-stage AD may exhibit compensatory mechanisms with increased connectivity, while later stages may be characterized by widespread decreases in connectivity as neurodegeneration advances [2]. Unfortunately, there is only a small range of data that is inspected, however it might be interesting to check whether such a difference can be detected in the different stages.

AD patients will exhibit altered functional connectivity patterns, including increased connectivity in certain regions and decreased connectivity in others which might correlate with cognitive decline.

For the analysis the coherence measure is employed to quantify connectivity patterns and compare them between groups. Additionally, a correlation analysis of the cognitive impairment score will be included.

IV. Methods

In the last section the different hypotheses were elaborated. Based on that, in the next section, there will be a detailed description of the methods that were used for the analysis.

A. Power Spectral Density

PSD is an appropriate measure for the distribution of signal power across different frequency components [3]. The idea is to gain insights into the differences in brain activity between AD patients and healthy controls. Expected are increased theta power and decreased alpha power values.

1) Single subject analysis: In figure IV-A1 the overall PSD of the whole range of frequency bands of 2 AD patients with participant ID 001 and 020, as well as one healthy control with participant id 041 is depicted. The x-axis denote the Frequency in Hz, and the y-axis the Power per Frequency in dB/Hz. Additonally the figure also contains plots only showing the extract of the alpha band frequency range. The idea was to first of all try to capture an overview, if there can be seen structural differences in the overall PSD when comparing AD patients and healthy controls. Next to that, the second plot was meant to go into more detail and focus on the alpha band power since a reduced value is associated with cognitive impairment which is what is aimed to depict. What can be observed is that the overall PSD of both AD patients is weaker compared to the healthy control. The main power range for the AD patient 020 lies in between 10 and 30 dB/Hz, whereas for the AD patient 001 the main power range is in between 50 and 70 dB/Hz. For the healthy control group, the main power range is between 50 and 80 dB/Hz. Moreover, around frequency range 50 for the AD patient 020 the PSD was particularly low compared to the healthy control. For the AD patient 001, the PSD is lower compared to the healthy control, but stronger compared to the other AD patient 020. This could be connected to the fact that the AD patient 020 has a higher MMSE score, namely 4 and the AD patient 001 a score of 16. Since the frequency range 30-100 Hz is assigned to the gamma wave, the observation would suggest that the gamma wave activity is impaired in the given AD patients and the effect increases for the patient with a higher MMSE score. Altered gamma activity may indicate disrupted neural synchronization and information processing [2] and would therefore be a sensible finding. Generally speaking, the overall PSD of the subjects manifests that the PSD of AD patients is lower as well as the range of activity is reduced compared to the healthy control. The second type of plot depicting the PSD of the alpha frequency band (8-13 Hz) in more detail shows that PSD of the two AD patients is highly reduced compared to the healthy control. Especially for the AD patient 020 the difference is the highest. Again, this could potentially be related to the higher MMSE score indicating a higher degree of cognitive impairment. For the AD patient 020 the main power range is between 10 and 20 dB/Hz, whereas for the AD patient 001 this is a range of 50-60 dB/Hz and for the healthy control this is 50-70 dB/Hz. An interesting observation is that at 9.5 Hz the power is especially decreased for the AD patient 020 in the context of the whole alpha frequency band for this patient. For the AD patient 001, it can be observed that at 9.5 Hz there is also one of the lowest values in the frequency range, however the significance is not as strong as in the AD patient 020. The healthy control exhibits in this frequency of 9 Hz a relatively low value in the context of the whole frequency band, however, this biomarker is not perceptible here. In other words, a specifically low value in the range of 9 Hz can be observed in both AD patients, however not in the control which suggests that this could be a potential AD artifact. The fact that this effect is stronger for the AD patient with a higher MMSE score supports this assumption.

