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Review on Fundamental of Electroencephalography and Detection Techniques in Substance and Behavioral Addiction

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Abstract:

Addiction is a chronic relapsing brain disease associated with substantial individual and societal burden. It includes complex phyco-physiological conditions and can be recognized by compulsive and harmful behaviors towards a substance or activity, despite negative consequences or "bio-psycho-social-spiritual" disorder. This paper aims to comprehensively review previous research which applied machine learning to electroencephalography (EEG) signals for automatic detection of addiction. A systematic search was done on 200 relevant papers published between 2018 - 2023. The previous studies applied a variety of machine learning algorithms including support vector machines, neural networks, logistic regressions and brain networks to classify addicted individuals against controls based on resting state, cue-reactivity paradigms and neurofeedback tasks. The features utilized spectral power, functional and effective connectivity, graph theoretical measures and event-related potentials. The output accuracy often exceeded 95% across multiple substances including alcohol, nicotine, cannabis and opioids. In this review, the challenges and open questions around data quality, model interpretation and transition to clinical settings were also discussed. In overall, automatic EEG analysis shows significant potential as an objective and accessible tool for addiction diagnosis, treatment monitoring and relapse prevention that projects worthy of continued refinement and validation.

Keywords: Detection; EEG; Drug addiction; Chronic relapsing; Brain disease.

1. INTRODUCTION

Addiction is the dependence on external abusive material that causes neuroadaptation or out-of-control behaviors (1). It often involves both physical and psychological dependencies, leading to a loss of control and an overwhelming desire to continue engaging in addictive behavior. Addiction can have devastating effects on individuals, their relationships, and society as a whole (2). There are several factors that influence a person's risk of developing an addiction, such as an individual's drug susceptibility, which is counted under the genetic-related problems, especially those who live with families who have a history of using addictive materials will be more likely to be addicted persons themselves.

There are also environmental factors such as stress levels, economic problems, and physical inabilities which make the young people tend to escape from their real life by drugs (3). Other conditions such as the availability of substances, influences of friends and public figures, and early exposure to drugs or alcohol during puberty especially during the brain's early development also play important roles and increase addiction risks as well (4). Some studies suggest that there are some effects of amphetamine to weaken the prefrontal cortex circuitry connections, where the prefrontal cortex is responsible for rational thinking and decision making. The combination of the strength of sub-cortical automatic memories associated with drug use, will lead to addicted individuals experience with intense drug craving in the presence of environmental conditions (5).

2. HUMAN BRAIN AND ADDICTIONS

2.1 Structure of Brain



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The human brain can be divided anatomically into three parts, namely hindbrain, midbrain, and forebrain. The forebrain in turn consists of many parts including cerebrum, thalamus, hypothalamus, pituitary gland, limbic system, and olfactory bulb (6, 7). The outer layer of the cerebral cortex has four major lobes which are frontal, occipital, parietal, and temporal (8). The top portion of the brain is called the cortex. It plays an important role in the functionality of the brain and is considered as a cognitive part (9). The cortex consists of many areas that reflect multiple jobs including the sensory areas, motor areas, and association areas, and has interactions with the subcortical areas. With all these components and relationships, the cortex is in charge of the majority of our daily functions (10). High-order cognitive tasks arise primarily from the combination of various sensory inputs, processed by subcortical areas and association areas. The association functions include generally everything related to language such as analyzing information, memorizing, planning, and conscious thinking (creativity) (11). Different lobes are given different tasks by the brain cortex. In general, personality, emotions, and higher order cognitive abilities are associated with frontal lobe functions. The temporal lobe has many functions the main function involves the processing of hearing and other senses. On the other hand, the occipital lobe is primarily involved in vision (12), whereas the parietal lobe appears to be particularly involved in language, subjective feelings, and concentration (13).

2.2 Various Additions and Their Types

Drug addiction is a chronic neuronal and behavioral regression disorder; craving and negative affect on behavior are the most common symptoms of this retreat; these effects with emotional tests are used to diagnose the degree of drug intake (14). Substance abuse occurs when the substance used affects the central and peripheral nervous systems, causing abnormalities in their functioning, which can be noticed in their behavior and brain- related biosignals, such as EEG (15). Drug addiction has been associated with a broad range of cognitive deficits, including emotional regulation and motivation, attention and flexibility, working memory, learning, and decision making (16). For example, cocaine addiction can cause psychotic behavior, brain damage, and death from overdose (17). Emotional effects on rational decisions such as competition for fortunate and illegal markets, terrorizing neighborhoods, wreck political and judicial systems, and in severe cases even can cause many violent deaths (18). Addictive drugs have to connect to some proteins in the human brain in order to be active, based on that we can classify them according to their types and effects into major categories as below (19).

Opioids - There are three types of opioids, natural opioids like morphine and codeine, semi- synthetic opioids such as oxycodone and hydrocodone, and synthetic opioids such as fentanyl and methadone which are the most addictive ones. Opioids work includes binding to the pain and reward receptors in the brain and create a reward increment mechanism. Their effects are similar to endogenous endorphins (20).

