

Crazy Minds in Courts: An Analysis of Forensic Neuropsychology

Ava Wang, Ana Corra, Polygence

ABSTRACT

This paper demonstrates how forensic neuropsychology, especially mental disorders, is used in legal cases by providing some basic information and giving two examples, schizophrenia and dissociative identity disorder (DID), explaining both biological and legal aspects. This paper offers information on the history of forensic neuropsychology, tests needed for an insanity defense, and specialists involved. It also puts forward symptoms, etiology, neuroanatomy, pathophysiology, treatments, and some legal cases for both schizophrenia and DID. This paper also makes a connection between the biological and legal aspects of the disorders, concluding that mental disorders may help to argue for retaining rights but unlikely for getting an acquittal.

Keywords: forensic neuropsychology, schizophrenia, dissociative identity disorder

Introduction

Forensic neuropsychology has evolved rapidly in the past few decades in courts, and mental or psychological tests are also introduced as testimony (Hom, 2003). Forensic neuropsychology refers to the application of neuropsychological and cognitive assessments and evaluations for a party in a civil or criminal lawsuit (University of South Florida, 2017). The assessment is executed by a forensic neuropsychologist hired by the court or attorney to determine whether the litigant has mental disorders, suffers enough severity from the disorder or cognitive/functioning disability when the case happened, and needs treatment or therapy (University of South Florida, 2017). The assessment can be utilized as a determination of the ability to complete the trial, evidence to indicate the liability of the crime, or assistance in conviction or commutation (University of South Florida, 2017). This paper focuses on two mental disorders – schizophrenia and dissociative identity disorder (DID) – and discusses how these two disorders have been used in the previous court cases. This paper first addresses the history of forensic neuropsychology, pertinent court cases, and the main tests and specialists used in the field. The paper then examines the biological and legal aspects of schizophrenia. For the biological aspect, the paper inspects the symptoms, etiology, neuroanatomy, pathophysiology, and treatment of schizophrenia—a mental disorder causing people to misinterpret reality and have delusions, hallucinations, or disorderly extreme behaviors that influence their lives severely (Mayo Clinic Staff, 2020). For the legal aspect, the paper provides several past case examples using schizophrenia in court: Harper sued the Washington state for the forceful use of antipsychotic medications to treat his schizophrenia (*Washington v. Harper*, 1990); Clark used schizophrenia as a defense when he was sued for first-degree murder (*Clark v. Arizona*, 2006); Connelly was unable to relinquish the Miranda rights despite his claim to have waived the rights, he was diagnosed with schizophrenia and therefore it was ruled that he was “forced” to confess due to the disorder (*Colorado v. Connelly*, 1986). Next, the paper studies the biological and legal aspects of dissociative identity disorder (DID). For the biological aspect, the paper inspects the symptoms, etiology, neuroanatomy, pathophysiology, and treatment of DID. People with DID have two or more separated identities and may experience amnesia, depression, or delusions (Mitra & Jain, 2022). For the legal aspect, the paper demonstrates two case examples using DID in court: Badger was found competent to stand the trial even though it was his alternate identity attempted burglary (*State v. Badger*, 1988); Grimsley was found guilty of driving under alcohol violation while she used DID as an insanity defense (*State v. Grimsley*, 1982).

Background

History and Statistics of Forensic Neuropsychology

Forensic neuropsychology is defined as “the application of the science of brain-behavior relationships to legal decision making” (Horton & Hartlage, 2010). Since the appearance of clinical neuropsychology in the 1940s (Horton & Hartlage, 2010), its use in legal matters has been delayed (Horton & Hartlage, 2010). Neuroscience and psychology were already used in courts in the early 20th century (Aono et al., 2019). The first recorded case involving psychology was *State v. Driver* in 1921, where a psychologist was presented as an expert witness, but due to the lack of study of psychology in that time period, this testimony was rejected in the court (Dye, 2011). Neuroscience was introduced in court around the 1940s, where electroencephalography (EEG) was first used to defend a litigant with epilepsy (Aono et al., 2019). Later in 1981, in the trial of John W Hinckley, computed tomography (CT) was used as an argument to prove Hinckley’s schizophrenia; the evidence was permissible, and as a result, Hinckley was “found not guilty by reason of insanity (NGRI)” (Aono et al., 2019). In *People v. Weinstein*, 1992, positron emission tomography (PET) was used to prove Weinstein experienced brain function disruption and abnormal mental state while committing the murder, and Weinstein was found guilty but with and alleviated sentence (Aono et al., 2019). The legal systems relating to neurological/psychological testimonies were further improved in later years. For example, *Daubert v. Merrell Dow Pharmaceuticals, Inc.* in 1993, “Congress had passed the Federal Rules of Evidence (FRE), which offered a more liberal standard for allowing scientific testimony to enter trial” and set this case as the new standard, the “*Daubert* standard” (Aono et al., 2019). As the studies of neuropsychology advanced, a greater frequency of neurological/psychological testimonies appeared in court. According to the courts in England and Wales, from the years of 2005 to 2012, the number of cases reported using neuropsychological evidence increased significantly, from minimum 15 cases in 2005 to 32 cases in 2012 with maximum 45 cases in 2010 (Catley & Claydon, 2015). Among 204 cases using neuropsychological defense, 57 were homicides (27.9%), 44 of which were first degree murder (21.6%); 37 were violent crimes (18.1%) and among these, 24 were more severe crimes; 33 were crimes of dishonesty (16.2%); 30 were sexual offenses (14.7%); and the rest involved driving, drug, and other offenses (Catley & Claydon, 2015). Also, the evidence was used in different circumstances: “appeal against conviction (61 cases, 29.9% of the cases), appeal against sentence (92 cases, 45.1%), appeal against both conviction and sentence (20 cases, 9.8%), resisting extradition (11 cases, 5.4%), and resisting [increased] sentences (8 cases, 3.9%)” (Catley & Claydon, 2015).

Reasons/Tests for Insanity Defense

To determine a litigant’s mental state, test examinations are needed. There are often four types of tests used for an insanity defense: the M’Naghten rules, the irresistible impulse test, the Model Penal Code, and Durham test (Letman, 1982). The M’Naghten Insanity Defense is named after Daniel M’Naghten, who committed murder under the paranoid delusion that the Prime Minister of England intended to kill him, so he was found NGRI (*Queen v. M’Naghten*, 1843). The M’Naghten Insanity Defense is a cognitive test, which first requires that the person suffer from a mental defect when the crime is committed, and due to this defect, the person is unable to recognize the nature or quality or the wrongfulness of the criminal act (University of Minnesota, 2015). Irresistible Impulse Insanity Defense is a supplementation of M’Naghten Insanity Defense which also requires the person to suffer from a mental defect, but focuses on cognition and the ability to control conduct and involves volition: if the person does not have the capacity to control his/her conduct due to the defect, then the person is excused despite he/she is aware that the act is wrong (University of Minnesota, 2015). The Model Penal Code was completed in 1962 and created the Substantial Capacity Test, which argues that “[a] person is not responsible for criminal conduct if at the time of such conduct as a result of mental disease or defect he lacks substantial capacity either to appreciate the criminality [wrongfulness] of his conduct or to conform his conduct to the requirements of law” (University of Minnesota, 2015). The Model Penal Code also requires

the mental defect and both cognitive and volitional standard, but instead of losing the total capacity, the person is lacking substantial capacity (University of Minnesota, 2015). The Durham Insanity Defense, also called the product test, is currently only used in New Hampshire, and was named after *Durham v. U.S.*, which found that “an accused is not criminally responsible if his unlawful act was the product of mental disease or mental defect” (*Durham v. U.S.*, 1954). The Durham Insanity Defense requires a more psychological/objective standard mental defect and causation, in which the person’s criminal act resulted from his/her mental defect (University of Minnesota, 2015).