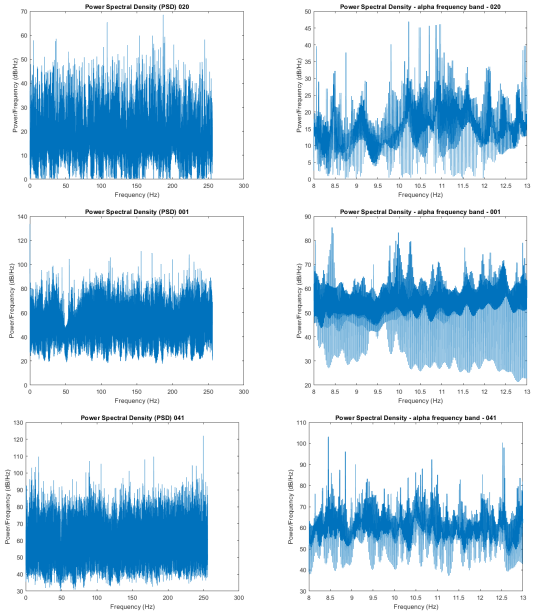


Fig. 3. Overall PSD and PSD in the range of alpha frequency band of 1 healthy control and 2 AD patients)

After inspecting the overall and alpha frequency band PSD in single subjects, the next fig IV-A1 displays the power density of an EEG signal within the alpha and theta frequency range per 1 Hz of bandwidth. This measurement helps quantifying the intensity of alpha activity at a specific point in time and is used to analyze changes in brain activity. The x-axis denotes the Frequency in Hz, and the y-axis represents the amplitude or power of the EEG signal within the specified frequency band. It's squared because power is often measured as the square of the signal amplitude. Microvolts (μV^2) are a unit of electrical potential or voltage, and squaring it provides a measure of the signal's intensity. The colorful lines denote the 19 different channels. The figure contains 6 plots, 3 plots showing the alpha frequency power from 2 AD patients and one healthy control, and 3 plots showing the theta frequency power of the same subjects. In the figure the upper left plot represents the plot for the AD patient 020, the plot in the middle is the plot of the healthy control 041 and the last graph represents the plot for the AD patient 001. The same order applies for the 3 plots right next to them. What can be observed in the alpha band frequency range is that for the healthy control there are high power values for several channels and they reach up to a Microvolt value of 10-14. These observed peaks cannot be found in the AD patients 020 and 001. What is also evident in the given plots is that for

the AD patient 020 there is also a lower Log Power Spectral Density mainly being around a density of 3 to maximum 6 ($\mu V^2/Hz$). For the AD patient 001 the main power spectrum is in between 4 and 8 ($\mu V^2/Hz$). By contrast, the healthy control exhibits a power spectrum around 4 to 12 ($\mu V^2/Hz$). Additionally, it can be observed in both AD patients mostly the lines are falling. For the healthy control group, most of the time there is a rise in the graph which is the most obvious contrast. Since these observations hold true for the alpha band, one may suggest that the lower values are connected to the cognitive impairment of the AD patients, or structural change of connectivity. Furthermore, for the theta frequency band there are different results. Namely, the graph for the healthy control shows a frequency spectrum from 6 to 13 ($\mu V^2/Hz$). The graph is mainly falling until a 6 Hz rapidly and from there on a bit slower. For the AD patient 020 it can be observed that the graph is also mainly falling rather slowly than rapidly, and exhibits a frequency power spectrum of 7 to 15 ($\mu V^2/Hz$). The graph of the AD patient 001 clearly indicates a fall of the graph as well, however it can be observed that there is a mixture of channels falling more rapidly, or more slowly. Compared to the other two plots it seems like there is more like a mixture of a dynamic. Moreover, for the AD patient 001, it is important to note that at the frequency 6 Hz there is also for a few of the channels a more slower fall like it is also for the healthy control. The frequency band power range is in between 7 to 15 $\mu V^2/Hz$). Compared to the healthy control, both AD patients show a higher Log Power Spectral Density. The value is the highest over a long period since the graph is falling slower than the other graphs. The different behavior of the PSD of the channels may correlate with AD symptoms and may exhibit connectivity changes, as well as the higher PSD may indicate coping mechanisms due to lower power in other regions. The statement of the hypothesis, that the theta power is increasing in a low MMSE score and when reaching high MMSE scores the power has fallen again compared to a healthy control, could not be observed. For both AD patients, having a MMSE score of 22 and 4, the theta frequency band power was similar.