Stimulants - Their work is related to the neurotransmitters dopamine, serotonin and norepinephrine in the brain's reward system, they work, when taken, to increase levels of these neuro- transmitters which leads to improved mood, focus, and energy in the short term (21). Stimulants available in different ways such as Cocaine, amphetamines like Adderall and methamphetamine, and even caffeine can be addictive stimulants (22).

Nicotine - Nicotine is one of the most commonly addictive substances available in our life, it can be found in tobacco products. Its work is related to dopamine neurotransmission which when increases activates neural circuits whichenhances pleasure and habit formation (23).

Cannabis - Cannabis, when consumed, mimics the effects of endogenous cannabinoids produced naturally in the body like anandamide. It changes the mood including brings relaxation, effect the sensory conceives such as increase the appetite, and increase the color brightness. Cannabis comes in many forms such as marijuana, hashish and tetrahydrocannabinol (THC) ex- tracts, these products carry addiction risk for chronic levels. The cannabinoid works to bind with CB1 receptor, where the CB1 receptor is one of the main cannabinoid receptors located throughout the nervous system and plays a role in addiction-related neural plasticity effects. Cannabis is available in different forms such as smoking or vaping of dried cannabis flowers.

Alcohol - Ethanol has the risk of disrupting structure and function of some parts of the brain, especially the prefrontal cortex reward pathways when used regularly and heavily with time. It works on GABA receptors and produces calming effects (24).

Benzodiazepines - Normally, benzodiazepines is a psychoactive drug used in cases of anti-anxiety, social phobia, insomnia, alcohol withdrawal, seizures, panic attacks, and muscle contraction. They include medicines such as Xanax work via GABA receptors like alcohol. Generally, they are not very addictive, if used properly and under the supervision of doctors, yet carry high abuse potential if misused outside of medical oversight (25).

Social media addiction, smartphones addiction, and food addiction may seem to be less harmful, yet they affect patients' life and can cause some behavioral and emotional disorders (26, 27).

3. EFFECTS OF ADDICTION ON THE BRAIN

Addiction originates in the brain reward system, where the brain reward system controls our motivations and drives survival requirements like water, food, shelter, and sex. A natural development occurs later when the brain creates a mechanism to reinforce these behaviors, which has been called the mesolimbic reward system (28). The mesolimbic pathway, sometimes referred to as the reward pathway, is a dopaminergic pathway in the brain (29). Anatomically, see Figure 1, it

is a pathway that connects the ventral tegmental area in the midbrain to the ventral striatum of the basal ganglia in the forebrain (30).

The ventral striatum includes the nucleus accumbent and the olfactory tubercle (31). When dopamine is released into the nucleus accumbens, the mesolimbic pathway controls incentive salience, or motivation and desire for rewarding stimuli. It also promotes reinforcement and reward-related motor function learning (1). Dopamine (DA), which is secreted from the vental tegmental area (VTA), affects the human behavior, cognitive behavior, decision making, and emotional reactions through the of VTA projections to the prefrontal cortex (PFC), nucleus accumbes (NAc), and amygdala which plays a vital role in the reward mechanism of the brain including drug addiction (32).

Besides dopamine, another neurotransmitter plays an important role in the reward system of the brain (33). Serotonin (5-hydroxytryptamine; 5-HT) plays a crucial role in the development and regulation of addiction. 95% of the body's serotonin, however, is produced in the intestine, where it has been increasingly recognized for its hormonal, autocrine, paracrine, and endocrine actions (34). Serotonin pathways originate from the raphe nuclei in the brainstem and project widely through the cortex and limbic regions. This includes projections to the nucleus accumbens reward center (35). That makes it involved in a wide range of physiological activities, brain functions, and human behaviors. For example, physiological activities include stress response, sleep, the modulation of reward, cardiovascular and endocrine functions, emotions, and mood. Behaviors such as aggression, pain, anger, appetite, and sexuality, as well as neuro-psychological processes like perception, attention, and memory (36). Such functionality means this neurotransmitter will be available in almost every lobe and region of the brain (36).

Serotonin was identified as a neurotransmitter by Vittorio Erspamer and Irvine H. Page in 1952. But it was not until the 1980s and 1990s that significant research on the subject of serotonin and addiction became well-known, emphasizing the neurotransmitter's function in a number of addictive behaviors (37). This is only one explanation out of three theories that have been proposed to explain the alterations in the brain's reward system that underline addiction. First, the impulsive theory proposes that addiction is characterized by excessive sensitivity to reward and failure of inhibition. Individuals with a hyperactive brain reward system may show a strong response to even non-drug-related incentive cues, which can explain the continuous drive and motivation to obtain pleasure-inducing substances or other potentially rewarding stimuli (38). The reward-deficiency theory, however, proposes that addiction is characterized by a reduced response to rewards, which reduces the impact of rewarding experiences and results in increased drug-seeking behaviors (39).