Types of Specialists to Make Diagnoses/Defense

A third party often hires neuropsychologists, neurologists, psychiatrists, or psychologists as an expert witness to help perform an independent forensic neuropsychological examination, present a professional measurement, or prove the presence of brain injury (McWilliams et al., 2020). According to the survey of the members of the National Academy of Neuropsychology and the Association of Trial Lawyers of America, 52% of attorneys hire neuropsychologists and 46% of attorneys hire neurologists in most or all (76%-100%) of their cases involving brain injury (Essig et al, 2001). According to the 2005 Mental Capacity Act of England and Wales, among 114 of these brain specialist professionals hired, 39 are psychiatrists (34.2%) and 33 are psychologists (28.9%) (McWilliams et al., 2020). Other professionals may be hired in a few circumstances, such as rehabilitative medicine doctors, practitioners, therapists, etc. (McWilliams et al., 2020). The experts may be asked to manage examinations, write reports, give depositions and testimony, and instruct attorneys with knowledge of neuropsychology (Essig et al, 2001). The result of the assessments must be used in an appropriate way by the third party: if the information is used improperly, such as sharing the same information with the litigant or litigant’s family, the test result may be invalid and discreditable in court (Essig et al, 2001). It is the expert’s responsibility to ensure such examination is recommended and taken appropriately; if the third party insists in misusing the information, the expert may consider taking a referral (Essig et al, 2001). The experts may also serve as the third-party observer, helping to ensure the litigant is not questioned on improper matters, such as liability, and to check the validity of the administration of examination procedures as well as other expert witnesses’ opinions (Essig et al, 2001).

Schizophrenia

Schizophrenia is a serious and enfeebling mental disorder. Schizophrenia often shows positive symptoms, such as hallucination and delusion, and negative symptoms, such as anhedonia, anergia, alogia, avolition, and affective flattening (Woo, 2014). People with schizophrenia often experience lack of pleasure, energy, speech, desire, and emotional reaction. Cognitive deficits may come along with the disorder, disturbing working memory, attention, and executive functions (Woo, 2014). Schizophrenia affects about 1% of the population worldwide (Woo, 2014). Among the people who are diagnosed, half of them suffer from sporadic but long-term mental problems, and one fifth of them have disabilities and chronic symptoms (Owen et al., 2016). Suicide is the most serious symptoms: suicide rate of a schizophrenic during his/her lifetime is 4%-13%, average 10%, while suicide attempt rate is high to 18%-55% (Sher & Khan, 2019).

There are usually three phases of schizophrenia: prodromal, psychotic, and residual (Girdler et al., 2019). The prodromal phase occurs 4.3-6.7 years before the first onset and has symptoms of function decline and sensation/language/motor disturbance; the psychotic phase is the first onset of psychosis, lasting for 5-10 years and having increased symptoms and risk of suicide; the residual phase is chronic, 5-10 years after the first onset, having decreased disorder progression and residual “eruptive” symptoms (Girdler et al., 2019). Schizophrenia’s onset typically begins in late adolescence and early adulthood (18-30 years old) (Woo, 2014). People with schizophrenia at a younger age may experience more negative symptoms, more relapses, and poorer social functioning (Immonen et al., 2017). Schizophrenic people need to get diagnosed as soon as possible, which may alleviate the symptoms and other deficits. According to the Diagnostic or Statistical Manual of the American Psychiatric Association, to be diagnosed with

schizophrenia, the patient must at least show some of the symptoms for a duration each month for six months, which may be the prodromal or residual phases (Girdler et al., 2019).

Etiology

The etiology (cause) of schizophrenia is unclear, but there are some factors involved (Girdler et al., 2019). 1) Neuro-development: schizophrenia may develop in utero, but onset often happens during adolescence due to the pruning of gray matter in the prefrontal cortex (Woo, 2014). 2) Genetics: inherent genes consist of 80% of the risk of having schizophrenia (Girdler et al., 2019). Schizophrenia is polygenic, affected by over 100 genetic loci. The risk of having it is pleiotropic, which may also cause autism spectrum disorder (ASD), attention-deficit/hyperactivity disorder (ADHD), bipolar disorder, depression, etc. (Owen et al., 2016). 3) Other environmental factors: pregnancy, parenting, social class, urbanity, trauma/social relation, migration, drug use, disease/virus, etc. (Stilo & Murray, 2019).

Neuroanatomy

The brain of people with schizophrenia is impaired in many different regions. People with schizophrenia have a smaller volume of frontal lobe (Wible, 2001), which is in charge of high-level critical thinking and managing other parts of the brain. The damage of the prefrontal cortex causes the deficits in working memory and executive functions (Owen et al., 2016). Also, the brain of schizophrenic people is found to have less gray matter in the temporal lobe (Vita et al., 2012), which includes hippocampus, thalamus, auditory cortex, and language center. Hippocampus is in charge of memory and sorting it into long-term memory. The damage of hippocampus and closely-related prefrontal cortex causes the deficit in working memory (Harrison, 2004) and forming more false memory (Fairfield et al., 2016). Thalamus primarily sorts and sends “data” to other parts of the brain, and its damage may cause cognitive deficit (Andreason, 1997). Auditory cortex dysfunctions take part in producing auditory hallucination along with the frontal lobe (Shi, 2007). Wernicke’s area and Broca’s area (in the frontal lobe) are the language center and are both damaged due to schizophrenia, causing language deficit and also taking part in the auditory hallucination (Curcic-Blake, 2013).

Pathophysiology

Schizophrenia is caused by abnormal levels of certain neurotransmitters. Dopamine is an excitatory neurotransmitter involved in reward mechanisms, attention, and motor control. The hyperactivity of dopamine due to the disturbance of the cortical pathway of the nucleus accumbens increases the activation of D2 receptors, which lead to the positive symptoms, delusions and hallucinations (Brisch et al., 2014). The low dopamine in the prefrontal cortex leads to decreased activation of D1 receptors and reduced activation of the nucleus caudatus, which trigger the negative symptoms (Brisch et al., 2014). Serotonin is primarily an inhibitory neurotransmitter involved in mood, sleep and appetite. People with schizophrenia have high-levels of serotonin which cause the negative symptoms (Risch, 1996). Norepinephrine is an excitatory neurotransmitter involved in alertness, wakefulness, and memory consolidation, and the research has shown that people with relapsed schizophrenia have higher levels of norepinephrine than that of people without relapses (Nagamine, 2020). Other excitatory neurotransmitters like glutamate are also at a high-level in schizophrenia (McCutcheon et al., 2020). Ablation of N-methyl-D-aspartate (NMDA), a glutamate receptor, on parvalbumin (PV) neurons, an inhibitory neuron, causes impaired working memory (Woo, 2014). Low levels of inhibitory neurotransmitters, gamma-aminobutyric acid (GABA), are also seen, so treatments may involve increasing GABA to alleviate the symptoms of schizophrenia (Yang & Tsai, 2017).

