T.C.
ÜSKÜDAR UNIVERSITY
INSTITUTE OF HEALTH SCIENCE

NEUROSCIENCE DEPARTMENT

MASTER'S PROGRAM IN NEUROSCIENCE

MASTER'S THESIS

THE RELATIONSHIP BETWEEN IMPULSIVITY AND RESTING STATE QEEG AMONG PATIENTS WITH SIMULANT USE

Esra TORLAK

Thesis Advisor

Prof. Dr. Cemal Onur NOYAN

İSTANBUL-2024

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ÖZET

STİMULANT KULLANAN HASTALARDA DÜRTÜSELLİK VE RESTİNG STATE EEG İLİŞKİSİNİN İNCELENMESİ

Bu çalışmamızda Uyarıcı Madde Kullanan ve dürtüsellikle ilişkili nörobiyolojik yollar üzerindeki etkileri incelenmiştir. Bu çalışma, uyarıcı kullanan yüksek dürtüselliğe sahip hastaların EEG bulgularında frontal bölge Delta ve Beta aktivitesi olup olmadığını test etmeyi amaçlamaktadır. Özellikle, dürtüselliğin EEG dalga boyları üzerindeki etkisini ortaya koyarak, beyin aktivitesinin dürtüsellik düzeyleriyle ilişkisi incelenmiştir. Bu çalışma, uyarıcı kullanımı ile dürtüsellik arasındaki korelatif ilişkiyi ve dürtüsellik ile beynin frontal bölgesindeki belirli EEG bulguları arasındaki ilişkiyi araştırmaktadır. Bu çalışmada toplam 72 katılımcı hastalar (n=36) ve sağlıklı kontrol (n=34) dahil edildi. Katılımcılara sosyo-demografik veri formu, Barrat dürtüsellik ölçeği uygulandı. Katılımcılar anketi tamamladıktan sonra EEG absolute power değerlendirmesi yapıldı. Barratt Dürtüsellik Ölçeği Toplam puanı ile EEG dalga boyu arasında istatistiksel olarak anlamlı bir ilişki bulunmuştur. Delta dalga boyları ile Barrat Dürtüsellik Testi toplam puan arasında negatif bir ilişki bulunmuştur. Bu çalışma uyarıcı kullananlarda dürtüsellik ile beyin dalgası uzunluğu arasındaki ilişkiyi ortaya koymaktadır ve dürtüsellik düzeyinin beyin dalgası aktivitesi üzerinde bir ilişki olduğunu olduğunu söyleyebiliriz. Özellikle, yüksek dürtüselliğe sahip bireylerde düşük Delta ve Theta dalga aktivitesi gözlenmiştir.

Anahtar Kelimeler: Uyarıcı, Dürtüsellik, EEG, Stimulant Kullanımı Bozukluğu

ABSTRACT

THE RELATIONSHIP BETWEEN IMPULSIVITY AND RESTING STATE QEEG AMONG PATIENTS WITH STIMULANT USE

Naturally occurring or synthetically produced substances can either stimulate the brain's pleasure centers to achieve a desired euphoric effect (e.g. cocaine) or depress them to find solace and courage to counter the negative events of everyday life (e.g. alcohol), which can evolve over time into a substance use problem which can consequently have profound negative effects on normal cerebral neurochemical/neurobiological functionality. This paper will examine Stimulant Use Disorder (SUD) and their effects on the associated neurobiological pathways related to impulsivity. This study aims to test whether patients with high impulsivity who use stimulants have frontal region Delta and Beta activity in their EEG findings. Specifically, by revealing the effect of impulsivity on EEG wavelengths, we examined how brain activity is related to impulsivity levels. This study investigates the correlative relationship between stimulant use and impulsivity, as well as the relationship between impulsivity and specific EEG findings in the frontal region of the brain. A total of 72 participants were involved in this study (Patients (n=36) and Health Control (n=34). After completing a questionnaire, the participants underwent EEG tracings from which absolute power was determined. A statistically significant relationship was found between the Barratt Impulsivity Scale Total score and EEG wavelength. This reveals the relationship between impulsivity and brainwave length in stimulant users and shows that impulsivity level has a significant impact on brainwave activity. In particular, low Delta and Theta wave activity was observed in individuals with high impulsivity.

Keywords: Stimulant, Impulsivity, EEG, Simulant Use Disorder,

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DECLARATION FORM

I hereby declare that all data and documents used in this study were obtained according with the relevant academic rules and regulations. Furthermore, I confirm that all visual, auditory, and written information and results were presented in a manner that is consistent with the principles of scientific ethics. I also confirm that I did not falsify any data ı used and that I have provided appropriate citations for all sources.

Esra TORLAK

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SYMBOLS AND ABBREVIATIONS

ACC: Anterior Cingulate Cortex

ADHD: Attention -Deficit / Hyperactivity Disorder

AUD: Alcohol Use Disorder

BPD: Borderline Personality Disorder

CNS: Central Nervous System

CT: Computed Tomography

DA: Dopamine

DBT: Dialectical Behavioral Therapy

DLPFC: Dorsolateral Prefrontal Cortex

DSM-5: The Diagnostic and Statistical Manual of Mental Disorders

EEG : Electroencephalograph

FDA : Food and Drug Administration

GABA: Gamma-Aminobutryic Acid

GABAergic: Relating to Neuronal GABA Pathways

GI: Gastrointestinal

HIV: Human Immunodeficiency Virus

MRI: Magnetic Resonance Imaging Spectroscopy

MUD: Methamphetamine Use Disorder

NAcc: Nucleus Accumbens

OFC: Orbitofrontal Cortex

OUD: Opioid Use Disorder

PET: Positron Emission Tomography

PFC: Prefrontal Cortex

SNS: Sympathetic Nervous System

SSRIs: Selective Serotonin Reuptake İnhibitors

SUD: Substance Use Disorder

VTA: Ventral Tegmental Area

1. INTRODUCTION

The use of narcotics is of great global concern as substance abuse has reached epidemic proportions. Over 250 million individuals abuse substances (such as stimulants), and 0.6% of them have substance use disorders (SUDs). The substance abuse of stimulants and impulsivity, and their consequent associated effects on the brains of these patients (short-term usages as well as long-term usages (developing into psychopathologies)), as evidenced by electroencephalographic (EEG) investigations and resultant normal versus abnormal waveform trace findings, will be the main investigative focus of examination of this thesis.

There are various forms of narcotics which are prevalent and include stimulants and depressants, which can have a predilection to develop into a SUD. There is a strong correlation between SUD and impulsivity. An interesting pondering point is…does impulsivity lead to SUD or does SUD contribute to an already existent impulsive behavior? Examining the neurobiological pathways and subsequent EEG results may help to better understand this relationship.

2. GENERAL INFORMATION

2.1. Overview Of Stimulant Use

2.1.1 Definition of a stimulant:

Stimulants, also known as psychostimulants, are drugs that increase alertness, attention, and energy levels. These medications are widely misused for their euphoric effects; however, they can be used therapeutically to treat disease state disorders including attention deficit hyperactivity disorder (ADHD), depression and narcolepsy. Stimulants are a class of chemicals that work on the central nervous system to improve alertness, focus, and energy while also promoting pleasant mood and arousal. Their mode of action is to promote the activation of natural stimulation pathways in the brain, specifically the catecholamines (monoamine neurotransmitters) noradrenaline/norepinephrine, adrenaline/epinephrine, dopamine (a contraction of 3,4-dihydroxyphenethylamine), and serotonin (5-hydroxytryptamine) and their resultant functions. It should be noted that structurally norepinephrine (the most important nuclei in the brain where it's produced is in the locus coeruleus, located within the pons) (PubChem., 2015), epinephrine (mainly produced by the adrenal glands and by some neurons in the medulla oblongata and plays a vital role in the body's fight-or-flight response) (DermNet, 2023), and dopamine are catecholamines (of which dopamine is the simplest form but makes up 80% of the catecholamine content within the brain as it plays a major role in the motivational component of reward-motivated behavior) (Berridge, 2007). Serotonin on the other hand, is produced mainly in the brainstem's raphe nuclei in the central nervous system (CNS) where 90% of that produced is located within the GI tract's enterochromaffin cells (a type of enteroendocrine and neuroendocrine cells), where it regulates intestinal motility. It's also stored in blood platelets. (CalTech, 2015), (King, 2009), (Berger, 2009) These monoamines are in charge of the sympathetic response to stress, the metabolic correlates of aggression and fear, and the motivational system's rewarding mechanisms.

Pharmacological stimulants, in particular, can enhance the synaptic level of stress monoamines, promoting their release, limiting re-uptake by brain cells, and increasing their ability to excite receptors. The persistent and extensive stimulation of the stress response caused by these pharmaceutical drugs may result in a depletion of

natural stimulants and a reduction in the sympathetic response (Ghodse, 2010 ; Nestler 2012).

Stimulant use poses a significant public health concern, with consequent implications regarding cognitive and neurophysiological functioning. Impulsivity, a core feature of addictive behaviors, is known to play a crucial role in development and maintenance of stimulant use disorders.

Stimulant use and stimulant use disorder are linked to a variety of health and social consequences, including mental and cardiovascular morbidity, infectious disease transmission (including HIV and hepatitis C), drug-related crime, suicides, and homelessness. Opioid and CNS stimulant use may also lead to the development of serotonin syndrome (a group of symptoms caused by the use of two or more serotonergic medications or drugs with symptoms ranging from mild to severe, and are potentially fatal) (Volpie-Abadie, 2013). Over the last decade, the global incidence of stimulant usage has increased dramatically, with an alarming surge in the use of amphetamine-type stimulants in several jurisdictions (Ronsley et al., 2020).