Finally, another competing theory, the incentive-sensitization theory, posits that addiction is characterized by hypersensitization of the reward anticipation process (i.e., the "wanting" process) without necessarily affecting the reward outcome process (i.e., the "liking" process) (40). Based on this, addiction can be redefined as the dependence on any external material or stimulus that can pharmacologically" hijack" the reward circuitry conciliated by its effect on the brain and body (the neuromimetic effect of drug administration). However, it could be suggested that any stimuli (drug or behavior) that transform basic drives required for survival (natural rewards like feeding, thirst, and reproduction) into actions of craving or seeking behaviors or repetitive out-of-control behaviors may make it plausible that addiction can occur even in the absence of drug taking. Thus, behavioral addictions may share many of the same pathways associated with chemical dependence (1, 41).

Where craving is the reflection of drug-acquisitive state which motivates drug use, or the subjective experience of drug craving, defined as a strong desire or urge to use drugs, is a prominent and common clinical phenomenon for individuals diagnosed with opioid use disorder (OUD) (42). Since craving is a complex psychological phenomenon, assessment of craving is not an easy method at all (43). The huge ability of these addictive materials to target the neurouvs system and especially the human brain returns to the fact that they are lipophilic, which means that they can easily make bonds with lipids, which are abundant in the brain. These effects can be noticed and studied using EEG signals, and this and many other reasons have led researchers to be oriented towards using EEG in automatic detection of different types of addiction for many decades (44).

4. DIAGNOSIS OF ADDICTIONS

Diagnosis of addiction requires a synchronized operation in order to assets various factors, including behavioral patterns of EEG (biomarkers), physical symptoms, and psychological indicators, in addiction to self-reporting, and the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), to have a solid base diagnosis of addiction.

4.1 Biomarkers

EEG biomarkers play a crucial role in automatic diagnosis of addiction using EEG signals, since They represent specific patterns of electrical activity in certain regions and areas of the brain that might be involved in the reward cycle (or addiction cycle). In this way, biomarkers are valuable identifiers and help in diagnosis, treatment, and monitoring of different types of addictions. Some probabilistic biomarkers are:

Alpha/beta power: Increased alpha and beta power in frontal and central regions is seen in addiction during drug cue exposure and withdrawal. This reflects heightened arousal and attentional processing of addiction-related stimuli.

Gamma oscillations: Abnormal gamma band synchronization in frontal-striatal-limbic networks disrupts cognitive control over drug seeking. Cue-induced gamma changes predict relapsed risk.

P3 event-related potential: Amplitude of the P3 ERP component elicited by drug cues is enhanced in addiction. It indexes attentional allocation and salience attribution to addiction cues.

Resting state connectivity: Addiction is linked to hyperconnectivity of brain regions involved in reward, motivation and emotion processing. Hypoconnectivity between these and cognitive control regions impairs self-regulation.

Frontal theta activity: Elevated frontal midline theta power in addiction may index conflict monitoring and impulse control deficits when resisting drug cues. It normalizes with protracted abstinence.

Alpha asymmetry: Relative left frontal hypoactivation (more right alpha power) is seen in addiction, implicating approach motivation biases toward drug rewards.

Oscillatory phase coupling: Abnormal phase-amplitude coupling between low and high frequencies may disrupt optimal information transfer in addiction networks.

4.2 Psychological Assessment

Psychologically, to evaluate a person with addiction we need some tests. Different methods had been used for assessments, yet nowadays The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) is the best test to make such an assessment, which can give as a scale of craving from 'no craving at all' to 'strong intensive craving' (45). It was published for the first time by the American Psychiatric Association (APA) in 1994 and served as a comprehensive guide for mental health professionals to identify and classify various mental health conditions. The DSM-IV contained 17 major diagnostic classes covering over 300 specific mental health conditions and disorders (46). It provided descriptive text for each disorder including essential features, subtypes, prevalence rates, course, risk and prognostic factors, differential diagnosis, and more (47, 48).

Another famous one was developed in the late 1990s by psychiatrist Kimberly Young and has been validated through multiple studies The Implicit Association Test (IAT) (49). It consists of 20 questions rated on a 5-point Likert scale. The questions evaluate how much a per- son's daily activities, responsibilities, emotions, and psychological health are negatively impacted by excessive internet use. Sample items include "How often do you find that you stay online longer than you intended?" and "How often do you feel preoccupied with the Internet when off-line, or fantasize about being online?" (50). Scoring involves summing the responses, with an overall range between 20-100 points. Higher scores indicate more severe addiction symptoms. Scores between 20-30 points suggest average internet use without problematic behavior. Scores between 31-49 signify mild-moderate addiction issues. Scores 50-79 are moderate-severe, and 80-100 indicate severe problems requiring professional help (51).

