

# The Use of Machine and Deep Learning Algorithms in the Diagnosis of Obsessive-Compulsive Disorders: An Analysis of Meta study.

Nasra Bashir Mohamed

Department of neuroscience, uskudar University, Turkey Lecturer, Department of neuroscience, uskudar University, Turkey.

---

**Abstract:** Schizo-obsessive disorders are described by the clinical syndrome in which related obsessive compulsive disorders companions in schizophrenia. This study examined the number of neuropsychological and clinic different between schizophrenia, Schizo-obsessive compulsive, and obsessive-compulsive disorders. While using different neuroimaging machine to study methods for meta-analysis of complex brain image data, as it includes many dimensions related in brain alterations in obsessive compulsive disorders and schizophrenia patients. This research will emphasize the different between structural and functional abnormalities in the brain including Gray matter and white matter in Schizo-obsessive compulsive disorders and schizophrenia patients by doing the abnormality in the different region of the brain. Schizophrenia, Schizo-obsessive and obsessive-compulsive patients are distinguished by age, gender, methods used in different reports of the patients in order to obtain more reliable performance results.

**Keywords:** schizophrenia, Schizo-obsessive, obsessive-compulsive disorders, a meta-analysis, neuroimaging, machine learning algorithm.

---

Date of Submission: 17-06-2020

Date of Acceptance: 03-07-2020

---

## I. Introduction

Obsessive compulsive disorders is an anxiety disorder wherein an individual suffers from recurrent obsessions or compulsions, or both, Obsessions are persistent, involuntary thoughts, images, or impulse that invade consciousness and cause a person great distress. People with obsessions might worry about contaminations by germs or about whether they performed a certain acts. A person with compulsion feels a persistent, irresistible, irrational urge to perform an act or ritual repeatedly. The individual knows such acts are senseless but cannot resist performing them without experiencing an intolerable build-up of anxiety-which can be relieved only by yielding to the compulsions. The aim of this research is to explain abnormalities in the neuroanatomical and functional variations between schizophrenia, and obsessive-compulsive disorders involving gray matter and white matter tracts that connect in the brain regions. We will analysis of brain abnormalities in these disorders' express involvement of similar and different regions, with support of epidemiological studies and clinical symptoms are present in both early and chronic stages of the illness.

The Clinical and neuropsychological studies are to diagnose in the different symptoms between patient disorders in neuro-structural and neurological disorder. we categorize this study in several section, first will explain the relation and difference between schizophrenia and obsessive compulsive disorders which relate how it impact the context of neuroanatomical, neurofunctional abnormalities; Second I will clarify Gray matter and white matter differences between schizophrenia and obsessive compulsive patients, finally will emphasize the Schizo-obsessive compulsive; obsessive-compulsive disorder and schizophrenia using different neuroimaging and machine learning algorithm.

### Objectives of the study:

To find the main general information regarding to the use of machine and deep learning algorithms diagnosis of obsessive-compulsive disorders: an analysis of Meta study.

### SPECIFIC OBJECTIVES

- To assess compulsive disorders and schizophrenia which affect the brain in human and brings about irrational thoughts.
- To determine the effect of structural and functional brain region in OCD.
- To maintain actual information about the use of machine and deep learning algorithms diagnosis of obsessive-compulsive disorders: an analysis of Meta study.

## II. Review Of Related Literature And Studies

This chapter primarily presents the different kinds of literature and studies from both foreign and local researchers, which have a significant bearing on the present study.

### 2.1. Review of Relevant Literature

Obsessive-compulsive disorder (OCD) is one of the most common psychiatric disorders that affect the brain regions, it usually causes abnormality issues and it is the results of a negative emotion that affects the normal thoughts and behavior of an individual's personals, social and professional life according to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5: American Psychiatric Association "APA", 2013), the criteria of obsessive-compulsive disorders includes; obsessions (e.g., thoughts, feelings or impulses which are generally unwanted or intrusive) that are suppressed by performing a compulsion (repetitive behavior's such as washing, checking or repeating words silently) that the individual feels driven to perform (APA, 2013). These obsessions and/or compulsions are often time consuming (in excess of one hour per day) and cause significant distress and impairment to the individual's life. OCD is probably one of the most disabling psychiatric disorders with a consistent cross-cultural lifetime prevalence of about 2%, typical onset during adolescence and a female prevalent ratio of 1.5-1.0 (Sassano and pato ,2015).

**Risk factors** include a history of child abuse or other stress -inducing event. Some cases have been documented to occur following infections. The diagnosis is based on the symptoms and requires ruling out other drug-related or medical causes. Rating scales such as the Yale-brown Obsessive-compulsive Scale (Y-BOCS) can be used to assess the severity. Other disorders with similar symptoms include anxiety disorder, major depressive, eating disorders, tic disorders, and obsessive-compulsive personality disorders.