2.1.1. Overview of stimulant types

Stimulants are a type of psychoactive stimulant that increases central nervous system activity, resulting in increased alertness, arousal, and improved cognitive performance. These medications primarily influence neurotransmitters like dopamine and norepinephrine (which has important systemic circulatory effects such as cardiac contractility and vasoconstriction). Stimulants are classified into several kinds based on their origin and pharmacological effects:

a. Amphetamines

Amphetamines, such as Adderall and Dexedrine, are synthetic stimulants that increase dopamine and norepinephrine release. They are frequently used to treat diseases such as attention deficit/hyperactivity disorder (ADHD) and narcolepsy (Smith, 2018).

b. Methylphenidate

Methylphenidate-based drugs, such as Ritalin and Concerta, act by preventing dopamine and norepinephrine reuptake and so boosting their levels in the brain.

Because they can increase dopaminergic transmission activity, methylphenidate can be used in late-life, treatment–resistant unipolar major depression. This class is also used to treat attention deficit hyperactivity disorder (ADHD) (Johnson & Miller, 2020).

c. Cocaine

Cocaine is a natural stimulant derived from the coca plant that blocks dopamine reuptake. It has been linked to increased mood and vitality, but it also has a significant risk of abuse and addiction (National Institute on Drug Abuse, 2021). Cocaine is used clinically as a topical anesthetic (particularly in ENT settings (oral/nasal)).

d. Nicotine

Nicotine, which is contained in tobacco, stimulates nicotinic acetylcholine receptors, which contributes to enhanced alertness. Nicotine intake is generally associated with smoking, however it is available in a variety of delivery modalities to help people quit smoking (World Health Organization, 2019). It can increase blood pressure and heart rate.

e. Caffeine

Caffeine, found in coffee, tea and various cola drinks, promotes wakefulness by inhibiting adenosine receptors. While caffeine is generally regarded safe in moderation, excessive consumption might have negative consequences such as increased blood pressure and heart rate (World Health Organization, 2019).

f. Ephedrine

Ephedrine, a naturally occurring stimulant, promotes norepinephrine release and is used in asthma and nasal congestion treatments. Because of the potential for misuse, regulatory procedures are in place (Smith, 2018).

2.1.3 Type of stimulants and prevalence:

Stimulant Types and Prevalence Stimulants include both prescription drugs like methylphenidate and amphetamines and illegal narcotics like cocaine and methamphetamine. Prescription stimulants are routinely used for attention deficit / hyperactivity disorder (ADHD) (Johnston et al., 2021). In addition, a new non-stimulant

medication (clonidine hydrochloride) has recently been approved by the US Food and Drug Administration (FDA) for the treatment of ADHD.

2.2. Impulsivity

2.2.1. Definition of Impulsivity

The personality psychologists J.P. and Ruth Guilford first used impulsivity in psychological theory in their work. They described rhathymia as "freedom from care or concern; a lack of serious-mindedness and an impulsiveness" and listed it as a key personality feature.

Impulsivity has been studied from a variety of perspectives, with resultant definitions thus having been incorporated. Moeller and colleagues approach impulsivity through a bio-psycho-social lens, integrating several cognitive-social elements and its characteristic features. They contend that each of the following elements ought to be included in a thorough description of impulsivity:

(1): reduced awareness of the detrimental effects of one's actions.

(2): spontaneous, instinctive response to stimuli before fully digesting the information.

(3): disregard for a behavior's long-term effects (Moeller at all,2001).

Impulsivity is a multidimensional construct, incorporating state and trait classifications, where there is a predisposition of impulsivity towards maladaptive, risky behavior relative to normal behavioral responses.

Impulsivity can be conceptualized as a failure of the inhibitory process, given its involvement in the frontostriatal circuitry, which in turn leads to dysfunction of top-down cognitive control. Behavioral (motor) impulsivity is associated with the inability

to inhibit impulsive actions, which is linked to dorsolateral prefrontal lobe activity that is comparable to response inhibition.

Impulsivity is defined by the DSM-5 as dysfunctional decision-making that includes hazardous conduct and a sense of urgency in emotionally charged settings.

According to Eysenck's characterization theory, impulsivity is defined by rash decisions and unplanned, risky acts. According to Dickman, dysfunctional impulsivity is typified by acting without giving it as much thought as most people with an equivalent degree of expertise and understanding. Subsequently, Dickman identified another factor known as the inhibitory component, which shows up as insufficient attention and is a contributing factor to impulsivity (Bakhshani, 2014). Barratt defined three distinct aspects of impulse: Factors that are non-planning (a reduction in future orientation), cognitive (rapid cognitive decision making), and motor (activity without thought) (Barratt,1994).

2.2.2. The neurobiological basis of impulsivity

Impulsivity, characterized by rash actions and decisions with disregard for potential consequences, is a complex behavior with roots in both neurobiology and environmental factors. While the precise mechanisms remain under investigation, growing evidence suggests abnormalities in specific brain regions where neurotransmitter systems play a significant role.

Three factors arise when the strong association and complex relationship between impulsivity and a substance use disorder (SUD) are considered:

1). the trait effect of impulsivity, centering on decreased cognitive and response inhibitions;

2). the state effect resulting from either acute or chronic substance use on brain structure and function; and

3). the genetic and environmental factors (such as age and sex) may influence impulsive behavior associated with SUDs.

Impulsivity is a marker associated with substance use disorders (SUDs) and is characterized by an imbalance of bottom-up and top-down neural systems. The brain utilizes the cognitive process of attention (which affects the mean neuronal firing rate as

well as its variability and correlation across neurons) to direct neural resources in accordance with the situational contingencies of the moment. This can be categorized into two functions: bottom-up and top-down attention. The bottom-up attention refers to attentional guidance purely on externally driven factors to stimuli that are salient due to their inherent properties relative to the background, whereas the top-down attention refers to internal guidance of attention based on prior knowledge, willful plans, and current goals (the frontoparietal network (a large-scale brain network primarily composed of the dorsolateral prefrontal cortex and the posterior parietal cortex, around the intraparietal sulcus, and is involved in sustained attention, complex problem-solving and working memory) is essential in both of these types of attentional processes). Neurotransmitter systems and brain circuits involved in impulsivity and addiction risks includes three neurobiological systems:

1). the regulatory system mediated by the medial and ventral prefrontal cortices;

2). the reward system via the ventral striatum and midbrain dopaminergic system; and

3). the threat system via the amygdala.

With regards to impulsivity, urgency (such as the tendency of responding to negative emotions irrationally resulting in problematic outcomes) has been associated with excessive recruitment of lateral prefrontal cortex (PFC) activity resulting in self-regulatory failure (such as substance misuse). The absence of forethought is linked to a reduction in the volume of grey matter in specific regions, including the insula and putamen, which are associated with decision-making efficacy. A lack of persistence or conscientiousness has been linked to a reduction in the function of the anterior cingulate cortex (ACC), as well as the left ventrolateral and left anterior prefrontal cortices, which are associated with risky behaviour. Sensation-seeking is associated with the activation of regions implicated in motivation, arousal, and reinforcement. Inadequate control, powerful reward, and poor harm-avoidance signals may all contribute to substance abuse, resulting in an imbalance between the prefrontal cortex (PFC) top-down cognitive control systems and the subcortical bottom-up incentive-reward system, which may lead to dangerous behaviors (e.g. drug experimentation).

As previously mentioned, the control/regulatory, reward, and threat systems, which are mediated by the medial and ventral prefrontal cortex, the ventral striatum and midbrain dopaminergic system, and the amygdala, respectively, form an overlapping pathway that connects brain circuitries and neurotransmitter systems associated with impulsivity and addiction risk.

Elevated glutamate levels due to a synaptic-nonsynaptic imbalance are related with dysregulation between the PFC and the nucleus accumbens (NAcc), which is observed in drug dependence. Impulsivity is linked to lower levels of gamma-aminobutryic acid (GABA), an inhibitory transmitter, in the dorsolateral prefrontal cortex.

A fundamental predisposition towards addictive and impulsive behaviors is underpinned by a cluster of genes that enhance emotional well-being by releasing dopamine (DA) from NAcc neurons via neurotransmitter interactions within the mesolimbic system. Furthermore, the reward cascade includes serotonin release, which stimulates encephalin (a pentapeptide that regulates pain feeling (nociception)) in the hypothalamus, as well as GABA inhibition in the substantia nigra, which fine-tunes the release of DA from the NAcc. Genetic variations that induce dysfunction in the brain reward cascade may result in a hypo-dopaminergic drive, which is reflected in increased impulsivity and, as a result, increased drug-seeking behavior. Norepinephrine has been associated with impulsive behaviors and addictions that mediate the effects of stimulants, such as drug-seeking behavior. Serotonin levels, like glutamate and GABA, have been connected to substance use disorders, with low levels of serotonin transmission being linked to addictions and impulsive decisions.

A biological predisposition to impulsivity and substance use disorders (SUD) is associated with dysregulated frontal control over corticolimbic circuitry. This is contributed by dopaminergic projections from the ventral tegmental area (VTA) to the nucleus accumbens (NAc), as well as serotonergic, GABAergic, and glutamatergic processes. The recognition of excessive impulsive behavior may facilitate the prediction of the onset and progression of SUD, thus representing an efficacious approach to therapy.

There are two related personality traits are associated with impulsive behaviour, including substance abuse. These traits, positive and negative urgency, refer to the disposition to act rashly when experiencing extremely powerful positive or negative emotions. Impulsive activity, including drug usage, can be triggered by intense emotions. Extreme intensive negative and positive emotions can undermine rational decision making by interfering with the patient's orientation toward the pursuit of their long-term goals and increasing focus on their short-term needs.