2) Group analysis: In the last section we have looked into PSD's of single subjects. For now, the different groups, namely healthy (C) and AD patient (AD) will be compared. In fig IV-A2 4 different plots can be observed. The average alpha and theta power of 3 AD patients and 3 healthy controls is depicted there as a topoplot. The different colors denote the amplitude or power of the EEG signal in $\mu V^2/Hz$. The topoplot depicts the channels with their responsive positioning. The first two plots show the average alpha power for both groups, and the second two show the average theta power for both groups. What can be observed is that the average Alpha power for the AD patients lies in between 0.8 and 43.2 $\mu V^2/Hz$. For the healthy control group this range is in between 19.7 and 80 $\mu V^2/Hz$. Next to a clear difference in the power spectrum, it can also be observed that the location of the relative stronger activity for the AD patients is more present in the occipital

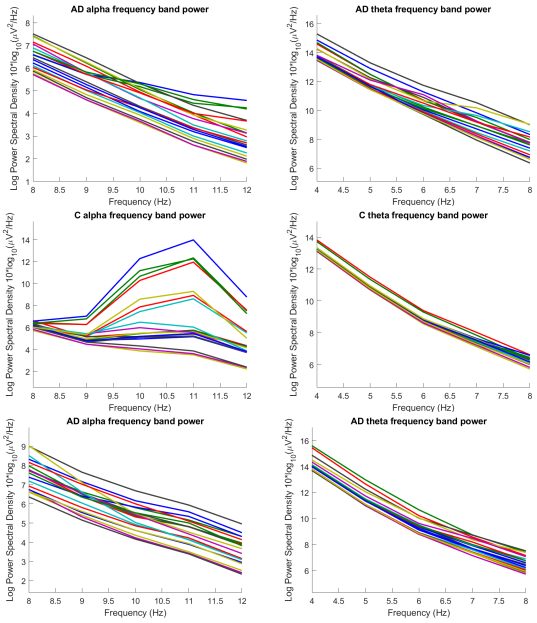


Fig. 4. The average Alpha and Theta Power from 2 AD patients and one healthy control across all channels

region with a deformation. The channel 'O1' seem to be more inactive whereas the 'P10' and 'P9' channel seem to be more active. The graph of the healthy control group exhibits also activity in the frontal region, whereas for the AD group the activity in the frontal region is diminishing low. Next to that for the control group it can be observed that compared to the AD group, that the channel'P10' shows less activity. Moreover, the channel 'O1' shows strong activity compared to the AD group. Generally speaking, what becomes evident is first of all a smaller power spectrum for the AD group and also a different activity pattern in the posterior region. The average theta power shows different results. The AD group has a power spectrum of 215-657 $\mu V^2/Hz$, whereas the power spectrum for the healthy group lies in between 192-286 $\mu V^2/Hz$. The regions of activity also differ from each other. For the healthy control group the main activity can be located in frontal and occipital regions of the brain. For the AD group, the main activity can be located only in the posterior region. Moreover, for the AD group there can be observed way more activity which may suggest a coping mechanism, since the alpha power is pretty low, so that it is tried to compensate for that through higher activity in the theta frequency. The hypothesis could be supported by the findings, since reduced alpha frequency band power and increased theta band power could be found in the AD group compared to healthy controls.