Treatments

Due to the high-level of activation of dopamine D2 receptors occurring in schizophrenia, typical treatment often blocks the D2 receptors, such as fluphenazine and haloperidol (Girdler et al., 2019). There are also the atypical antipsychotics, primarily D2 antagonists, such as quetiapine and risperidone; however, these do not solely target dopamine pathways and may have effects on serotonin and other important dopamine receptors (Girdler et al., 2019). Therefore, extrapyramidal and endocrine side effects are involved, such as rigidity, dystonia, tremor, akathisia, galactorrhea, and hyperprolactinemia (Girdler et al., 2019). For treatment resistant schizophrenia (TRS), clozapine is “shown to be superior to all other antipsychotics,” superseding both typical and atypical treatments (Nucifora, 2019). For those 30% of people who do not respond to clozapine, clozapine augmentation, utilizing medications such as risperidone, may enhance the weak D2-antagonistic properties of clozapine, but the result is limited and more research is needed (Nucifora, 2019). Other therapies like “electroconvulsive therapy (ECT), repetitive transcranial magnetic stimulation (rTMS), [...] deep brain stimulation (DBS)[,] cognitive behavioral therapy (CBT)[, and] cognitive remediation (CR)” may help along with clozapine in alleviating the symptoms (Nucifora, 2019). Treatment of the high-level of NMDA, such as phencyclidine, may also help to reduce the symptoms (Woo, 2014).

Legal Cases

Washington v. Harper

Washington v. Harper was argued on October 11, 1989 and was decided on February 27, 1990. The petitioner was Washington state and was advocated by William L. Williams and Paul J. Larkin, Jr.; the respondent was Walter Harper and was advocated by Brian Reed Phillips. The opinion was delivered by Justice Anthony M. Kennedy. In 1976, Harper was imprisoned for robbery. Initially, Harper was diagnosed with manic depression while first imprisoned, but later in December 1983 was diagnosed with schizoaffective disorder. During imprisonment, he was receiving antipsychotic drugs, which are given to treat mental disorders such as schizophrenia. These medications would affect the chemical balance in the brain in order to assist the patient to retain his/her thinking process and rational mind state. Due to the violent actions during parole, Harper was sent to the Special Offender Center (SOC) where they treat convicted felons with mental illnesses. In November 1982, Harper refused to take medications. In SOC Policy, an inmate is subject to a hearing with psychiatrists, and the inmate must comply with prescribed medication if a majority of psychiatrists in the hearing agree that the inmate “(1) suffers from a ‘mental disorder’ and (2) is ‘gravely disabled’ or poses a ‘likelihood of serious harm’ to himself, others, or their property” (*Washington v. Harper*, 1990). In the case of Harper, the treating physician insisted on the medication process despite the respondent’s objections. Since November 23, 1982, Harper unwillingly received medication for a year, and the medication continued after he was transferred to SOC the second time due to his deteriorated condition without treatment in Washington State Reformatory. In February 1985, Harper sued several of the State’s defendants under 42 U. S. C. § 1983 and declared that “the failure to provide a judicial hearing before the involuntary administration of antipsychotic medication violated the Due Process, Equal Protection, and Free Speech Clauses of both the Federal and State Constitutions, as well as state tort law” (*Washington v. Harper*, 1990). The trial court refused his suit though they acknowledged his right to not be subjected to involuntary antipsychotic medications. However, the Washington Supreme Court reversed and remanded, concluding that according to the Due Process Clause of the Fourteenth Amendment—which prohibits arbitrarily depriving “life, liberty, or property” unless authorized by law—it is possible for the State to involuntarily give antipsychotic medication to a capable inmate if, “in a judicial hearing at which the inmate had the full panoply of adversarial procedural protections, the State proved by ‘clear, cogent, and convincing’ evidence” (*Washington v. Harper*, 1990).

Clark v. Arizona

Clark v. Arizona was argued on April 19, 2006 and was decided on June 29 in the same year. The petitioner was Eric Clark and was advocated by David I. Goldberg; the respondent was Arizona state and was advocated by Paul D. Clement and Randall M. Howe. The opinion was delivered by Justice David H. Souter. In the early morning of June

21, 2000, Clark was pulled over by Officer Jeffrey Moritz due to circling around a residential area with loud music. Clark soon shot Officer Moritz after the officer informed him to remain at the same spot. Officer died soon after calling for help from the police dispatcher. Clark was arrested later on an on-foot escape, and gunpowder residue was found on his hands, and the gun that killed the officer was found nearby. Clark was charged for first-degree murder. Clark received treatment at a state hospital in March 2001 because he was incompetent to stand a trial, and two years later, he was released and asked to stand the trial for restored competence. Clark admitted the shooting and the death of Officer Moritz, but he denied having certain intention to kill a law enforcement officer or acknowledging he took such action. In the defense case, Clark claimed to have mental illness, which was paranoid schizophrenia. He first proposed an affirmative defense of insanity, which claimed that “at the time of the commission of the criminal act [he] was afflicted with a mental disease or defect of such severity that [he] did not know the criminal act was wrong” (*Clark v. Arizona*, 2006). He then refuted the evidence of the requisite *mens rea*—criminal intent or intentional and knowing murder—brought by prosecution. To prove his schizophrenia, Clark’s attorneys used the testimony from his family, schoolmates, local officials, and experts to demonstrate he had displayed abnormal behavior over the year before the crime. For example, he had delusions that made him believe there were aliens in town which he had to defend using guns, and that he was affected by the delusions of aliens when the crime happened. The experts concluded that Clark was morally incapable and cognitively incapable to lure the officer nor understand the right or wrong due to insanity when the crime happened, which fulfilled the M’Naghten rule by being unable to know “the nature and quality of the act” (*Clark v. Arizona*, 2006). However, a psychiatrist of the State argued that Clark was still able to acknowledge the wrongfulness of his act under schizophrenia, which could be proven by his act preceding or following the crime: playing loud music, escaping, and hiding the gun. At the close of the defense case, Clark and his team were unable to prove that Clark was not aware that the victim was an officer, and therefore the judge issued a verdict of first-degree murder and the sentence of life imprisonment without release under 25 years. Though Clark suffered from paranoid schizophrenia, the judge noted, the mental disorder “did not . . . distort his perception of reality so severely that he did not know his actions were wrong” (*Clark v. Arizona*, 2006).

