Heliyon 7 (2021) e07984

Contents lists available at ScienceDirect

Heliyon

journal homepage: www.cell.com/heliyon

Research article

Complexity analysis of the brain activity in Autism Spectrum Disorder (ASD) and Attention Deficit Hyperactivity Disorder (ADHD) due to cognitive loads/demands induced by Aristotle's type of syllogism/reasoning. A Power Spectral Density and multiscale entropy (MSE) analysis

Anastasia G. Papaioannou [a,b,*], Eva Kalantzi [a], Christos C. Papageorgiou [c], Kalliopi Korombili [a], Anastasia Bokou [a], Artemios Pehlivanidis [a], Charalabos C. Papageorgiou [a,b,**], George Papaioannou [d,***]

[a] 1st Department of Psychiatry, National University of Athens, Medical School, Eginition Hospital, Athens, Greece
[b] University Mental Health, Neurosciences and Precision Medicine Research Institute "COSTAS STEFANIS", (UMHRI), Athens, Greece
[c] 251 Air Force General Hospital, Athens, Greece
[d] Center for Research of Nonlinear Systems (CRANS), Department of Mathematics, University of Patras, Patra, Greece

ARTICLE INFO

Keywords:
Multiscale entropy
Power Spectral Density
Aristotle's syllogism
ASD-ADHD
Systems of thinking I&II
Cognitive load

ABSTRACT

Objective: We aim to investigate whether EEG dynamics differ in adults with ASD (Autism Spectrum Disorders), ADHD (attention-deficit/hyperactivity disorder), compared with healthy subjects during the performance of an innovative cognitive task: Aristotle's valid and invalid syllogisms. We follow the Neuroanatomical differences type of criterion in assessing the results of our study in supporting or not the dual-process theory of Kahneman, 2011) (Systems I & II of thinking).

Method: We recorded EEGs from 14 scalp electrodes in 30 adults with ADHD, 30 with ASD and 24 healthy, normal subjects. The subjects were exposed in a set of innovative cognitive tasks (inducing varying cognitive loads), the Aristotle's four types of syllogism mentioned above. The multiscale entropy (MSE), a nonlinear information-theoretic measure or tool was computed to extract features that quantify the complexity of the EEG.

Results: The dynamics of the curves of the grand average of MSE values of the ADHD and ASD participants was significantly in higher levels for the majority of time scales, than the healthy subjects over a number of brain regions (electrodes locations), during the performance of both valid and invalid types of syllogism. This result is seemingly not in accordance of the broadly accepted 'theory' of **complexity loss** in 'pathological' subjects, but actually this is not the case as explained in the text. ADHD subjects are engaged in System II of thinking, for both Valid and Invalid syllogism, ASD and Control in System I for valid and invalid syllogism, respectively. A surprising and 'provocative' result of this paper, as shown in the next sections, is that the Complexity-variability of ASD and ADHD subjects, when they face Aristotle's types of syllogisms, is higher than that of the control subjects. An explanation is suggested as described in the text. Also, in the case of invalid type of Aristotelian syllogisms, the linguistic and visuo-spatial systems are both engaged ONLY in the temporal and occipital regions of the brain, respectively, of ADHD subjects. In the case of valid type, both above systems are engaged in the temporal and occipital regions of the brain, respectively, of both ASD and ADHD subjects, while in the control subjects only the visuo-spatial type is engaged (Goel et al., 2000; Knauff, 2007).

Conclusion: Based on the results of the analysis described in this work, the differences in the EEG complexity between the three groups of participants lead to the conclusion that cortical information processing is changed in ASD and ADHD adults, therefore their level of cortical activation may be insufficient to meet the peculiar cognitive demand of Aristotle's reasoning.

* Corresponding author.
** Corresponding author.
*** Corresponding author.
E-mail addresses: anas.papaioannou24@gmail.com (A.G. Papaioannou), chpapag@med.uoa.gr (C.C. Papageorgiou), gpthespies@gmail.com (G. Papaioannou).

https://doi.org/10.1016/j.heliyon.2021.e07984
Received 2 February 2021; Received in revised form 13 May 2021; Accepted 8 September 2021

Significance: The present paper suggest that MSE, is a powerful and efficient nonlinear measure in detecting neural dysfunctions in adults with ASD and ADHD characteristics, when they are called on to perform in a very demanding as well as innovative set of cognitive tasks, that can be considered as a new diagnostic 'benchmark' in helping detecting more effectively such type of disorders. A linear measure alone, as the typical PSD, is not capable in making such a distinction. The work contributes in shedding light on the neural mechanisms of syllogism/reasoning of Aristotelian type, as well as toward understanding how humans reason logically and why 'pathological' subjects deviate from the norms of formal logic.

1. Introduction

The main aim of the present work is to shed light on the connection between two of the 'special' types of reasoning, namely the *Aristotle's valid and invalid types of syllogisms* with the dual-process theory of thinking, according to which humans have a unique ability to engage in different modes of thinking: the intuitive (System I) and the analytical (System II) (Kahneman, 2011, Stavovich and West, 2000). Support for dual-process theories originate from a wide range of studies (e.g categorization, judgment and decision making, problem solving, inductive, deductive and probabilistic reasoning (Osman, 2004). Based on such a range, this present work focuses on evidence from syllogisms (other types of evidence are the selection task and the conjunction problem). The findings from various studies following one of the above types of evidences are assessed on the base of four criteria: *Criterion S (Sloman's criterion S), the individual differences, the Implicit vs. explicit processing* and finally the *Neuroanatomical differences*, on which the assessment of the results of our study is based (Osman, 2004).

More specifically, in this paper, by using EEG recordings from *healthy (control) and 'pathological' subjects (ASD and ADHD)* and combining two approaches (working in a complementary mode), a linear (power spectral density analysis, PS) and a nonlinear (Multiscale entropy, MSE), we examine possible Neuroanatomical differences or equivalently differences in the performance of the subjects, due to their exposure in cognitive loads of varying difficulty induced by the 'peculiarities' of the Aristotelian syllogisms. This examination or analysis, as a consequence, necessitates the examination of the involvement or coupling of the following 'concepts': *a) the systems of thinking I & II, b) The Aristotle's types of syllogism c) the complexity-variability of EEG signals of healthy and 'pathological' subjects, as they are quantified by PS and MSE, d) the primary cognitive processes (cognitive control, attention, working memory etc.) and their associated changes in alpha, theta, beta, delta and gamma rhythms.* The interaction of the above 'concepts' will provide valuable information towards answering the question of whether or not the main cognitive processes in subjects exposed in Aristotelian syllogisms engage System I or II of thinking, i.e. supporting or not supporting the dual-process theory of reasoning.

Therefore, in order to facilitate the interpretation of the results of this work, in answering the above question, it is necessary to provide a short but also concise introduction and literature review.

1.1. System I & II of thinking

Daily decisions we make range from fast, intuitive responses to slow deliberations. Broadly the models of thinking are classified in two categories, the intuitive (System I) and analytical (System II). Whereas *System I is fast, automatic and effortless*, *System II is slow, contemplative and effortful* (Kahneman, 2011; Evans and Stanovich, 2013a). System I is the main Operator of the brain, however in some cases, it is interrupted by the System II which takes the control in order to explore alternative decision options that *require significant mental effort (increased cognitive load)*.

No general consensus exists about the cognitive processes involved in these two systems, however System I is attributed to autonomous processing while System II to high-level cognitive mechanism (Evans and Stanovich, 2013a; Pennycook, 2017).

Systems I and II are linked to:

- Cognitive control (Kahneman, 2011; Pennycook, 2017)
- Attention (Brush et al., 2017)
- Working memory (Evans and Stanovich, 2013b)
- Long-term memory (Brush et al., 2017)

Evans et al., (2013b), argue also that all above cognitive processes are the outcome of activation of *cognitive networks that interact*, as the EEG signals reveal (significant power in the theta and alpha brain rhythms). For example, increased frontal theta activity (event-related synchronization) is linked to cognitive control (Cavanagh and Frank, 2014) and working memory (Hsieh and Ranganath, 2014), while the recruitment of attention is linked to decreased parietal alpha activity (event-related synchronization) (Klimesch, 2012). Long-term memory is associated with increased parietal alpha activity (Klimesch, 2012). A very interesting finding is that *novelty conflict, punishment and error* are associated with a cognitive *control under uncertainty* (Cavanagh and Frank, 2014). The mental effort in remembering increasing number of things, facts etc. is associated with *increased theta activity* with which maintenance and manipulation are also linked. Computational demands are linked to all components of working memory. Attention to a task-relevant information is associated with a decreased alpha activity. Access of knowledge systems (including long-term memory) is linked to increased alpha activity, a finding that is in compliance with the broadly accepted notion that *long-term memory is an automatic process* (Brush et al., 2017).

In summary, *System I operation may reflect autonomous access to long-term memory and System II operation may involve the recruitment of cognitive control, working memory and focused attention.* This premise is supported by the robust *interconnectivity* between cognitive control, working memory, attention and long-term memory (Mathewson et al., 2014; Klimesch, 2012).

A natural and challenging question that arises is *how the above mentioned important cognitive processes are connected to (so be assessed within System I and II framework) to Aristotle's system of syllogism,* a question, the answer of which, is one of the main objectives of the present paper. The evaluation of the four important cognitive processes within the framework of System I and II may not be simple.

One of the first attempts for such an evaluation is the study Complex word or semantic problems (tasks) that are difficult to implement in neuroimaging research. However, a limited number of EEG studies, based on ERPs, provide good examples of the linking of cognitive processes with Systems I and II (Banks, 2017). Evans and Stanovich (2013b), provide a relatively good number of various tasks that can be used towards assessing Systems I and II mode of thinking in relation to cognitive process. We mention here the seminal work of Kahneman et al. (1968), in which manipulated thinking mode, induced by having subjects make computations under time pressure, was measured by pupillometer (a tool for measuring the dilation of the pupil in an eye). They found that increased pupil size in the *more demanding cognitive task* (add-one condition, in their famous experiment), was analogous to *increased processing load and therefore System II or analytical thinking.*

1.2. The structure of Aristotle's syllogism and its relation to cognitive processes

We briefly introduce at this point the syllogistic reasoning task and orthodox Aristotelian classification. This information is considered

necessary in order to interpret easily the cognitive loads that the Aristotelian syllogisms impose on subjects trying to 'handle' them during experiments. Syllogisms are constructed with two premises and one conclusion. Each preposition or statement belongs to a group of four forms called ***moods*** (Smith and Translator, 1989; Owen et al., 2015). These moods, traditionally, are labeled A, I, E and O, as below (Hattori, 2016):

A: All X are Y
I: Some X are Y
E: No X is Y
O: Some X are not Y

The subject (S) (in a preposition) and predicate (P) in the conclusion are called *end terms,* while a term not present in the conclusion is a *middle term (M).* There are four arrangements or possibilities of end and middle terms, since each premise has two possibilities. The four possibilities mentioned above are called figures. Therefore, there are 4X4X4 = 64 possible types of premises for logical syllogisms, because each of two premises can be one of the four moods and 4 possibilities regarding the location of terms. Only 19 syllogisms out of 64 have a logically conclusion (they are ***valid***), even though the validity of a syllogism is a relatively controversial concept (Hattori, 2016). Reasoning is intended to derive reasonable conclusions from premises and is carried out in working memory. Human *performance on syllogistic reasoning* is based on mental representations, as the mental model theory (one of the earliest comprehensive psychological theories of syllogism) explain (Jhonson--Laird and Bara, 1984). As the authors in this paper claim, the *difficulty of syllogistic reasoning is a function of the number of mental models that must be constructed to derive a logically valid conclusion.* Also, a task that requires more models to be constructed to reach a correct answer, increases the probability of errors in the inference process, resulting possibly to a failure. As the mental model theory claims, the difficulty of (or cognitive load exerted in) syllogisms is determined primarily by the number of models and the Aristotelian *figure* (one of the four likely possibilities mentioned above). Mental model theory of syllogistic reasoning, incorporates explicitly the *working memory capacity*, an important component of cognitive process (Baddeley, 2007). According to *Sample Mental Model (SMM),* a probabilistic approach in a mental representation (Hattori, 2016), *people 'use' or sample six or seven instances in working memory to derive a conclusion to a syllogism.* However, Halford et al. (2007) argue that the *limit of working memory capacity* is actually three to five chunks ('fat pieces'), and this limit reflects human's capacity for attention and constraints the relational representations, enabling making inferences.

All the preceding information lead to the conclusion that the 'architecture' or structure of Aristotelian syllogisms, reflected by *modes and figures*, in combination with the difficulty of syllogistic reasoning (which depends on the number of mental models that must be constructed before reaching a valid conclusion) and the limited capacity of working memory constraining the ability of working inferences, may be considered as the sources of cognitive loads, exerted on participants that are tested in Aristotelian syllogisms, that are responsible for shaping the dynamics of the EEGs recorded during relevant experiments. The aforementioned sources of cognitive loads may be located at, and activated by different brain regions, with different ways depending on task conditions or 'pathological' condition of a subject. It is challenging therefore to examine how normal, ASD and ADHD subjects 'react' when facing Aristotelian valid and invalid syllogisms, since the loads these syllogisms exert on subjects belonging to groups previously referred, differ as described above. Aristotle's method of deduction is probably the first to analyze the logical reasoning or syllogism (also called valid reasoning). In his famous work 'ORGANON – Prior analytics' (Smith, 1989; Owen et al., 2015), the great philosopher presents a series of statements (the 'building blocks') in the process of reasoning that leads to a *valid conclusion with absolute certainty*. The *dual processing* model for the logical reasoning (De Neys, 2009; Goel, 2007; Williams C. et al., 2019), is developed to help understanding how the brain functions when a subject is 'engaged' in this type of reasoning. A natural question that is generated is whether an Aristotle's valid and invalid syllogism induce the same or different mental processes in the brain. This is a current, still open and challenging research question, aiming at shedding light in the fundamental operation of reasoning, in its two extreme conditions.

In this work, we focus on an experiment called *'Aristotle's experiment'*, in which EEG signals of participants belonging to three groups (control, ASD and ADHD) are recorded, when these subjects applied valid, invalid, paradox and illusions type of reasoning. Special care was taken during the experiment, to induce the *working memory* (WM) of each participant, as WM is a very crucial cognitive activity that helps humans to retain information 'alive', not just for memorizing purposes but also for other very important cognitive tasks, like reasoning, problem solving, decision making, planning etc.

One of the main objectives of this work is to find out whether different modes of electro-physiological activity are activated when healthy (controls), ASD and ADHD patients are exposed in *Aristotle's valid and invalid syllogisms*. The contribution of this paper is that it sheds light in how critical aspects of reasoning process, related to attention, perception and cognitive behavior, differs between the aforementioned groups of participants. The difference in EEG signals in the case of healthy participants exposed in valid and paradoxes syllogisms has been analyzed in case of a single subject in the work of Papaodysseus et al. (2016). Therefore, the present work can be seen as a natural extension of the previous paper, answering some of the questions suggested as future considerations, however it examines the complexities of EEGs taken from subjects of three different groups when they are 'exposed' in Aristotle's valid and invalid cognitive loads, so it differs from aforesaid work in various ways.

1.3. Syllogistic reasoning and linguistic and visuo-spatial systems. Connection with systems I&II

Goel et al. (2000) provide strong evidence that syllogistic reasoning implicates a widespread network involving occipital, temporal and parietal lobes, prefrontal cortex, and surprisingly, cerebellum and basal ganglia nuclei. According to Goel et al. (2000), syllogistic reasoning is implemented in two distinct systems, whose engagement is mainly a function of the presence or absence of *semantic* content. In fact, the *temporal* system (left hemisphere, LH) is recruited during *content-based* reasoning, while the *parietal* system is activated when the reasoning lacks semantic content. The two systems however, share common components (bilateral basal ganglia nuclei, right cerebellum, bilateral fusiform gyri and left prefrontal cortex). In addition, the *right prefrontal cortex* is recruited when logical argument results in a *belief-logic conflict*. What is doubtable in Goal's et al. work (Knauff, 2007), is the conclusion these authors draw from their findings. Specifically, the conclusion that the *frontal-temporal system* is more *'basic'*, and effortlessly engaged (i.e. 'corresponds' to *System I)*, while the parietal system is effortfully engaged (i.e. 'corresponds' to System II) only when the frontal-temporal route is due to a lack of familiar content. Under the current perspective, the question of how mental logical reasoning is implemented in the human brain is a question of *formal reasoning*. Goel et al. (2000) and Knauff (2007) argue that in syllogistic reasoning, both linguistic and visuo-spatial systems are engaged. Frontal cortex place central role in logical reasoning. Since Aristotelian Syllogism 'belongs' to the model-based reasoning theoretical context, which suggests that reasoning is a visuo-spatial process it is natural to deduce that parietal and occipital cortices are essential brain structures for Aristotelian syllogism. More specifically, Goel et al. (2000) used problems with semantic content (e.g. 'All A are B; all B are C; so all A are C). Based on the discussion section 4, tables 15 and 16, we attempted a connection of our results with the above information.

1.4. Complexity and MSE in normal and 'pathological' conditions

A novel approach of analysis and description towards investigating normal or typical and pathological states, degenerative or developmental, is the Physiological Complexity, a term which combines physiology with complexity, the later developed and used extensively in the fields of physics and mathematics. In complexity perspective, seemingly irregular dynamic evolution of physiological signals may contain a significant amount of nonrandom (of 'nonlinear deterministic' or stochastic type) fluctuations over multiple time scales, 'hidden' in the signals and not easily or never revealed by using 'typical' or linear tools of analysis (Glass and Mackey, 1992; Manor et al., 2010). Adopting the complexity approach, Costa et al. (2002, 2005), introduced the entropy analysis in biological and physiological signals or time series, more specifically the *Multiscale entropy* (the tool that is also used in this work), while Fallani Fde et al. (2010), used graph theory, a nonlinear approach, to study brain functional networks.

To investigate the differences in complexity of EEGs for the participated groups of subjects, the multiscale entropy (MSE) is adopted in this study. Entropy is a measure to quantify the complexity or order of a system. Systems exhibiting periodic or regular dynamic behavior are said to have low values of entropy. Irregular or random noise-like dynamics have high values of entropy. Regularity and complexity are not necessarily correlated. For example, white noise (a random process), even though has a high value of entropy does not have the characteristics of a complex system since does not possess the *structural – informational richness over multiple temporal scales* that a genuine complex system exhibits. A measure of complexity to be able distinguish an EEG signal from a linear or nonlinear stochastic signal, behaving the 'same way' (for example in view of the autocorrelation function etc.) as an EEG one, the MSE developed by Costa et al. (2002,2005), is applied here and it is a powerful tool in detecting the multiple time-scales present in a physiological signal, as the EEG, using a *coarse-graining procedure*. This type of procedure suggests that *optimally operating biological systems are modulated* by multiple mechanisms which *interact over multiple temporal scales. Therefore for these (optimally) functioning systems, the MSE is expected to have a high value, sustained for increasingly coarser time-scale.* On the opposite, the signals generated by *random processes will have MSE or entropy values that decrease as the timescales increase.* This is actually an expected result since the information in random noise remains only on the shortest timescale (information is lost), due to the fact that no new structure in the signal is revealed, as the timescale increase. In fact, the variance of the signal decreases, resulting in a decreasing value of the entropy.

A number of articles published relatively recently, provide an evidence that a plethora of pathological processes, like in ASD and in ADHD that are examined in this work, are 'linked' to atypical and often, but not always, to the phenomenon of *reduced levels of physiological complexity* regardless the developmental and clinical situations, that could attribute a different understanding on the changes in complexity encountered in such conditions. In the case of detecting differences in EEG complexity between normal and patients with schizophrenia, Takahashi et al. applied the multiscale entropy method (Takahashi et al., 2010).

Brain activity complexity, in particular, measured by EEGs is an appealing area for research, because they incorporate the simultaneous action of a number of factors-sources that interact with each other, are nonlinearly coupled and induce feedback loops. This structure has the ingredients of a high-dimensional, nonlinear 'deterministic' or stochastic system that exhibits complex dynamic behavior capture in the EEGs (Sakkalis et al., 2008). The fine temporal resolution the EEGs provide, make them suitable for analyzing their inherited nonlinear or chaotic characteristics, which originate from complex functioning of the brain, when the subject is exposed in various cognitive loads. *The aim of this paper is the detection of differences in the response of subjects in the control, ASD and ADHD groups, when they are tested in cognitive tasks and conditions within the frame of the Aristotelian syllogisms (described in section 2).* The *Aristotelian* type of valid and paradox reasoning was applied in a new method of classification of event related potential (ERPs) (Papaodysseus et al., 2010). In the present work, we follow the same 'way of thinking', regarding the cognitive loads that are 'imposed' on the subjects under examination, but now focusing on the differences in the EEGs of ASD, ADHD and normal groups, in the cases of valid and invalid types.