The experience of intense positive emotions has been demonstrated to increase distractibility and engender an undue sense of optimism regarding the potential positive outcomes of various situations amongst patients. Given the considerable inter-individual variability in the extent to which these phenomena occur, they can serve as highly significant predictors of the onset and progression of substance use disorders and other addictive behaviours.

Negative urgency (the tendency to respond impulsively when upset in the setting of negative affect) (Cyders and Smith, 2008; Whiteside and Lynam, 2001) is a powerful transdiagnostic risk factor for maladaptive behaviors frequently seen clinically by patients of both psychiatrists and psychologists (Dir et al., 2013). Examples of these types of behaviors are intimate partner violence along with reactive aggression (Derefinko et al., 2011), prolonged excessive abuse of narcotic substances (Fischer et al., 2012; Kaiser et al., 2012), and high risk deviant sexual practices (Deckman and Nathan DeWall, 2011; Derefinko et al.). Furthermore, the literature indicates that the phenomenon of self-injury, including self-injury of a non-suicidal nature (Bresin et al., 2012; Peterson et al., 2014), behavior demonstrating actual suicidal intent (Anestis and Joiner, 2011), and excessive eating (binge type) with or without accompanying voluntary purging (Fischer et al., 2013; Wenzel et al., 2014), may also be associated with the phenomenon of reactive aggression.

The perception of urgency is a significant contributing factor to the likelihood of engaging in risk-taking behaviors that may have adverse consequences. While sensation seeking is associated with the frequency of substance abuse, negative urgency is a more significant predictor of problematic alcohol consumption, particularly ethanol (ETOH). Negative urgency is the strongest (predominant) impulsivity-related predictor of problematic ETOH consumption and bulimic symptomatology.

Regarding the two urgency features, high neuroticism, low conscientiousness, and low agreeableness are the characteristics of negative and positive urgency. These two traits are related and are facets of an overall urgency domain. There is little variance which they share regarding other impulsivity-related traits (i.e. lack of perseverance, sensation seeking, lack of planning, fun seeking, reward responsiveness, and drive).

The perception of urgency can influence a patient's psychosocial learning about risk-taking behaviours, potentially leading to an increased propensity for engaging in risky activities. The theory of person-environment transaction, which is based on the idea that people are differently prepared to acquire certain learning experiences depending on their personalities, is extended in the Acquired Preparedness model of risk (AP Model; Smith and Anderson, 2001). According to this model, urgency increases the likelihood of addictive behaviors because it biases learned associations for risk-taking, which over time increases the likelihood of taking risks.

Negative urgency is generally connected with negativity sensitivity (neuroticism). Emotion-related impulsivity may be promoted by activation of the sympathetic nervous system (SNS), whereas tonic SNS activity may moderate the association between neuroticism and negative urgency. A minor relationship exists between impulsive behavior which is emotion-related and negative affect that is mediated by the sympathetic branch of the autonomic nervous system (ANS). The pathophysiology of emotion-driven impulsivity appears to be facilitated by elevated SNS activation. A decrease in executive cognitive function in response to stress is associated with the activation of beta-adrenergic receptor sites by the SNS, which, in addition, yields an increased reactivity of the amygdala (Hurlemann et al., 2010; van Stegeren et al., 2005), (Ramos and Arnsten, 2007). Since SNS activity is decreased by beta-adrenergic receptor inhibition, it can also lower the sensitivity of the amygdala to emotional stimuli (Hurlemann et al., 2010) and, during stressful events, modify executive cognitive functioning in a positive manner (Alexander et al., 2007; Roozendaal et al., 2004). Beta-adrenergic blockade, at the behavioral level, has been demonstrated to reduce aggressiveness. (Goedhard et al., 2006; Haspel, 2009).

Higher levels of SNS arousal have been linked to conditions such as emotional impulsivity. Emotional impulsivity is a clinical feature that can alter SNS activity. Patients with borderline personality disorder (BPD) have higher tonic sympathetic

activity than control groups (Ebner-Priemer et al., 2007; Kuo and Linehan, 2009; Weinberg et al., 2009). BPD patients also demonstrate extremely elevated levels of neuroticism (Mullins-Sweatt et al., 2012) as well as increased impulsive behaviour in a reactive response to negative emotions (Chapman et al., 2010; Peters et al., 2013). Therefore, individuals with higher levels of tonic SNS activity and a predisposition to negative affect may be more likely to act impulsively when they're upset or anxious. Clinical groups with non-affective impulsivity, such as ADHD, conduct disorder and antisocial personality disorder, show lower levels of tonic SNS activity and reduced SNS reactivity (Beauchaine et al., 2013; Lorber, 2004).

‘’From a biological and neuropsychological perspective, impulsivity is characterized by failure in inhibiting a potentially risky impulse for the individual or the others around. From a cognitive viewpoint, impulsivity is the inability to inhibit behavioral impulses and thoughts. It considers impulse control as an important component of executive functions. It plays an important role in one's social and personal functioning

The factor analysis of Barratt impulsivity scale indicates three factors: increased motor activity, decreased attention, and decreased planning. Decreased attention and planning are the main factors of impulsivity. The psychiatric disorders that come along impulsivity are probably related to different patterns of these basic mechanisms. For instance, frontal lobe lesions that impair attention and planning lead into the emergence of personality disorder symptoms, and a high level of motor activity is observed in mania (Bakhshani, 2014)’’.

In clinical and scientific settings associated with dangerous behaviors and certain mental diseases, the concept of impulsivity is crucial. Although impulsivity is listed as a diagnostic criterion for a number of diseases in the DSM and ICD (1), it is unclear how impulsivity affects mental health issues and the emergence of risky behaviors. Risk factors for impulsivity can include:

1. Age (younger patients rather than older ones)
2. Family history
3. Trauma (i.e. violence, abuse or neglect)
4. Substance abuse

Some possible causes for impulsivity include psychological factors (i.e. parental upbringing) and genetic factors (i.e. mutations affecting dopamine and serotonin production impacting mood and cognition).

Underlying conditions that may be associated with impulsivity can include physical (i.e. neurodegenerative disorders) or mental (i.e. bipolar mania, impulse control disorders (kleptomania, pyromania), antisocial personality disorders, ADHD).

There are treatment regimens for this disorder which include psychotherapy (i.e. Dialectical Behavioral Therapy (DBT) and associated mindfulness meditation) and conjunctive medication (i.e. selective serotonin reuptake inhibitors (SSRIs) mood stabilizers, atypical antipsychotics).

Forming a complete theory addressing the development of impulsivity and its role in psychopathology is at present very difficult given the current nature of ongoing professional disagreements over the definition, components, and methods of measuring impulsiveness. However, it must be understood that impulsivity is a characteristic of other conditions such as ADHD, bipolar disorder or borderline personality disorder (BPD) (which are characterized as hallmarks in DSM-5). Presentation depends on the patient's diagnosis and condition severity. There is no single test to confirm whether impulsive behavior is linked to a specific mental disorder, rather a variety of psychological testing must be performed to satisfy the suspected DSM-5 diagnostic.

2.2.3 EEG overview

Although Dr. Richard Caton in England was the first to describe the electrical phenomena of the exposed cerebral hemispheres of rabbits and monkeys in 1875, it wasn't until 1924 when Dr. Hans Berger invented and recorded the first human electroencephalograph (EEG) (L F et al, 2003). Since the 1930's it's been incorporated in the study for numerous biomedical purposes.

When EEG signals are obtained they must be evaluated. This EEG analysis extracts information from EEG signals using a combination of mathematical signal analysis approaches (time domain, frequency domain, time-frequency domain, and nonlinear) and computer technology (Rajerdra Acharya, Vinitha, Swapna, Joy Martis, & Suri, 2013).

The EEG offers millisecond-range temporal resolution, which is not possible with other high-resolution anatomical imaging techniques (CT, PET, MRI) (Bakhshani, 2014; Barrat,1994). Signals are primarily generated by cortical pyramidal neurons, which send postsynaptic potentials to the apical dendrites perpendicular to the cortical

surface. Neurons are polarized (electrically charged) by membrane transport proteins that pump ions across their membranes. Neurons continuously exchange ions with the extracellular environment (e.g. maintaining resting potentials and propagating action potentials). Ions of similar charge repel each other in a forward wave (volume conduction). When the waves of ions reaches the electrodes on the scalp, they can push or pull on the metal on the electrodes. Since metal easily conducts the push or pull of electrons, a voltmeter can measure the difference in push or pull voltages between any two electrodes. Thus recording these voltages over time produces the EEG. (Tatum WO, et al (2008)) Scalp EEG activity yields oscillations at a variety of frequencies. EEG signals can be transformed into a topographic map associated with respective cortical activity. The topographic images thus generated can provide a guide in epileptic patients to remove epileptogenic zones during neurosurgery. It is possible for there to be irregular variations in the graphic waves, which may result in alterations to the net change between inhibitory and excitatory postsynaptic potentials in a manner that is dependent on both time and space.