Colorado v. Connelly

Colorado v. Connelly was argued on October 8, 1986 and was decided on December 10 the same year. The petitioner was Colorado state and was advocated by Nathan B. Coats and Andrew J. Pincus; the respondent was Francis Connelly and was advocated by Thomas M. Van Cleave, III. The opinion was delivered by Chief Justice William H. Rehnquist. In August 18, 1983, Connelly found the Denver Police Department and confessed to Officer Patrick Anderson about him committing a murder. Anderson immediately warned Connelly he had Miranda rights, which is “to remain silent, that any statement he does make may be used as evidence against him, and that he has a right to the presence of an attorney, either retained or appointed” (*Miranda v. Arizona*, 1966). Connelly waived his rights, meaning he insisted on confessing after knowing and understanding the rights. Later he continued admitting to the murder of Mary Ann Junta to Anderson and Detective Stephen Antuna and showed them the crime scene though he was warned again about the Miranda rights. However, after he was held for a night, he seemed to be disoriented and claimed that he was guided to confess. Connelly was psychologically assessed and was deemed unfit to stand trial, and could not hold his defense until March of 1984. At the preliminary hearing, Connelly asked to suppress all his statements. The state hospital hired psychiatrist Dr. Jeffrey Metzner, who testified that at least the day before confession, Connelly suffered from chronic schizophrenia and had “command hallucinations” where he heard a “god voice” ordering him to either confess or commit suicide. Through this reasoning, Dr. Metzner argued that Connelly confessed involuntarily. Dr. Metzner indicated that schizophrenia did not impair Connelly’s cognitive abilities and that he was still able to understand the Miranda rights, but the disorder interfered with his “volitional abilities; that is, his ability to make free and rational choices” (*Colorado v. Connelly*, 1986). According to the evidence, the court affirmed that Connelly’s statements must be suppressed because it was not the “the product of a rational intellect and a free will” (*Colorado v. Connelly*, 1986). Although his confession was suppressed, Connelly was later found guilty and sentenced to five years and ten months

in prison, followed by one year of parole. This was a shorter sentence than normal, due to his schizophrenic defense (Pizzi, 2009).

Discussion

Schizophrenia plays different roles in the three cases listed, and having a better biological understanding of the disorder can help to improve and expand its relevance in the legal system. In *Washington v. Harper*, schizophrenia was the cause of Harper's aggressive and violent behaviors, and Harper indeed illustrated positive symptoms, such as attacking the nurses in the hospital (*Washington v. Harper*, 1990). This may be due to the high-level of D2 dopamine which caused Harper to be over energized and aggressive. The antipsychotic medication can block the D2 receptors and help to suppress the symptoms; therefore, Harper reacted logically and cooperated to take the medications when he took it, but after Harper stopped taking medicines, his symptoms deteriorated. In *Clark v. Arizona*, Clark used schizophrenia as an insanity defense, more particularly, the M'Naghten Insanity Defense, the most common insanity defense. Clark claimed he had delusions of aliens due to schizophrenia, which might be true because delusions are one of the positive symptoms and are caused by the high-level D2 dopamine. The high dopamine may also cause poor impulse control, so this may explain why Clark murdered the officer without reason and plan. Playing loud music at night may also be a result of schizophrenia, due to the trouble of sleep caused by an abnormal level of serotonin. He may also have the impaired frontal lobe and thalamus, which causes cognitive incapability. In *Colorado v. Connelly*, Connelly had command hallucinations of god voices, this involved the malfunction of the frontal lobe and auditory cortex, causing him to hear fake voices and forcing Connelly to confess. In the later time, Connelly became logical again and appealed to suppress the statements. This may be explained by his chronic schizophrenia. During this phase, the symptoms are decreased and sometimes show "eruptive" symptoms (Girdler et al., 2019). In this case, the "eruptive" symptoms is the action of confession, and the reason for this eruption may be triggered by the stress after murder (Girdler et al., 2019). Dopaminergic neurons may be excited due to this acute stress, causing the positive symptoms, delusions and hallucinations (Bloomfield, 2019). Schizophrenia successfully helped Connelly to suppress his statements, somewhat demonstrated a possibility of acquittal for Clark, and was the main cause that held Harper from being free from involuntary medications. Schizophrenia is largely used in court to aid an insanity defense, and the complex laws surrounding it are constantly enhanced as a result of its increased use in courts.

Dissociative Identity Disorder

Dissociative Identity Disorder (DID), known as Multiple Personality Disorder (MPD) before 1994, is a post-traumatic disorder that is often portrayed as having alternate identities with multiple voices in the head, distinctive changes in behavior, etc. (Mitra & Jain, 2022). The sign of an altered state may be eye rolling, eye blinking, changes in posture, and trancelike behavior, and the switch is often triggered by high stress level or other stimulations of extreme emotions, depression, and sexual activities (Gentile et al., 2013). According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), DID is described as "an identity disruption indicated by the presence of two or more distinct personality states [...] with discontinuity in sense of self and agency, and with variations in affect, behavior, consciousness, memory, perception, cognition, or sensory-motor functioning" (Brand et al., 2016). Dissociative disorders affect about 1%-5% of the global population, and DID is a rare disorder diagnosed in around 1.5% of the population (Mitra & Jain, 2022). In some research, women are twice as likely to be diagnosed with dissociative disorders than men (Şar, 2011). DID is the most complex, severe, and chronic type of dissociative disorders (Şar, 2011). Other dissociative disorders include dissociative amnesia, depersonalization disorders, and dissociative fugue, which are different or too mild to meet the criteria of DID—for example, dissociative amnesia is not severe enough to be DID (Şar, 2011). DID has symptoms of dissociative disorders: a sense of detachment from the body and emotions, a blurred sense of self-identity, and amnesia, which cause DID to be commonly misdiagnosed as borderline personality disorder (Mitra &

Jain, 2022). DID may also show positive symptoms—new personalities and derealization—and negative symptoms—examples such as paralysis and autism (Mitra & Jain, 2022). Though the report shows 1%-2% of the patients completed suicide, people with DID may more frequently show suicide attempts and self-injurious behavior (Şar, 2011). Evaluations for DID are variable: Dissociative Experiences Scale, Dissociation Questionnaire, Difficulties in Emotion Regulation Scale, Multidimensional Inventory of Dissociation, etc. (Mitra & Jain, 2022).

Etiology

DID has multiple factors in its etiology, which includes developmental traumatization, sociocognitive sequelae, and biological factors (Şar et al., 2017). Though there is no direct proof, genetics are highly possible being involved in DID (Şar et al., 2017). DID is highly relative to childhood trauma and abusive experience exposed before the age of five (Mitra & Jain, 2022). DID shares similar dynamics with Post-Traumatic Stress Disorder (PTSD), but the trauma is discretized into multiple first-person perspectives in DID only, where the consciousness is broken between the alternate identities (Şar et al., 2017). The cause of milder DID symptoms may be a dysfunctional relationship between family members, such as emotional neglect (Şar et al., 2017). Childhood is an important period to develop self and social recognition and integration; the abundant imagination and fantasy increases the risk of dissociation (Gentile et al., 2013). Abuse and neglect increase the sense of isolation, loneliness, and alienation, which may prevent the development of ability processing the abuse (fail to integrate the abuse with other experiences) and construction of the “sense of self-in-relation-to-others” forming dissociative identities (Şar et al., 2017). Disorganized attachment style, which is developed by the child being frightened and abused by the parents who the child is seeking safety from, causes contradictory impressions of the parents being both safe and dangerous, motivating protection from abuse when the safety is sought from the parents, and this attachment style helps to establish and maintain DID (Şar et al., 2017). Alternate identities and DID are developed mainly as a means of coping with traumatic events.