It seems very reasonable to assume that ASD subjects (exhibiting behaviors reflecting the autism spectrum conditions), may be associated with atypical patterns of brain complexity. This is because the core social and ***cognitive*** characteristics of ASD, according to the American Psychiatric Association, 2000 (restricted repetitive range of behaviors, interests and activities, impairments in social interactions and qualitative disturbances in communication), as well as recently recognized characteristics as atypical patterns of sensory and motor functioning and interaction (Simmons et al., 2009), atypical visual perception (Kaiser et al., 2010), auditory perception (Hitoglou et al., 2010) and the reduced adaptability to environmental changes (Russo et al., 2007), suggest that they are possibly connected with EEG signals of atypical complexity. Brain functioning models aiming to explain the above mentioned features of ASD, include those suggesting disturbances in the underlying brain complexity, atypical neural connectivity (Barttfeld et al., 2011), and *disrupted temporal integration of information* (Rippon et al., 2007).

The idea that in autism an atypical functional complexity may exists, is enhanced by the observation that *subjects without ASD* exhibit *improved adaptability to cognitive demands or loads* associated with increasing variability (or volatility), as it is manifested by greater complexity in scalp EEG (Sitges et al., 2010). Therefore, it seems natural to adopt a *complexity measure* in order to 'measure' the possible differences in the amplitude and frequency dynamics of EEGs taken from ASD, ADHD adults patients and a matched typically developing control (normal) group of subjects. *A surprising and 'provocative' result of this paper, as shown in the next sections, is that the Complexity-variability of ASD and ADHD subjects, when they face Aristotle's types of syllogisms, is higher than that of the control subjects. An explanation is suggested as described in the discussion section.*

ADHD and ASD are the most prevalent neurodevelopmental disorders. ADHD is characterized by developmentally inappropriate inattention, impulsiveness, and/or hyperactivity that remain relatively persistent over time and result in impairment across multiple domains of life activities. ASD is characterized by persistent deficits in social interaction and communication as well as restrictive, repetitive patterns of behavior or interests American Psychiatric Association (2013). These disorders in most cases persist into adult life. There is a significant unmet clinical and research need to understand the persistence into adulthood (Kooij et al., 2019; Lai and Baron-Cohen, 2015; Baron-Cohen et al., 2001).

An extensive literature exists for the assessment of EEG characteristics related to ADHD, revealing a continuous substantial interest of the researchers. The majority of the literature concerns measures or indicators of frequency-domain, as estimates of absolute and relative power for frequency bands (Barry et al., 2003; Swartwood et al., 2003). These two works, and especially the last one, are very connected –related to the focus of our work, since it describes the EEG differences in ADHD during baseline and cognitive tasks. It therefore helped us toward finding an appropriate way to link Aristotle's syllogisms with cognitive tasks of varying cognitive difficulty (load), imposing different demands on the brain of the examined subjects with ADHD, resulting in EEG with different complexity levels.

In typical (normal) development from childhood to adult, brain activity is associated with increasing MSE values (McIntosh et al., 2008). Patients with schizophrenia show an *increase MSE* in *fronto-central and parietal* brain regions (Takahashi et al., 2009). The same researchers have shown that treatment of schizophrenia with antipsychotics is associated with *reduced entropy*. A more relevant to ours work is the one by Bosl et al. (2011), which shows a decrease in resting state EEG complexity, during several phases of development, for infants at high risk of ASD, in

Table 1. Main findings in studies examining the link of disorders with various cognitive tasks extracted from literature review.

Neuropsychiatric Disorder	Study	Subjects	Analysis Methods	Condition	Main Findings
ADHD	Li et al. (2016)	13 ADHD 13 control	δ, θ, α, β MSE	Multi-source interference task (MSIT)	Increased complexity of EEG data in delta, theta frequency bands and decreased bands in ADHD children
	Ke et al. (2014)	14 healthy adults	Θ, β MSE	Visual attention Resisting No attention	Higher level of visual attention is correlated to greater values of MSE. Classification of recognition of 3 levels of attention is superior when using MSE feature extraction than by using classical θ/β ratio.
	Khoshnoud et al. (2017)	12 ADHD 12 age-matched healthy children	α, β, θ, δ mDFA, PCA	During rest (eyes-closed)	Average LLE of the ADHD group was significantly higher than the Control. Mean ApEn in ADHD subjects was lower than the control group, at all 19 channels
		11 ADHD adolescent boys 12 healthy boys	Θ, α, β, γ ApEn, PSD	Rest During CPT (continuous performance test)	Mean ApEn in ADHD patients was lower than the healthy subjects over the frontal region Fp2, F8 during CPT, but not at rest.
ASD	Heunis et al. (2018)	16 ASD 46 TD (typically developing)	RQA Recurrence Quantification analysis SVM	Resting	RQA differentiate ASD from TD, in the age-matched sample, leave-one-subject-out classification with a nonlinear Support Vector Machine (SVM). It shows 92.9% accuracy, 100% sensitivity and 85.7% specificity).
	Bosl et al. (2017)	18 ASD 26 CAE 47 controls	Modified MSE RQA	Resting	In the frontal, occipital and left temporal areas, ASD subjects showed higher MSE
	Takahashi et al. (2016)	43 ASD 72 TD	MSE	Video-watching	Increased Complexity (MSE) in MEG signals from ASD subjects (typical age-related). In younger children with ASD the complexity in MEG is enhanced.
	Catarino et al. (2011)	15 adult with ASD 15 normal (control)	MSE PSD	EEG in a face and chair detection tasks	During tasks, a reduction of EEG Complexity over temporal-parietal and occipital regions, in ASD subjects was observed, compared with typical controls, using MSE measure. No changes in PSD observed.
	Bosl et al. (2011)	46 HRA 33 Normal (Controls)	Modified MSE	Resting	Different complexity profiles in HRA and control infants were detected. SVM was used for classification.

-Note: EMD: Empirical Mode Decomposition, IMF: Intrinsic Mode Fuction, PSD: Power Spectral Density, - LLE: Largest Lyapunov Exponent, ApEn: Approximate Entropy, mDFA: multi fractal detrended fluctuation analysis, - PCA: Principal Component analysis δ, θ, α, β, γ –delta, theta, alpha, beta and gamma frequency ranges.E9, - CAE: childhood absence epilepsy, - RQA: Recurrence qualification analysis, - HRA: High- Risk autism.

Table 2. Linking of Systems I and II way of thinking with cognitive processes and frequency bands (α, β γ, θ, δ), found in the literature.

Cognitive Control Process	System I Frequency Bands		System II Frequency Bands	
	α	θ	α	θ
Cognitive Control		Frontal Fz	Parietal CPz Recruitment of CC	Frontal
Working Memory	Parietal CPz Release of WM	Frontal Fz	Parietal Recruitment of WM	
Attention	Parietal CPz Release of A	Frontal Fz	Parietal CPz focused A	
Long- Term Memory	Parietal PCz Recruitment of autonomic LTM	Frontal Fz	Parietal CPz no need to access LTM	

Note: WM = Working Memory, A: Attention, LTM: Long Term Memory, CC = Cognitive Control.

comparison with infants at low risk of ASD. As Green (1996) has shown for the case of schizophrenia, the functional consequences of neuro-cognitive deficits in ASD and ADHD conditions are 'translated' to reduced complexity of the brain functioning. *Sustained cognitive operations* such as *logical reasoning, thought continuity and working memory*, are known to be affected in ASD and ADHD disorders (Catarino et al., 2011; Ponomarev et al., 2014). This kind of operations are related to long-time scales (tens of seconds) and correspond to temporal patterns of neuronal activities resembling random-like processes. ASD and ADHD can be deemed as dynamic processes neuronal activities, evolving in time that have not addressed adequately, as the literature reveals. These processes are associated with highly volatile neuronal states as abnormal functional

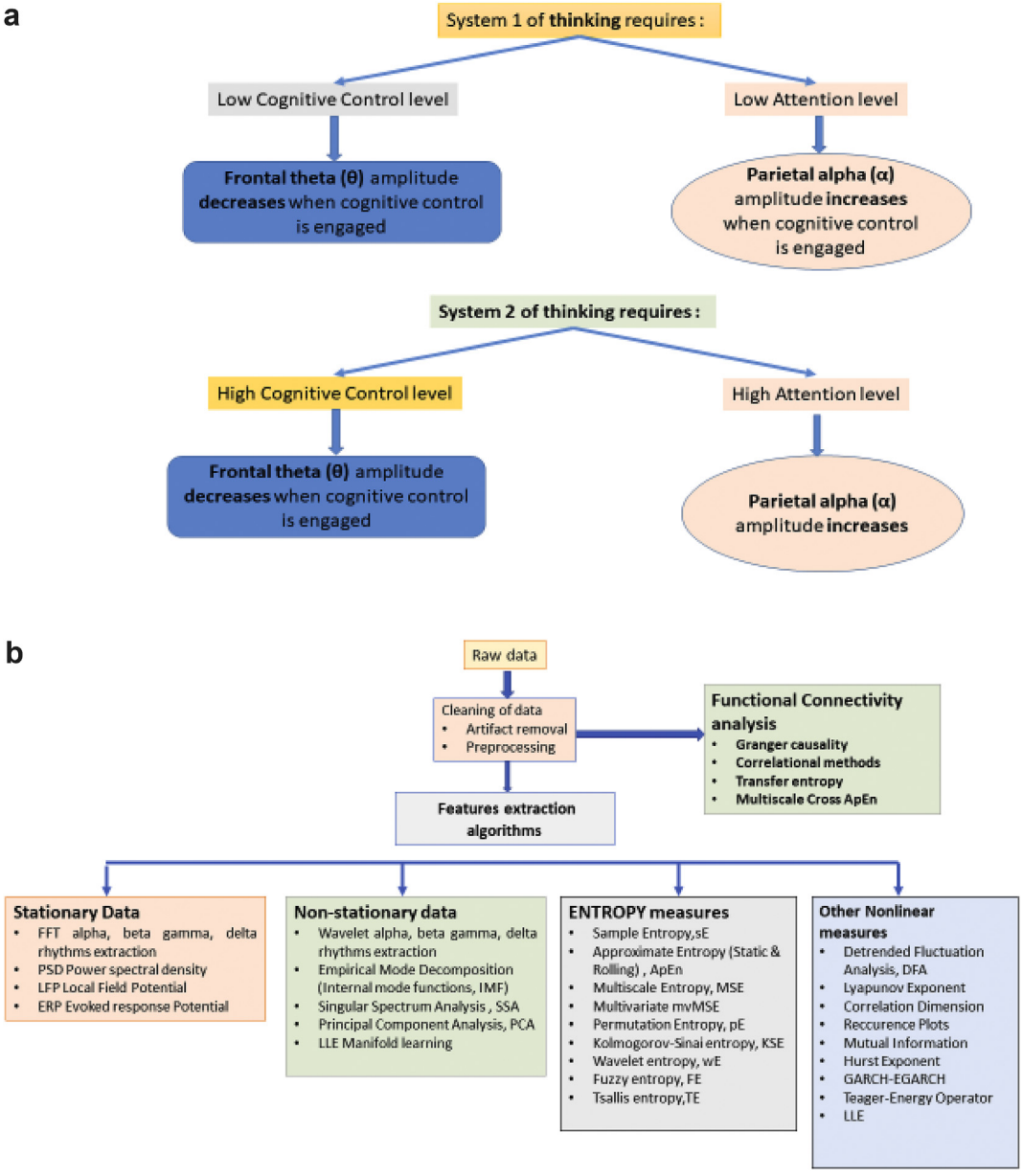

Figure 1. a: Systems of thinking and their connection to Cognitive Control and Attention. b: An indicative workflow. Our approach (path) is from raw data to preprocessing, Multi Scale Entropy (MSE) in combination with Power Spectral Density.

Table 3a. Groups of Subjects engaged in Systems 1 and 2 of Thinking based on Theta and Alpha amplitudes, for the valid type of syllogism.

Group	High (increased) frontal Theta (θ) (4–8 Hz)	Low (decreased) parietal alpha (α) (8–13 Hz)	Engagement in System of Thinking I or II
ASD	Yes, in 3 channels		1
ADHD	Yes, in 7 channels	Yes, in channel P7	2
CONTROL	Yes, in 3 channels	Yes, in channel P8	2
	Cluster 1: ASD + Control Cluster 2: ADHD		

Table 3b. Groups of Subjects engaged in Systems 1 and 2 of Thinking based on Theta and Alpha amplitudes, for the invalid type of syllogism.

Group	High (increased) Frontal Theta (θ) (4–8 Hz)	Low (decreased) Parietal alpha (α) (8–13 Hz)	System of Thinking
ASD	Yes, in 1 channel	Yes, in channel 8	2
ADHD	Yes, in 6 channels	Yes, in channel 7	2
CONTROL	Yes, in 1 channel		1
	Cluster 1: ASD + Control Cluster 2: ADHD		

connectivity which allows irregular and random-like jumps or switching between different brain regions or neuronal populations.

ASD and ADHD pathologies are linked to abnormal brain activities, therefore they can be viewed on the basis of a *dynamical system*, in which the neuronal phase or state space is the so-called *attractor*, a geometric structure encompassing the 'attracted' trajectories or paths of the system (describing the dynamics of neuronal activities), due to reduction in excitations and inhibitory synaptic transmission, inducing change in the neuronal states**.** Table 1 summarizes key findings in studies reviewed in literature that examine the link of disorders with various Cognitive tasks extracted from literature review. We have included also in this table (first entry), the main findings of the present work, in an attempt to show where its contribution is positioned. Table 2 below, presents the linking of Systems I and II way of thinking with cognitive processes and frequency bands [alpha (α), beta (β), gamma (γ), theta (θ), delta (δ)], found in the literature.

1.5. Linking cognitive processes with systems I & II of thinking and alpha, beta, theta rhythms

Going form System I to System II way of thinking, due to a *systematically increasingly cognitive load*, is associated with an *increased frontal theta (θ) power* and decreased *parietal alpha (α) power* (Williams et al., 2019). *Does the 'switching' from one type of syllogism, during an experiment (for example from valid type of syllogism to invalid), corresponds to such a systematically increasing cognitive load ?;* and, if the answer is yes then which regions of the brain are activated and interacting due to this cognitive load escalation, the dynamic behavior of which can be detected by *measuring the power spectral density* of alpha, beta, theta etc. waves (a typical or main stream approach) or by *quantifying the complexity (for example via entropy, an innovative approach)* of the EEGs signals taken from brain regions, the main approach adopted in this work. Figure 1a below shows pictorially the link between Systems of thinking and Cognitive Control and Attention.

To the best of our knowledge, no works in EEG analysis exists that examine the *neural substrate* that sustain *deductive syllogism*. However, the *categorical syllogism* (the syllogism in which, in contrast to the *conditional or hypothetical syllogism),* the premises are categorical propositions or statements ('all men are mortal, Socrates is a man, Socrates is a mortal), a dual-path system was found in imaging studies (fMRI), consisting of a left frontal-temporal network, mainly in memory and language-dependent regions and a visuospatial network, located at right parietal regions (Goel et al., 2000).

In the case of checking for deductive validity of categorical syllogistic arguments**,** Osherson et al. (1998), have found a *right-hemispheric network*, also covering language-homologue regions (Parsons and Osherson, 2001), supported the above results in the case of *Conditional syllogism.* Activation of fronto-parietal-occipital network, for the solution of linear systems (problems consisting of three terms) have been shown in the work of Knauff et al. (2003).

A 'switching' from posterior brain regions to left prefrontal cortex, in the case of a *conditional* syllogism task, was observed as described in the paper of Houde et al. (2000). In a more recent work, in problems of relational spatial syllogisms**,** Knauff (2006), provide evidence for activation of bilateral prefrontal, occipital and parietal regions.

The above literature review, albeit limited, provide evidence about the connection between types of reasoning (syllogism) in general, with

Table 4. Linking Aristotle's type of syllogism and systems of thinking, for ASD, ADHD and control groups.

Model of Thinking	Aristotle's types of Syllogism	
	Valid	Invalid
Intuitive System I • Fast • Automatic • Effortless	ASD	CONTROL
Analytic System II • Slow • Contemplative • Effortful	ADHD CONTROL	ASD ADHD

Table 5. Channel index, name and location.

Channel index	Channel name	location
1	AF3	Anterio-frontal, left
2	F7	Frontal-temporal, left
3	F3	Frontal, left
4	FC5	Frontal-central, left
5	T7	Temporal, left
6	P7	Parietal, left
7	01	Occipital, left
8	02	Occipital, right
9	P8	Parietal, right
10	T8	Temporal, right
11	FC6	Frontal-central, right
12	F4	Frontal, right
13	F8	Frontal-temporal, right
14	AF4	Anterio-frontal, right

various brain regions and even more, about how this connection is associated, in consequence, with the cognitive processes and their EEG α, β, δ, θ, γ rhythms. However, it does not provide any information of how Systems I and II of modes of thinking are connected with all mentioned above 'typical' types of reasoning. If such a connection has not been provided by the existed literature, the connection with Aristotelian syllogisms is even more difficult to be found. Therefore, to the best of our knowledge, there is no work found to show the connection between Aristotelian types of reasoning (syllogism) with Systems I and II, and even more how this connection is associated, in consequence, with various crucial cognitive processes and the dynamics and complexities of their associated EEG signals. *The present work is an effort, as well as contribution, towards shedding light in the connection between Systems I &II of thinking, primary Cognitive Processes and Aristotelian type of Syllogism (reasoning), focusing in particular to the differences in the structure of this connection in subjects belonging to ASD and ADHD and control groups.*

1.6. Key-findings in this work

In Tables 3a and 3b and Table 4 we provide in advance some of the main findings of the paper, regarding the groups of Subjects that are engaged in Systems 1 and 2 of Thinking, based on Theta and Alpha amplitudes of their EEGs, for the valid type of syllogism, as well as the linking of Aristotle's type of syllogism with the systems of thinking, for ASD, ADHD and control groups. The entries in the table are extracted from tables 13 and 14 respectively (see discussion, section 4) (see Table 5).

From the above table, *the required combination of high Frontal θ and low parietal α is satisfied by ADHD and Control group so these two groups engage in System 2 of thinking, while ASD in System 1 of thinking, when the subjects face valid Aristotelian syllogisms.*

For the invalid type of syllogism, *ASD and ADHD groups are engaged in System 2 of thinking and Control in System 1 of thinking.*

Therefore, we observe from the two tables above that ADHD is engaged in Systems 2 of thinking for both valid and invalid while ASD and Control, in system 1 for valid and invalid respectively.

So, our results (Table 1) are in accordance with the results shown on the above table for cognitive control and attention. Furthermore, the intense activity in parietal channels shown in our results could be linked also to the release of working memory and recruitment of autonomic LTM. For example, ADHD and control groups exhibit high variability in parietal lobe so they can be linked with release of working memory and recruitment of autonomic LTM during the valid type of syllogism task.

The rest of this paper is structured as follows. In section 2 the materials and methods are described (participants, the EEG recording and the Aristotelian types of syllogism and the experiment procedure is explained). Also, in the same section, the analysis of signals and the MSE method is provided, as well as the results of power spectral analysis (on alpha, beta etc. waves), and finally the statistical measures for assessing the results. Detail results are given in section 3, followed by an extensive discussion (section 4) and a conclusion (section 5).

2. Materials and methods

2.1. The workflow

For the purpose and the objectives of the present work, there is an extensive spectrum of tools of analysis that could be used. Figure 1b presents an indicative workflow that one can follow to study how the dynamic behavior of recorded EEGs of subjects, in different groups (control and 'pathologic') is affected, when the subjects are 'tested' in valid and valid Aristotelian syllogisms. In this paper we follow the path from raw data analysis and preparation to the 'simultaneous' usage of two different approaches: the linear PSD analysis (allowed by the found stationarity in the data) and nonlinear MSE analysis, that provide an efficient and effective tool towards detecting the complexities and their changes, at various timescales that are 'linked' with the 'main-stream' power rhythms [alpha (α), beta (β), theta (θ), delta (δ)].