Since addiction is related to brain activity, which can be measured by measuring its electrical activity, the electroencephalogram (EEG) has been used to record, for later analysis, the electrical activity of the brain of addicted people, and a horde of EEG investigations have shed light on the neuroplasticity associated with addiction. The study has specifically looked at how drugs affect the post-synaptic potentials, with a focus on their influence on the mesolimbic dopamine (DA) pathway. In this way, it is evident that EEG contributes to the characterization of systemic alterations that promote compulsive drug seeking and impair self-control in addiction. It reveals limbic and cognitive control imbalances that might last long after substance use stops. Additionally, EEG exhibits potential as a supplemental biomarker for assessing the severity of addiction and tracking the effectiveness of treatment over time. With high-density EEG and combined neuroimaging techniques, future research can deepen our understanding of the mechanisms involved (52).

PHYSIOLOGICAL ASSESSMENT 5.

Physiological signs of addiction can manifest in various ways across different substances or behaviors. These signs are related to the physiological changes that occur in the body due to prolonged substance use or engagement in addictive behaviors. Here are some common physiological signs of addiction:

Tolerance: With continued substance use, the body may develop tolerance, requiring higher doses of the substance to achieve the desired effect. This occurs as the body adapts to the substance and becomes less responsive to its effects. Withdrawal symptoms: When the substance is abruptly discontinued or reduced, individuals may experience withdrawal symptoms. These symptoms can vary depending on the substance but may include physical discomfort, cravings, irritability, anxiety, insomnia, nausea, and sweating.

Physical dependence: Prolonged substance use can lead to physical dependence, where the body becomes reliant on the substance to function normally. When the substance is removed, the body experiences withdrawal symptoms.

Changes in vital signs: Substance use can impact vital signs such as heart rate, blood pressure, and respiratory rate. Stimulant drugs like cocaine or amphetamines can increase heart rate and blood pressure, while depressant drugs like opioids or sedatives can lower these parameters.

Disrupted HPA axis: Addiction dysregulates the hypothalamic-pituitary-adrenal axis responsible for stress response. This contributes to cravings triggered by stress and negative reinforcement of continued use.

Cardiovascular/pulmonary issues: Smoking/vaping and stimulant abuse produce respiratory effects while various drugs stress the heart through arrhythmias, changes in blood pressure, etc.

Nutritional deficiencies: Damage to appetite regulation, poor food choices, money diverted from necessities for drugs deplete micronutrients over prolonged use.

Biosignals: All the psychological symptoms mentioned can be measured through biosignals and especially the electrical activity of the brain (EEG). Electroencephalography (EEG) is a technique that measures electrical brain activity and can be utilized in the detection and assessment of addiction. By analyzing the patterns and frequencies of brainwaves, EEG can provide insights into the neural correlations of addiction and help identify abnormal brain activity associated with addictive behaviors. This method has the potential to contribute to the early detection and monitoring of addiction, aiding in personalized treatment approaches.

6. BRAIN SIGNALS AND ARTIFACTS

EEG is a non-invasive, an easy, rapid, unexpensive method that has been used to examine the electrical activity in the brain, which gives indications of the brain states and responses to different stimuli (53). It has high temporal resolution and can detect delicate changes in brain waves or oscillations associated with different cognitive and emotional states, specially the synchronised activity of exitatory and inhibitory post-synamptic potenails in the brain cortex (54). The non-invasibility of EEG allowed and eased the work of scientists to study brain behaviour. In addiction to that, EEG can also offer meaning-rich signals with a high temporal resolution that is accessible even when using cheap, portable EEG devices which made it even more easy to make practical applications such as detecting and tracking the progress of different mental illnesses and cases (55).

That's made EEG the best choice for research, since 1929 when Berger published the results of his work, to use it in many research fields related to brain like monitoring and diagnosing seizures, dementia, brain tumors, encephalitis, obstructive sleep apnea, depth of anesthesia, coma, and stroke. Besides this use, it is well recognized that analyses of signals have the potential to identify human emotions, which would have great potential for a great range of practical uses (56). However, the impurity of the recorded EEG signals due to other biosignals and external environmental noises will reduce the efficacy of detecting the physiological and pathological information of the brain. Such impurities can be listed according to their sources to internal or biological, and external sources as follows (57):

Eye movement artifacts: One of the most common problems in EEG recording process is the eye movements artifacts, which will add a rapid signal with ~100 μ V to our signal. The eye movement artifacts appear whenever the patient blinks or moves his eye or eyelids. Normally the closure the recording electrodes to the eye the stronger the recorded noise will be.

Cardiogenic Artifacts: The artifacts which result from the electrical activity of the heart are much larger than the EEG with nearby frequencies. The good thing about ECG is that it can be detected by bare eyes since it has a fixed rhythm (QRS). **Muscle movements artifacts:** EMG waves also can be interfered with EEG signals, especially those in the cranial (head and neck) region since these movements are irregular and normally have low frequency values, which will interfere with the low frequency components of the recorded EEG signal.