Neuroanatomy

DID may cause by a “smaller hippocampal volume in patients with a history of exposure to traumatic stress and an accompanying stress-related psychiatric disorder” (Vermetten et al., 2006). In DID, smaller volumes of amygdala (emotions), hippocampus (memory), and parahippocampus (spatial memory) are found, which are majorly caused by the childhood trauma, similar with the volumes in PTSD, and are in charge of dissociative amnesia and emotional detachment (Krause-Utz et al., 2017). Dissociative amnesia in DID is significantly related to the cornu ammonis 1 (CA1) region, which is a hippocampal subfield and important to autobiographical memories (Dimitrova et al., 2021). The decreased volume in CA1 along with childhood traumatization causes the obstruction of identity development, scattered sense of self, and fragmentation of mind (Dimitrova et al., 2021). Moreover, people with both PTSD and dissociative subtype have less gray matter volume (GMV), compared to those with PTSD and no dissociative subtype, in the right inferior temporal gyrus (cognition, memory, and sensory integration). The “severity of depersonalization and derealization” is associated with increased GMV in the right middle frontal gyrus (attention). In addition, studies have shown “positive associations between trait dissociation and GMVs in medial/lateral [prefrontal cortex], orbitofrontal, temporal polar, parahippocampal, and inferior parietal cortices”, which are related to emotion regulations (Krause-Utz et al., 2017). These deficiencies further explained symptoms of failure of self-integration and emotional indifference and dissociation. Furthermore, increased activation in the dorsolateral prefrontal and parietal cortex, mainly associated with memory, and functional and blood-flow reduction in the orbitofrontal cortex (decision making in emotions, tastes, and rewards) may explain the symptoms of changes and forgetfulness of alternate identities (Rutkofsky et al., 2017).

Pathophysiology

Hippocampus is the target of glucocorticoids, which is released when stressed (Vermetten et al., 2006), and is sensitive to the release of stress hormones, which may explain the identity change when experiencing a high level of stress (Krause-Utz et al., 2017). There is a hypothesis that long exposure to glucocorticoids may cause the atrophy in the hippocampus (Vermetten et al., 2006). And this is also the reason for the high similarity between DID and other stress-related psychiatric disorders (Vermetten et al., 2006). Moreover, ketamine, the antagonist of NMDA, is highly concentrated in the hippocampus as well, resulting in dissociative symptoms, such as feeling detached from the body and amnesia (Vermetten et al., 2006). Other neurotransmitters, such as glutamate (major excitatory neurotransmitter), serotonin (mood), and endogenous opioids (analgesia, euphoria induction, stress resilience) (Shenoy & Lui, 2022) may also affect on the volume of hippocampus and amygdala, and these neurotransmitters may associate with the dissociation, but more study is needed to clarify the relationships between DID and these neurotransmitters (Vermetten et al., 2006).

Treatments

The goal of treatment of DID is not to eliminate alter identities but to integrate and adapt them to be a strong overall personality structure (Gillig, 2009). Therapy for DID often has three stages. Stage one is to establish safety and reduce suicide attempts and self-mutilate behaviors; stage two is to cope and ameliorate symptoms of trauma, sharing the memory with alter identities; stage three is to develop and improve self and social integration and rehabilitate (Huntjens et al., 2019). There are also three major therapies: cognitive behavioral therapy (CBT), dialectical behavioral therapy (DBT), and Eye Movement Desensitization and Reprocessing (EMDR) (Mitra & Jain, 2022). CBT helps to recognize and cope with negative thoughts (NAMI, 2015). DBT helps to control suicidal actions and regulate emotions, and is primarily used to treat borderline personality disorder (Foote & Van, 2016). EMDR is not suggested when the patient has sufficient coping skills; it allows the patient to alternate identities and replaces the negative memory with positive beliefs or reactions (Mitra & Jain, 2022). Hypnosis is also considered as a treatment, with which can access and facilitate the appearance of alternate identities, and people with DID are easier to be hypnotized than other clinical populations (Mitra & Jain, 2022). Medications, such as mood stabilizers, antipsychotic medications, and stimulants, are not superior and highly effective in treating DID (Mitra & Jain, 2022).

Legal Cases

State v. Badger

State v. Badger was decided on October 13, 1988. The petitioner was New Jersey state and was advocated by Denyse Coyle Galda; the respondent was Christopher J. Badger and was advocated by Daniel Baer. The opinion was delivered by Justice Kuechenmeister. Christopher Badger attempted burglary and was arrested on May 1, 1986, which was on the day after his release from Southern State Correctional Facility, where he served a sentence for seven years due to the similar charges. Badger was diagnosed with MPD since the age of seventeen, and he had completely no control and memory of his actions whenever the alternate identity was in control. At the time of attempted burglary, Badger's alternate identity "Philip" was controlling the body and could remember and describe the night, while the dominant identity "Christopher" had no control nor the recollection of the actions committed by "Philip." Badger was incarcerated after the arrest, and he was transferred to the forensic unit of the Bergen Pines Hospital in January 1987 due to attempted suicide in jail. In the hospital, he was evaluated by Dr. Peter Martindale and was found that he genuinely had two competent, distinctive identities, "Christopher" and "Philip," who both knew right from wrong. On August 10, Badger was found incompetent to complete trial; however, when he was later in Greystone, he was found competent despite his MPD because he acted non-delusional, coherent, and organized. Badger clearly acknowledged the charges against him, the facts of the arrest, the pleas available, and the potential penalties. Nonetheless, Martindale was still hesitant about whether Badger was capable of standing the trial because first, only "Philip" could recall the

event on May 1 and properly aid his attorney in the defense, and second, there was a possibility that Badger would switch identities between “Christopher” and “Philip” during the trial, causing either of them to not know what had transpired before the switch. The court found that both his identities had “the mental capacity to appreciate his presence in relation to time, place and things,” so the problem following the switch may be resolved by Badger’s attorney explaining the events prior to the switch (*State v. Badger*, 1988). Also, the court found this case to be highly similar with cases of amnesia, which “would not ‘render a defendant unable to comprehend his position or to consult intelligently with counsel in preparation of his defense’,” so the same principle must apply, that was, the respondent Christopher Badger had the capability to stand the trial (*State v. Badger*, 1988).