2.2. Participants

Eighty four (84) subjects (30 ASD and 30 ADHD patients, and 24 typical normal) were recruited in this study. The 84 subjects (58 males and 26 females) were split in three (3) groups: control (24), ASD (30) and ADHD (30).

The study was part of a larger research project on de novo diagnosed adults with ADHD and ASD (Pehlivanidis et al., 2020). The multi-disciplinary team that carries out all assessments consists of: psychiatrists who have extended experience in the diagnosis and treatment of Neurodevelopmental Disorders in adults and are trained in ADOS (Lord et al., 2012; Papanikolaou et al., 2009), ADI-R (Le Couteur et al., 2003; Papanikolaou et al., 2009) and DIVA (Kooij et al., 2019a; 2019b); and clinical psychologists. In order to be included in the study subjects had to be adults with normal intelligence and fluent phrase speech and to be assessed for the first time in their life for a possible ADHD and/or ASD diagnosis. Exclusion criteria were a previous ADHD and/or ASD diagnosis, the presence of acute psychopathology requiring urgent psychiatric treatment, current substance abuse disorder, IQ < 70 according to WAIS and a known genetic cause. Diagnosis regarding the presence of ADHD and/or ASD is given during a consensus meeting of the multidisciplinary team and is based on DSM-5 criteria while taking into consideration all available information.

Written consent was obtained from all participants and the study was approved by the Ethics Committee of the National and Kapodistrian University of Athens, Eginition Hospital (10549/17.10.2016).

2.3. EEG recordings

In this work, For EEG signals recording, the Emotive EPOC system was used, consisting of 14 channels (plus CMS/DRL references), following the 10–20 International system of locations. The channel names are AF3, AF4, F3, F4, F7, F8, FC5, FC6, P7, P8, T7, T8, O1 and O2. Sampling was sequential, with single ADC, and rate 128 Hz (2048 Hz interval). All electrodes were placed over subject scalp. Band pass was filtered between 0.05 Hz. Ground and reference electrode were placed on left and right ear lobes. The electrode impedance below 5KΩ was kept. The scalp locations for the Emotion EPOC system are shown in Figure 2.

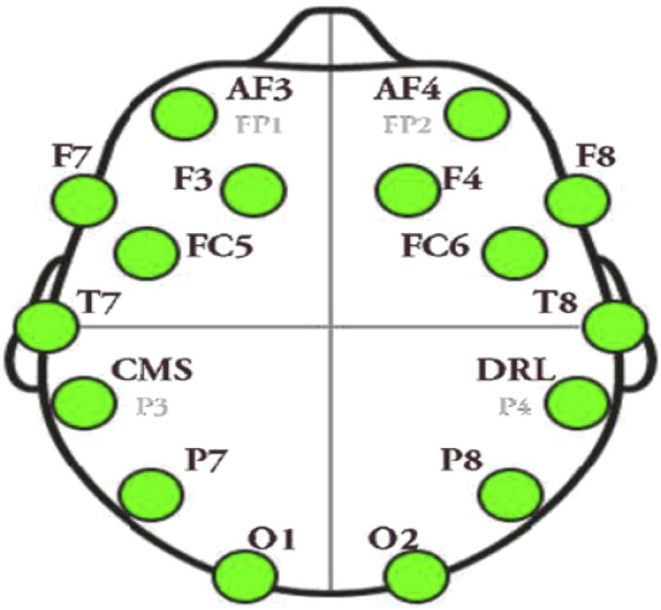

Figure 2. Scalp locations covered by Emotive EPOC.

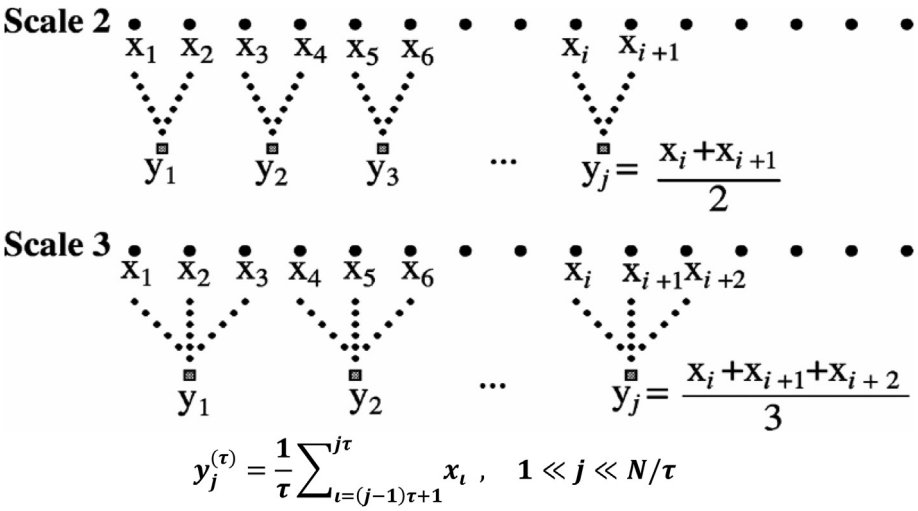

Figure 3. The procedure for coarse-graining (Adapted from Costa et al., 2005).

In the case of our study, the Emotiv EPOC device has provided several important benefits (i) compared with more expensive multichannel equipment: the setting up time of the Emotiv EPOC system is significantly shorter than that of an expensive EEG system, (ii) additionally, recent research assessing the reliability of the EMOTIV Epoc EEG device provides converging evidence indicating their capacity to measure consistently EEG signals (Debener et al., 2012; Papageorgiou et al., 2017).

After cap fitting, good conductivity was confirmed with Emotiv software through wet saline electrodes (Ramirez et al., 2015).

2.3.1. Tasks description. The Aristotle experiment

Using EEG signals, we aim to isolate the particular brain regions involved in Aristotelian types of reasoning or syllogisms, and to differentiate their engagement during the different types of syllogism: valid, invalid, illusions and paradox. In addition, we will try to answer how this differentiation is accounted for in the case of three groups of subjects (control, ASD and ADHD).

Care has been taken so the sentences or prepositions in the above types of syllogisms were presented visually (on a computer screen), in order to dissociate brain regions related to syllogism processing from those related to sensory process and low-level reasoning. This is a crucial stage in the process of analysis since the isolation of substrates associated with this high-level syllogisms (activating cross-modal cognitive systems), is not an easy task. In the experiment the syllogism statements consisted of **categorical type,** and the participants were informed to reach to a conclusion from the premises provided by the instructor, and indicate whether the given instruction was 'True' or 'False' (see below).

Aristotle's experiment is based on the study of reasoning or syllogism process, based on logical rules and concerns the way we reach to a conclusion (reasoning process) and on the way we take decisions. The process starts with assumptions (hypothesis) that ideally lead to a valid conclusion. The most known theoretical model ever proposed so far is that of dual process theory. Type or system I of the process is consider old, fast and automatic, while type or system II is a newer, slower and allows reasoning based on logical rules. In type I we are aware only for the result (conclusion), while in type II both, of the way and the result. Usually, there is an interaction between the two types. The question that arises often is whether personality or psychopathology enter the reasoning process. The Aristotle experiment is a process consisting of four stages. Valid, invalid syllogisms are used, together with paradoxes and illusions-visual paradoxes (not considered in this study).

The subjects under test seated in front of a computer screen. Instructions were presented to the subjects, before the start of the experiment, so they can be familiar with the process and its requirements. Then, syllogisms or arguments started to appear on the screen, in sets of 39, and every one of them was accompanied by a question 'right', or 'wrong'. The time duration of each slide is proportional to the number of letters in the syllogisms and then the slide disappears. Just immediately the next stimulus is appeared which may be a right answer or wrong. The subject is called to give answer. Answers to each one of the 'valid' syllogisms are considered right if subject considers them as right, while answers to 'invalid' syllogisms are considered right if subject considers them wrong. Each subject's answer is accompanied by his percent (%) of certainty given, which reflects his certainty in the answer he provides (100 % absolutely certain and 0 % not at all certain).

In parallel with the above process, a recording of the emotional condition of each of the tested subject is taking place (its intensity, control and mood of the emotions he feels). Also, EEG signals of alpha, beta, delta and theta frequency bands (and their sub-bands), are simultaneously recorded, using a wireless system of 14 electrodes (EMOTIV PRO, see above).

2.4. Sample entropy and Multiscale entropy (MSE)

Multiscale entropy (MSE) analysis is the procedure of calculating an entropy measure, as the $S_{E,}$ for each coarse-grained time series, plotted as

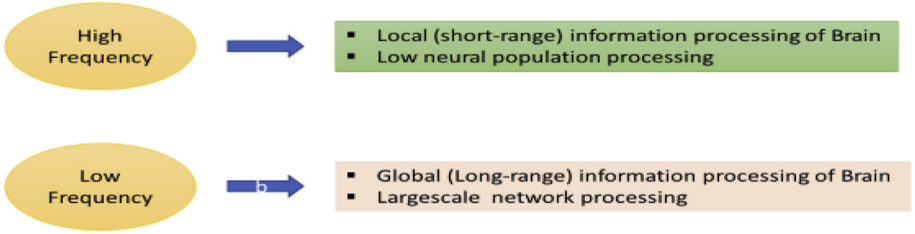

Figure 4. Linking high and low frequency in PS with local and global information processing in the brain.

Figure 5. Linking of fast and slow frequencies in PS with fine and Coarser time scales in MSE.

a function of the scale factor τ, as described below. For the computation of MSE, the original signal $\{X\} = \{x_1, x_2, .., x_i \ldots, x_N\}$ is transformed to a coarse-grained signal $\{y^{(\tau)}\}$, where τ is the scale factor (SF). The procedure for coarse-graining is shown in Figure 3, adopted from Costa et al. (2005). The original series is divided into non-overlapping windows of length τ, and the points inside the window are averaged, so a coarse-grained series is obtained as

$$y_j^{(\tau)} = \frac{1}{\tau} \sum_{i=(j-1)\tau+1}^{j\tau} x_i,\ 1 \le j \le \frac{N}{\tau} \tag{1}$$

S_E is computed for each time-series $y_j^{(\tau)}$.

Sample entropy is a modification of the approximate entropy, $A_E(m, r)$, and has the ant vantage of being less dependent on the time series length. S_E also shows relative consistency over a wider range of the parameters, *r, m* and *N,* define below. Let $\{X\} = \{x_1, x_2, x \ldots, x_i \ldots, x_N\}$ is a time series of length *N.* We form the *m-length* vectors: $U_m(i) = \{x_i, x_{i+1}, \ldots, x_{i+m-1}\},\ 1 \le i \le n - m + 1$. Now let n_i^m indicate the number of vectors that satisfy $d[u_m(i),\ u_m(j)] \le r$, where *d* is the Euclidean distance, so *r* is the tolerable distance between the vectors. The quantity

$$C_i^m(r) = \frac{n_i^m(r)}{N - m + 1} \tag{2}$$

represents the probability that any vector $u_m(j)$ is close to the vector $u_m(i)$. The quantity

$$C^m(r) = \frac{1}{N - m + 1} \sum_{i=1}^{N-m+1} C_i^m(r) \tag{3}$$

Is the average of the $C_i^m(r)$, the probability that any two vectors are *within r* of each other. Then, using the above, the K_2 entropy, the lower bound of the Kolmogorov-Sinai, KS, entropy (Grassberger and Procaccia, 1983) is written as

$$K_2 = \log_{N\to\infty} \log_{m\to\infty} \log_{r\to\infty} - \ln\left(C^{m+1}(r) - C^m(r)\right) \tag{4}$$

Following the same direction, Eckmann and Ruelle (1985) define the function $\Phi^m(r) = \frac{1}{N-m+1} \sum_{i=1}^{N-m+1} lnC_i^m(r)$, and $\Phi^{m+1}(r) - \Phi^m(r) \approx \sum_{i=1}^{N-m+1} \frac{lnC_i^m(r)}{C_i^{m+1}(r)}$, *the average of the natural logarithm of the conditional probability that sequences that are close to each other for m consecutive data points will shall be close to each other when one point is known.* Thus, Eckman and Ruelle suggested a new quantity for KS, as follows

$$H_{ER} = \log_{N\to\infty} \log_{m\to\infty} \log_{r\to\infty} \left(\Phi^m(r) - \Phi^{m+1}(r)\right)$$

The above formula, although useful in characterizing low dimensional chaotic systems, it does not have a practical application in experimental data, since for a noisy process (as the majority of real data), the above formula gives an infinity. For this reason, Pincus (1991) introduced the approximate entropy, $A_E(m,\ r) = \log_{N\to\infty}[\Phi^m(r) - \Phi^{m+1}(r)]$, and A_E is calculated by the ***regularity statistics*** $A_E(m, r, N) = (\Phi^m(r) - \Phi^{m+1}(r))$. This entropy measure applies to 'real life' time series and has been extensively used in physiology and medicine (Pincus, 2001).

Regular (e.g. periodic) time series have lower A_E values, while ***irregular, less predictable*** time series, have higher A_E values. Richman et al. (2004), defined Sample entropy as the parameter

$$S_E(m, r) = \log_{N\to\infty} - ln\frac{U^{m+1}(r)}{U^m(r)}$$

which is computed by the statistics

$$S_E(m, r, N) = -ln\frac{U^{m+1}(r)}{U^m(r)} \tag{5}$$

S_E is exactly equal to the negative natural logarithm of the conditional probability that sequences close to each other for *m* consecutive data points will remain also close to each other when one more point is added to each sequence. S_E and A_E both measure the ***degree of randomness*** (or inversely the ***degree of orderliness***) of a signal. S_E is computed for the coarse-grained time series $y_j^{(\tau)}$ in (1). The following term,

$$U^{m+1}(r) = \frac{\{number\ of\ pairs(i,j) with\ \left|y_k^{(\tau)m} - y_l^{(\tau)m}\right| < r,\ k \ne l\}}{\{number\ of\ all\ probable\ pairs\ in\ the\ coarse - grained\ time\ series\}} \tag{6}$$

therefore, symbolizes the distance between vectors $y_k^{(\tau)m}$, $y_l^{(\tau)m}$, formed from the coarse-grained time series, with scale factor τ, and having length m, and *r* the tolerable distance between the two groups.

The relative complexity of the normalized time series (same variance for scale factor $\tau = 1$), is detected by comparing the MSE curves (see Figures 9 and 10), based on the guidelines: a) if for the majority of the scales (in our study we used 20 scales) the entropy values are higher for one time series than for another, the former is said to be more complex than the latter b) a decrease of the entropy values, following a monotonic mode, indicates that the original time series ($\tau = 1$) contains information only in the smallest scale. Also, the value of the parameter *r* is a percentage of the standard deviation, SD, of the time series (we used $r = 0.15$ SD).

Based on previous studies S_E has a good statistical validity for parameters $m = [1,2]$ and $0.1 \le r \le 0.25$ (Richman et al., 2004). In the present study $m = 2$, $N = 60000$ samples and $\tau = 20$ scale factors, so $N/\tau = 3000$ samples, therefore, enough to obtain a reliable estimation of S_E (Richman et al., 2004).

2.5. Linking power spectral analysis (alpha, beta, theta, and delta rhythms) and MSE's parameters

According to Takahasi et al. (2016), differences in the MSE (or S_E) may be correlated with differences in the EEG power spectra. We investigated this possibility in our, clean from artifacts, data by computing the PSD (power spectral density), in the first 40 s (or 5120 samples for sampling rate $f_s = 128$ Hz), by using the EEGLAB (which is based on the *pwelch* function of MATLAB, ver. 2019b, and uses a Hamming window, 8 segments with 50% overlapping). *Five typical band frequencies were analyzed:* ***delta (1–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta 1*** *(13–19 Hz),* ***beta 2*** *(20–30) and* ***gamma*** *(30–60 Hz).*

The evolution of dynamics in EEGs, over different time scales, is a crucial information for understanding the overall functioning of the brain and requires the detection of temporal correlations on such scales. The detection however is not a trivial work. The structure of volatility or variability of EEGs at ***short time scales*** or equivalently ***higher frequencies*** are linked to ***local neural populations processing,*** while at ***longer time scales*** or ***lower frequencies*** are linked to ***large scale network processing*** (McIntosh et al., 2014). Figure 4 below shows schematically these two

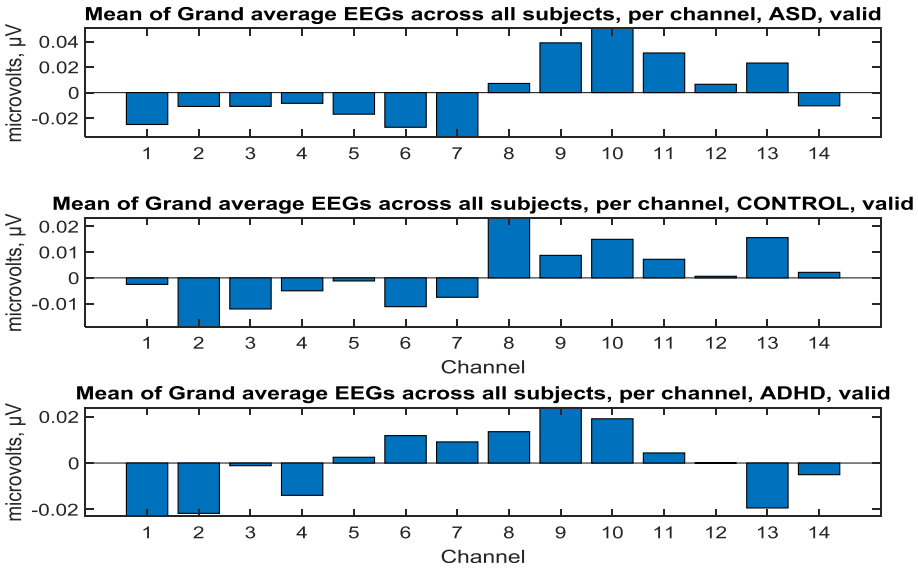

Figure 6. Mean of Grand average EEGs across all subjects for valid type, all groups.

important links, on which the present work will refer frequently, towards interpreting the results.

The nature of the linking of **frequency domain** (High/Low) with the **spatial scales** of neuronal processing is considered more neurophysical than neurophysiological: (Cognitive) **Communication** and **information** processing in brains involve different frequencies *across bands* and *spatial scales*, respectively (Postle B., 2015)**.** Cross-frequency coupling (CFC) methods quantify network information flow, in 'switching' from **synchronization** to **desynchronization** structures, and although they capture 'sufficiently' the complexity of communication, the linking of its long-range form with ***delta (δ), theta (θ) and alpha (α) rhythms*** (***lower frequencies***) and its **short-range** form with **higher frequencies** (***beta β and gamma γ***), can be realized on the basis of error propagation encountered in these two forms of communication. Faster oscillations are more detrimentally affected by variance in this error (Jirsa and Muller, 2013). In addition, when applying MSE as a measure to differentiate EEGs complexities among various groups of subjects (in our case control, ASD and ADHD), it is not clear how to interpret the MSE patterns or curves, beyond the naïve conclusion that some EEGs are more or less complex (entropic) than other. *More importantly, how the MSE curves are explicitly related to neurophysiological processes.*

Table 6. Correspondence between PS and MSE parameters (from scale factor to frequency content).

Scale factor, τ_{sf} In MSE	Frequency, Hz $f_{N,\tau_{sf}} = \frac{f_{s,1}}{2\tau_{sf}}$ $f_{s,1} = 128\ Hz$	Time Scale $\frac{\tau_{sf}}{f_{s,1}} x1000$ In ms (millisecond)	Log (Time Scale)	Signal Rhythms
1	64	7.81	2.05	gamma
2	32	15.6	2.75	
3	21.3	23.4	3.15	beta
4	16	31.25	3.44	alpha
5	12.8	39.06	3.66	
6	10.6	46.87	3.84	
7	9.14	54.68	4.00	
8	8	62.50	4.13	theta
9	7.11	70.31	4.25	
10	6.4	78.12	4.35	
11	5.8	85.93	4.45	
12	5.33	93.75	4.54	
13	4.92	101.56	4.62	
14	5.57	109.37	4.69	
15	4.26	117.8	4.76	
16	4	125	4.82	
17	3.76	132.8	4.88	delta
18	3.55	140.6	4.94	
19	3.36	148.4	5.00	
20	3.2	156.2	5.05	

Local and **Global** brain networks are very important concepts in understanding the functioning of the brain, so it is very useful to shed light on the linking between MSE's time scales and frequency contents of the EEGs. A literature review we made (Bruce et al., 2009), 'revealed' a correlation between single-scale (no coarse grained) entropy and power spectrum. More specifically, it was found a ***negative correlation (Corr) with delta power and positive with beta power:***

$$\text{Corr(entropy, } \delta) < 0 \text{ and Corr(entropy,}\beta) > 0 \tag{7}$$

With both correlations attributed mainly on changes is power spectrum rather than in regularity. This finding revealed the weakness of using single scale (Sample) entropy methods. In later studies, however, MSE was used to relate entropy timescales to frequency content of the underlying signal dynamics (McIntosh et al., 2014), as shown in the Figure 5 below (see Figure 6).