The external noises are the noise added from the electromagnetic signals outside the human body:

Environmental noise: Since the EEG signal is an electromagnetic signal, any kind of signal available in the outside environment will affect the EEG test. Especially those that have a relatively low value of frequency like the 50 Hz powerlines, other electronic devices like laptop PCs and other medical devices in the hospital room can also make a noise interference to our EEG measurement. We can get rid of such noises by using notch filters.

Problems due to electrode-scalp contact: It is of great importance to make sure that all the electrodes have good contact with the scalp, otherwise a capacitive effect will occur and make an additional noisy effect to the main EEG signal.

Other important factors to notice when recording the EEG signal are personal differences, like the type of skin, whether it is greasy or dry, the type and direction of the hair on the patient's head, and so on (58).

7. STUDY REMARKS, GAPS AND FUTURE PERSPECTIVE

Addiction is a chronic relapsing disorder characterized by the loss of inhibitory control over drug- seeking and taking, and maintenance of drug use despite negative consequences, drug can affect the patient mind changing its neuronal wiring and causing an addiction neuronal path (59). Addiction can be diagnosed by many means, such as, Biomarkers, psychological Assessment, and physiological assessment. Where the Diagnostic and Statistical Manual of Mental Disorders (DSM), has been considered as the tool for diagnosing mental illness used by the American Psychiatric Association, has highly evolved from the eighties to the nineties in the criteria used to diagnose addiction (47). However, depending only on psychological methods in diagnosis, treatments, and follow up with the patient after the withdrawal and its accompanied symptoms. In this way, the need for using EEG has increasing importance due to many reported limitations in this test (60-62). As a result, more advanced EEG analysis methods are needed, like the use of Deep Learning in this field (63).

AUTHORSHIP CONTRIBUTION STATEMENT

Sadeem Nabeel Saleem Kbah: writing - original draft; Tan Tian Swee: supervision, conceptualization; Jahanzeb Sheikh: writing - review & editing; Noor Kamal Al-Qazzaz: writing - review & editing; Maheza Irna Mohamad Salim: writing - review & editing; Hum Yan Chai: writing - review & editing; Michael Loong Peng Tan: writing - review & editing; Fujie Qiu: writing - review & editing

DATA AVAILABILITY

Data are available within the article and/or its supplementary materials.

DECLARATION OF COMPETING INTEREST

The authors declare no conflict of interest.