State v. Grimsley

State v. Grimsley was decided on February 10, 1982. The petitioner was Ohio state and was advocated by Mr. Simon L. Leis, Jr.; the respondent was Ms. Grimsley and was advocated by Mr. John W. Hauck. The opinion was delivered by Justice Black. Ms. Grimsley was convicted of driving under alcohol violation, and she was tested having 0.21% of alcohol in blood. She claimed four assignments of error, and two of them related to MPD. In Grimsley’s second assignment of error, she contended that she was not responsible for the violation because the alternate identity “Jennifer” was in control at the time of violation, and the dominant identity “Robin” was dissociated. Therefore, she stated she was acting unconsciously and involuntarily, which cannot be charged with guilt. It was true that, according to the expert psychiatric testimony admitted by the prosecution, Grimsley indeed was diagnosed with MPD and was having psychotherapy since June 1977. She claimed that the development of “Jennifer,” who was angry, impulsive, anxious, fearful, and having a drinking problem, was due to the psychological trauma that she was informed about a lump on her breast. Grimsley pleaded for acquittal, not an insanity defense, because during the violation, “Jennifer” was in control, “Robin” had no memory nor control of “Jennifer’s” actions, so the actions were committed involuntarily and unconsciously. However, the court disagreed. Assuming “Jennifer” was distinct to “Robin”, there was still no evidence indicating that “Jennifer” was committing the offense unconsciously and involuntarily. As long as she was conscious in one state, the actions were the product of her own offense. Grimsley’s other contention was that “Robin” had minimal memory of “Jennifer’s” actions, so “Robin” was unable to answer the questions about the violation and was not liable to the conduct. The court was not persuaded because “[if the court was] to allow the bare existence of a defendant’s [MPD] to excuse criminal behavior, [the court] would also relieve from responsibility for their criminal acts all defendants whose memories are blocked” (*State v. Grimsley*, 1982). The second assignment of error was found to have no merit. In Grimsley’s third assignment of error, she claimed that it was erroneous that the court found that she failed to argue the insanity defense, because it was undeniable that “Robin” did not acknowledge the wrongfulness of “Jennifer’s” actions nor could prevent the actions. The court claimed that the evidence could not prove that MPD impaired Grimsley’s reason that either or both identities “either did not know that her drunken driving was wrong or did not have the ability to refrain from driving while drunk” (*State v. Grimsley*, 1982). The third assignment of error was found to have no merit. The case, in the end, was reversed and remanded (*State v. Grimsley*, 1982).

Discussion

DID was considered when deciding competency to stand a trial and was used as an insanity defense in the cases listed, and the biological aspect may explain the details within these legal cases. In *State v. Badger*, Badger was unable to recall the attempted burglary, and this might be caused by the decreased volume in the hippocampus, especially CA1 region, which is associated with dissociative amnesia (Dimitrova et al., 2021). Other regions, dorsolateral prefrontal, parietal, and orbitofrontal cortex may also be involved for the same reason (Rutkofsky et al., 2017). During incarceration, Badger attempted to commit suicide, which illustrates the high suicide-attempting rate of people with DID. Martindale’s worry of an identity switch during court is reasonable because an identity switch is indeed uncontrollable and may be caused by the high stress level due to the sensitivity of glucocorticoids in the atrophied hippocampus (Vermetten et al., 2006). In *State v. Grimsley*, dissociative amnesia also occurred since “Robin” could not recall the

actions of “Jennifer”, which is also highly associated with the reduced volume in the hippocampus and other significant regions mentioned and high concentration of ketamine in the hippocampus relating to the change of identities and amnesia (Vermetten et al., 2006). An interesting point in this case is that DID of Grimsley was developed due to the surprise acknowledgment of a lump in her breast in adulthood, which is different from the popular etiology of DID which is childhood trauma and long-term abuse. “Jennifer” may have been developed in order to cope with the traumatic event; leading “Jennifer” to become alcoholic and have other negative emotions. In both cases, the amnesia aspect of DID seemed to be the major concern in court rather than the logical and moral acknowledgment of the crime, and these two cases were treated in the principle of amnesia that though people cannot remember the action, people are still conscious and voluntary, and are therefore competent to stand the trial and unlikely to be acquitted.

Besides the cases listed, there are other cases involving DID, such as the case of Billy Milligan. In October, 1977, 23-year-old Milligan committed three rapes in Ohio State University, and prior to those, he also committed a couple of robberies. After he was arrested, police observed several times of Milligan’s identity changes, such as accents, speech patterns, and movement habits. Later, after multiple assessments and interviews, Milligan was diagnosed with MPD. The identity that committed robbery was “Ragen”, a Yugoslavian age 23, and the identity that committed three campus rapes lesbian “Adalana” age 19. The other identities did not have memory of these crimes at the time of being committed. In fact, including the primary identity “Billy”, there were 24 identities inside the body of Milligan. The cause of Milligan’s scattered identities was his abusive childhood, based on Milligan’s memory: his biological father committed suicide when Milligan was 4 years old, and after his mother remarried and divorced a couple of times, Milligan was sodomized and tortured by his new step father, who denied all those allegations and was not charged. His life was later ruined due to the MPD until he was eventually arrested. When provided treatment after his arrest, it was discovered that Milligan contained an already fused identity “The Teacher.” “The Teacher” helped other identities to learn their talents, and when “The Teacher” emerged in December, 1978, Milligan was interviewed basing *The Minds of Billy Milligan*, and “The Teacher” receded after the publicity and criticism in March 1979, and alternate identities reappeared. After a few more disturbances, Milligan was finally released and faded away from the public eye (Phillips, 2007). Again, since identities do not share memories and behavior patterns except the fused identity “The Teacher,” Milligan’s hippocampus and CA1 region may have smaller volume, and dorsolateral prefrontal, parietal, and orbitofrontal cortex and ketamine may be also affected (Rutkofsky et al., 2017). Clearly, Milligan’s alternate identities were developed due to long-term childhood abuse, which derived from a disorganized attachment style and emotional neglect, and he displayed symptoms such as dissociative amnesia and blurred sense of self-identities, which are likely to be more severe due to the rejections he experienced in his later life. Moreover, the retreatment of “The Teacher” is most likely due to the public criticism, which induced Milligan to have a high stress-level, further exacerbated by a high sensitivity to glucocorticoids, and cause an identity switch to occur. In addition, “Billy” attempted several suicides when he was in control of the body (Keyes, 1981). This again suggests that people with DID have a tendency to commit suicide. In order to protect Milligan from “Billy’s” radical behavior, other identities often keep “Billy” asleep, and there is a “David” developed to endure pain for other identities and a “Danny” who holds the memory of being buried alive by Milligan’s stepfather. This suggests that DID is indeed a coping mechanism with traumatic events. Still, there are skeptics about the truthfulness of the story of Billy Milligan and more research on DID is needed.

Conclusion

In today’s federal court cases, forensic neuropsychology is more developed, and the appearance of mental disorders is more frequent. The appearance of forensic neuropsychology is quite late compared to other methods used in courts, since the clinics and technology involved developed late. In courts, forensic neuropsychology is often used in an insanity defense, and four major rules for the defense are: the M’Naghten insanity defense, the irresistible impulse insanity defense, the Model Penal Code, and Durham insanity defense. Within the defense, neuropsychologists and other types of experts are hired to give testimony and conduct the assessments for the litigant, which is to identify the

presence of mental disorders. This paper picks two of the disorders to elaborate: schizophrenia and DID. Schizophrenia, often caused by an abnormal level of dopamine and smaller volume of the frontal lobe, triggers the person to have symptoms such as hallucinations, delusions, avolition, anhedonia, etc. In the legal examples, schizophrenia serves as the cause of involuntary medication, an insanity defense, and the reason to suppress a waiver of rights. DID, often caused by a traumatic childhood and smaller volumes of hippocampus and amygdala, triggers the person to have symptoms such as presence of alternate identities, distinctive change of behaviors, dissociative amnesia, etc. In the legal examples, DID serves as a concern of capability to stand a trial and an insanity defense. Both of the disorders may still argue for retaining the litigants' rights but are unlikely to successfully argue for not guilty by reason of insanity. Future studies on both of the disorders are needed, especially the etiology of schizophrenia and pathophysiology of DID, and the legal systems involving mental disorders may be revised and improved. This paper simply demonstrates how mental disorders are used in legal cases by providing some basic information and giving two examples explaining both biological and legal aspects. This paper may introduce the fundamental knowledge of forensic neuropsychology in courts and encourage the reader to explore this area.