There are different wave patterns that can be exhibited with the EEG. These include delta waves, theta waves, alpha waves, beta waves, gamma waves, and mu waves. Of the wave patterns the delta waves (frequeny range up to 4 Hz) are generally the highest in amplitude and the slowest waves. It's normally seen frontally in adults in slow-wave sleep as well as posteriorly in infants. It can also be seen in some neuropathologies (e.g. subcortical lesions, diffuse lesions, or deep midline lesions) (Kirmizi-Alsan et al.,2006). Theta waves (frequency range 4-7 Hz) are seen in young children. It can also occur in older children and adults experiencing drowsiness, meditation or arousal (Cahn BR, et al, 2006). As with delta waves, theta waves can also be seen in various neuropathologies (e.g. metabolic encephalopathy, sometimes with hydrocephalus). Alpha waves (frequency range 8-12 Hz) are seen in the posterior regions of the head bilaterally, with the dominant side producing a higher amplitude. They are observed with closing the eyes, relaxed/reflective states, and are associated with inhibition control. An abnormal presentation of diffuse alpha waves is seen in patients who are in a coma. Beta waves (frequency range 13-30 Hz) are mainly seen frontally and bilaterally in symetrical distribution. It's seen in low amplitude in active thinking, being anxious, being focused, whereas it's attenuated in active motor behavior and movements. It can be associated pathologically with the effects of benzodiazepines

and in Dup15q syndrome (the most common genetic cause of autism caused by the partial duplication of the proximal long arm of chromosome (Hao et al.,2023). This variant also confers a strong risk for the development of epilepsy) (Frochlich et all., 2016). Gamma waves (frequency 30-100 Hz) are seen in the somatosensory cortex. It's shown during cross-modal sensory processing of perceptions that combine two different senses (i.e. auditory and visual), as well as during short-term memory matching of recognized objects, sounds, or tactile sensations. A decrease in gamma activity may be pathologic when associated with cognitive decline. Patients with chronic nicotine use can present with an increase in gamma activity while patients with chronic cannabis use can present with a decrease in gamma activity. Mu waves (frequency 8-13 Hz) display the synchronous firing of motor neurons in rest state. A deficit in mu wave suppression may be associated with autism.

EEG is used to study opiod use disorders (OUD), methamphetamine use disorders (MUD), and alcohol use disorders (AUD), sometimes collectively referred to as substance use disorders (SUD). Gamma-aminobutyric acid (GABA) can be altered by opioid abuse in that postsynaptic excitatory potentials, such as cortical pyramidal neurons, may become less sensitive to GABAergic inhibitory control (Baldo et al., 2016). This may result in a change in the electrical synchronisation between cortical neurons. An examination of delta, theta, alpha, beta, and gamma waves revealed that all five spectral powers were elevated with near equipotency in the frontal, central, temporal, parietal, and occipital subregions of OUD patients. Additionally, selective elevation was observed in patients with MUD and AUD. Prolonged methamphetamine exposure can cause a decrease in cerebral dopamine transporters and that only the delta and theta bands were elevated nearly globally in the cortical subregions (Newton et al, 2003). Alcohol is inhibitory, paralelly resembling GABA's effect on postsynaptic GABA receptors. In AUD patients the gamma powers bands were elevated across their cerebral cortex.

It should be noted that unlike positron emission tomography (PET) and nuclear magnetic resonance spectroscopy (MRS), the EEG can not identify exact specific locations in the brain at which various neurotransmitters, drugs, etc. can be found. Since the EEG can be altered by drugs that affect brain functions the science of pharmaco-

electroencephalography has developed methods to identify substances that systematically alter brain functions for therapeutic and recreational use.

Though EEG's have been used extensively to estimate mental health, there doesn't appear to be a general acceptance/consensus on spectral powers altered in SUD patients.

3. METARIAL AND METHOD

3.1. Type of Research

The research is designed as an observational – descriptive type of study.

3.2. Project Goals

The goal of this thesis is to examine patients who abuse narcotic stimulants and to show an expected increased cerebral impulsivity. We can expect their electroencephalographic (EEG) tracings to exhibit an increase in neuronal firing of Delta and Beta wave forms within the frontal/medial prefrontal cortex region. This group will be concurrently compared to a control group. The study will investigate the relationship between stimulant usage and the corresponding impulsivity, as well as the associated impulsivity and its specific EEG findings in that frontal region of the cerebrum. The participants will be asked to complete the Barrat Impulsivity Scale and undergo a separate resting-state EEG. This study will thus explore the degree of impulsivity based on their stimulant usage and their corresponding resting-state EEG activity in the expected region of the brain.

3.3. Hypotheses of the Study

1. My initial hypothesis was that there would be among SUD patients an across the board increase in EEG wave form activity in that there would be an expected decreased activity of theta and beta firings and revealing an underlying increase in impulsive behavior.
2. Given the EEG trace results that I would initially have expected, to then find a corresponding correlation utilizing the Barratt Impulsiveness Scale (BIS) showing an abnormal result where there would be a negative relationship between the EEG and Barratt, specifically in delta frequency and increased levels of impulsivity.
3. When compared to the control group, the Delta and Beta activity can be expected to be more prominent.

3.4. Assumptions of the Study

1. It is assumed that participants provided sincere answers to the questions in the assessment tools used in the study.
2. It is assumed that the selected assessment tools, sample, and research techniques are appropriate for the purpose, subject, and problem identification of this study.

3.5. Place and Time of Research

This research was conducted at NP Istanbul Brain Hospital from September 2023 to February 2024. The necessary permissions were obtained from the Üsküdar University Ethics Committee and the hospital administration.

3.6. Population and Sample

The research sample included 72 participants (patient [N =36], Health control [N= 36]) who had received or were receiving treatment at NP Istanbul Brain Hospital for stimulant use. Of the 72 participants in the study (36 patients and 36 control) there was an attrition of two from the control group when their EEG tracings were found to be defective, thus decreasing the participants in the control group to 34.

3.6.1 Inclusion criteria for the research studies

To be between the ages of 18 and 60 and be able to read and understand scales. Inclusion criteria are patients who are right-handed, those who have not previously participated and agree to the study, and patients who are not under the influence of alcohol.

3.6.2. Exclusion criteria for the research studies

Exclusion criteria for the study would be patients who experienced head trauma, patients with schizophrenia, patients with bipolar disorders, and patients with epilepsy.

3.7. Data Collection Tools

Sociodemographic Data Form: The sociodemographic data form was utilized to assess the participants' age, marital status, degree of education, existence of a mental problem in their medical history, and any extra general medical conditions.

Barratt Impulsiveness Scale: The Barratt Impulsiveness Scale (BIS) is a psychological measure that is used for the purpose of assessing the personality and behavioural

construct of impulsiveness. The BIS was developed by Dr Ernest Barratt and has undergone several revisions, with the BIS-11 being the most widely used version. The scale comprises 30 items, which are rated on a 4-point Likert scale, ranging from "Rarely/Never" to "Almost Always/Always". The BIS-11 assesses impulsivity along three principal dimensions: The three primary factors are attentional impulsiveness, motor impulsiveness, and non-planning impulsiveness. The total score may range from 30 to 120. A higher score on the BIS is indicative of a higher level of impulsivity**.** (Lau et. all., 2022).

EEG resting State: QEEG data was collected from a total of 72 participants (36 patients and 36 control group) using a 19-electrode cap and measures for absolute, relative power, and coherence will calculate or each of the 4 frequency bands. The EEG recordings were taken while participants were in a wakeful-resting condition with their eyes closed, and technicians monitored them to prevent drowsiness (Ergüzel et. all., 2019).

We established a sampling rate of aquisition of 125 Hz, band-pass filtering the aquired signals at a frequency between 0.15 Hz-70 Hz (utilizing a 50 Hz notch filter), while we manually eliminated (off-line) any detected segmental data artifacts (e.g. muscle or eye movement, electrode popping) for each and every patient, by inspecting and thus removing and consequently excluding them from cumulative data analysis. We incorporated a Fast Fourier Transform (FFT) analysis by taking the average across 2-second epochs. We ultimately derived our computed absolute power for the wave bands delta (1-4 Hz), theta (4-8 Hz), alpha (8-12 Hz), and beta (12-25 Hz). The software we utilized for the qEEG analysis study was NeuroGuide Delux v.2.5.1 (Applied Neuroscience, Largo, FL).

3.8. Data Analysis

The study's data was analyzed using the SPSS 25.0 package application. Descriptive statistics on the subjects' sociodemographic information were presented as frequency tables.

The data from the study were examined in order to evaluate the assumptions of normality. The Kolmogorov-Smirnov values were determined to be less than 0.05, indicating that the data did not meet the criteria for normality. Consequently, a Spearman correlation analysis, a non-parametric test, was conducted to determine the relationship between the scale/survey scores. Additionally, Mann-Whitney U test and Kruskal-Wallis H test, non-parametric tests, were applied to determine whether there was a significant difference between the participants' sociodemographic data and the scale/test scores. In the case of a significant difference between groups, post-hoc tests were performed to determine between which groups the significance existed. Due to the non-homogeneous distribution of variance and the unequal sample sizes, the Games-Howell Post-Hoc test was selected (Sparks, 1963). A p-value of <0.05 was considered statistically significant**.**

4. RESULT

Table 1: Sociodemographic characteristics of the participants

Demographic Variables		**N**	**%**
Age	20-32 Years	16	47,1
	33-50 Years	18	52,9
Marital Status	Single	18	52,9
	Married	14	41,3
	Divorced	1	2,9
	Widowed	1	2,9
Education Level	Primary School	2	5,9
	High School	12	35,3
	University	20	58,8
Smoking	No	31	91,2
	Yes	3	8,8
Alcohol Use	No	33	97,1
	Yes	1	2,9

Tablo 1 shows the distribution of participants' social demographic variables.

Table 2: Comparison of participants' scale and subscale scores by age

Variables	Age	N	Mean±SS	Z	P
Barrat Impulsiveness Score	20-32	16	44,70±13,30	-0,864	0.387
	33-50	18	43,45±12,07		
Attentional Impulsiveness Score	20-32	16	17,49±6,59	-0,765	0.444
	33-50	18	16,98±5,81		
Non-planning Impulsiveness Score	20-32	16	17,17±5,39	-0,070	0.944
	33-50	18	17,35±5,05		
Motor Impulsiveness Score	20-32	16	10,04±2,79	-0,087	0.931
	33-50	18	9,12±2,77		

Mann Whitney U Test, p<0.05

As Table 2. shows, no statistically significant differences were observed between scale/subscales and age.