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REFERENCES

- Karim JT, Aksel C, Kharas N, Reves-Vasquez C, Dafny N. Caudate nucleus neurons participate in methylphenidate (1) function: Behavioral and neuronal recordings from freely behaving adolescent rats. Brain Res Bull. 2018; 142:241-52. https://doi.org/10.1016/j.brainresbull.2018.07.008
- Levy YZ, Levy D, Meyer JS. Computational hypothesis for maturing out of addiction and mindfulness-based cognitive (2)techniques. In: Klimov VV, Kelley DJ, editors. Biologically inspired cognitive architectures 2011. IOS Press: Netherland; 2011. p. 222-231. https://doi.org/10.3233/978-1-60750-959-2
- (3) Morales AM, Jones SA, Kliamovich D, Harman G, Nagel BJ, Identifying early risk factors for addiction later in life: A review of prospective longitudinal studies. Curr Addict Rep. 2020; 7:89-98. https://doi.org/10.1007/s40429-019-00282
- Yap JX, Che Amat MA. Social media addiction and young people: A systematic review of literature. J Crit Rev. 2020; (4) 7(13):537-541. https://doi.org/10.31838/jcr.07.13.97
- Ferrucci M, Limanaqi F, Ryskalin L, Biagioni F, Busceti CL, Fornai F. The effects of amphetamine and (5) methamphetamine on the release of norepinephrine, dopamine and acetylcholine from the brainstem reticular formation. Front Neuroanat. 2019; 13:48. https://doi.org/10.3389/fnana.2019.00048
- Forebrain (n.d.). BYJU'S. [internet]. [cited 2024 Jan 9]. Available from: https://byjus.com/neet/difference-between-(6) forebrain-midbrain-andhindbrain/#:~:text=The%20forebrain%20is%20the%20largest,the%20cerebrum%2C%20thalamus%20and%20hypo thalamus.
- Forebrain (n.d.). Vedantu. [internet]. [cited 2024 Jan 9]. Available from: https://www.vedantu.com/biology/forebrain
- (8) Nacy SM, Kbah SN, Jafer HA, Al-Shaalan I. Controlling a servo motor using EEG signals from the primary motor cortex. Am J Biomed Eng. 2016; 6(5):139-146. https://doi.org/10.5923/j.ajbe.20160605.02.
- Muñoz-Castañeda R, Zingg B, Matho KS, Chen X, Wang QX, Foster NN, et al. Cellular anatomy of the mouse primary (9) motor cortex, Nature, 2021; 598(7879);159–166, https://doi.org/10.1038/s41586-021-03970-w
- (10) Kbah SN, Abushaeer MT. Studying the emotional behavior using an orbitocortical-amygdalo computational model. 2019 Fifth International Conference on Advances in Biomedical Engineering (ICABME). 2019; 1-4. https://doi.org/10.1109/ICABME47164.2019.8940342
- (11) Sunavsky A, Poppenk J. Neuroimaging predictors of creativity in healthy adults. Neuroimage. 2020; 206:116292. https://doi.org/10.1016/j.neuroimage.2019.116292
- (12) Kbah SN. A computational model of the brain cortex and its synchronization. Biomed Res Int. 2020. https://doi.org/10.1155/2020/3874626
- (13) Kbah SN. Investigation of a moderate cortical model synchronization created using Brian simulator. 2019 Medical Technologies Congress (TIPTEKNO). 2019; 1-4. https://doi.org/10.1109/TIPTEKNO.2019.8895185
- (14) Chen XJ, Wang DM, Zhou LD, Winkler M, Pauli P, Sui N, Li YH. Mindfulness-based relapse prevention combined with virtual reality cue exposure for methamphetamine use disorder: Study protocol for a randomized controlled trial. Contemp Clin Trials. 2018; 70:99-105. https://doi.org/10.1016/j.cct.2018.04.006
- (15) Acharya U, Sree S. Automated EEG analysis of epilepsy: A review. Semant Scholar. 2012; 45:147-165. https://doi.org/10.1016/j.knosys.2013.02.014
- (16) Verdejo-Garcia A, Garcia-Fernandez G, Dom G. Cognition and addiction. Dialogues Clin Neurosci. 2019; 21(3):281– 290. <u>https://doi.org/10.31887/DCNS.2019.21.3/gdom</u>
 (17) Pelloux Y, Giorla E, Montanari C, Baunez C. Social modulation of drug use and drug addiction. Neuropharmacology.
- 2019; 159:107545. https://doi.org/10.1016/j.neuropharm.2019.02.027
- (18) Turnip A, Esti KD, Amri MF, Simbolon AI, Suhendra MA, Iskandar S, Wirakusumah FF. Detection of drug effects on brain activity using EEG-P300 with similar stimuli. IOP Conf Ser Mater Sci Eng. 2017; 220(1). https://doi.org/10.1088/1757-899X/220/1/012042
- (19) Lüscher C, Ungless MA. The mechanistic classification of addictive drugs. PLoS Med. 2006; 3(11):e437. https://doi.org/10.1371/journal.pmed.0030437
- (20) Ersek M, Cherrier MM, Overman SS, Irving GA. The cognitive effects of opioids. Pain Manag Nurs. 2004; 5(2):75-93. https://doi.org/10.1016/j.pmn.2003.11.002
- (21) Arnsten AFT. Stimulants: Therapeutic actions in ADHD. Neuropsychopharmacology. 2006; 31(11):2376-2383. https://doi.org/10.1038/sj.npp.1301164
- (22) Rubia K, Alegria AA, Cubillo AI, Smith AB, Brammer MJ, Radua J. Effects of stimulants on brain function in attentiondeficit/hyperactivity disorder: A systematic review and meta-analysis. Biol Psychiatry. 2014; 76(8):616–28. https://doi.org/10.1016/j.biopsych.2013.10.016