B. Coherence

Coherence is a statistical measure commonly used in EEG (Electroencephalogram) analysis to assess the degree of linear relationship or synchronization between two EEG signals recorded from different brain regions or electrodes [4]. It quantifies the consistency of phase and amplitude relationships

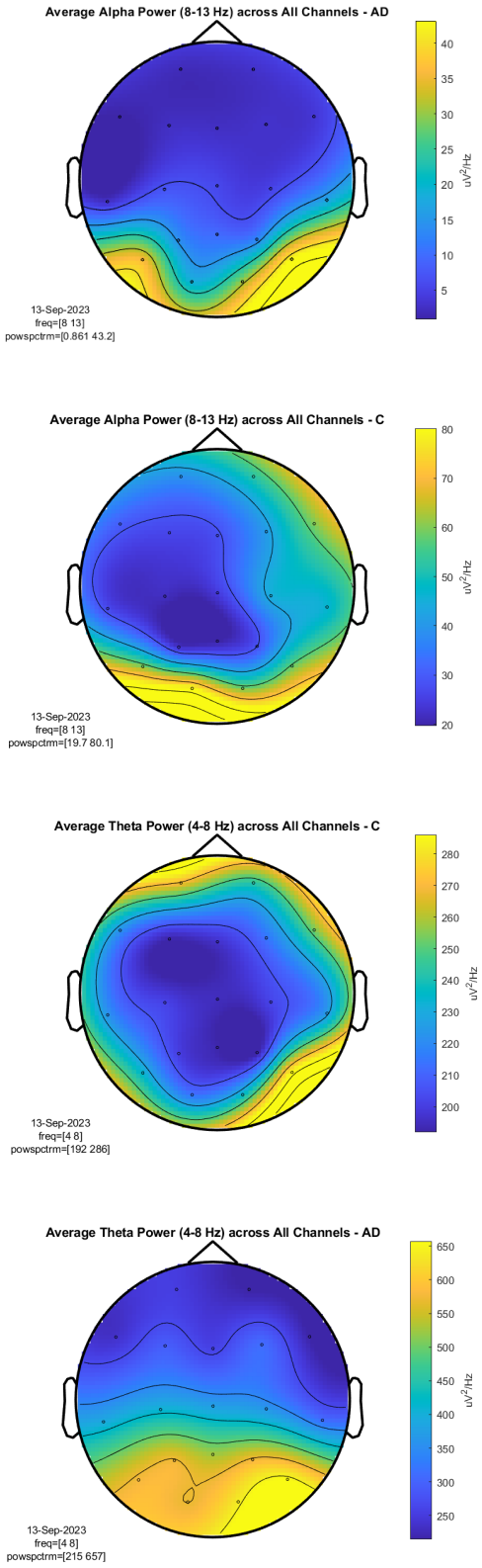


Fig. 5. The average Alpha and Theta Power for AD and C group in a topoplot.

between the signals at a specific frequency. Thus, coherence is an appropriate method in order to analyze the connectivity changes of AD patients [4]. It is expected to observe different structural patterns of connectivity compared to healthy controls.

1) Single analysis: In fig IV-B1 the average coherence of certain channel pairs in the alpha frequency range for different subjects is depicted. The x-axis denotes the channel pairs and the y-axis the average coherence. The channel pairs contain a range of frontal, temporal location and also location in between different hemispheres. Channel pairs between frontal and frontal regions are of interest because they are associated with executive functions and working memory, which are often impaired in AD [4]. Channel pairs of frontal and temporal regions are involved in memory processes. AD patients often exhibit progressive memory impairments, and EEG measures can help elucidate the neural basis of these deficits. The channel pairs of the different hemispheres are interesting because they may exhibit an impaired communication between each other due to AD [4]. What can be observed in the figure is that the average coherence of all the channels is highly reduced in AD patient 020. In particular the channel pair Fp2-F4 is reduced with a coherence of 0.1. For the AD patient 005 the coherence of the value is reduced to around 0.65 whereas for the healthy control the coherence is about 0.9. Here a correlation to the cognitive decline can be observed that the effect is stronger in the higher MMSE score patient, however, still present in the lower MMSE score patient and not at all present in the healthy control. In other words, it can be observed that the working memory is impaired in the AD patients quite significantly.