In the present work, we have applied the guidelines given in the paper by Courtiol et al. (2016), in order to attain a strong and consistent relation between MSE's and Power Spectrum's parameters, making the interpretation of the results of the two ***complementary*** approaches more easy to understand. For this purpose we provide Table 6 that shows the aforementioned linking of parameters.

In the table above, f_s is the sampling frequency and is related to the highest frequency in the data via the Nyquist-Shannon's sampling theorem as $f_N = \frac{f_s}{2}$. The link between frequency and scale factor τ_{sF} is given by

$$f_{N,\tau_{sF}} = \frac{f_{s,1}}{2x\tau_{sF}} \tag{8}$$

where $f_{s,1}$ corresponds to the sampling frequency at time scale 1 (the one of the original signal), so in our case $f_{s,1} = 128$ Hz. The above link becomes clearer by converting the MSE's parameter ***scale factor*** to ***time scale***, τ as

Table 7a. Descriptive statistics of Grand average EEG values for valid syllogism, ASD group.

	Summary Statistics of Grand average EEG values (across all subjects per channel) Valid ASD (ARISTOTLEs experiment)													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
	AF3	F7	F3	FC5	T7	P7	1	2	P8	T8	FC6	F4	F8	AF4
Mean	0.089641511	0.015187433	-0.059346519	0.060755502	0.039833553	0.010227663	-0.0030163529	0.14914748	0.2910209	-0.01862349	-0.05348425	-0.12772317	-0.41123942	-0.1484074
St.dev	129.99092	63.073463	172.72287	91.861076	106.78784	92.234344	122.06396	133.46136	169.01474	112.62379	118.38284	102.02227	191.14389	108.55531
skewness	0.035868894	-0.012462092	0.080187865	0.022107847	-0.043885704	-0.00853455581	0.0367455511	-0.029339498	-0.021495968	-0.00733534674	0.057055853	0.048469231	-0.0024089515	0.056308951
kurtosis	8.7665367	8.0388765	8.0039215	8.0340652	5.6680207	6.8667493	8.7865543	4.910893	8.3625879	6.7510624	8.0389271	6.0464764	7.1712489	6.2936935
ADF h	1	1	1	1	1	1	1	1	1	1	1	1	1	1
ADF pvalue	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001
ADF Statistic	-183.13119537708 1	-182.42174268294	-184.25319786671 5	-183.74696390261 4	-181.97151880074	-181.50996435375 7	-181.54739424910 6	-182.59001802024 5	-181.89579274678 4	-181.77101331348 5	-183.57114507250 1	-182.32726006061	-182.46276972422 2	-182.33380006318 7

$$\tau = \frac{\tau_{sF}}{f_{s,1}} \times 1000 \tag{9}$$

where the factor 1000 is for expressing all parameters in milliseconds (ms). In order to capture all possible changes in the distribution of both structure and amplitude of EEG signals, the figures in the results of the analysis are presented in log-log graphs. The difference in the MSE patterns between the groups of subjects becomes clearer compared with those when using linear x and y scales in 2-d graphs.

MSE is an extension of S_E *(SampEn)* (Richman et al., 2004), using temporal coarse -graining procedure (Costa et al., 2002, 2005bib_Costa_et_al_2002bib_Costa_et_al_2005). SampEn can capture effectively the structure of variability (or volatility) of a signal and has been applied on biological systems. It can distinguish regular (predictable-less complex) signals from irregular (less predictable, more complex). MSE evaluates SampEn on multiple time scales, via the temporal coarse-graining procedure, which is equivalent to filtering the original signal with a moving average filter (by down-sampling it successively) in the time-domain, and equivalent to a low-pass filter in the frequency domain (Govindan et al., 2007). Because coarse-graining alters the frequency components of a signal, the clarification for the link between MSE and PS parameters given in Table 6 above is very informative and extremely useful.

2.6. Statistical evaluation

Average values are expressed as mean and SD. Significant differences in behavioral as well as computational parameters (i.e extracted features after applying measures -in our case MSE-on the cleaned data), between the control, ASD and ADHD groups, are determined either by Independent sample t-test, in case the extracted features signal are normally distributed (checked by Q-Q plots and other statics), or the non -parametric Mann-Whitney U test, in case they are not normally distributed. A **6-way ANOVA** was applied with within and between subjects' factors, as described in section 3.2, where the independent Variable in ANOVA is the MSE (extracted feature). For all above statistical tests, the SPSS, version 20.0 for the Windows is used. A p-value$<$0.05 is considered statistically significant.

For the MSE values, the distribution normality was tested via Kolmogorov-Smirnov test, and via optical control of the Skewness and Kurtosis values, for each channel (1–14) and each group (ASD, ADHD, and control).

For all analysis, as ANOVA method requires, the Greenhouse-Geisser adjustment was applied to the degrees of freedom, and the Bonferroni correction was applied for all post hoc tests (Catarino et al., 2011).

We have also performed Independent t-test, Paired Sample test and a Kruskal-Wallis, one-way between subjects, as described in section 3.2.

3. Results

3.1. Some useful notes on multiscale entropy (MSE)

- For simulated *white and 1/f noises* (see section 3.5), we observe that both the mean value of sample entropy SE and standard deviation (st.dev) ***increase*** as the length of the time series ***decrease*.**
- The required minimum number of data depends on the level of accepted uncertainty. In the present work we use 40.000 data points, so the shortest coarse-grained time series, for 20 scale factors, is 40000/20 = 2000 samples (or 15.6 s, with a sampling rate of 128 Hz)
- ***Stationarity*** is also a crucial consideration when applying MSE. In calculating S_E, a parameter must be fixed depending on the st.dev of the signal. The value of SE is greatly affected by non-stationarities due to the presence of outliers, artifacts. The structure of the signal is not modified when removing local artifacts and a small percentage of outliers (<2%), but is significantly affected over multiple time scales when removing trends. In our case, an outlier is a data point larger than the mean +/- 3.5 x (standard deviation) of the time series.

Table 7b. Descriptive statistics of Grand average EEG values for invalid syllogism, ASD group.

Summary Statistics of Grand average EEG values (across all subjects per channel) invalid ASD (ARISTOTLEs experiment)

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
	AF3	F7	F3	FC5	T7	P7	1	2	P8	T8	FC6	F4	F8	AF4
Mean	0.18682604	-0.015813814	-0.53897434	-0.17039201	0.14008445	-0.099464081	-0.28451654	0.25416258	-0.035371911	0.025764728	0.14175573	0.039659519	0.028510032	0.074675709
St.dev	131.77226	59.953854	160.14217	84.083015	96.343819	86.497856	118.35937	116.95061	154.98761	101.4311	105.73117	92.322853	181.07526	98.982399
skewness	-0.051170535	0.001715614 2	-0.00550729 42	0.0038642667	0.0220776342	0.0097626317	0.060023934	0.0015328329	0.043709766	-0.01902844	0.051398199	0.0060317395	0.040607121	-0.0039996309
kurtosis	11.330706	9.5341167	9.557497	9.4103308	6.910634	8.5714483	11.664709	6.0850892	11.051902	8.0081329	9.7260208	7.31599	7.8763881	7.3848586
ADF h	1	1	1	1	1	1	1	1	1	1	1	1	1	1
ADF pvalue	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001	0.001
ADF Statistic	-176.46449225 3586	-175.94760495 1575	-181.33490986 1759	-180.81334470 7042	-180.76291704 8159	-178.26557702 8434	-176.28463847 3261	-181.72552294 5624	-180.66272156 3697	-179.48023955 8871	-178.99404170 1536	-179.42220006 8588	-178.84540256 2859	-180.17726173 4619

- When a biological system is forced to work under *stressful conditions*, even in the typical (control) systems, *the dynamic of the system is constrained or limited, to evolve within a subset of the state space.* Therefore, it is anticipated that under a variety of stressed conditions (in our case under the cognitive loads included to control subjects due to their exposure to -testing in- Aristotelian syllogism information processing), ***control systems will generate less complex outputs than under being tested in more usual reasoning loads*** (Costa et al., 2003).
- *The reduced MSE exhibited by subjects of control group, over a number of timescales, compared to the larger-complexities of ADHD and ASD subjects, may be explained by the above argument. During the Aristotle's Syllogisms, the dynamic evolution of neural waves 'emitted' by ADHD and ASD subjects continues to travel all over the high-dimensional state space of the brain regions that have been activated by these types of reasoning, so the trajectories of the underlying dynamic system visit the whole space, not just a small region as it is in the case of a healthy system.*

3.1.1. MSE, nonlinear dynamics and 'pathology-related' differences in EEG signals

One of the main advantages of using MSE is its strong capacity in differentiating time series of weak nonlinearity from those with strong nonlinearity, as well as from stochastic ones. In, fact MSE is much more sensitive to the interactions of stochastic dynamical components with linear autocorrelations than the power spectrum. Therefore, in case of existence of such components in EEGs, MSE patterns (curves) are expected to reflect such dynamical interactions and to differentiate them in different groups of subjects (in our work control, ASD and ADHD). If the nonlinearity content in EEGs is weak, then its influence in differentiating EEGs from different groups, as it is measured by MSE, is also weak.

3.2. Descriptive statistics, stationarity and normality tests

In the Tables 7a and 7ba and b below we provide summary statistics information for the Grand averages EEG values, for the ASD group and in supplementary materials for the rest groups of subjects and channels. EEG are biomedical data that are regarded as the stochastic phenomena of biological systems. It is necessary, the statistical properties of such time series to be often examined because most of the statistical analysis processed in the frequency and the time domain is based on the assumption that the time series is weakly stationary and normally distributed. Therefore, it is necessary to know whether the EEGs analyzed in this study satisfy the conditions of weak stationarity and normality, since the methods used here, in a *complementary* mode, are both linear (Power spectral analysis, an FFT approach that assumes both stationary and normally distributed data) and nonlinear (MSE).

Actually, EEG signals are '3N' – Nonstationary, Nonlinear, and Noisy, since brain activity is essentially nonstationary, not because of casual external influences of the stimuli on the brain mechanisms, but due to the fact that of switching of the inherent metastable states of neural assemblies during brain functioning. Therefore, to confirm that EEGs of grand averages are indeed stationary, we also conducted the *ADF (Dickey-Fuller test for a unit root in a univariate time series) stationarity tests* (Kwiatkowski et al., 1992), the results of which are shown on the Table 7a and b (and in tables of Appendix A). In the tables the logical variable h = TRUE indicates rejection of the unit-root null hypothesis in favor of the alternative model (no unit root present, so the time series is stationary). Therefore, all signals found to be stationary, rendering possible the power spectral analysis (based on FFT, a linear method which assumes stationarity). For the normality testing of our data, we performed q-q plots (plot of quantiles of each EEG vs. the quintiles of normal distribution). All signals found to be linearly distributed, thanks again to the averaging of EEG amplitudes (across all subjects in a group, per each channel separately). Figure 7 presents the results of the tests, for an indicative channel, AF3, for valid and invalid types of syllogism, for all groups of subjects. The Kolmogorov-Smyrnov normality test (K–S test)

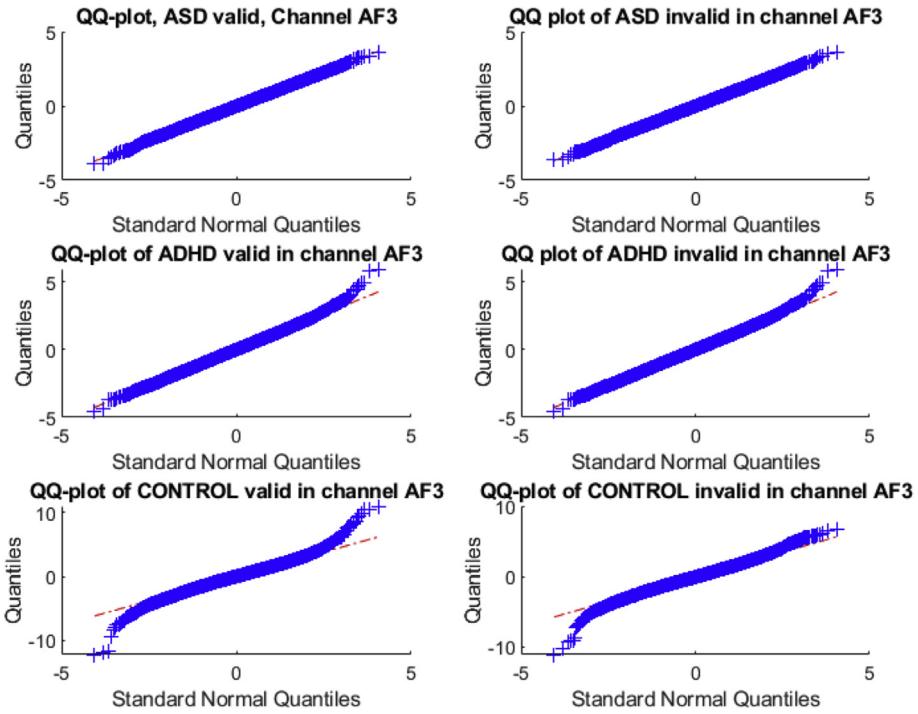

Figure 7. Q-Q plot of Grand average EEGs, for valid & invalid types, for each group, at channel AF3, Vs. normal distribution. The linearity of the points suggests that the data are normally distributed.

was also applied, the results of which (not shown here) confirm as well, the normal distribution of EEGs analyzed.

3.3. Analysis of the kurtosis of EEGs amplitudes

The calculations show that the kurtosis of the ADHD and ASD patients' EEG are positive and much higher than that of the controls. Kurtosis is a statistical quantity which measures the complexity of an EEG data set, and indicates how intensely the tails of a distribution compared to tails of normal distribution (it determines if the EEG signal has a peak or rather flat at the mean point of the signal (Brijil C., et al., 2010). Higher values of kurtosis indicate that the signal has a sharp peak at the mean point of an EEG signal data set and low values of kurtosis indicate that the signal has a flat nature at the mean point of the signal. The kurtosis for a signal $x(t)$ is given by (Brijil C., et al., 2010),

$$k = \frac{1}{N}\sum_{t=1}^{N}\left(\frac{x(t)-\mu}{\sigma}\right)^4 \tag{10}$$

where σ is the standard deviation and μ is the mean of the signal. The kurtosis for EEG signals from ADHD, ASD subjects and control subjects, for valid and invalid syllogisms, are calculated. The values for the ADHD, ASD subjects and control subjects are compared and shown in the Figures S2 and S3, in Supplementary material. From the figures, it is observed that the kurtosis for ASD for both valid and invalid syllogisms remains small and constant for all channels. For the valid type, the kurtosis of the control subjects is large at channels 3(F3) and 10 (T8) and very large at channel 4 (FC5), while for ADHD the kurtosis is extremely high at channel 12 (F4) and large at channel 6(P7). For the invalid type, the variability (kurtosis) of Control subjects is high at channel 4(FC5), while for ADHD is extremely high at channel 12(F4) and high at channel 6(P7).

In summary, *control and ADHD subjects have similar kurtosis at frontal right (extremely high variability in EEGs),* and at *parietal left (high variability)*. So *there is a significant difference between the variability of ASD subjects for both types of syllogisms (small and constant at all channels) and the variability of ADHD and Control subjects (which is extremely high at right frontal and high at left parietal).*

3.4. Behavioral results. Comparing MSE values across groups and brain regions and syllogism types. An ANOVA and related tests

We used first an Independent t-test to compare the 'performance' in MSE of the participant in the ASD, ADHD and control groups, since the MSE scores were obtained using an independent groups design. It was hypothesized that the complexity, i.e. the average MSE values recorded in frontal, parietal, temporal and occipital regions of the brain, for both types of valid and invalid syllogisms, are significantly different between participants of the above (independent) groups.

In the test regarding *ASD and ADHD*, the Levene's test for equality of variances showed a value $p > 0.05$, indicating a quality of variances in all combinations of syllogism and brain regions (e.g. valid-frontal, valid-parietal, invalid-temporal etc.), except in the valid-occipital combination ($p > 0.05$), where the variances were found different. So, we conclude that the mean difference of MSE in all, but one, combinations of syllogism and brain regions is significant, specifically we report the following results: significant in valid-frontal ($t = -2.432$, $df = 58$, $p = 0.018$), valid-parietal ($t = -3.025$, $df = 58$, $p = 0.004$), valid-temporal ($t = -2.841$, $df = 58$, $p = 0.006$), valid-occipital ($t = -4.966$, $df = 58$, $p = 0.000$) and invalid-occipital ($t = -4.079$,$df = 58$,$p = 0.000$), while not significant the combinations invalid-frontal ($t = -1.268$, $df = 58$, $p = 0.210$), invalid-parietal ($t = -1.965$, $df = 58$, $p = 0.054$), and invalid-temporal ($t = -1.444$, $df = 58$,$p = 0.154$).

In a similar test for difference in mean values of MSE between *ADHD and control* participants, the equal variances assumption is not valid ($p < 0.05$ in Levene's Test), and the difference was found to be significant for the combinations valid-frontal ($t = 1.692$, $df = 49$, $p = 0.097$), valid-parietal ($t = 2.156$, $df = 49$, $p = 0.036$), valid-occipital ($t = 2.572$, df

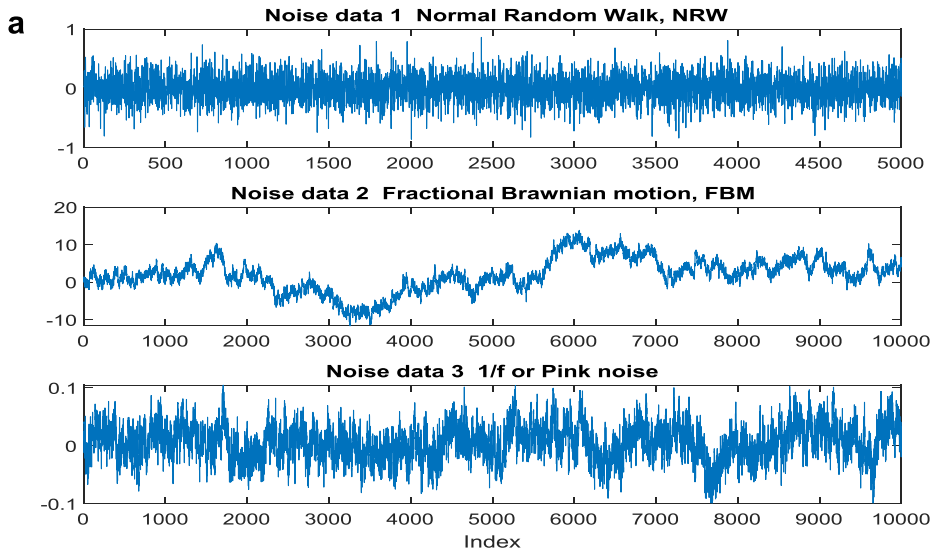

Figure 8a. Time series of normal random walk (white noise), fractional Brownian motion (FBM) and 1/f or Pink noise.

= 49, p = 0.013) and invalid-occipital (t = 2.498, df = 49,p = 0.016), while not-significant were found the combinations valid-temporal (t = 1.810, df = 49, p = 0.076 (.0.05)), invalid-frontal (t = 0.683, df = 49, p = 0.498), invalid-parietal (t = 1.227,df = 49, p = 0.229) and finally invalid-temporal (t = 0.835, df = 49, p = 0.407).

The same test for the difference in mean MSE values for *ASD and Control* subjects, we report the results as follows: the assumption of equal variances is satisfied in all syllogism-brain region combinations (Levene's $p > 0.05$), and not significant differences were found, in all combinations considered. *Therefore, there is no difference between the complexity values recorded in all brain regions and syllogism types considered in this study, between ASD and Normal subjects, by using an independent samples test.*

Then, we also tested for differences in mean MSE scores, as above, by using Paired Sample Test.