Table 3: Comparison of Participants' Scale and Subscale Scores by Marital Status

Variables	Marital Status	N	Mean±SS	KV	p	Post-Hoc
Barratt Impulsiveness Scale Total Score	(1) Single	18	70,83±10,9	4,432	0.218	-
	(2) Married	14	68,43±6,37			
	(3) Divorced	1	120±0			
	(4) Widowed	1	76±0			
Attentional Impulsiveness Score	(1) Single	18	17,61±5,09	8,162	**0.043**	-
	(2)Married	14	14,57±4,80			
	(3)Divorced	1	28±0			
	(4)Widowed	1	21±0			
Non-planning Impulsiveness Score	(1) Single	18	37,44±8,43	4,352	0.226	-
	(2) Married	14	35,21±6,49			
	(3) Divorced	1	56±0			
	(4) Widowed	1	35±0			
Motor Impulsiveness Score	(1) Single	18	26,50±9,28	4,167	0.244	-
	(2) Married	14	23,71±3,52			
	(3) Divorced	1	36±0			
	(4) Widowed	1	20±0			

Kruskal-Wallis H, p<0.05

As shown in Table 3, participants' scale and subscale scores were compared with their marital status. A statistically significant difference was observed between marital status and the "Attentional Impulsiveness" subscale score of the Barratt Impulsiveness Scale. The "Attentional Impulsiveness" score was found to be higher in single participants compared to married participants (KW=8.162, p=0.043).

Table 4: Comparison of Participants' Scale and Subscale Scores by Education Level

Variables	Education Level	N	Mean±SS	KV	p	Post-Hoc
Barratt Impulsiveness Scale Total Score	(1)Elementary	2	69,50±6,36	1,730	0.421	-
	(2)High	12	67,25±9,64			
	(3)University	20	74,15±13,82			
Attentional Impulsiveness Score	(1)Elementary	2	14±2,83	2,896	0.235	-
	(2)High	12	15,17±5,29			
	(3)University	20	18±5,49			
Non-planning Impulsiveness Score	(1)Elemantary	2	33,50±2,12	0,754	0.686	-
	(2)High	12	35,42±8,13			
	(3)University	20	38,30±8,43			
Motor Impulsiveness Score	(1)Elementary	2	22±1,41	1,892	0.388	-
	(2)High	12	25,08±9,71			
	(3)University	20	26±6,32			

Kruskal-Wallis H, p<0.05

As seen in Table 6, no statistically significant differences were observed between the scale and subscales and education level.

Table 5: Comparison of Participants' Scale and Subscale Scores by Smoking Status

Variables	Smoking Status	N	Mean±SS	Z	P
Barratt Impulsiveness Scale Total Score	No	31	69,71±9,29	-1,552	0.121
	Yes	3	89,33±26,63		
Attentional Impulsiveness Score	No	31	16,48±5,27	-0,98	0.327
	Yes	3	19,67±7,23		
Non-planning Impulsiveness Score	No	31	36,68±7,66	-0,246	0.806
	Yes	3	40,33±13,65		
Motor Impulsiveness Score	No	31	24,1±5,44	-2,566	**0.01**
	Yes	3	39,33±12,34		

Mann Whitney U Test, p<0.05

Participants' scale and subscale scores were compared with their marital status, as presented in Table 5. A statistically significant difference was observed between the 'Motor Impulsivity' score of the Barrat Impulsivity Scale and smoking status. The 'Motor Impulsivity' score from the Barrat Impulsivity Scale was found to be higher in smokers compared to non-smokers (Z=-2.566, p=0.001

Table 6: Comparison of Participants' Scale and Subscale Scores in Terms of Alcohol Use

Variables	Alcohol Use	N	Ort±SS	Z	P
Barratt Impulsiveness Scale Total Score	No	33	71,88±12,3	-1,481	0.139
	Yes	1	57±0		
Attentional Impulsiveness Score	No	33	16,42±5,11	-1,542	0.123
	Yes	1	28±0		
Non-planning Impulsiveness Score	No	33	36,42±7,5	-1,546	0.122
	Yes	1	56±0		
Motor Impulsiveness Score	No	33	25,33±7,53	-1,128	0.259
	Yes	1	29±0		

Mann Whitney U Test, p<0.05

In Table 6, no statistically significant differences were observed between alcohol status and the scales/subscales.

Table 7: Descriptive statistics of scale and subscale scores and EEG wave frequency means (n=34)

	Min.	Max.	Mean	SD
Barratt Impulsiveness Scale Total Score	45,00	120,00	71,4412	12,38
Motor Impulsivity Score	8,00	28,00	16,7647	5,41
Attentional Impulsivity Score	20,00	56,00	37,0000	8,11
Non- planning Impulsivity Score	16,00	53,00	25,4412	7,44
DELTA_L	-1,84	2,86	0,2038	1,08
THETA_L	-8,67	2,39	0,1553	1,82
ALPHA_L	-0,97	1,62	0,1594	0,67
BETA_L	-1,82	1,97	-0,2968	0,81
DELTA_R	-1,25	2,25	0,2529	0,96
THETA_R	-1,30	2,46	0,4168	0,93
ALPHA_R	-0,94	1,63	0,1212	0,66
BETA_R	-1,63	1,37	-0,3351	0,76
DELTA_C	-2,22	2,31	0,1312	1,05
THETA_C	-1,75	2,72	0,3438	1,02
ALPHA_C	-0,95	2,22	0,1768	0,71
BETA_C	-2,64	2,34	-0,0709	1,01

Table 7 presents descriptive statistics for the scale and subscale scores.

Table 8: Correlation Results of the Relationships Between the Scale and Subscales

		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1.Barrat İmpulsiveness Scale Total Score	r	1,000															
	p																
2.Attentional Impulsivity Score	r	0,191	1,000														
	p	0.280															
3. Non-planning Impulsivity Score	r	0,127	0,100	1,000													
	p	0.474	0.575														
4.Motor Impulsivity Score	r	,419*	,454**	,446**	1,000												
	p	**0.014**	**0.007**	**0.008**													
5.DELTA L	r	-,457**	-0,106	0,159	-0,181	1,000											
	p	**0.007**	0.551	0.371	0.306												
6.THETA L	r	-0,180	-0,055	-0,004	-,397*	,618**	1,000										
	p	0.307	0.757	0.982	**0.020**	**<0.001**											
7. ALPHA L	r	0,035	-0,037	-0,120	-0,178	0,153	,426*	1,000									
	p	0.846	0.834	0.501	0.314	0.389	**0.012**										
8.BETA L	r	0,152	0,083	0,011	0,035	-0,001	0,202	,565**	1,000								
	p	0.391	0.642	0.949	0.845	0.994	0.253	**<0.001**									
9.DELTA R	r	-,442**	-0,028	0,109	-0,235	,851**	,572**	0,097	0,053	1,000							
	p	**0.009**	0.876	0.538	0.180	**<0.001**	**<0.001**	0.585	0.765								
10. THETA R	r	-0,192	0,001	-0,008	-,349*	,617**	,969**	,443**	0,192	,613**	1,000						
	p	0.277	0.998	0.965	**0.043**	**<0.001**	**<0.001**	**0.009**	0.277	**<0.001**							
11. ALPHA R	r	0,011	0,004	-0,140	-0,182	0,229	,520**	,965**	,527**	0,186	,563**	1,000					
	p	0.949	0.984	0.430	0.302	0.193	**0.002**	**<0.001**	**0.001**	0.291	**0.001**						
12. BETA R	r	0,113	0,201	0,033	0,105	0,033	0,217	,526**	,941**	0,088	0,240	,535**	1,000				
	p	0.525	0.253	0.854	0.555	0.855	0.218	**0.001**	**<0.001**	0.619	0.171	**0.001**					
13. DELTA C	r	-,431*	-0,144	0,184	-0,281	,838**	,522**	-0,004	0,096	,843**	,533**	0,073	0,156	1,000			
	p	**0.011**	0.418	0.298	0.107	**<0.001**	**0.002**	0.982	0.590	**<0.001**	**0.001**	0.681	0.377				
14.THETA C	r	-0,245	-0,117	0,039	-,438**	,546**	,855**	,339*	0,259	,537**	,847**	,439**	0,304	,645**	1,000		
	p	0.163	0.511	0.827	**0.010**	**0.001**	**<0.001**	**0.050**	0.139	**0.001**	**<0.001**	**0.009**	0.080	**<0.001**			
15. ALPHA C	r	0,036	-0,134	-0,125	-0,239	0,117	,389*	,917**	,531**	0,055	,388*	,907**	,491**	0,039	,448**	1,000	
	p	0.841	0.449	0.481	0.173	0.509	**0.023**	**<0.001**	**0.001**	0.757	**0.024**	**<0.001**	**0.003**	0.827	**0.008**		
16. BETA C	r	0,106	0,023	0,026	-0,080	-0,044	0,170	,368*	,880**	-0,008	0,167	,368*	,861**	0,154	,373*	,427*	1,000
	p	0.549	0.896	0.885	0.651	0.805	0.335	**0.032**	**<0.001**	0.964	0.346	**0.032**	**<0.001**	0.386	**0.030**	**0.012**	

In Table 8, the relationships between the scores obtained from the scale and subscales applied to the participants are shown using Spearman correlation analysis.