Acknowledgments

I would like to thank my advisor for the valuable insight provided to me on this topic.

References

- Andreasen NC. The role of the thalamus in schizophrenia. *Can J Psychiatry*. 1997 Feb;42(1):27-33. doi: 10.1177/070674379704200104. PMID: 9040920.
- Aono, D., Yaffe, G. & Kober, H. Neuroscientific evidence in the courtroom: a review. *Cogn. Research* 4, 40 (2019). <https://doi.org/10.1186/s41235-019-0179-y>
- Bloomfield, M. A., McCutcheon, R. A., Kempton, M., Freeman, T. P., & Howes, O. (2019). The effects of psychosocial stress on dopaminergic function and the acute stress response. *eLife*, 8, e46797. <https://doi.org/10.7554/eLife.46797>
- Brand, B. L., Sar, V., Stavropoulos, P., Krüger, C., Korzekwa, M., Martínez-Taboas, A., & Middleton, W. (2016). Separating Fact from Fiction: An Empirical Examination of Six Myths About Dissociative Identity Disorder. *Harvard review of psychiatry*, 24(4), 257–270. <https://doi.org/10.1097/HRP.000000000000100>
- Brisch R, Saniotis A, Wolf R, Bielau H, Bernstein H-G, Steiner J, Bogerts B, Braun K, Jankowski Z, Kumaratilake J, Henneberg M and Gos T (2014) The role of dopamine in schizophrenia from a neurobiological and evolutionary perspective: old fashioned, but still in vogue. *Front. Psychiatry* 5:47. doi: 10.3389/fpsy.2014.00047
- Catley, P., & Claydon, L. (2015). The use of neuroscientific evidence in the courtroom by those accused of criminal offenses in England and Wales. *Journal of law and the biosciences*, 2(3), 510–549. <https://doi.org/10.1093/jlb/lsv025>
- Clark v. Arizona*, 548 U.S. 735, 766 (2006).
- Colorado v. Connelly*, 479 U.S. 157, 167, 107 S.Ct. 515, 93 L.Ed.2d 473 (1986).
- Criminal Law*. Minneapolis, MN: University of Minnesota Libraries Publishing. (2015).
- Curcic-Blake, B., Liemburg, E., Vercammen, A., Swart, M., Knegtering, H., Bruggeman, R., & Aleman, A. (2013). When Broca goes uninformed: reduced information flow to Broca's area in schizophrenia patients with auditory hallucinations. *Schizophrenia bulletin*, 39(5), 1087–1095. <https://doi.org/10.1093/schbul/sbs107>
- Dimitrova, Lora I; Dean, Sophie L; Schlumpf, Yolanda R; Vissia, Eline M; Nijenhuis, Ellert R S; Chatzi, Vasiliki; Jäncke, Lutz; Veltman, Dick J; Chalavi, Sima; Reinders, Antje A T S (2021). A neurostructural biomarker

- of dissociative amnesia: a hippocampal study in dissociative identity disorder. *Psychological Medicine: Epub ahead of print*.
- DOI: <https://doi.org/10.1017/S0033291721002154>
- Durham v. U.S.*, 214 F.2d 862, 875 (1954), accessed July 5, 2022, http://scholar.google.com/scholar_case?case=1244686235948852364&hl=en&as_sdt=2&as_vis=1&oi=scholar.
- Dye, S. (2011, June 13). *Court Cases*. Retrieved July 5, 2022, from <https://forensicpsych.umwblogs.org/psychology-and-law/court-cases/>
- Fairfield, B., Altamura, M., Padalino, F. A., Balzotti, A., Di Domenico, A., & Mammarella, N. (2016). False Memories for Affective Information in Schizophrenia. *Frontiers in psychiatry*, 7, 191. <https://doi.org/10.3389/fpsyt.2016.00191>
- Foote, B., & Van Orden, K. (2016). Adapting Dialectical Behavior Therapy for the Treatment of Dissociative Identity Disorder. *American journal of psychotherapy*, 70(4), 343–364. <https://doi.org/10.1176/appi.psychotherapy.2016.70.4.343>
- Gentile, J. P., Dillon, K. S., & Gillig, P. M. (2013). Psychotherapy and pharmacotherapy for patients with dissociative identity disorder. *Innovations in clinical neuroscience*, 10(2), 22–29. DOI: 10.1002/da.20874
- Gillig P. M. (2009). Dissociative identity disorder: a controversial diagnosis. *Psychiatry (Edgmont (Pa. : Township))*, 6(3), 24–29.
- Girdler, S. J., Confino, J. E., & Woesner, M. E. (2019). Exercise as a Treatment for Schizophrenia: A Review. *Psychopharmacology bulletin*, 49(1), 56–69.
- Harrison, P.J. The hippocampus in schizophrenia: a review of the neuropathological evidence and its pathophysiological implications. *Psychopharmacology* 174, 151–162 (2004). <https://doi.org/10.1007/s00213-003-1761-y>
- Hom, J. (2003). Forensic Neuropsychology: are we there yet? *Archives of Clinical Neuropsychology*, 18(8): 827–845, [https://doi.org/10.1016/S0887-6177\(03\)00076-3](https://doi.org/10.1016/S0887-6177(03)00076-3)
- Horton, A. M., Jr., & Hartlage, L. C. (2010). *The handbook of forensic neuropsychology* (2nd ed.). Springer.
- Huntjens, R., Rijkeboer, M. M., & Arntz, A. (2019). Schema therapy for Dissociative Identity Disorder (DID): rationale and study protocol. *European journal of psychotraumatology*, 10(1), 1571377. <https://doi.org/10.1080/20008198.2019.1571377>
- Immonen, J., Jääskeläinen, E., Korpela, H., & Miettunen, J. (2017). Age at onset and the outcomes of schizophrenia: A systematic review and meta-analysis. *Early intervention in psychiatry*, 11(6), 453–460. <https://doi.org/10.1111/eip.12412>
- Iritani S. (2013). What happens in the brain of schizophrenia patients?: an investigation from the viewpoint of neuropathology. *Nagoya journal of medical science*, 75(1-2), 11–28.
- Keyes, D. (1981). *The Minds of Billy Milligan*. New York: Random House.
- Krause-Utz, A., Frost, R., Winter, D., & Elzinga, B. M. (2017). Dissociation and Alterations in Brain Function and Structure: Implications for Borderline Personality Disorder. *Current psychiatry reports*, 19(1), 6. <https://doi.org/10.1007/s11920-017-0757-y>
- Letman, S. T. (1982). *Future of the Defense of Legal Insanity*. <https://www.ojp.gov/ncjrs/virtual-library/abstracts/future-defense-legal-insanity-future-criminal-justice-p-178-186#:~:text=There%20are%20several%20tests%20for,Law%20Institute's%20Model%20Penal%20Code>.
- Mayo Clinic Staff. (2020, January 7). *Schizophrenia*. Mayo Clinic. Retrieved June, 2022, from <https://www.mayoclinic.org/diseases-conditions/schizophrenia/symptoms-causes/syc-20354443#:~:text=Schizophrenia%20is%20a%20serious%20mental,with%20schizophrenia%20require%20lifelong%20treatment>.
- McCutcheon, R. A., Krystal, J. H., & Howes, O. D. (2020). Dopamine and glutamate in schizophrenia: biology, symptoms and treatment. *World psychiatry : official journal of the World Psychiatric Association (WPA)*, 19(1), 15–33. <https://doi.org/10.1002/wps.20693>