The difference in mean score measured in frontal and parietal brain regions, for the valid type of syllogism (i.e. valid-frontal and valid-parietal combinations), was found *significant* (t = 5.10, df = 80, p = 0.00025, one-tailed). The difference for the valid-temporal and valid-occipital combinations were found *not significant.* Similar results were found for the invalid-type of syllogism and brain regions (significant differences in invalid-frontal and invalid-parietal, and not significant in invalid-temporal and invalid-occipital). We also computed the correlation between the MSE scores in the frontal and parietal regions, for all groups considered (ASD, ADHD and control), for both valid and invalid syllogism, and we found it to be significant (0.926, $p < 0.00025$ and 0.914, $p < 0.00025$, respectively). We also compare the difference in the mean MSE values, for all brain regions considered, for both valid and invalid types, considering the whole data set (all groups included), by

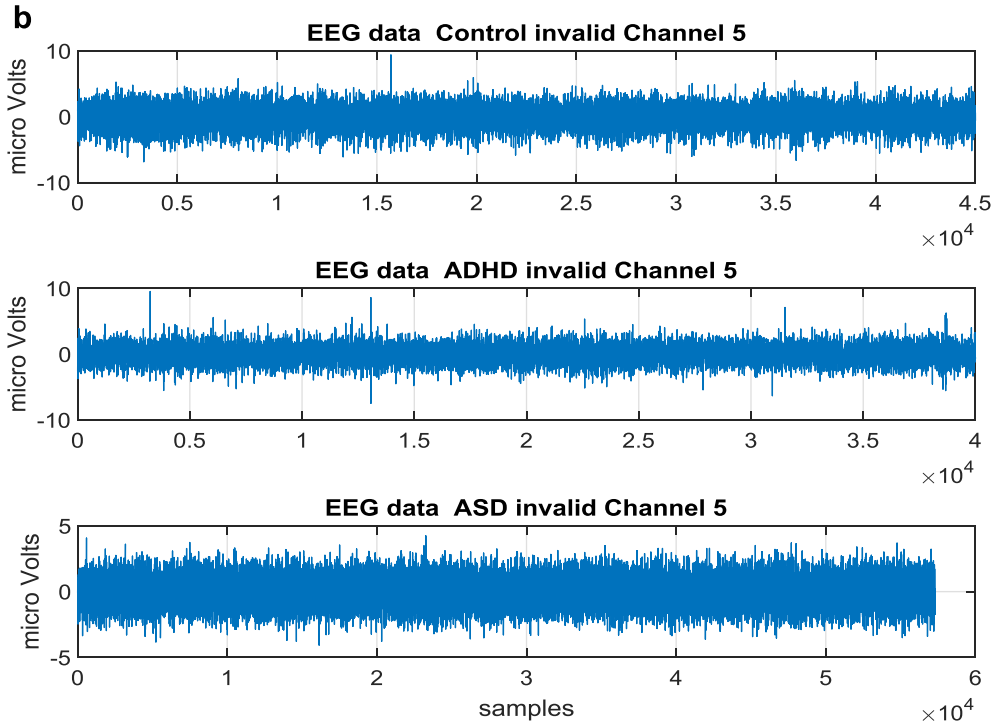

Figure 8b. Grand average EEGs (of each second in each condition-type of syllogism, across all subjects), at channel 5 (T5) taken from ADHD, ASD and control groups, for the invalid type of syllogism in 'Aristotle's' experiment.

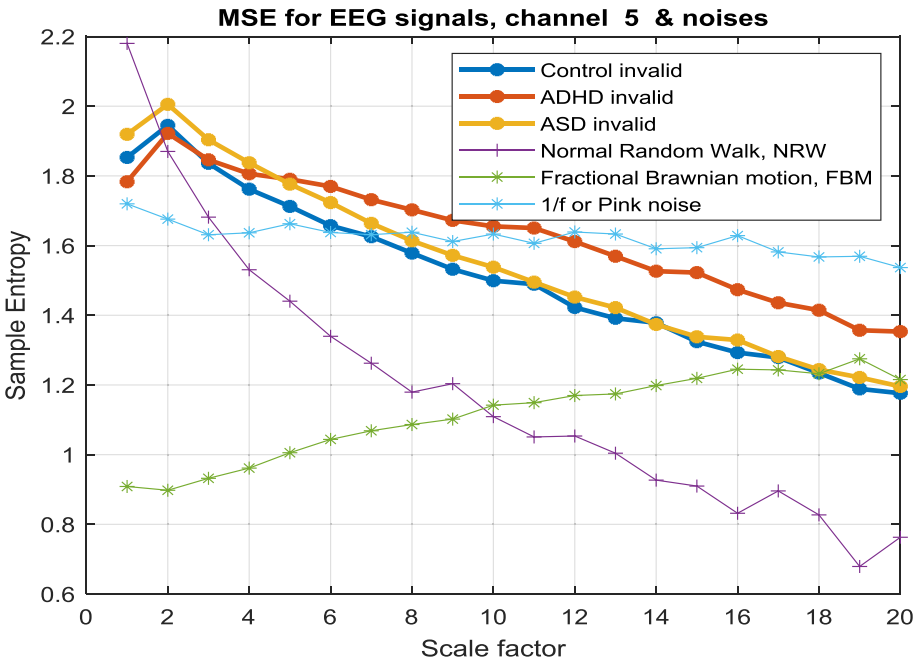

Figure 9. Comparison of MSE curves (linear x y scales) for the three stochastic noises and three EEGs at channel 5 (indicative), for the invalid type of syllogism, and for the three groups of subjects (control, ASD and ADHD).

using paired t-test. All combinations were found to be not significant, however their (linear) correlations were found significant indicating that in the same brain regions, the scores of MSE values of the subjects, tasted on both valid and invalid syllogisms, on the average 'go the same way or change in the same direction'.

We have also tested for differences in the *distribution of percentage values of certainty* of the participants that they have reported in answering which of the thirty nine (39) questions presented to them (see section 2.3.1), are valid or invalid, using the *Kruskal-Wallis, One-Way between subjects test*. According to the test, the distribution is similar across all categories (ASD, ADHD and control).

Finally, we perform a 6-way ANOVA or 8X3X2X2X2X2 (syllogism-BrainRegion*group*smoking*handedness*health issues*health treatment), where group, smoking, handedness, health issues and health treatment are **between subjects** factors and syllogism-BrainRegion is a within subjects factor. Since the Mauchly's test of sphericity was found significant ($W = 0.007$, $p = 0.000$), we adopted the multivariate approach and the statistics are as follows:

- The *main effect* of syllogism_BrainRegions is significant by Pillai's Trace ($F_{(7,48)} = 5.184$, $p = 0.000$)
- The *syllogism_BrainRegions by group (interaction)* is significant by Pillai's Trace ($F_{(14,98)} = 1.942$, $p = 0.034$)
- The *syllogism_BrainRegions by group (interaction) by health_treatment* is significant by Pillai's Trace ($F_{(7,48)} = 1.955$, $p = 0.008$)
- All other interactions were found to be not significant.

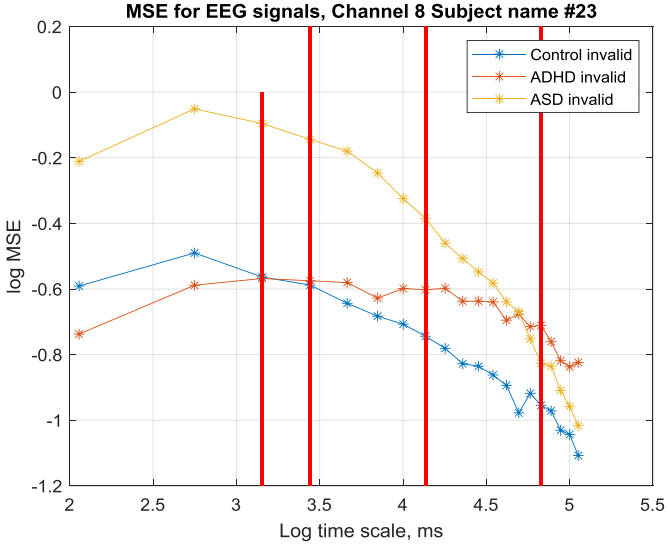

Figure 10. MSE curves vs. time scales, in log-log scales, for subject #23 and channel 8, for **invalid** type of syllogism. The red line set the borders of frequency bands (gamma, beta, alpha, theta and delta).

3.5. MSE analysis

In this section we present the results from applying MSE and Power Spectrum analysis on our data, emphasizing once more that the two approaches work complementarily. Since the number of results, expressed mainly as Figures, is large (30 data sets corresponding to 30 subjects for each ASD and ADHD groups, and 24 for control group, for two types of syllogism, valid and invalid i.e. $84 \times 2 = 164$ figures), we present only some indicative but representative results. Specifically, for a single subject namely #23 in all groups (selected arbitrarily), at channel 8 (02 occipital location of electrodes) we provide all the results for valid and invalid types of syllogism. This gives the opportunity to see the EEG's MSE patterns or curves for each time scale, as well as the EEG's power spectra at channel 8, for each type of syllogism, an information that is both representative and informative of the rest of all other results. Then, the MSE curves and PS for some selected Grand EEGs (across all subjects, for each group at each channel separately) are presented (the majority of the results is in the supplementary material, figures S4–S10**).** In order to facilitate both presentation and interpretation of the results, we provide summary Figures 19 and 20 in section 4, that incorporates, we believe, all essential information extracted by the analysis.

Before proceeding to the presentation of MSE for the recorded EEGs, it is very insightful to compare MSE curves computed on simulated data of three types of noise with those computed on the EEGs, following the work of Courtiol J. et al. (2016). The need for such a comparison will be obvious as we proceed further into our analysis. Figure 8a shows the time

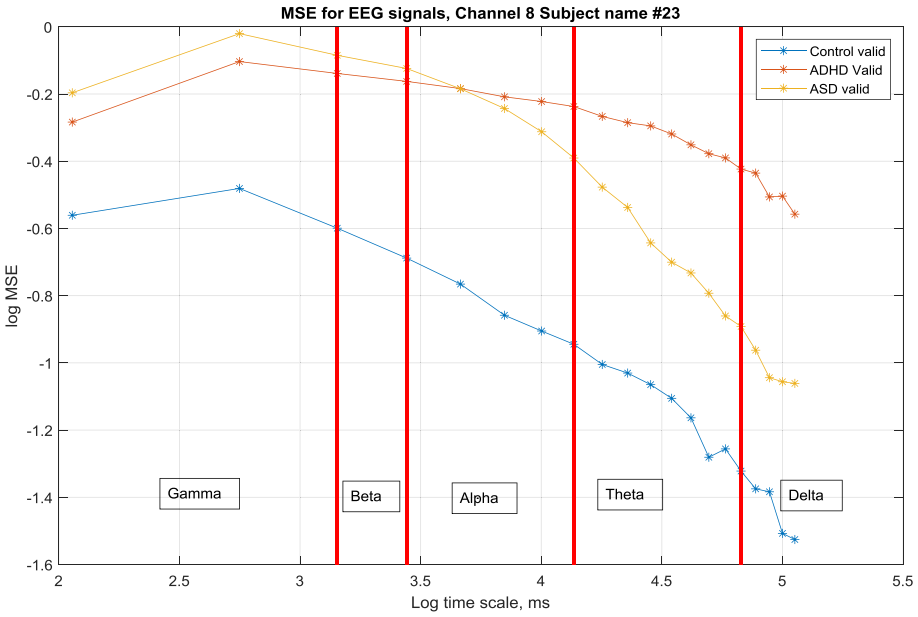

Figure 11. MSE curves vs. time scales, in log-log scales, for subject #23 and channel 8, for **valid** type of syllogism. The red line set the borders of frequency bands (gamma, beta, alpha, theta and delta).

series of *normal random walk (white noise), fractional Brownian noise* and *1/f (where f frequency)*, and Figure 8b shows the time series of grand average EEGs, at channel T5, for all groups for invalid syllogism. The behavior of MSE varies significantly for these noises, and as we observe in Figure 9, MSE of white noise 'resembles' that one estimated on the EEGs of our study. The figure shows that uncorrelated random signals (white noise) are less complex than the correlated random signals (colored noise). For scale one (corresponding to the original signal, before coarse-graining), a higher value of entropy is assigned to normal walk (white noise) time series, in comparison with 1/f time series. The coarse-grained 1/f time series however remains almost constant for all scales, while the coarse-grained white noise decreases monotonically and for scales >4 becomes smaller than the corresponding values for 1/f noise. Fractional Brownian noise on the other hand, increases with scale. Coarse-grained signals are progressively 'smoothed out' and the standard deviation decreases monotonically with the scale factor, reflecting the fact that such signals have information only on the shortest scales.

As we see in Figure 9, the three EEGs in our study (for the invalid type of syllogism, and for the three groups of subjects, at channel 5) 'generate' MSE curves like the ones extracted from white noise, i.e. after the 2nd scale they decrease monotonically (become less complex, they lose the information content). Most importantly, up to scale 5 the complexity of EEG from ASD subjects is larger than both ADHD and control ones, while after scale 5 the complexity of ADHD is higher. *This indicates the advantage of using multiscale entropy measure instead of using the 'typical', simple, one-scale entropy that does not take into account that the* dynamics of EEGs are dependent also on the time-scale (new mechanisms activated at different timescales come on the 'scene' and affect the dynamics of EEG).

In the next Figure 10 the MSE curves vs. time scales, in log-log scales, for subject 23 and channel 8, for all groups and valid and invalid types of syllogisms, are shown. As it is shown in the figure, in *gamma* frequency region (gamma rhythm), group ASD present the highest value in MSE for invalid type as also in the case of valid type (see next Figure 11), in which MSE of ADHD is larger than that of ASD. In *beta* region, MSE values for

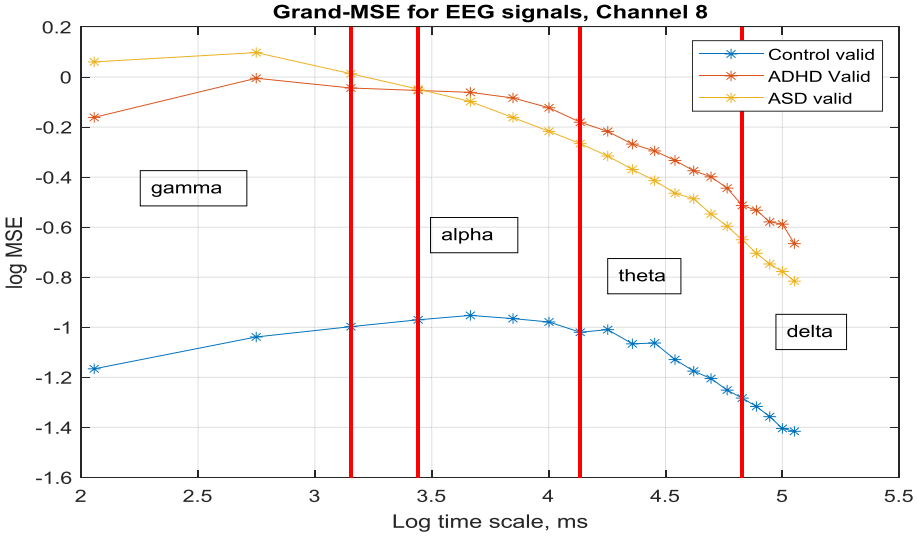

Figure 12. MSE curves vs. time scales, in log-log scales, for Grand-average of EEGs at channel 8, for **valid** type of syllogism. The red line set the borders of frequency bands (gamma, beta, alpha, theta and delta).

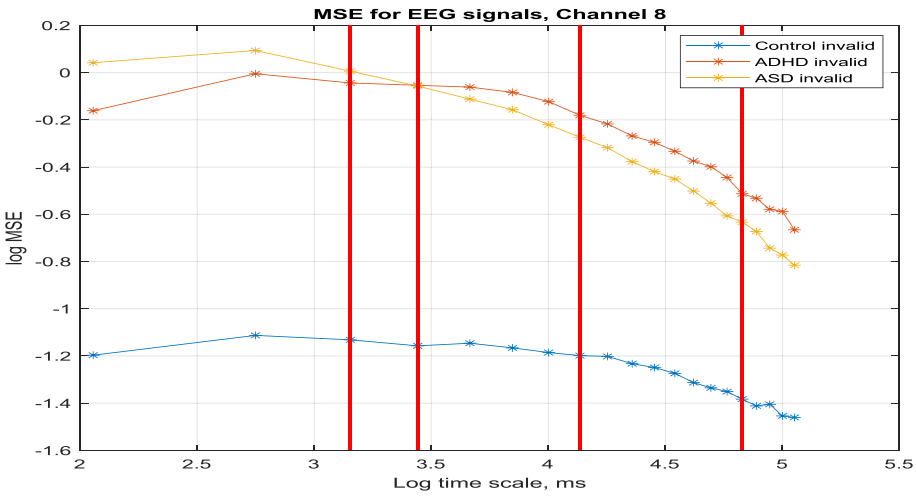

Figure 13. MSE curves vs. time scales, in log-log scales, for Grand-average of EEGs at channel 8, for invalid type of syllogism. The red line set the borders of frequency bands (gamma, beta, alpha, theta and delta).

valid and invalid are different. In this figure both control and ADHD EEGs have identical MSE values, while in the valid type MSE od ASD is larger than that od ADHD which is larger that of control. In the most interesting region of frequencies *alpha (α),* MSE of ASD is larger than both the ADH and control, while the MSE curve of the ADHD group becomes faster larger (almost just before the onset of the alpha region but also within beta region) than that of the control group, in comparison with the behavior in the valid type (see Figure 11). Therefore, this may be an indication that mechanisms that are activated mainly by frequencies stimulating factors 'working' within region alpha and secondarily less within region beta, in the case of the invalid type of syllogism the mechanism start operating earlier and affect the dynamics of EEG

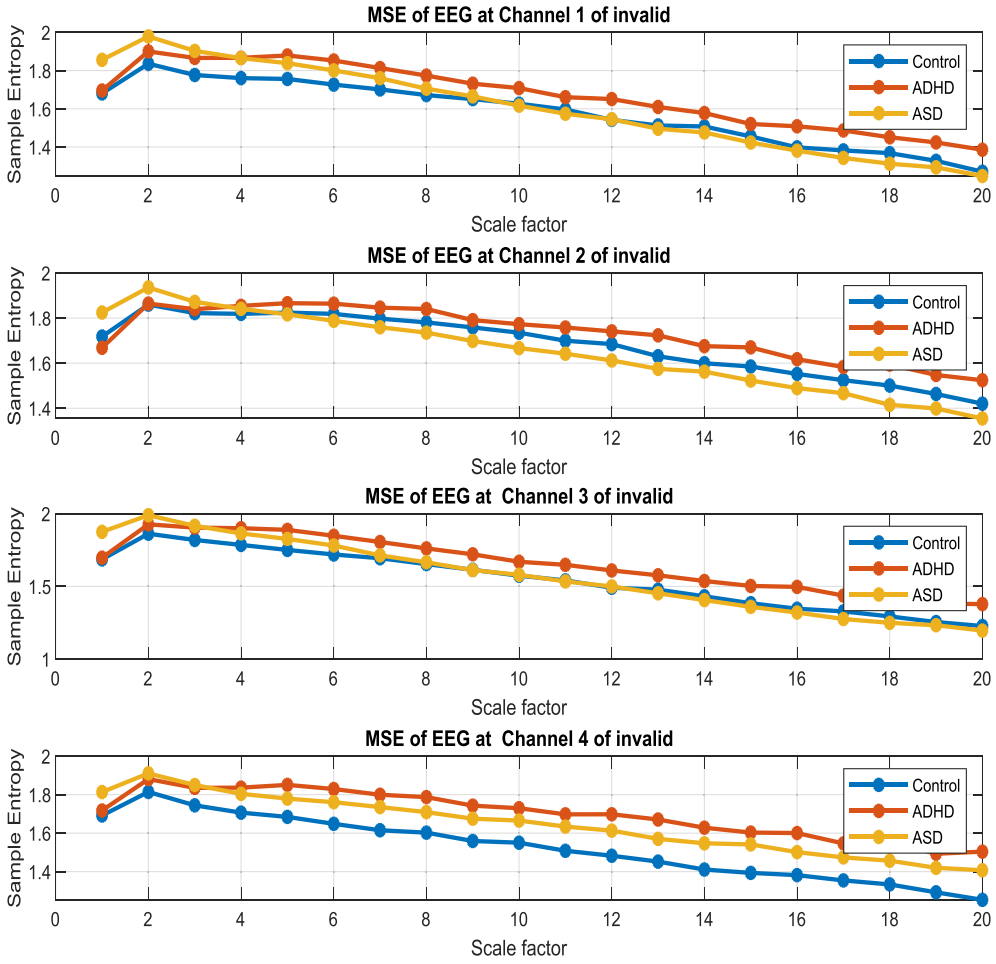

Figure 14. *MSE vs scale factor (linear scale)* at channels 1 to 4, computed on EEG grand averages (across all subjects of each group, for each separate channel), for the invalid type of syllogism.