- (23) Dwyer JB, McQuown SC, Leslie FM. The dynamic effects of nicotine on the developing brain. Pharmacol Ther. 2009; 122(2):125–139. <u>https://doi.org/10.1016/j.pharmthera.2009.02.003</u>
- (24) Lovinger DM, Roberto M. Synaptic effects induced by alcohol. Curr Top Behav Neurosci. 2013; 13:31–86. https://doi.org/10.1007/7854_2011_143
- (25) Lim B, Sproule BA, Zahra Z, Sunderji N, Kennedy SH, Rizvi SJ. Understanding the effects of chronic benzodiazepine use in depression: A focus on neuropharmacology. Int Clin Psychopharmacol. 2020; 35(5):243–53. https://doi.org/10.1097/YIC.00000000000316
- (26) Escamilla-Ocañas CE, Albores-Ibarra N. Current status and outlook for the management of intracranial hypertension after traumatic brain injury: decompressive craniectomy, therapeutic hypothermia, and barbiturates. Neurología (English Edition). 2023; 38(5): 357–363. <u>https://doi.org/10.1016/j.nrleng.2020.08.024</u>
- (27) Wang D, Zhou Ć, Zhao M, Wu X, Chang YK. Dose-response relationships between exercise intensity, cravings, and inhibitory control in methamphetamine dependence: An ERPs study. Drug Alcohol Depend. 2016; 161:331–339. <u>https://doi.org/10.1016/j.drugalcdep.2016.02.023</u>
- (28) Liu Y, Chen Y, Fraga-González G, Szpak V, Laverman J, Wiers RW, Ridderinkhof KR. Resting-state EEG, substance use and abstinence after chronic use: A systematic review. Clin EEG Neurosci. 2022; 53(4):344–366. https://doi.org/10.1177/15500594221076347
- (29) Serafini RA, Pryce KD, Zachariou V. The mesolimbic dopamine system in chronic pain and associated affective comorbidities. Biol Psychiatry. 2020; 87(1):64–73. <u>https://doi.org/10.1016/j.biopsych.2019.10.018</u>
- (30) Baik JH. Stress and the dopaminergic reward system. Exp Mol Med. 2020; 52(12):1879–1890. https://doi.org/10.1038/s12276-020-00532-4
- (31) Ciccocioppo R, de Guglielmo G, Li HW, Melis M, Caffino L, Shen Q, Domi A, Fumagalli F, Demopulos GA, Gaitanaris GA. Selective inhibition of phosphodiesterase 7 enzymes reduces motivation for nicotine use through modulation of mesolimbic dopaminergic transmission. J Neurosci. 2021; 41(28):6128–6143. https://doi.org/10.1523/JNEUROSCI.3180-20.2021
- (32) Kazemi T, Huang S, Avci NG, Waits CMK, Akay YM, Akay M. Investigating the influence of perinatal nicotine and alcohol exposure on the genetic profiles of dopaminergic neurons in the VTA using miRNA–mRNA analysis. Scientific Reports. 2020; 10. <u>https://doi.org/10.1038/s41598-020-71875-1</u>
- (33) Reward Foundation. (n.d.). Reward system [internet]. [cited 2024 Jan 9]. Available from: https://rewardfoundation.org/brain-basics/reward-system/
- (34) Terry N, Margolis KG. Serotonergic mechanisms regulating the gi tract: experimental evidence and therapeutic relevance. Handb Exp Pharmacol. 2017; 239:319–342. <u>https://doi.org/10.1007/164_2016_103</u>
- (35) Carhart-Harris RL, Nutt DJ. Serotonin and brain function: A tale of two receptors. J Psychopharmacol. 2017; 31(9):1091–1120. <u>https://doi.org/10.1177/0269881117725915</u>
- (36) Coray R, Quednow BB. The role of serotonin in declarative memory: A systematic review of animal and human research. Neurosci Biobehav Rev. 2022; 139:104729. <u>https://doi.org/10.1016/j.neubiorev.2022.104729</u>
- (37) Whitaker-Azmitia PM. The discovery of serotonin and its role in neuroscience. Neuropsychopharmacology. 1999; 21(1):2–8. <u>https://doi.org/10.1016/S0893-133X(99)00031-7</u>
- (38) Garami J, Moustafa AA. Delay, probability, and effort discounting in drug addiction. In: Moustafa AA, editor. Cognitive, clinical, and neural aspects of drug addiction. Academic Press: United States; 2020. p. 61–83. https://doi.org/10.1016/B978-0-12-816979-7.00004-2
- (39) Blum K, Bowirrat A, Braverman ER, Baron D, Cadet JL, Kazmi S, Elman I, Thanos PK, Badgaiyan RD, Downs WB, et al. Reward deficiency syndrome (RDS): A cytoarchitectural common neurobiological trait of all addictions. Int J Environ Res Public Health. 2021; 18(21):11529. <u>https://doi.org/10.3390/ijerph182111529</u>
- (40) Morales I, Berridge KC. 'Liking' and 'wanting' in eating and food reward: Brain mechanisms and clinical implications. Physiol Behav. 2020; 227:113152. https://doi.org/10.1016/j.physbeh.2020.113152
- (41) Robbins TW, Clark L. Behavioral addictions. Curr Opin Neurobiol. 2015; 30:66–72. https://doi.org/10.1016/j.conb.2014.09.005
- (42) Bruneau A, Frimerman L, Verner M, Sirois A, Fournier C, Scott K, Perez J, Shir Y, Martel MO. Day-to-day opioid withdrawal symptoms, psychological distress, and opioid craving in patients with chronic pain prescribed opioid therapy. Drug Alcohol Depend. 2021; 225:108787. <u>https://doi.org/10.1016/j.drugalcdep.2021.108787</u>
- (43) Zheng R, Hao L, Li Y, Zhang T, Bai D, Zhang L, Li D, Hao W. Commentary: Craving in opioid use disorder: From neurobiology to clinical practice. Front Psychiatry. 2021; 12:615921. <u>https://doi.org/10.3389/fpsyt.2021.615921</u>
- (44) Zhao D, Zhang M, Tian W, Cao X, Yin L, Liu Y, Xu TL, Luo W, Yuan TF. Neurophysiological correlate of incubation of craving in individuals with methamphetamine use disorder. Mol Psychiatry. 2021; 26(11):6198–6208. <u>https://doi.org/10.1038/s41380-021-01252-5</u>
- (45) Vafaie N, Kober H. Association of drug cues and craving with drug use and relapse: A systematic review and metaanalysis. JAMA Psychiatry. 2022; 79(7):641–650. <u>https://doi.org/10.1001/jamapsychiatry.2022.1240</u>
- (46) Chmielewski M, Clark LA, Bagby RM, Watson D. Method matters: Understanding diagnostic reliability in DSM-IV and DSM-5. J Abnorm Psychol. 2015; 124(3):764. <u>https://doi.org/10.1037/abn000006</u>
- (47) Grant BF, Shmulewitz D, Compton WM. Nicotine use and DSM-IV nicotine dependence in the United States, 2001–2002 and 2012–2013. Am J Psychiatry. 2020; 177(11):1082–1090. <u>https://doi.org/10.1176/appi.ajp.2020.19090900</u>
- (48) Livne O, Shmulewitz D, Stohl M, Mannes Z, Aharonovich E, Hasin D. Agreement between DSM-5 and DSM-IV measures of substance use disorders in a sample of adult substance users. Drug Alcohol Depend. 2021; 227:108958. <u>https://doi.org/10.1016/j.drugalcdep.2021.108958</u>
- (49) Schimmack U. The implicit association test: A method in search of a construct. Perspect Psychol Sci. 2021; 16(2):396–414. <u>https://doi.org/10.1177/1745691619863798</u>