2) Group analysis: In IV-B2 There can be seen a group analysis for the average coherence of 3 AD patients and a healthy control group of 3.The axis are the same as in fig IV-B1.Again, the alpha frequency band range is depicted. What can be seen is that the Frontal to frontal channel pairs (Fp1-F3, Fp2-F4) are significantly reduced for the AD group. The difference in AD and control group is about 0.2. F3-T3 and F4-T4 channel pairs are a bit reduced, for the AD patients a coherence of 0.6 and 0.7, and for the control group the coherences are both about 0.8. The C3-C4 and F3-F4 channel coherence is also reduced for the AD group by around 0.2 coherence difference each. Generally speaking, it can significantly be observed that all of the channel pairs for the AD group exhibit reduced coherence. This may represent cognitive decline and the impairment of working memory. Concerning the hypothesis, decreased connectivity could be observed, however no increased connectivity. This is probably the case because a certain channel pairs were selected that are associated with memory processes. Maybe another selection of channel pairs may have shown a broader variety.

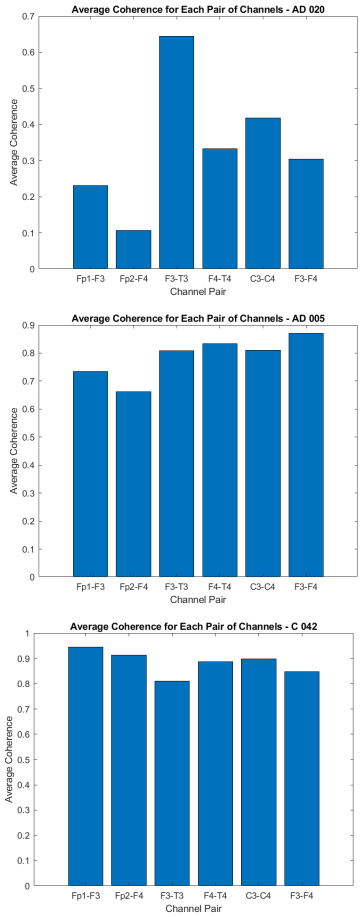


Fig. 6. The average coherence of selected channel pairs for 2 AD patients and one healthy control.)

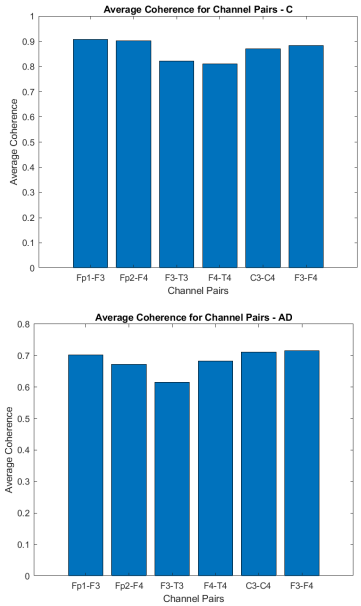


Fig. 7. Average coherence of selected channel pairs for group AD and C.)

V. Conclusion

The paper analyzed hypotheses driven by 6 different EEG datasets of 3 AD patients and of 3 healthy controls. The first hypothesis, that AD patients will show reduced alpha power and increased theta power could be supported by the results. There could be observed a correlation of cognitive decline and reduced alpha power, however no significant correlation for the theta power. The second hypothesis, namely that AD patients will exhibit altered functional connectivity patterns could be approved in terms of significant reduction of coherence, however not in terms of an increase. In terms of a reduction of the coherence, a correlation with the cognitive decline was evident. Concerning the central question it is important to note that EEG findings in AD are not uniform across all patients, and individual variations can occur. The two biomarkers elaborated in this paper, namely frequency band power and connectivity patterns, may serve as an indication, however they are not sufficient to make a diagnosis. More neural imaging procedures and investigations are necessary. The individual patient has to be considered, as well as an overlap with other neurodegenerative illnesses has to be excluded. There is the need for a metric that contains several biomarkers and analysis methods, so that an interpretation for a diagnosis could be more appropriate since more factors are included. Furthermore, there is also the option to employ Machine learning approaches that are capable of recognizing patterns, maybe better and more efficiently than humans do. A research field opens where EEG data and ML approaches are interacting with each other.

VI. Pictures title page

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