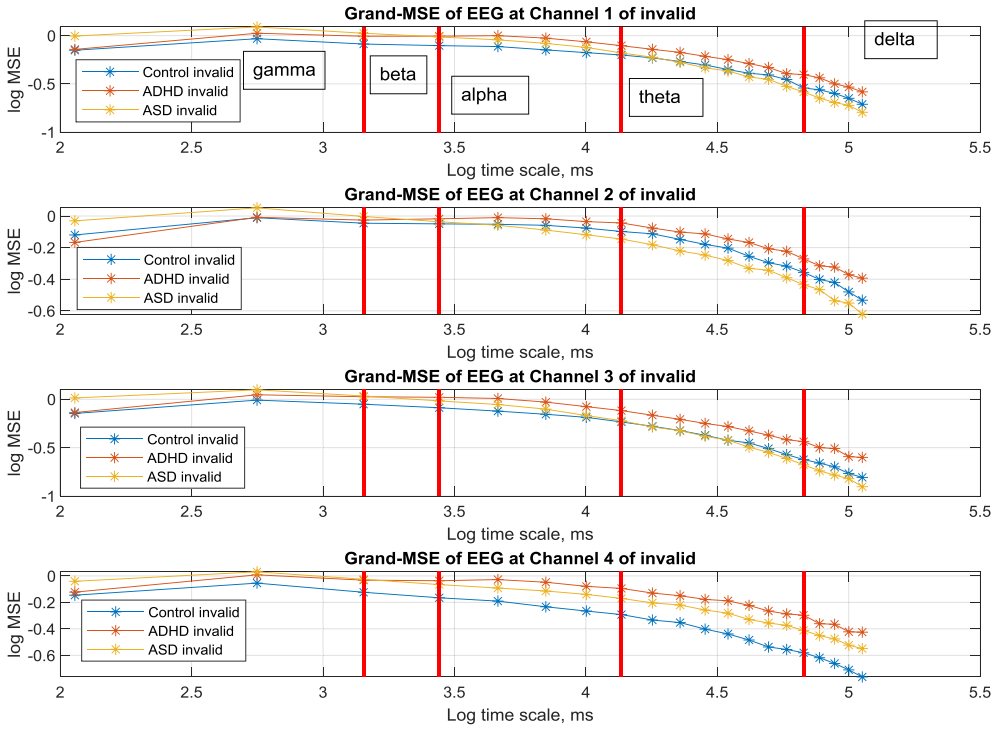

Figure 15. log MSE vs. log time scales at channels 1 to 4, computed on EEG grand averages (across all subjects of each group, for each separate channel), for the invalid type of syllogism. The red line set the borders of frequency bands (gamma, beta, alpha, theta and delta).

recorded at channel 8. A very interesting observation is the leveling off of MSE for ADHD group, which remains constant in that region. In *theta* (θ) band, the relative difference in levels of the curves remain as before, but here the slopes of MSE curves for ASD and control are steeper while the slope of ADHD is falling more slowly and finally at the end of theta band, is the same as the slope of the other two. At region *delta (δ)*, the complexities of all curves are small with same slope and series of relative levels.

In valid type of syllogism, the dynamic behavior of MSE curves is significantly different than that in invalid type. In the gamma region MSE values for ASD and ADHD are clustered together and are both more away than the values in the control group. In beta also, MSE values of ASD and control differ significantly, while in the middle of alpha region an intersection takes place, after which ADHD's entropy is larger than that of ASD's one, and completely different than that of control. *So, in this crucial region, the clustering of MSE values of both 'pathological' groups is clear and differ strongly than those of the control group. It is very important to examine further which underlying dynamic mechanism 'enters the scene' at the middle of the alpha region and causes the dynamics of the ADHD signal to be more complex than that of the ASD signal.* In delta region, the relative series of values of MSE remains as in the previous region, but with a large deviation of the ASD curves from ADHD curves and with larger slope.

Next, we present in Figures 12 and 13, the results of MSE curves evaluated for the Grand averages of EEGs (across subjects of each group, and for each channel). Here we show the result for channel 8, for valid and invalid groups, so we can compare the results with those computed for a single subject, at the same channel. As it is expected, the averaging across subjects 'smooths' the data but as it is also evident from the figures, the dynamic behavior of the curves is consistent.

In Figures 14 and 15, the *MSE vs scale factor (linear scale) and log scale,* respectively, is presented, at channels 1 to 4, computed on EEG grand averages (across all subjects of each group, for each separate channel), for the invalid type of syllogism. Similar results, for both valid and invalid types, at all channels, for all groups are provided at the supplementary material, in figures S4–S10.

3.6. Power spectrum analysis

In Figures 16, 17, and 18 we present the results of the power spectrum analysis for control, ADHD and ASD groups of subjects, for the invalid type, for delta, theta and alpha rhythms. Similar results for all groups, both type of syllogism and all rhythms, are presented in supplementary material (figures S11–S25). Figure 16 is concentrated in a single subject PSD analysis, actually subject c#23 (from Control group).

In order to have an insight on the behavior of PSD curves per each channel, and particularly on the spatial power distribution, this is implemented via the topographic maps at each electrode location. In Figure 16a, PSDs and topographic maps, of subject #23, for all frequency bands are shown. We observe that the peak of power values (the PSD intensity, reflecting a high level of variability in EEGs oscillations), for almost all frequencies, are highly concentrated mainly at the 02 and secondarily at P8 electrodes (right Occipital and Parietal), as shown also in figure 16b, a zoom at theta band. A low to moderately variability is also observed at AF4 electrode (anterio-frontal, right), as shown in the right top region of the topographic map, Figure 16b. The rest of the figures present the PSD of Grand average EEGs, for each type of syllogism and group of subjects. Combining the information contained in these figures and the results from MSE analysis, we have constructed the summary Figure 19, section 4, on which all results are 'synthesized' and facilitate a lot their interpretation as well as the extraction of final conclusions.

4. Discussion

In this paper, we attempt to detect whether typical controls and groups with ASD and ADHD have similar or differing patterns of complexity, as reflected by statistically significant different MSE values, as the coarse-graining is getting larger, compared to that in the controls. The stimuli used to activate the brain are cognitive tasks of varying complexity (inducing varying cognitive loads), four type of Aristotle's syllogisms or reasoning patterns. Based on the above literature, the

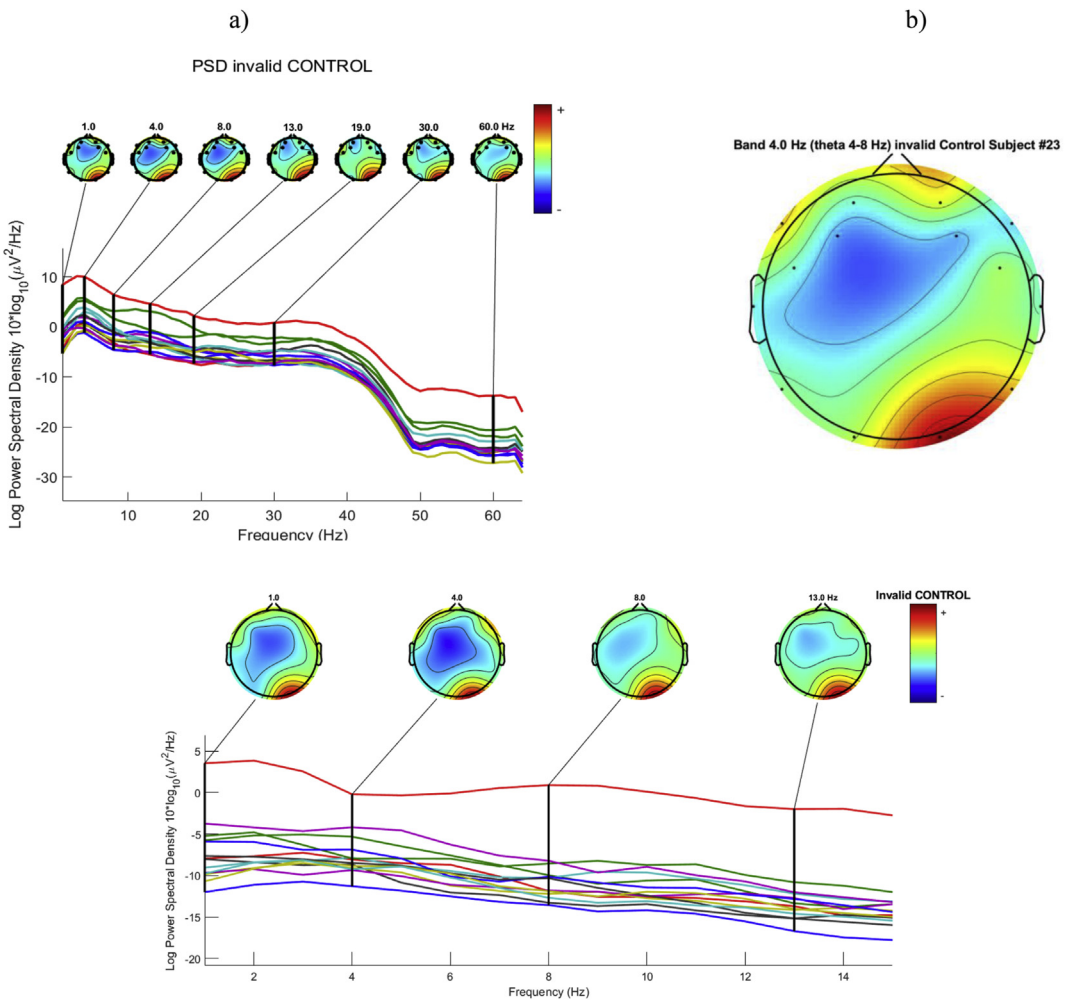

Figure 16. Channel spectra and topographical maps of EEGs, for the Invalid type of syllogism, from CONTROL group of subjects. a) PSD and topographic maps, of subject #23, for all frequency bands. b) Zoom of frequency band 4–8 Hz (theta). c) Delta, theta and alpha bands. Each colored trace represents the spectrum of the activity of one of the 14 channels. The maps show the scalp distribution of power at *1*–4 Hz *(delta rhythm), 4*–8 Hz *(theta rhythm), and 8*–13 Hz *(alpha rhythm)* (The small black dots on the map, indicate the locations of the electrodes).

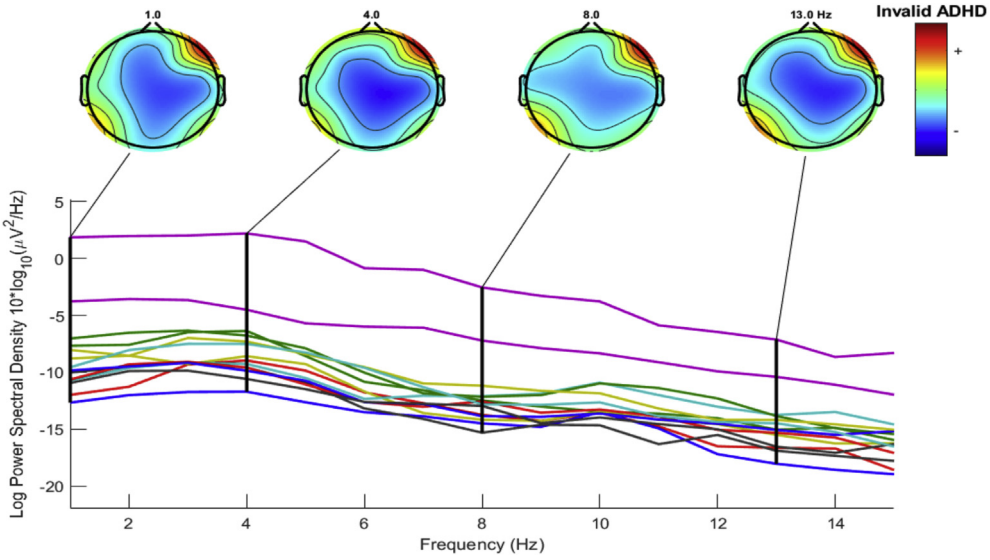

Figure 17. Channel spectra and topographical maps of EEGs, for the Invalid type of syllogism, from ADHD group of subjects. Each colored trace represents the spectrum of the activity of one of the 14 channels. The maps show the scalp distribution of power at *1*–4 Hz *(delta rhythm), 4*–8 Hz *(theta rhythm), and 8*–13 Hz *(alpha rhythm)*.

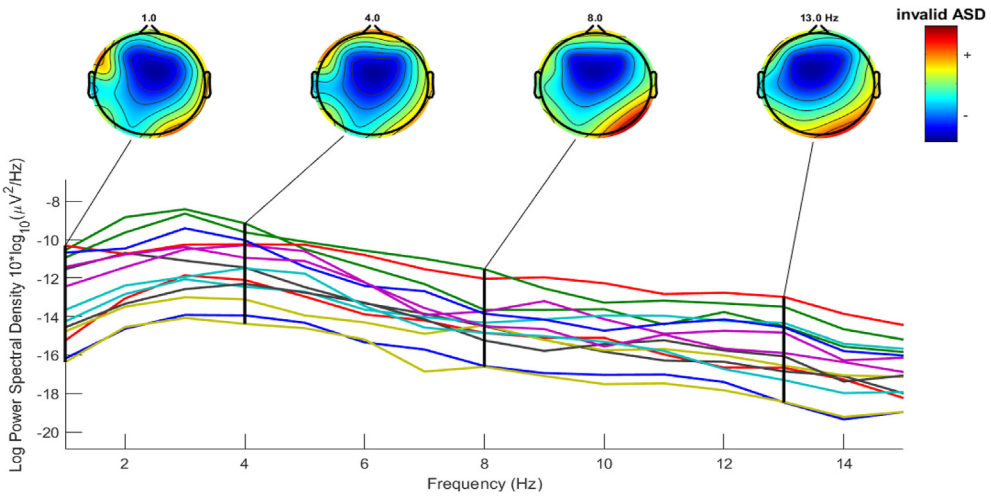

Figure 18. Channel spectra and topographical maps of EEGs, for the Invalid type of syllogism, from ASD group of subjects. Each colored trace represents the spectrum of the activity of one of the 14 channels. The maps show the scalp distribution of power at *1*–4 Hz *(delta rhythm), 4*–8 Hz *(theta rhythm), and 8*–13 Hz *(alpha rhythm).*

Valid Syllogism		Identified groups of subjects with peak values in Power spectra (upper row) Identified groups of subjects with peak values in Multiscale Entropy MSE (lower row)					
Channel Index	Channel Name	Frequency Bands					
		gamma γ (30-60 Hz)	beta1 β1(13-19 Hz)	beta 2 β2 (19-30)	Alpha α (8-13 Hz)	Theta θ (4-8 Hz)	Delta δ (1-4Hz)
1	AF3	ASD	ASD+ADHD	ASD+ADHD	ADHD	ADHD	ADHD
2	F7	ASD					
		ASD	ALL	ALL	ADHD	ADHD	ADHD
3	F3						
		ASD	ALL	ALL	ADHD	ADHD	ADHD
4	FC5						
		ALL	ASD+ADHD	ASD+ADHD	ADHD	ADHD	ADHD
5	T7						
		ALL	ALL	ALL	ADHD+ASD	ADHD	ADHD
6	P7	ADHD			ADHD		ADHD
		ALL	ASD	ASD	ASD+CONTROL	ASD	ASD
7	O1	ASD+CONTROL	ADHD+ASD	ADHD+ASD	ASD		
		ALL	ASD+ADHS	ASD+ADHD	ADHD+CONTROL	ADHD+CONTROL	ADHD
8	O2	ASD+CONTROL	ASD+AONTROL	ASD+CONTROL	ASD+CONTROL	CONTROL	CONTROL
		ASD	ASD+ADHD	ASD+ADHS	ASD+ADHD	ADHD	ADHD
9	P8		CONTROL	CONTROL	ASD		
		ASD	ASD+ADHD	ASD+ADHD	ASD+ADHD	ADHD	ADHD
10	T8	ASD		ADHD			
		ALL	ALL	ALL	ALL	ADHD	ADHD
11	FC6						
		ASD	ALL	ALL	ALL	ALL	ADHD
12	F4						
		ASD	ASD	ASD	ASD	ASD+CONTROL	ASD+CONTROL
13	F8	ADHD	ADHD	ADHD	ADHD	ADHD	ADHD
		ASD	ASD	ASD	ASD+CONTRL	ASD+CONTROL	ALL
14	AF4						
		ASD	ASD	ASD	ASD+ADHD	ADHD	ADHD

Figure 19. Summary of results of MSE and PS analysis for the valid type of syllogism.

research hypothesis in this challenging work is that MSE will be reduced over coarser time scales in the ASD and ADHD groups, when compared to MSE in the control group, during performance of *Aristotle's valid and invalid sets of types of syllogisms (cognitive tasks).*

The results of the application of the combination of a linear (power spectrum) and a nonlinear (MSE) tool, are summarized in the Figures 19 and 20. Starting with the valid type of syllogism, in AF3 channel in the left anterio-frontal region of the brain, only the groups ASD and ADHD are observed to show significant complexity-variability in the EEGs. In rhythms (frequency bands) alpha and theta, this region is dominated by EEGs of the ADHD group, showing large values of entropy and peak values in the power spectrum. EEGs of the ASD group appear to have large values of complexity-variability in gamma and beta, in which ADHD is present. In summary, ADHD and ASD groups differ from control group in left anterio-frontal region. The *increased*, high value in *frontal theta amplitude* combined with the ***decreased*** – ***small value*** of *parietal (P7, P8) alpha* amplitude, demonstrate the engagement of *enhanced cognitive control and attention levels,* conditions that correspond to *System II of thinking* (Cavanagh and Frank, 2014; Cavanagh & Shankman, 2015).

As we have seen in section 1.3, when a subject engages in *System I of thinking*, cognitive control and attentional resources are required to a

Invalid Syllogism		Identified groups of subjects with peak values in Power spectra (upper row) Identified groups of subjects with peak values in Multiscale Entropy MSE (lower row)					
Channel Index	Channel Name	Frequency Bands					
		gamma γ (30-60 Hz)	beta1 β1(13-19 Hz)	beta 2 β2 (19-30)	Alpha α (8-13 Hz)	Theta θ (4-8 Hz)	Delta δ (1-4Hz)
1	AF3						
		ASD	ASD+ADHD	ASD+ADHD	ADHD	ADHD	ADHD
2	F7				ASD		
		ASD	ASD+ADHD	ASD+ADHD	ADHD	ADHD	ADHD
3	F3						
		ASD+ADHD	ASD+ADHD	ASD+ADHD	ADHD	ADHD	ADHD
4	FC5						
		ASD+ADHD	ASD+ADHD	ASD+ADHD	ADHD	ADHD	ADHD
5	T7						
		ASD	ASD+ADHD+CONTROL	ASD+ADHD+CONTROL	ADHD	ADHD	ADHD
6	P7	ADHD	ADHD	ADHD			
		ASD+CONTROL	ASD	ASD	ASD	ASD	ASD
7	O1						
		ASD	ALL	ADHD+CONTROL	ADHD+CONTROL	ADHD+CONTROL	ADHD+CONTROL
8	O2	ASD+CONTROL	ASD+CONTROL	ASD+CONTROL	ASD+CONTROL	ASD+CONTROL	CONTROL
		ASD	ALL	ALL	ADHD	ADHD	ADHD
9	P8						
		ASD	ALL	ALL	ADHD	ADHD	ADHD
10	T8	ASD		ADHD+ASD			
		ALL	ASD+ADHD	ASD+ADHD	ADHD	ADHD	ADHD
11	FC6						
		ASD	ALL	ALL	ALL	ADHD+CONTROL	ADHD+CONTROL
12	F4						
		ASD	ASD	ASD	ASD	ASD+CONTROL	ASD+CONTROL
13	F8	ADHD	ADHD	ADHD	ADHD	ADHD	ADHD
		ASD	ASD	ASD	ASD+CONTROL	CONTROL	CONTROL
14	AF4			ADHD			
		ASD	ASD	ASD	ADHD	ADHD	ADHD

Figure 20. Summary of results of MSE and PS analysis for the invalid type of syllogism.

lesser extend (*decreased frontal theta* amplitude, increased parietal alpha amplitude), reflecting a reliance on automatic or routinized systems of the brain (Evans, 2010a, 2010b; Evans & Stanovich, 2013a, 2013b; Kahneman, 2011; Varga & Hamburger, 2014). The above literature has shown also that *cognitive control (theta) and attentional mechanisms (alpha) are not independent and work in conjunction to result in System I and System II thinking.*

4.1. Valid type of syllogism

In channel F7 (frontal temporal left), ADHD subjects have the largest values primarily in α and θ bands, as well as lesser in β1 and β2, while ASD in γ and control in β1 and β2.