According to this analysis, a statistically significant positive relationship was observed between the Barratt Impulsiveness Scale Total score and the Motor Impulsiveness score, one of the Barratt Impulsiveness Scale subscales (r=0.419, p=0.014). A statistically significant negative relationship was observed between the Barratt Impulsiveness Scale Total score and the EEG wave frequencies averages: DELTA L (r=-0.457, p=0.007), DELTA R (r=-0.442, p=0.009), and DELTA C (r=-0.431, p=0.011).

A statistically significant positive relationship was observed between the Attentional score, one of the Barratt Impulsiveness Scale subscales, and the Motor Impulsiveness score, one of the Barratt Impulsiveness Scale subscales (r=0.454, p=0.007).

A statistically significant positive relationship was observed between the Non-planning score, one of the Barratt Impulsiveness Scale subscales, and the Motor Impulsiveness score, one of the Barratt Impulsiveness Scale subscales (r=0.446, p=0.008).

A statistically significant negative relationship was observed between the Motor Impulsiveness score, one of the Barratt Impulsiveness Scale subscales, and the EEG wave frequencies averages: THETA L (r=-0.397, p=0.020), THETA R (r=-0.349, p=0.043), and THETA C (r=-0.438, p=0.010).

A statistically significant positive relationship was observed between DELTA L and the following EEG wave frequencies averages: THETA L (r=0.618, p<0.001), DELTA R (r=0.851, p<0.001), THETA R (r=0.851, p<0.001), DELTA C (r=0.838, p<0.001), and THETA C (r=0.546, p=0.001).

A statistically significant positive relationship was observed between THETA L and the following EEG wave frequencies averages: ALPHA L (r=0.426, p=0.012), DELTA R (r=0.572, p<0.001), THETA R (r=0.969, p<0.001), ALPHA R (r=0.520, p=0.002), DELTA C (r=0.522, p=0.002), THETA C (r=0.855, p<0.001), and ALPHA C (r=0.389, p=0.023).

A statistically significant positive relationship was observed between ALPHA L and the following EEG wave frequencies averages: BETA L (r=0.565, p<0.001), THETA R (r=0.443, p=0.009), ALPHA R (r=0.965, p<0.001), BETA R (r=0.526, p=0.001),

THETA C (r=0.339, p=0.050), ALPHA C (r=0.917, p<0.001), and BETA C (r=0.368, p=0.032).

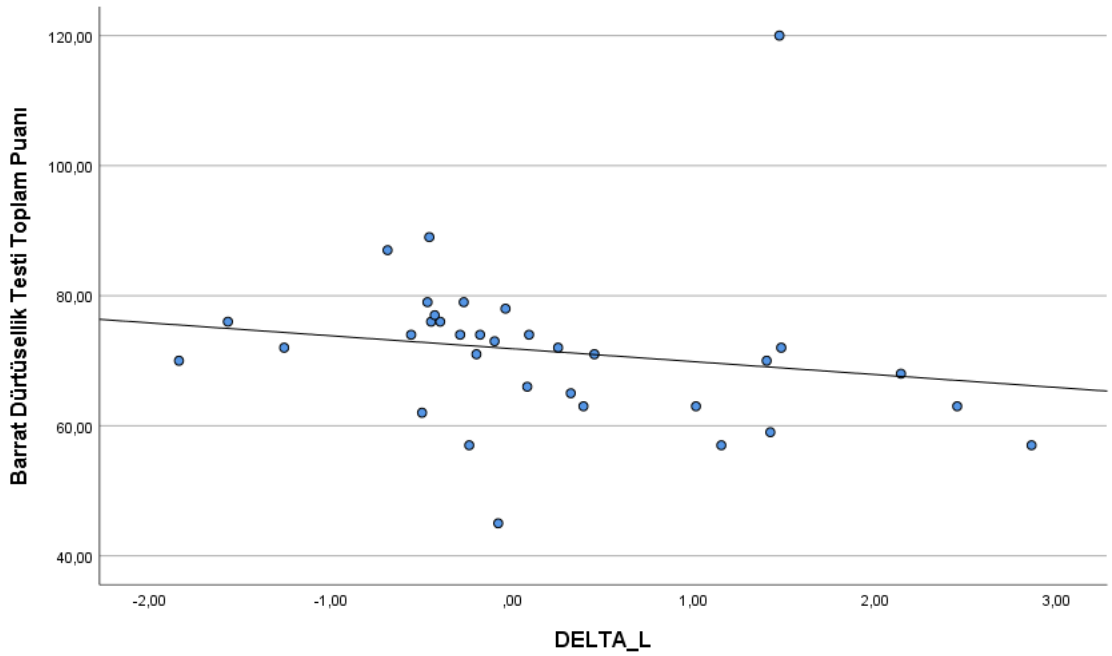
A statistically significant positive relationship was observed between BETA L and the following EEG wave frequencies averages: ALPHA R (r=0.527, p=0.001), BETA R (r=0.941, p<0.001), ALPHA C (r=0.531, p=0.001), and BETA C (r=0.880, p<0.001).

A statistically significant positive relationship was observed between DELTA R and the following EEG wave frequencies averages: THETA R (r=0.613, p<0.001), DELTA C (r=0.843, p<0.001), and THETA C (r=0.537, p<0.001).

A statistically significant positive relationship was observed between THETA R and the following EEG wave frequencies averages: ALPHA R (r=0.563, p=0.001), DELTA C (r=0.533, p=0.001), THETA C (r=0.847, p<0.001), and ALPHA C (r=0.388, p=0.024).

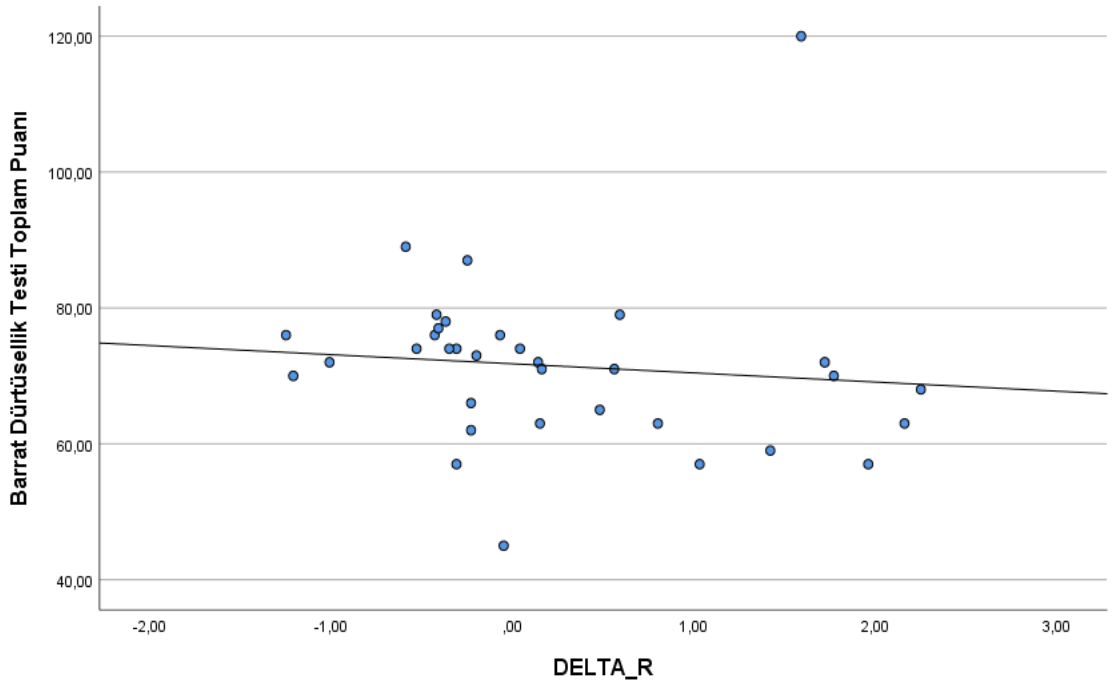
A statistically significant positive relationship was observed between ALPHA R and the following EEG wave frequencies averages: BETA R (r=0.535, p=0.001), THETA C (r=0.439, p=0.009), ALPHA C (r=0.907, p<0.001), and BETA C (r=0.368, p=0.032).

Figure 1: Data plot graph



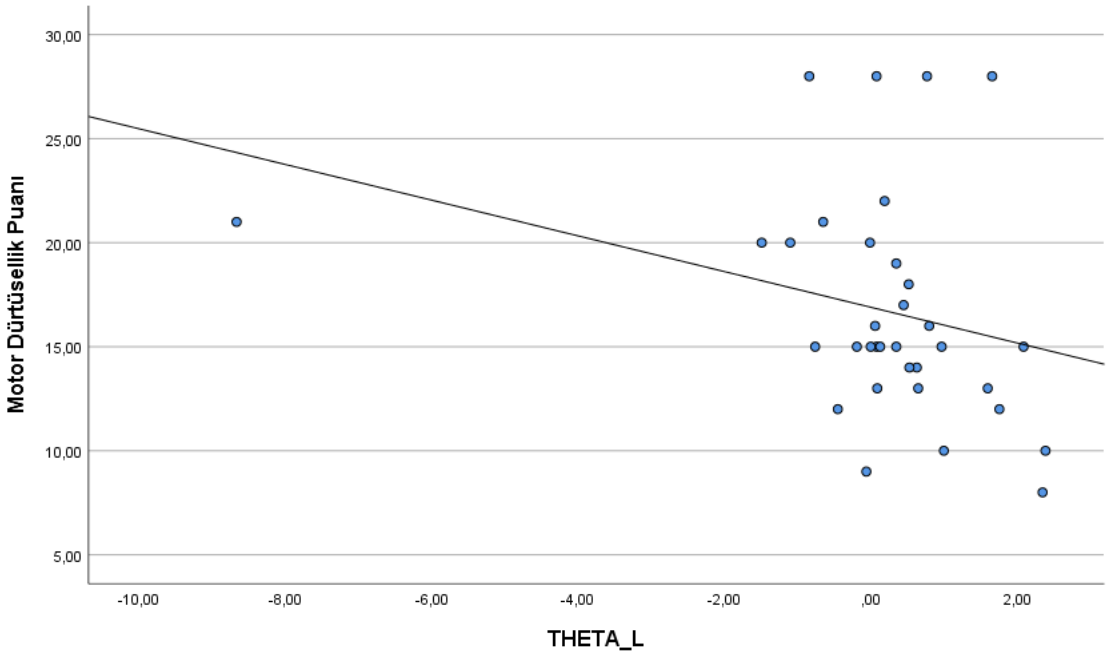
There is a noticeable negative relationship between DELTA_L and the Barratt Impulsiveness Scale Total Score, but this relationship is quite weak. The wide dispersion of data points indicates that the relationship between the two variables is not strong.