- (50) Vianello M, Bar-Anan Y. Can the implicit association test measure automatic judgment? The validation continues. Perspect Psychol Sci. 2021; 16(2):415–421. <u>https://doi.org/10.1177/1745691619897960</u>
- (51) Hogenboom SAM, Schulz K, van Maanen L. Implicit association tests: Stimuli validation from participant responses. Br J Soc Psychol. 2023; 63(2):975–1002 <u>https://doi.org/10.1111/bjso.12688</u>
- (52) Kbah SN, Sengor NS. Neuronal synchronization and the sparseness of the cortico-cortical connections. 2020 Medical Technologies Congress (TIPTEKNO). 2020; 1–4. <u>https://doi.org/10.1109/TIPTEKNO50054.2020.9299299</u>
- (53) Lopes Da Silva F. EEG: Origin and measurement. In: Mulert C, Lemieux L, editors. EEG-fMRI: Physiological basis, technique, and applications. Springer Berlin: Heidelberg; 2023. p. 23–48. https://doi.org/10.1007/978-3-540-87919-0
- (54) Mercier RM, Dubarry AS, Tadel F, Avanzini P, Axmacher N, Cellier D, Del Vecchio M, Hamilton LS, Hermes D, Kahana MJ, et al. Advances in human intracranial electroencephalography research, guidelines and good practices. Neuroimage. 2022; 119438. https://doi.org/10.1016/j.neuroimage.2022.119438
- (55) Huang X, Xu Y, Hua J, Yi W, Yin H, Hu R, Wang S. A review on signal processing approaches to reduce calibration time in EEG-based brain-computer interface. Front Neurosci. 2021; 15:733546. https://doi.org/10.3389/fnins.2021.733546
- (56) Kbah SN, Al-Qazzaz NK, Jaafer SH, Sabir MK. Epileptic EEG activity detection for children using entropy-based biomarkers. Neurosci Inform. 2022; 2(4):100101. https://doi.org/10.1016/j.neuri.2022.100101
- (57) Chaddad A, Wu Y, Kateb R, Bouridane A. Electroencephalography signal processing: A comprehensive review and analysis of methods and techniques. Sensors. 2023; 23(14):6434. <u>https://doi.org/10.3390/s23146434</u>
- (58) Etienne A, Laroia T, Weigle H, Afelin A, Kelly SK, Krishnan A, Grover P. Novel electrodes for reliable EEG recordings on coarse and curly hair. 2020 42nd Annual International Conference of the IEEE Engineering in Medicine & Biology Society (EMBC). 2020; 6151–6154. <u>https://doi.org/10.1109/EMBC44109.2020.9176067</u>
- (59) Pickard H. Addiction and the self. Noûs. 2021; 55(4):737–761. https://doi.org/10.1111/nous.12328
- (60) Fabiano F, Haslam N. Diagnostic inflation in the DSM: A meta-analysis of changes in the stringency of psychiatric diagnosis from DSM-III to DSM-5. Clin Psychol Rev. 2020; 80:101889. <u>https://doi.org/10.1016/j.cpr.2020.101889</u>
- (61) Park SC, Kim YK. Anxiety disorders in the DSM-5: changes, controversies, and future directions. In: Kim YK, editor. Anxiety disorders: Rethinking and understanding recent discoveries. Springer: Singapore; 2020. p. 187–196. <u>https://doi.org/10.1007/978-981-32-9705-0_12</u>
- (62) Taylor E, Verhulst F, Wong JCM, Yoshida K. Mental health and illness of children and adolescents. Singapore: Springer; 2020. p. 3–15. <u>https://doi.org/10.1007/978-981-10-2348-4</u>
- (63) Hosseini MP, Hosseini A, Ahi K. A review on machine learning for EEG signal processing in bioengineering. IEEE Rev Biomed Eng. 2020; 14:204–218. <u>https://doi.org/10.1109/RBME.2020.2969915</u>