- McWilliams A, Fleming SM, David AS and Owen G (2020) The Use of Neuroscience and Psychological Measurement in England's Court of Protection. *Front. Psychiatry* 11:570709. doi: 10.3389/fpsy.2020.570709
- Miranda v. Arizona*, 384 U.S. 436, 479, 86 S. Ct. 1602, 1630, 16 L. Ed. 2d 694 (1966).
- Mitra P, Jain A. *Dissociative Identity Disorder*. [Updated 2022 Mar 9]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK568768/>
- Nagamine T. (2020). Role of Norepinephrine in Schizophrenia: An Old Theory Applied to a New Case in Emergency Medicine. *Innovations in clinical neuroscience*, 17(7-9), 8–9.
- Nucifora, F. C., Jr, Woznica, E., Lee, B. J., Cascella, N., & Sawa, A. (2019). Treatment resistant schizophrenia: Clinical, biological, and therapeutic perspectives. *Neurobiology of disease*, 131, 104257. <https://doi.org/10.1016/j.nbd.2018.08.016>
- Owen, M. J., Sawa, A., & Mortensen, P. B. (2016). Schizophrenia. *Lancet* (London, England), 388(10039), 86–97. [https://doi.org/10.1016/S0140-6736\(15\)01121-6](https://doi.org/10.1016/S0140-6736(15)01121-6)
- Phillips, J. (2007, October 28). Multiple-personality case of Billy Milligan still fascinates. *The Columbus Dispatch*. <https://www.dispatch.com/story/news/2007/10/28/multiple-personality-case-billy-milligan/984983007/>
- Pizzi, W.T. (2009). Colorado v. Connelly: What Really Happened? *7 Ohio St. J. Crim. L.*, 377-389. Available at <https://scholar.law.colorado.edu/articles/268>.
- Psychotherapy*. (2015, February 23). NAMI. Retrieved July 28, 2022, from <https://www.nami.org/About-Mental-Illness/Treatments/Psychotherapy>
- Queen v. M'Naghten*, 10 Clark & F.200, 2 Eng. Rep. 718 (H.L. 1843), accessed July 5, 2022, http://users.php.ufl.edu/rbauer/forensic_neuropsychology/mcnaghten.pdf.
- Risch SC. Pathophysiology of schizophrenia and the role of newer antipsychotics. *Pharmacotherapy*. 1996 Jan-Feb;16(1 Pt 2):11-4. PMID: 8778681. Rutkofsky IH, Khan AS, Sahito S, Aqeel N, Tohid H (2017) The Neuropsychiatry of Dissociative Identity Disorder: Why Split Personality Patients Switch Personalities Intermittently?. *J Cell Sci Ther* 8: 267. doi:10.4172/2157-7013. 1000267
- Sar, V., Dorahy, M. J., & Krüger, C. (2017). Revisiting the etiological aspects of dissociative identity disorder: a biopsychosocial perspective. *Psychology research and behavior management*, 10, 137–146. <https://doi.org/10.2147/PRBM.S113743>
- Vedat. (2011). “Epidemiology of Dissociative Disorders: An Overview”. *Epidemiology Research International*, vol. 2011, Article ID 404538, 8 pages. <https://doi.org/10.1155/2011/404538>
- Shenoy SS, Lui F. Biochemistry, Endogenous Opioids. [Updated 2022 Jun 20]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK532899/>
- Sher, L., & Kahn, R. S. (2019). Suicide in Schizophrenia: An Educational Overview. *Medicina (Kaunas, Lithuania)*, 55(7), 361. <https://doi.org/10.3390/medicina55070361>
- Shi W. X. (2007). The auditory cortex in schizophrenia. *Biological psychiatry*, 61(7), 829–830. <https://doi.org/10.1016/j.biopsych.2007.02.007>
- State v. Badger*, 229 N.J. Super. 288, 551 A. 2d 207 (1988).
- State v. Grimsley*, 3 Ohio App.3d 265, 268-69, 444 N.E.2d 1071, 1076 (1982).
- Steven M. Essig, Wiley Mittenberg, Randy S. Petersen, Silvia Strauman, Joan T. Cooper, Practices in forensic neuropsychology: Perspectives of neuropsychologists and trial attorneys, *Archives of Clinical Neuropsychology*, Volume 16, Issue 3, April 2001, Pages 271–291, <https://doi.org/10.1093/arclin/16.3.271>
- Stilo, S. A., & Murray, R. M. (2019). Non-Genetic Factors in Schizophrenia. *Current psychiatry reports*, 21(10), 100. <https://doi.org/10.1007/s11920-019-1091-3>
- University of South Florida. (2017, December 19). *Forensic Neuropsychology*. USF Health. Retrieved June 27, 2022, from <https://health.usf.edu/medicine/neurosurgery/neuropsychology/forensic>
- Vermetten, E., Schmahl, C., Lindner, S., Loewenstein, R. J., & Bremner, J. D. (2006). Hippocampal and amygdalar volumes in dissociative identity disorder. *The American journal of psychiatry*, 163(4), 630–636. <https://doi.org/10.1176/ajp.2006.163.4.630>

- Vita, A., De Peri, L., Deste, G., & Sacchetti, E. (2012). Progressive loss of cortical gray matter in schizophrenia: a meta-analysis and meta-regression of longitudinal MRI studies. *Translational psychiatry*, 2(11), e190. <https://doi.org/10.1038/tp.2012.116>
- Washington v. Harper*, 494 U.S. 210, 227 (1990).
- Wible, C. G., Anderson, J., Shenton, M. E., Kricun, A., Hirayasu, Y., Tanaka, S., Levitt, J. J., O'Donnell, B. F., Kikinis, R., Jolesz, F. A., & McCarley, R. W. (2001). Prefrontal cortex, negative symptoms, and schizophrenia: an MRI study. *Psychiatry research*, 108(2), 65–78. [https://doi.org/10.1016/s0925-4927\(01\)00109-3](https://doi.org/10.1016/s0925-4927(01)00109-3)
- Woo T. U. (2014). Neurobiology of schizophrenia onset. *Current topics in behavioral neurosciences*, 16, 267–295. https://doi.org/10.1007/7854_2013_243
- Yang, A. C., & Tsai, S. J. (2017). New Targets for Schizophrenia Treatment beyond the Dopamine Hypothesis. *International journal of molecular sciences*, 18(8), 1689. <https://doi.org/10.3390/ijms18081689>