Figure 2: Data plot graph



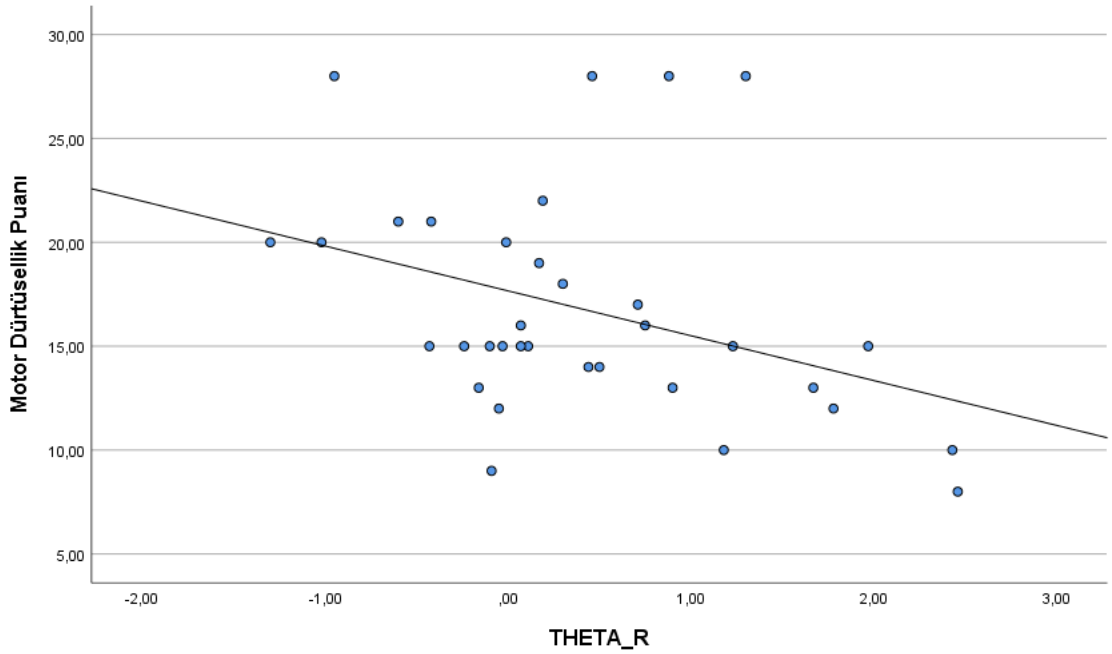
There is a noticeable negative relationship between DELTA_R and the Barratt Impulsiveness Scale Total Score, but this relationship is quite weak. The wide dispersion of data points indicates that the relationship between the two variables is not strong.

Figure 3: Data plot graph



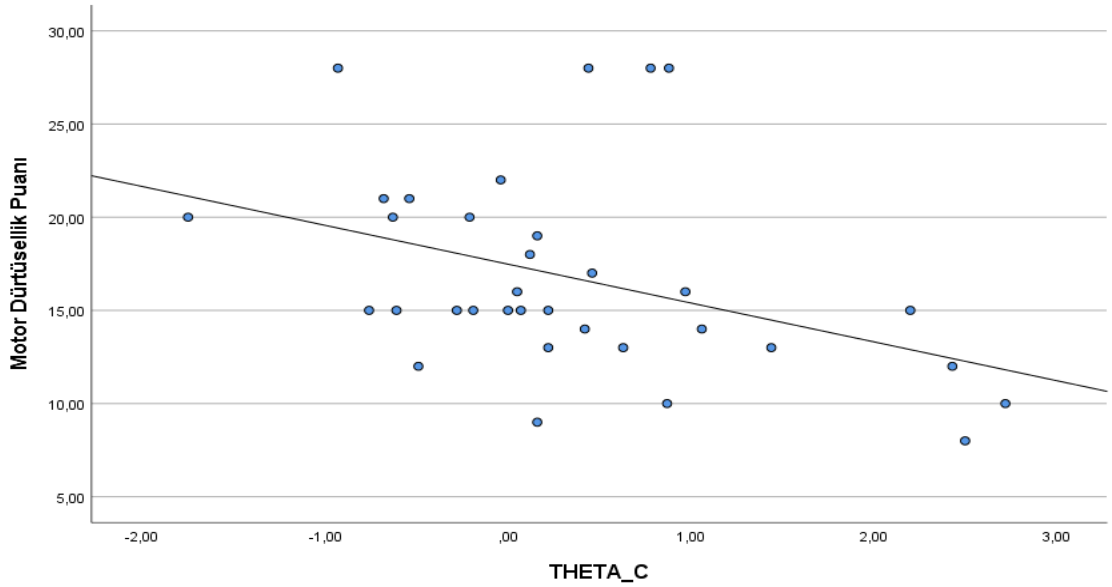
There is a noticeable negative relationship between THETA_L and the Motor Impulsiveness Score, but this relationship is quite weak. The wide dispersion of data points indicates that the relationship between the two variables is not strong.

Figure 4: Data plot graph



There is a noticeable negative relationship between THETA_R and the Motor Impulsiveness Score, but this relationship is quite weak. The wide dispersion of data points indicates that the relationship between the two variables is not strong.

Figure 5: Data plot graph



There is a noticeable negative relationship between THETA_C and the Motor Impulsiveness Score, but this relationship is quite weak. The wide dispersion of data points indicates that the relationship between the two variables is not strong.

Table 9: Comparison of the EEG Averages of Patient and Control Groups

Variables	Age	N	Ort±SS	Z	P
DELTA L	Patients	34	0,20±1,08	-1,601	0.109
	Controls	66	0,37±0,71		
THETA L	Patients	34	0,16±1,82	-0,284	0.777
	Controls	66	0,37±0,65		
ALPHA L	Patients	34	0,16±0,67	-0,666	0.506
	Controls	66	0,24±0,75		
BETA L	Patients	34	-0,30±0,81	-1,335	0.182
	Kontrol	66	-0,07±0,72		
DELTA R	Patients	34	0,25±0,96	-0,837	0.403
	Controls	66	0,31±0,77		
THETA R	Patients	34	0,42±0,93	-0,215	0.830
	Controls	66	0,31±0,69		
ALPHA R	Patients	34	0,12±0,66	-0,939	0.348
	Controls	66	0,23±0,75		
BETA R	Patients	34	-0,34±0,76	-1,212	0.226
	Controls	66	-0,15±0,69		
DELTA C	Patients	34	0,13±1,05	-0,044	0.965
	Controls	66	0,06±0,80		
THETA C	Patients	34	0,34±1,02	-0,746	0.456
	Controls	66	0,11±0,76		
ALPHA C	Patients	34	0,18±0,71	-0,211	0.833
	Controls	66	0,16±0,78		
BETA C	Patienst	34	-0,07±1,01	-0,953	0.340
	Controls	66	-0,22±0,82		

Mann Whitney U Test, p<0.05

As seen in Table 9, the comparison of the EEG averages of the patient and control groups did not reveal any statistically significant differences.

5. DISCUSSION

In this clinical study, the effects of impulsivity levels on EEG findings in patients using stimulants were examined. The findings of the study revealed that impulsivity has significant relationships with EEG wave frequencies. These relationships were particularly observed in decreased frequencies of Delta and Theta activities in the frontal region. In our study, the EEG brain waves of the patient group with measured Barratt Impulsiveness Scale scores were examined, and differences were observed in some subgroups of the brain waves.

When we examine Table 3, we find that there is a significant relationship between the total BIS score and the delta wave frequency, with a significant relationship being observed between the motor impulsive subscale and the theta wave frequency. There's no correlation between attentional non-planning impulsiveness and any wave activity. Since the BIS is the assessment tool used to measure the level of impulsivity in patients, we have found that the significant total BIS score and delta wave frequency suggest that a higher level of impulsivity may be exhibited more frequently in a clinical setting.

Our finding demonstrates that the relationship between THETA waves and motor impulsiveness score is weak, and we also see a similar weak relationship between Delta wave forms and BIS. These would be indicative of a negative correlative effect of cerebral neurobiological pathways.

The findings of increased beta and theta amplitude density across all scalp areas of patients with OUD suggests that there are distributed neuronal networks which are involved in problematic prescription opioid use. These indicate a possible dysfunction in distributed neural circuits, rather than local focal changes (Motlagh et al,2016; Fingelkurts et al., 2008). These findings correlate with those that can be seen with stimulant usage in generalized SUD patients.