In channel F3 (frontal left), exhibits the same behavior as channel F7.

In FC5 we have the same behavior as the previous channels but ASD shows large entropy values also in β1 and β2.

In T7 (temporal left) channel, all groups (control included) show large complexity and variability in γ, β1 and β2 while α, θ and δ are dominated by ADHD, while ASD in α.

In channel P7 (parietal left), ASD appears strong in α, θ, δ, β1 and β2 and control in α. ADHD group is absent in this channel. So, EEGs in left parietal are primarily activated by ASD.

In channel 01 (occipital left), control and ADHD dominate α and θ bands ASD and ADHD in β1 and β2, while δ is dominated by ADHD.

In channel 02 (occipital right), MSE analysis and PS indicates that ASD and ADHD are strong in γ, β1 and β2 (the PS shows also control in γ,

Table 8. Engagement of linguistic & visuo-spatial systems in temporal & occipital lobes, in the groups of subjects, for the valid type of syllogism.

Engagement in Syllogistic Reasoning	Alpha rhythm	Theta Rhythm
Linguistic System	Temporal Lobes	
	T7 (ASD + ADHD)	T7 (ADHD)
	T8 (ALL + ADHD)	T8 (ADHD)
Visuo-Spatial	Occipital lobes	
	01 (ADHD + CONTROL)	01 (ADHD + CONTROL)
	02 (ASD + ADHD)	02 (ADHD)

Table 9. Engagement of linguistic & visuo-spatial systems in temporal & occipital lobes, in the groups of subjects, for the invalid type of syllogism.

Engagement in Syllogistic Reasoning	Alpha rhythm	Theta Rhythm
Linguistic System	Temporal Lobes	
	T7 (ADHD)	T7 (ADHD)
	T8 (ADHD)	T8 (ADHD)
Visuo-Spatial	Occipital lobes	
	01 (ADHD + CONTROL)	01 (ADHD + CONTROL)
	02 (ADHD)	02 (ADHD)

β1, β2) while in α and θ ASD and ADHD (by MSE) and ASD and control (by PS). Thus MSE shows in the right Occipital, the EEGs of ASD and ADHD primarily show large variability and complexity in rhythms α and θ, and EEGs of control groups in a lesser level indicated by PS.

In channel P8 (parietal right), EEGs of ADHD and ASD show large variability in α (ASD + ADHD) and θ (ADHD), as also in all other bands. The EEGs of control observed to have very small variability. So, ASD and ADHD differ significantly from control in this region of the brain (right parietal).

In channel T8 (temporal right), all groups show the same complexity and variability in γ, β1, and β2, while ADHD dominates in α (together with control), θ and δ. So, control and pathological groups, show the same complexity in a band, in the right temporal region of the brain.

FC6 channel (frontal-central right), in the majority of frequencies (β1, β2, α, θ) the complexity-variability of the EEGs in the frontal-central right of the brain is the same in both control and pathological subjects.

F4 channel (frontal right), in the right frontal the complexity of ADHD is very small compared to that of ASD (in all frequency bands) and that of control (in θ band). This, in right frontal ASD EEGs complexity dominates.

In F8 channel (frontal-temporal right), also in this region, ASD EEGs dominate in all frequency bands, with control EEGs to be present in α and θ bands (MSE result) while PS also shows presence of ADHD EEGs.

In channel AF4 (anterio-frontal right) no control EEGs have large complexity. ASD and ADHD dominates the anterio-frontal right area, in all bands.

In summary, for the valid type of syllogism, out of 14 channels, EEGs of ADHD appears to have large complexity in 13 channels in alpha (α) frequency band (~93%), and in 12 channels in θ band (86%). Also, in these 3 important bands, ASD has large complexity in 10 channels (θ band) (71%), and only in 4 channels (28%) in alpha (α) band. Also, in these 3 bands, control has large complexity in 6 channels in alpha (α) band (43%) and in 5 channels in theta (θ) band (36%).

4.2. Invalid type of syllogism

Channel AF3 is dominated, in respect of complexity and variability by groups ASD and ADHD in all frequency bands. Control subjects do not appear in any of these frequency bands. More specifically ADHD is present in θ and α bands while ASD is present in γ and β and ADHD only in β. *Therefore, in the anterio-frontal region there is a significant difference in the EEGs complexity, between control and pathological participants.*

At channel F7 (frontal temporal left), only the pathological groups are present. Rhythms α and θ are dominated by ADHD. Pathological groups are present also in other rhythms, ASD in γ and β and ADHD in β1 and β2. *Therefore, at F7 there is complete distinction between ASD, ADHD and control.*

The above behavior is also present in F3 channel where control group is absent. The same also happens as FC5. At channel F7, control group is present at rhythms β1, β2 and ADHD group dominates frequency bands α and θ. At channel P7, control group appears only at γ frequency and ASD dominates α and θ frequency bands. At the left occipital 01 groups ADHD and control dominate in α and θ bands while control appears in β1, β2 and δ. At the right occipital the control group appears in α rhythms γ, β, δ, while ADHD in α and θ bands (MSE analysis). ASD is present at α and θ (PS analysis). At channel PH (right parietal) ADHD appears only in rhythms α and θ while ASD in γ and control in β and δ. At the frontal-central right region of the brain, the control group dominates in all rhythms while ADHD is present also at α and θ. At channel F4, ASD is present at α and θ, and control in θ and δ. Group ADHD is absent. At frontal temporal right (F8), ASD and control are present only in α and θ (MSE analysis) while PS also includes ADHD. ASD group dominates γ, β1, β2 rhythms while the control group appears in δ band. At F4 channel, ADHD dominates α, θ, δ bands while ASD in γ, β1 and β2.

In summary for the *invalid syllogism*:

1. *Rhythms α and θ are dominated heavily by ADHD group (at 10 out of 14 channels or 71%) while only 4 out of 14 channels (24%) by control group and only 1% by the ASD group.*
2. *Out of the 14 channel, control group shows the highest values in complexity and variability (independently from the method of analysis PS or MSE) only at 5 channels (33,3%) while the rest 66.6% of the channels is dominated by the pathological groups ASD and ADHD with the later to dominate completely at rhythms α and θ.*

Figure 19 shows that the Frontal channels at which Theta power is high are, for ADHD: AF3, F7, F3, FC5, FC6, F8 and F14 (7 channels). For ASD: FC6, F4 and F8 (3 channels). For CONTROL: FC6, F4 and F8 (3 channels). The Parietal channels at which alpha power is low, are for ASD: P7, and for CONTROL: P8.

We now describe our effort to connect our results found in temporal lobes (T7, T8 channels), and occipital lobes (O1, O2, channels) with the linguistic and visuo-spatial components in the Aristotelian syllogism, as described in section 1.3. The following table (using results in Figures 19 and 20) aims in summarizing the findings.

From Table 8 it is evident that the linguistic and visuo-spatial systems are strongly engaged in the 'pathological' subjects, as in ASD and ADHD groups the temporal and occipital lobes are heavily activated. We note that the engagement of linguistic system is very strong only in ASD subjects while the engagement of visuo-system is also present in the control group.

We observe in Table 9 that both the linguistic & visuo-spatial systems, in the case of invalid type, are only engaged in the ADHD subjects, with ASD in absence. In the control subjects the visuo-spatial system is also engaged.

Taken together, we found that during both valid and invalid experimental settings beta and gamma oscillations were significantly associated in patients with ASD compared to both ADHD and controls. These findings are compatible with studies reporting altered activities of beta oscillations, which are related to the continuance of the present sensorimotor or cognitive conditions in patients with ASD (Engel and Fries, 2010; Leung et al., 2014). In this context it should be noted that gamma oscillations are the highly frequently perceived brain activity in ASD (Maxwell et al., 2015). The gamma oscillations are believed to reflect the operation of inhibitory GABAergic interneurons, and a

predominant theory states that loss or reduction of inhibitory interneurons may result to compromised dealing with the social and emotional stimuli in ASD (Rubenstein and Merzenich, 2010).

The variability of the obtained patterns activity of the gamma and beta brain activity might reflect the heterogeneous nature of the disorder (Buzsaki and Draguhn, 2004; Uhlhaas and Singer, 2006).

Another balancing explanation regarding the observed results might be the widely acknowledged notion concerning the brain activity of individuals with ASD who mostly manifest a distributed network pattern with reduced activity in task-related areas and enhanced activity in task unrelated areas (Takarae et al., 2007; van Diessen et al., 2015).

As far as the observed EEG oscillations and ADHD group the present results appear to be compatible with studies investigating the relationship between ADHD and brain activity as it reflected by EEG bands. Indeed, there is evidence indicating that theta and alpha EEG activity would distinguish between healthy populations and adult ADHD (Adamou et al., 2020). Marzbani et al. (2016) also showed that people with ADHD disorder have slower brain wave activity (theta) and less beta activity compared to normal people. Similarly Egner and Gruzelier (2004) reported that beta activity is a good indicator for mental performance and inappropriate beta activity represents mental and physical disorders like depression, ADHD and insomnia (Egner and Gruzelier, 2004).

From the Reasoning Type (valid and invalid) the principal notice in the present study are the findings that ADHD and ASD groups differ from control group in left anterio-frontal region to greater degree and in a lesser degree the participation of visuospatial circuit. Probably to maintain and manipulate familiar information, together with a functional decoupling unfamiliar information. This assessment is compatible with the view regarding the anatomy of deductive reasoning. Indeed Goel (2007) claims 'Cognitive neuroscience data suggest a unitary system for logical reasoning and towards a fractionated system dynamically reconfigured in response to specific task and environmental cues'. It has been proposed that the left frontal-temporal system treats familiar material, while the parietal visuospatial system treats unfamiliar material. In a similar account Johnson-Laird PN (2010) suggests that reasoning is a reproduction of our around world obtainable through our knowledge, but not the logical frames of premises.

Regarding the, surprisingly, large complexity-variability in ASD and ADHD subjects (which is opposite to the 'main stream' theory of *complexity loss in 'pathological' brain systems*) we provide the following explanation. Since regularity and complexity *are not necessarily correlated, signals* exhibiting high complexity (high entropy) are not necessary, also, real complex systems i.e. they do not possess the structural-informational richness over multiple temporal scales. As we have seen (see section 3.3) *white noise* even though has a high value of entropy, is not a real complex system. In Figure 9 of section 3.3 we show that the MSE values of white noise resemble, in respect to their dynamic behavior (MSE carves), to the MSE values of ADHD and ASD. Therefore, the higher complexity-variability of these two "pathological" groups of subjects, relative to Control subjects, *does not undermines the broadly accepted argument that reduced levels of psysiological complexity is observed in 'pathological' groups.* This work, instead, supports further the above theory, since reveals, through a combination of linear and nonlinear tools (PS and MSE, respectively), that ASD and ADHD subjects do not possess the structural-informational richness of real complex and adaptive systems, as the control subjects do. Therefore, the increased complexity observed in ASD and ADHD is not due to their endogenous adaptive capacity to 'face' difficult demanding cognitive loads imposed by Aristotelian syllogism. The 'pathological' subjects, having low (inherently) cognitive and attentional capacity, during such syllogisms are in a stage of 'confusion', unable to recall any typical presentations in the brain that 'resemble' the ones required by syllogism. Their brain state of confusion incorporates a high number of brain subsystems located in various brain regions, making their brain a higher dimensional dynamical system with higher degrees of freedom (like a high dimensional white noise process), compared with the dynamics in the brains of control Subjects, which is concentrated in fewer regions of the brain, i.e. in a low dimensional *attractor,* but very adaptive to the external environment (generated by the syllogisms), making the brain with high cognitive control and attention capacity, as well as other cognitive qualities like working memory, STM, LTM that are necessary to face conditions with 'heavy' cognitive loads during reasoning processes.

The aforementioned discrepancy regarding the higher complexity-variability in ASD and ADHD subjects than the healthy controls could be also understood considering the cognitive efficiency theory. Cognitive efficiency (CE) has been described as the ability to reach optimal use of mental resources, such as cognitive and executive functions, in order to achieve the maximum performance on a task (Hoffman and Schraw, 2010).

This view is influenced by the following accounts. On the one hand frequent forms of cognition are easily learned through experience and thus might be developed automaticity (Hoffman, 2012). Additionally, human beings posit an evolutionary tendency to improve working memory load proceeding by demanding problem-solving duties (Evans, 2008; Stanovich, 2009). In this context, subjects solving cognitive tasks correctly are regarded cognitively efficient since use less brain energy consumption as verified by brain imaging technique whereas they actuate working memory resources (Neubauer and Fink, 2009).

Furthermore, there are convergent evidence suggesting that inefficient cognitive control of information processing is a fundamental deficit in both ASD and ADHD. Indeed Speirs et al. (2014); and Mackie MA, & Fan J, (2016) reported inefficient cognitive control of information processing in ASD which have implications for social functioning and interventions. Peterson et al. (2019) also demonstrated that children and adults with ASD exhibit reduced neural efficiency for cognitive and social functioning which in turn is correlated positively with higher hyperperfusion throughout frontal white matter and subcortical gray.

In the same way ADHD is characterized by various cognitive difficulties. In particular Segal et al. (2015), reported ineffective processing of semantic conflicts in adults with ADHD attributable to their inability to rely on executive attention and as an alternative, they may recruit higher (probably less effective) language mechanisms such as semantic mechanisms. In this sense Sheridan et al. (2007) revealed cognitive deficits in children and adolescents with ADHD which hypothesized to be related to low efficiency of prefrontal cortex (PFC) function. Equally Bédard et al. (2014) registered declined efficiency of DLPFC in ADHD for high-load visuospatial working memory and compensatory support of posterior spatial attention circuits to manage spatial position than healthy control.

In summary, in the case of invalid type of Aristotelian syllogisms, the linguistic and visuo-spatial systems are both engaged ONLY in the temporal and occipital regions of the brain, respectively, of ADHD subjects. In the case of valid type, both above systems are engaged in the temporal and occipital regions of the brain, respectively, of both ASD and ADHD subjects, while in the control subjects only the visuo-spatial type is engaged.

5. Conclusions

This investigation shows the ranking of cognitive workload using the EEG signals, imposed by Aristotle's valid and invalid types of syllogisms, and how this ranking is differentiated in three different groups of subjects: control, ASD and ADHD. For the differentiation within-subjects and within-groups, a combination of a linear tool (power spectral density analysis) and a nonlinear one (Multiscale entropy) was used. The main finding is that the level of complexity and variability of the EEG signals, reflecting the cognitive workload in various brain regions, differ significantly in respect to the location of the channel, as well as to the group

the subject belongs to. The cognitive load imposed by the different types of Aristotelian syllogism 'activate' different regions of the brain that may interact each other, depending on the difficulty the subject 'feels' in an effort to 'manage' the information content and the consequent processing in the syllogism cognitive task. The channels related to the *occipital and frontal lobes* attain the highest values of the complexity-variability, supporting the existing literature (Knauff, 2007) and further expanding it as the nonlinear tool MSE can detect-reveal nonlinear correlations and interactions of brain regions that cannot be by PS. In this respect, this work contributes toward understanding deeper how different subject groups, normal-'pathological', respond to these particular demanding cognitive workloads imposed by Aristotelian syllogisms.

Regarding the, surprisingly, large complexity-variability in ASD and ADHD subjects (which is opposite to the 'main stream' theory of *complexity loss in 'pathological' brain systems*) we provide the following explanation. Since regularity and complexity *are not necessarily correlated, signals* exhibiting high complexity (high entropy) are not necessary, also, real complex systems i.e. they do not possess the structural-informational richness over multiple temporal scales. As we have seen (see section 3.3) *white noise* even though has a high value of entropy, is not a real complex systems. In Figure 9 of section 3.3 we show that the MSE values of white noise resemble, in respect to their dynamic behavior (MSE carves), to the MSE values of ADHD and ASD. Therefore, the higher complexity-variability of these two 'pathological' groups of subjects, relative to Control subjects, *does not undermines the broadly accepted argument that reduced levels of psysiological complexity is observed in 'pathological' groups.* This work, instead, supports further the above theory, since reveals, through a combination of linear and nonlinear tools (PS and MSE, respectively), that ASD and ADHD subjects *do not possess the structural-informational richness* of real complex and adaptive systems, as the control subject do. Therefore, the increased complexity observed in ASD and ADHD is not due to their endogenous adaptive capacity to 'face' difficult demanding cognitive loads imposed by Aristotelian syllogism. The "pathological" subjects, having low (inherently) cognitive and attentional capacity, during such syllogisms are in a stage of 'confusion', unable to recall any typical presentations in the brain that 'resemble' the ones required by syllogism. Their brain state of confusion incorporates a high number of brain subsystems located in various brain regions, making their brain a higher dimensional dynamical system with higher degrees of freedom (like a high dimensional white noise process), compared with the dynamics in the brains of control Subjects, which is concentrated in fewer regions of the brain, i.e. in a low dimensional *attractor,* but very adaptive to the external environment (generated by the syllogisms), making the brain with high cognitive control and attention capacity, as well as other cognitive qualities like working memory, STM, LTM that are necessary to face conditions with 'heavy' cognitive loads during reasoning processes.

Declarations

Author contribution statement

Anastasia G. Papaioannou, George Papaioannou: Analyzed and interpreted the data; Wrote the paper.

Eva Kalantzi, Kalliopi Korombili: Performed the experiments.

Christos C. Papageorgiou: Contributed reagents, materials, analysis tools or data.

Anastasia Bokou: Performed the experiments; Analyzed and interpreted the data.

Pehlivanidis Artemios, Charalabos C. Papageorgiou: Conceived and designed the experiments.

Funding statement

This work was supported by the Regional Governor of Attica.

Data availability statement

The data that has been used is confidential.

Declaration of interests statement

The authors declare no conflict of interest.

Additional information

Supplementary content related to this article has been published online at https://doi.org/10.1016/j.heliyon.2020.e03459.

Acknowledgements

None.