The study of EEG variations in people with a history of cocaine dependency is an emerging field. Increased beta power (Herning et al., 1997; Noldy et al., 1994; Pascal-Leone et al., 1991) and increased alpha power are among the observed findings (the strength or amplitude of beta waves in the brain is referred to as beta power while

the strength or amplitude of alpha waves in the brain is referred to as alpha power). While some studies have found no significant differences, others have indicated increases in both alpha and beta power combined with decreases in delta power. Confounding variables including clinical depression and neuroleptic medication pose difficulties when interpreting EEG findings in cocaine-dependent patients (Costa Bauer, 1997).

This study demonstrated that AUD patients had lower functional connectivity, primarily in the beta and alpha bands, and higher absolute and relative beta power when compared to matched controls. In the alpha band, impaired connection was found at the fronto-central and occipito-parietal areas; in the beta band, it was found throughout the scalp electrodes. Additionally, we discovered a negative correlation between the non-planning dimension of impulsivity and reduced functional connectivity, notably in the alpha band at the fronto-central regions (Diaz, at al., 2016)

In the study conducted by Lee (Lee, at al., 2017) A total of 109 subjects with GD were assigned to one of three groups based on their level of impulsivity: high, middle, and low. The analysis of EEG absolute power differences was conducted using generalized estimating equations. The results demonstrated a reduction in theta absolute power in the high-impulsivity group, accompanied by a decrease in alpha and beta absolute power in the midline frontocentral regions.

In another study, the impact of smoking on resting EEG in chronic smokers was examined. Results reveal that daily smokers have reduced resting delta and alpha EEG power and higher impulsiveness compared to nondaily and non-smokers (Rass, al.,2016).

In the study conducted by Synder & Hall in (2006), regarding the identification of ADHD patients, it was determined that there is a correlative consistentcy with neurological evidence and a decrease in beta power. Specifically, observational findings in ADHD patients showed that there is a relationship to inattention and impulsivity with decreased beta power. There are possible neurophysiological similarities with ADHD when associated with decreased EEG beta power activity in patients with GD. That GD may have a candidate neurophysiological biomarker of behavioral addiction may thus be supported by this result (Lee,at al.,2017).

In sum from our study, as well as various correlative abuse/dependency disorder studies (e.g. smoking, gambling, alcohol, aggressive traits/tendencies, narcotics), it can ultimately be demonstrated that as a cerebral core universal neurobiological locus a generalized neural cause and effect relationship exists between impulsivity and abuse disorders whereby an inverse proportional ratio function shows that where there is an increase in impulsivity there will be a corresponding decrease in EEG neuroelectrical waveform activity.

6. CONCLUSION AND SUGGESTIONS

Demographic Variables

When examining the demographic characteristics of the participants, the age distribution ranged from 20 to 50, with diversity seen in variables such as marital status and education level. Data on smoking and alcohol consumption were also examined to analyze the effects of these factors on impulsivity and brain wave frequencies.

Relationship Between Impulsivity and EEG Frequency

A statistically significant relationship was found between the total score of the Barratt Impulsiveness Scale and EEG frequencies. Specifically, a negative relationship was found between the total score of the Barratt Impulsiveness Scale and the DELTA_L, DELTA_R, and DELTA_C frequencies. These results indicate that individuals with high levels of impulsivity have lower levels of delta wave activity in their brains. However, a negative relationship was found between the motor impulsivity score and the THETA_L, THETA_R, and THETA_C frequencies. These results show that as motor impulsivity increases, theta wave activity decreases.

Correlation Between EEG Frequencies

Correlation analysis between EEG frequencies showed how brain wave activity in certain regions interacts. A strong positive correlation was found between the DELTA and THETA waves. Specifically, a positive and significant relationship was found between DELTA_L and THETA_L, and between DELTA_R and THETA_R. These results indicate that brain activity is synchronized at certain frequencies.

Impulsivity and Demographic Factors

When examining the relationship between impulsivity levels and demographic factors, it was observed that variables such as age, marital status, education level, smoking, and alcohol consumption have certain effects on impulsivity. However, these effects were not statistically significant. Specifically, significant differences were found between marital status and attentional impulsivity scores; it was observed that individuals had higher levels of attentional impulsivity compared to married individuals.

EEG Frequency and Impulsivity

In the relationship between EEG frequency and impulsivity, the negative relationship between motor impulsivity and theta waves was found to be significant. This shows that individuals with high motor impulsivity have low theta wave activity. Similarly, the negative relationship between delta waves and general impulsivity scores indicates that individuals with high levels of impulsivity have lower delta wave activity in their brains.

Importance and Limitations of the Study

The results of this study provide significant contributions to understanding the relationship between impulsivity and brain wave activity in stimulant users. By revealing the effects of impulsivity on EEG frequencies, we show how brain activity is related to levels of impulsivity. However, this study has some limitations. The limited sample size and the study being conducted only stimulant users restrict the generalizability of the results. Future studies are recommended to consider larger sample groups and different substance usages to conduct more comprehensive analyses.

Conclusion

This study reveals the relationship between impulsivity and brain wave frequency in stimulant users and shows that the level of impulsivity has a significant impact on brain wave activity. Specifically, low DELTA and THETA wave activity was observed in individuals with high levels of impulsivity, thus validating the initial pre-research hypothesis. This in turn correlates with what is known in that there appears to be an “inverse ratio” of impulsivity to wave forms whereby an increased impulsivity will yield decreased neural wave forms. These results provide important insights into how brain activity interacts with levels of impulsivity. In clinical practice, if we can measure the resting EEG of patients and use stimulant as a biomarker we can relate it to impulsivity and prioritize the study of impulsivity in the treatment protocols of these patients. If these patients have changes in increased brain activity, we can create a rehabilitation protocol so as to modify these brain activities. As a treatment goal, ıf impulsivity can be decreased this will lead to an outcome where by the patients will be more relaxed and comfortable and thus result in better patient treatment compliance. Future studies are encouraged to consider larger sample groups and different variables

to more comprehensively examine the relationship between impulsivity and EEG activity (in addition to including short-term versus long-term comparative usage results among patients with SUDs).

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APPENDECIS

SURVEY

SOSYODEMOGRAFİK VERİ FORMU

1. Adı: **Ev Tel:** **Cep Tel:**

2. Yaşı:

4. Medeni Durum: Hiç evlenmemiş (1) Evli (2) Dul (3) Boşanmış (4)

5.Çocuk sayısı;

6.Çocukların yaşları;

7.Çocukların eğitim durumu;

8.Evde 65 yaş üstü yaşayan kişi varlığı; var (1) yok (2)

9. Öğrenim Durumu: İlk (1) Orta (2) Lise (3) Yüksek (4) OYY

10.Meslek;

11. Sigara kullanım var mı? Var (1) Yok (2)

12.Sigara kullanım varsa miktar belirtiniz……adet/ gün

13. Alkol kullanım var mı? var (1) yok (2)

14.Alkol kullanım varsa günlük /haftalık kullanım miktarı belirtiniz.

15.Kahve tüketimi var mı? Var (1) Yok (2)

16.Kahve tüketim miktarı belirtiniz……fincan /gün

17. Daha önce psikiyatrik öykü varlığı? var (1) yok (2)

18. Daha önce psikiyatrik tanı varsa belirtiniz

19.Ailede psikiyatrik hikâye var mı? var (1) yok (2)

Barratt Dürtüsellik Ölçeği -11 Türkçe

	Nadiren/ Hiçbir zaman	Bazen	Sıklıkl	Hemen herzaman/ Herzaman
İşlerimi dikkatle planlarım	☐	☐	☐	☐
Düşünmeden iş yaparım	☐	☐	☐	☐
Hızla karar veririm	☐	☐	☐	☐
Hiçbir şeyi dert etmem	☐	☐	☐	☐
Dikkat etmem	☐	☐	☐	☐
Uçuşan düşüncelerim var	☐	☐	☐	☐
Seyahatlerimi çok önceden planlarım	☐	☐	☐	☐
Kendimi kontrol edebilirim.	☐	☐	☐	☐
Kolayca konsantre olurum	☐	☐	☐	☐
Düzenli para biriktirim	☐	☐	☐	☐
Derslerde veya oyunlarda yerimde duramam	☐	☐	☐	☐
Dikkatli düşünen birisiyim	☐	☐	☐	☐
İş güvenliğine dikkat ederim	☐	☐	☐	☐
Düşünmeden bir şeyler söylerim	☐	☐	☐	☐
Karmaşık problemler üzerine düşünmeyi severim	☐	☐	☐	☐
Sık sık iş değiştiririm	☐	☐	☐	☐
Düşünmeden hareket ederim	☐	☐	☐	☐
Zor problemler çözmem gerektiğinde kolayca sıkılırım	☐	☐	☐	☐
Aklıma estiği gibi hareket ederim	☐	☐	☐	☐
Düşünerek hareket ederim	☐	☐	☐	☐
Sıklıkla evimi değiştiririm	☐	☐	☐	☐
Düşünmeden alışveriş yaparım	☐	☐	☐	☐
Aynı anda sadece bir tek şey düşünebilirim	☐	☐	☐	☐
Hobilerimi değiştiririm	☐	☐	☐	☐
Kazandığımdan daha fazla harcadım	☐	☐	☐	☐
Düşünürken sıklıkla zihnimde konuyla ilgisiz düşünceler oluşur	☐	☐	☐	☐
Şu an ile gelecekten daha fazla ilgilenirim	☐	☐	☐	☐
Derslerde veya sinemada rahat oturamam	☐	☐	☐	☐
Yap-boz/puzzle çözmeyi severimi	☐	☐	☐	☐
Geleceğini düşünen birisiyi	☐	☐	☐	☐