References

Adamou, M., Fullen, T., Jones, S.L., 2020 Aug 25. EEG for diagnosis of adult ADHD: a systematic review with narrative analysis. Front. Psychiatr. 11, 871.
American Psychiatric Association, 2000. Diagnostic and Statistical Manual of Mental Disorders: DSM-IV_TR. American Psychiatric Association, Washington, DC.
American Psychiatric Association, 2013. Diagnostic and Statistical Manual of Mental Disorders. (5th ed.) (DSM-5). American Psychiatric Publishing, Arlington, VA, USA.
Baddeley, A., 2007. Working Memory, Thought and Action. Oxford University Press.
Banks, A., 2017. Comparing dual process theories: evidence from event-related potentials. Dual Proc. Theory 2, 66–81.
Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J., Clubley, E., 2001. The autism-spectrum quotient (AQ): evidence from asperger syndrome/high-functioning autism, malesand females, scientists and mathematicians. J. Autism Dev. Disord. 31 (1), 5–17.
Barry, R.J., Clarke, A.R., Johnstone, S.J., 2003. A review of electrophysiology in attention-deficit/hyperactivity disorder: I. Qualitative and quantitative electroencephalography. Clin. Neurophysiol. 114 (2), 171–183.
Barttfeld, P., Wicker, B., Cukier, S., Navarta, S., Lew, S., Sigman, A., 2011. A big–world network in ASD: dynamical connectivity analysis reflects a deficit in long-range connections and an excess of short-range connections. Neuropsychologia 49, 254–263.
Bédard, A.C., Newcorn, J.H., Clerkin, S.M., Krone, B., Fan, J., Halperin, J.M., Schulz, K.P., 2014 Sep. Reduced prefrontal efficiency for visuospatial working memory in attention-deficit/hyperactivity disorder. J. Am. Acad. Child Adolesc. Psychiatry 53 (9), 1020–1030 e6.
Bosl, W., Tierney, A., Tager-Flusberg, H., Nelson, C., 2011. EEG complexity as a biomarker for autism spectrum disorder risk. BMC Med. 9 (1), 18.
Bosl, W.J., Loddenkemper, T., Nelson, C.A., 2017. Nonlinear EEG biomarker profiles for autism and absence epilepsy. Neuropsychiatr. Electrophysiol. 3 (1), 1.
Bruce, E.N., Bruce, M.C., Vennelaganti, S., 2009. Sampling entropy trucks changes in EEG power spectrum with sleep state and aging. J. Clin. Neurophysiolog. Off. Publ. Am. Electroencephalogr. Soc. 26, 257–266.
Brush, J.E., Sherbino, J., Norman, G.R., 2017. How expert clinicians intuitively recognize a medical diagnosis. Am. J. Med. 130, 629–634.
Buzsaki, G., Draguhn, A., 2004. Neuronal oscillations in cortical networks. Science 304 (5679), 1926–1929.
Catarino, A., Churches, O., Baron-Cohen, S., Andrade, A., Ring, H., 2011. Atypical EEG complexity in autism spectrum conditions: a multiscale entropy analysis. Clin. Neurophysiol. 122 (12), 2375–2383.
Cavanagh, J.F., Frank, M.J., 2014. Frontal theta as a mechanism for cognitive control. Trends Cognit. Sci. 18 (8), 414–421.
Cavanagh, J.F., Shackman, A.J., 2015. Frontal midline theta reflects anxiety and cognitive control: meta-analytic evidence. J. Pshychol. Paris 109 (1-3), 3–15.
Costa, M., Goldberger, A.L., Peng, C.K., 2002. Multiscale entropy analysis of complex physiologic time series. Phys. Rev. Lett. 89, 068102.
Costa, M., Peng, C.-K., Goldberger, A.L., Hausdorff, J.M., 2003. Phys. A 330, 53.
Costa, M., Goldberger, A.L., Peng, C.K., 2005. Multiscale entropy analysis of biological signals. Phys. Rev. E - Stat. Nonlinear Soft Matter Phys. 71, 021906.
Courtiol, J., Perdikis, D., Petkoski, S., Muller, V., et al., 2016. The Multiscale entropy: guidelines for use and interpretation in Brain signal analysis. J. Neurosci. Methods 273 (2016), 175–190.
De Neys, W., 2009. Dual processing in reasoning: two systems but one reasoned. Psychol. Sci. 17, 428–433.
Debener, S., Minow, F., Emkes, R., Gandras, K., de Vos, M., 2012 Nov. How about taking a low-cost, small, and wireless EEG for a walk? Psychophysiology 49 (11), 1617–1621.
Eckmann, J.P., Ruelle, D., 1985. Rev. Mod. Phys. 57, 617.
Egner, T., Gruzelier, J.H., 2004. EEG Biofeedback of low beta band components: frequency-specific effects on variables of attention and event-related brain potentials. Clin. Neurophysiol. 115 (1), 131–139.
Engel, A.K., Fries, P., 2010. Beta-band oscillations-signalling the status quo? Curr. Opin. Neurobiol. 20, 156–165.

Evans, J., 2008. Dual-processing accounts of reasoning, judgment, and socialcognition. Annu. Rev. Psychol. 59 (1), 255–278.
Evans, J.S.B.T., 2010a. Intuition and reasoning: a dual-process perspective. Psychol. Inq. 21, 313–326.
Evans, J.S.B.T., 2010b. Thinking Twice : Two Minds in One Brain. Oxford University Press, Oxford , England.
Evans, J.S.B., Stanovich, K.E., 2013a. Dual-process theories of higher cognition: advancing the debate. Perspect. Psychol. Sci. 8 (3), 223–241.
Evans, J.S.B., Stanovich, K.E., 2013b. Theory and metatheory in the study of dual processing: reply to comments. Perspect. Psychol. Sci. 8 (3), 263–271.
Fallani Fde, V., Costa Lda, F., Rodriguez, F.A., Astolfi, L., Vechiatto, G., Toppi, J., et al., 2010. A graph theoretical approach in brain functional networks. Possible implications in EEG studies. Nonlin. Biomed. 4 (Suppl. 1), s8.
Glass, L., Mackey, M.C., 1992. From Clocks to Chaos: the Rhythms of Life. Princeton University Press, Princeton.
Goel, V., 2007 Oct. Anatomy of deductive reasoning. Trends Cognit. Sci. 11 (10), 41–435.
Goel, V., Buchel, C., Frith, C., Dolan, R.J., 2000. Dissociation of mechanisms underlying syllogistic reasoning. NreuroImage 12, 504–514.
Govindan, R., Wilson, J., Eswaran, H., Lowery, C., Preiβl, H., 2007. Revisiting sample entropy analyisis. Physica A 376, 158–164.
Grassberger, P., Procaccia, I., 1983. Phys. Rev. A 28, 2591.
Green, M.F., 1996. What are the functional consequences of neurocognitive deficits in Schizophrenia? Am. J. Psychiatr. 153–330.
Halford, G.S., Cowan, N., Andrews, G., 2007. Separating cognitive capacity from knowledge: a new hypothesis. Trends Cognit. Sci. 11, 236–242.
Hattori, M., 2016. Probabilistic representation in syllogistic reasoning: a theory to integrate mental models and heuristics. Cognition 157, 296–320.
Heunis, T., Aldrich, C., Peters, J.M., Jeste, S.S., Sahin, M., Scheffer, C., De Vries, P.J., 2018. Recurrence quantification analysis of resting state EEG signals in autism spectrum disorder–a systematic methodological exploration of technical and demographic confounders in the search for biomarkers. BMC Med. 16 (1), 1–17.
Hitoglou, M., Ververi, A., Antoniadis, A., Zafeiriou, D.I., 2010. Childhood autism and auditory system abnormalities. Pediatr. Neurol. 42, 309–314.
Hoffman, B., 2012. Cognitive efficiency: a conceptual and methodological comparison. Learn. InStruct. 22, 133e144.
Hoffman, B., Schraw, G., 2010 Jan 21. Conceptions of efficiency: applications in learning and problem solving. Educ. Psychol. 45 (1), 1–4.
Houde, O., Zago, L., Mellet, E., Moutier, S., Pineau, A., Mazoyer, B., Tzourio-Mazoyer, N., 2000. Shifting from the perceptual brain to the logical brain: the neural impact of cognitive inhibition training. J. Cognit. Neurosci. 12 (5), 721–728.
Hsieh, L.T., Ranganath, C., 2014. Frontal midline theta oscillations during working memory maintenance and episodic encoding and retrieval. Neuroimage 85, 712–729.
Jhonson-Laird, P.N., Bara, B.G., 1984. Syllogistic inference. Cognition 16, 1–61.
Jirsa, V.K., Muller, V., 2013. Cross-frequency coupling in real and virtual brain networks. Front. Comput. Neurosci. 7, 78.
Johnson-Laird, P.N., 2010 Oct 26. Mental models and human reasoning. Proc. Natl. Acad. Sci. U. S. A. 107 (43), 18243–18250. Epub 2010.
Kahneman, D., 2011. Thinking, Fast and Slow. Macmillan.
Kahneman, D., Peavler, W.S., Onuska, L., 1968. Effects of verbalization and incentive on the pupil response to mental activity. Can. J. Psychol./Revue canadienne de psychologie 22 (3), 186.
Kaiser, M.D., Delmolino, L., Tanaka, J.W., Shiffrar, M., 2010. Comparison of visual sensitivity to human and object motion in autism spectrum disorder. Autism Res. 3, 191–195.
Ke, Y., Chen, L., Fu, L., Jia, Y., Li, P., Zhao, X., Ming, D., 2014. Visual attention recognition based on nonlinear dynamical parameters of EEG. Bio Med. Mater. Eng. 24 (1), 349–355.
Khoshnoud, S., Nazari, M.A., Shamsi, M., 2018. Functional brain dynamic analysis of ADHD and control children using nonlinear dynamical features of EEG signals. J. Integr. Neurosci. 17 (1), 17–30.
Klimesch, W., 2012. Alpha-band oscillations, attention, and controlled access to stored information. Trends Cognit. Sci. 16 (12), 606–617.
Knauff, M., 2006. A neuro-cognitive theory of relational reasoning with mental models and visual images. In: Held, C., Knauff, M., Vosgerau, G. (Eds.), Mental Models and the Mind: Current Developments in Cognitive Psychology, Neuroscience, and Philosophy of Mind. Elsevier, Oxford (UK), pp. 127–149.
Knauff, M., 2007. How our brains reason logically. Topoi 26 (2007), 19–36.
Knauff, M., Fangmeier, T., Ruff, C.C., Johnson-Laird, P.N., 2003. Reasoning, models, and images: behavioral measures and cortical activity. J. Cognit. Neurosci. 15, 559–573.
Kooij, S.J., Bijlenga, D., Salerno, L., Jaeschke, R., Bitter, I., Balázs, J., et al., 2019a. Updated European Consensus Statement on diagnosis and treatment of adult ADHD. Eur. Psychiatr. 56, 14–34.
Kooij, S.J., Francken, M.H., Bron, T.I., 2019b. Diagnostic interview for ADHD in adults (DIVA). Greek version. In: Pehlivanidis, A., Papanikolaou, K. (Eds.). DIVA Foundation, The Hague, the Netherlands.
Kwiatkowski, D., Phillips, P.C.B., Schmidt, P., Shin, Y., 1992. Testing the null hypothesis of stationarity against the alternative of a unit root. J. Econom. 54, 159–178.
Lai, M.C., Baron-Cohen, S., 2015. Identifying the lost generation of adults with autism spectrum conditions. Lancet Psychiatr. 2, 1013–1027.
Le Couteur, A., Lord, C., Rutter, M., 2003. The Autism Diagnostic Interview—Revised (ADI-R). Western Psychological Services, Los Angeles, CA, USA.
Leung, R.C., Ye, A.X., Wong, S.M., Taylor, M.J., Doesburg, S.M., 2014. Reduced beta connectivity during emotional face processing in adolescents with autism. Mol. Autism. 5, 51.
Li, C., Chen, Y., Li, Y., Wang, J., Liu, T., 2016. Complexity analysisnof brain activity in attention-deficit/hyperactivity disorder: a multiscale entropy analysis. Brain Res. Bull. 124, 12–20, 2016.
Lord, C., Rutter, M., DiLavore, P.C., Risi, S., Gotham, K., Bishop, S.L., 2012. Autism Diagnostic Observation Schedule, second ed. Western Psychological Services, Torrance, CA, USA.
Mackie, M.A., Fan, J., 2016 Mar. Reduced efficiency and capacity of cognitive control in autism spectrum disorder. Autism Res. 9 (3), 403–414. Epub 2015 Jul 14.
Manor, B., Costa, M.D., Hu, K., Newton, E., Starobinets, O., Kang, H.G., et al., 2010. Physiological complexity and system adaptability: evidence from postural control dynamics of older adults. J. Appl. Physiol. 109, 1786–1791.
Marzbani, H., Marateb, H.R., Mansourian, M., 2016 Apr. Neurofeedback: a comprehensive review on system design, methodology and clinical applications. Basic Clin. Neurosci. 7 (2), 143–158.
Mathewson, K.E., Beck, D.M., Ro, T., Maclin, E.L., Low, K.A., Fabiani, M., Gratton, G., 2014. Dynamics of alpha control: preparatory suppression of posterior alpha oscillations by frontal modulators revealed with combined EEG and event-related optical signal. J. Cognit. Neurosci. 26 (10), 2400–2415.
Maxwell, C.R., Villalobos, M.E., Schultz, R.T., Herpertz-Dahlmann, B., Konrad, K., Kohls, G., 2015. Atypical laterality of resting gamma oscillations in autism Spectrum disorders. J. Autism Dev. Disord. 45 (2), 292–297.
McIntosh, A.R., Kovacevic, N., Itier, R.J., 2008. Increased brain signal variability accompanies lower behavioral variability in development. PLos Compt. Biol. 4, e1000106.
McIntosh, A.R., Vakorin, V., Lovacevic, N., Wang, H., Diaconescu, A., Protzner, A.B., 2014. Spatiotemporal dependency of –age related changes in brain signal variability. Cerebr. Cortex 24, 1806–1817.
Neubauer, A.C., Fink, A., 2009. Intelligence and neural efficiency. Neurosci. Biobehav. Rev. 33, 1004–1023.
Osherson, D., Perani, D., Cappa, S., Schnur, T., Grassi, F., Fazio, F., 1998. Distinct brain loci in deductive versus probabilistic reasoning. Neuropsychologia 36 (4), 369–376.
Osman, M., 2004 Dec. An Evaluation of dual-process theories. Psychon. Bull. Rev. 11 (6), 988–1010.
Owen, O.F., Kenyon, F.G., Peters, F.H., translators, 2015. Aristotle's Organon, Complete Edition.
Papageorgiou, C., Manios, E., Tsaltas, E., Koroboki, E., Alevizaki, M., Angelopoulos, E., Dimopoulos, M.A., Papageorgiou, C., Zakopoulos, N., 2017. Brain oscillations elicited by the cold pressor test: a putative index of untreated essential hypertension. Int. J. Hypertens. 2017, 7247514. Epub 2017.
Papanikolaou, K., Paliokosta, E., Houliaras, G., Vgenopoulou, S., Giouroukou, E., Pehlivanidis, A., et al., 2009. Using the Autism Diagnostic Interview-Revised and the Autism Diagnostic Observation Schedule-Generic for the diagnosis of autism spectrum disorders in a Greek sample with a wide range of intellectual abilities. J. Autism Dev. Disord. 39 (3), 414–420.
Papaodysseus, C., Zannos, S., Giannopoulos, F., Arabadjis, D., Rousopoulos, P., Stathes, X., Papageorgiou, C., 2010. A new approach for the classification of event related potentials for valid and paradox reasoning. Biocybernet. Biomed. Eng. 30, 292–301.
Parsons, L.M., Osherson, D., 2001. New evidence for distinct right and left brain systems for deductive versus probabilistic reasoning. Cerebr. Cortex 11 (10), 954–965.
Pehlivanidis, A., Papanikolaou, K., Mantas, V., Kalantzi, E., Korobili, K., Xenaki, L.A., Vassiliou, G., Papageorgiou, C., 2020. Lifetime co-occurring psychiatric disorders in newly diagnosed adults with attention deficit hyperactivity disorder (ADHD) or/and autism spectrum disorder (ASD). BMC Psychiatr. 20, 423.
Pennycook, G., 2017. A perspective on the theoretical foundation of dual process models. Dual Proc. Theory 2, 34.
Peterson, B.S., Zargarian, A., Peterson, J.B., Goh, S., Sawardekar, S., Williams, S.C.R., Lythgoe, D.J., Zelaya, F.O., Bansal, R., 2019 Apr 1. Hyperperfusion of frontal white and subcortical gray matter in autism spectrum disorder. Biol. Psychiatr. 85 (7), 584–595. Epub 2018 Dec 21.
Pincus, S.M., 1991. Proc. Natl. Acad. Sci. U.S.A. 88, 22979.
Pincus, S.M., 2001. Ann. N. Y. Acad. Sci. 954, 245.
Ponomarev, V.A., Mueller, A., Candrian, G., Grin-Yatsenko, V.A., Kropotov, J.D., 2014. Group independent component analysis (gICA) and current source density (CSD) in the study of EEG in ADHD adults. Clin. Neurophysiol. 125 (1), 83–97.
Postle, B.R., 2015. Essentials of Cognitive Neuroscience. Wiley Blackwell, John Wiley & sons Ltd.
Ramirez, R., Palencia-Lefler, M., Giraldo, S., Vamvakousis, Z., 2015 Oct 2. Musical neurofeedback for treating depression in elderly people. Front. Neurosci. 9, 354.
Richman, J.S., Lake, D.E., Moorman, J.R., 2004. Sample entropy. Methods Enzymol. 384, 172–184.
Rippon, G., BrockJ, Brown, C., Boucher, J., 2007. Disordered connectivity in the autismbrain: challenges of the 'new psychophysiology'. Int. J. Psychophysiol. 63, 164–172.
Rubenstein, J.L.R., Merzenich, M.M., 2010. Model of autism: increased ratio of excitation/inhibition in key neural systems. Gene Brain Behav. 2 (5), 255–267.
Russo, N., Flanagan, T., IarocciG, Berringnger, D., Zelazo, P.D., Burack, J.A., 2007. Deconstructingexecutive deficits among persons with autism; implications for cognitive neuroscience. Brain Cognt. 65, 77–86.
Sakkalis, V., Tsiaras, V., Michalopoulos, k, Zervakis, M., 2008. Assessment of neural dynamics coupling and causal interactions between independent EEG components from cognitive tasks using linea and nonlinear methods. Conf. Proc. IEEE Eng. Med. Biol. Soc. 2008, 3767–3770.

Segal, D., Mashal, N., Shalev, L., 2015 Dec. Semantic conflicts are resolved differently by adults with and without ADHD. Res. Dev. Disabil. 47, 416–429. Epub 2015 Oct 27.
Sheridan, M.A., et al., 2007. Efficiency of the prefrontal cortex during working memory in attention-deficit/hyperactivity disorder. J. Am. Acad. Child Adolesc. Psychiatry. PMID: 17885578.
Simmons, D.R., Robertson, A.E., McKay, L.S., Total, E., McAleer, P., Pollick, F.E., 2009. Vision in autismspectrum disorders. Viss. Res. 49, 2705–2739.
Sitges, C., bornas, X., Llabres, J., Noguera, M., Montoya, P., 2010. Linear and nonlinear analyses of EEG dynamics during non-painful somatosensory processing in chronic pain patients. Int. J. Psychophysiol. 77, 176–183.
Smith, Robin, Translator, 1989. Aristotle's Prior Analytics. Hacket Publishing Company, Indianapolis/Cambridge.
Speirs, S.J., Rinehart, N.J., Robinson, S.R., Tonge, B.J., Yelland, G.W., 2014 Nov. Efficacy of cognitive processes in young people with high-functioning autism spectrum disorder using a novel visual information-processing task. J. Autism Dev. Disord. 44 (11), 2809–2819.
Stanovich, K., 2009. What Intelligence Tests Miss: the Psychology of the Rational Mind. The Reflective Mind, the Algorithmic Mind, and the Autonomous Mind. University Press, New Haven, CT, p. 20e44.
Stavovich, K.E., West, R.F., 2000. Individual differences in reasoning: implications for the rationality debate? Behav. Brain Sci. 23, 645–665 discussion 665-726.
Swartwood, J.N., Swartwood, M.O., Lubar, J.F., Timmermann, D.L., 2003. EEG differences in ADHD-combined type during baseline and cognitive tasks. Pediatr. Neurol. 28 (3), 199–204.
Takahashi, T., Cho, R.Y., Mizuno, T., Kikuchi, M., Mizukami, K., et al., 2009. Age-related variation in EEG complexity to photic simulation : a multiscale entropy analysis. Clin. Neurophysiol. 120, 476–483.
Takahashi, T., Cho, R.Y., Mizuro, T., KiKuchi, M., Murata, T., Takahashi, K., et al., 2010. Antipsychotics reverse abnormal EEG complexity in drug-naïve schizophrenia: a multiscale entropy analysis. Neuroimage 51, 173–182.
Takahashi, T., Yoshimura, Y., Hiraishi, H., Hasegawa, C., Munesue, T., Higashida, H., et al., 2016. Enhanced brain signal variability in children with autism spectrum disorder during early childhood. Hum. Brain Mapp. 37 (3), 1038–1050.
Takarae, Y., Minshew, N.J., Luna, B., Sweeney, J.A., 2007. Atypical involvement of frontostriatal systems during sensorimotor control in autism. Psychiatr. Res. 156, 117–127.
Uhlhaas, P.J., Singer, W., 2006. Neural synchrony in brain disorders: relevance for cognitive dysfunctions and pathophysiology. Neuron 52 (1), 155–168.
van Diessen, E., Senders, J., Jansen, F.E., Boersma, M., Bruining, H., 2015 Sep. Increased power of resting-state gamma oscillations in autism spectrum disorder detected by routine electroencephalography. Eur. Arch. Psychiatr. Clin. Neurosci. 265 (6), 537–540.
Varga, A.L., Hamburger, K., 2014. Beyond types 1 vs. type 2 processing: the tri-dimensional way. Front. Psychol. 5, 2–5.
Williams, C.C., Kappen, M., Hassall, C.D., Wright, B., Krigolson, O.E., 2019. Thinking theta and alpha: mechanisms of intuitive and analytical reasoning. Neuroimage 189, 574–580.