**The cause** is unknown. There appear to be some genetic components, with both identical twins more often affected than both non-identical twins *according* (The National Institute of Mental Health 2016). Disease onset is normally gradual cause (Abramowitz, 2009). On the other hand, money owed of patients developing obsessive-compulsive disorders like symptoms inside after traumatic brain injury (TBI) have been published for a popular overview of traumatic brain injury related with the improvement of obsessive-compulsive disorder (Rydon and Coetzer 2015 In USA, the common age of onset is 19.5 years with greater than 25% of all patients reporting an onset as early as 14 years or youthful (Rusci, 2010). Results from a massive global collaboration study pronounced the average onset to be about 18 years according (Brakoulias, 2017). The distribution of age of onset typically reveals bimodal probability distribution an early peak in adolescence and second peak in early adulthood and there is an ongoing debate regarding a sensible cut-off to define early and late onset obsessive-compulsive disorders (Anhol, 2014). Interestingly, late onset obsessive-compulsive disorders (age at onset >35 years) was once observed to be as a substitute exclusive with fewer than 15% of all cases (Grant, et al. 2007).

**2.2. Symptoms:** The common symptom of OCD entails cognitive dysfunction which include executive function, attention, working memory, non-verbal memory, and visuospatial abilities, main to useful impairment in home, school, and social settings (Nakao, 2009 and Weber ,2014). Although a few researches have published working reminiscence impairment in OCD. The carried-out acts can be mental and/or behavioral in nature and common goal at reducing feelings of anxiety and distress regularly induced by way of obsessions. Furthermore, the obsessions are identified as products of the own mind. According to American psychiatric Association, 2013. While a small proportion of patients may also suffer from obsessions or compulsions, the majority (>95%) record each symptom to co-occur. According to (Shavit, 2014). Clinically, the most normal symptom dimensions appear to be related to contamination and cleaning; doubt about harm and checking; unacceptable ideas and intellectual rituals; symmetry and ordering; cleanliness and washing as properly as well as hoarding according to (Mataix, Rosario & leckman, 2005). The disease presents with a broad variety of signs and symptoms from distinct symptoms dimensions and is consequently regularly viewed to be as an alternative heterogeneous. Despite the truth that diagnostics of obsessive-compulsive disorders is commonly based totally on the presence of obsessions, compulsions, and some shape of distress or anxiety, a current study factors in the direction of impairments in more than a few neuropsychological domains that partly predict differential effects in response to cognitive behavioral therapy (CBT) or pharmacological treatments (ALcant, 2012).

**2.3. Structure – Symptom Relationship in obsessive-compulsive disorders:** - The descriptive findings of structural alterations, various researches assessed whether these modifications are going alongside with clinical measures such as symptom severity or symptoms. Meta-analytic effects by revealed relationship between symptoms, severity and thalamic volumes, according to (Fontenelle,2013; Alvarenga, 2012). Report the symptoms dimension "aggression" to positively correlate with Gray matter volumes of left parietal cortex, whilst volumes of left insula, putamen and inferior OFC were negatively correlated. Scores for the "sexual/religious" dimension positively correlated with right lateral OFC as well DLPFC and negatively correlated with ACC. For evaluations on Gray matter modifications, for combined review of Gray matter and white matter alterations primarily based on voxel-based morphometry (VBM) research. Here, proportions of Gray matter volumes in caudate as properly as white matter volumes in parietal regions correlated with contamination/

washing symptoms. Harm/checking signs and symptoms correlated negatively, with Gray matter and white matter volumes in temporal pole. Additionally, symmetry/ordering signs and symptoms correlated negatively with Gray matter extent in right motor cortex, insula and parietal cortex, while a positive correlated with temporal Gray matter and white matter used to be found according to (Gilbert, 2008). Symmetry/ordering signs have been reported to correlate with common Gray matter and white matter volumes. He provides evidence for a negative correlation between washing symptoms and Broadman locations 6, i.e. frontal cortex. Clearly, studies use distinctive measures to assess symptoms and record various brain regions to be related with distinctive symptoms and moreover in sometimes contradicting directions. Also, whilst various studies do record such correlations (Fontenelle et al., 2011; Ha et al., 2009; Lochner et al., 2012). meta-analysis on subcortical volumetric differences in OCD sufferers performed, the data suggested differences to be discovered in the hippocampus as properly as pallidum whilst other subcortical regions commonly assumed to play a main role in OCD have been no longer discovered to display altered volumes (Boedhoe, Schmaal, Abe, Ameis, et al., 2017).

**2.4. Epidemiology and Comorbidity:** with a one-year incidence of 1.2% among adult and lifetime prevalence of 2.3% according to Ruscio, 2010, obsessive-compulsive disorder is ranked as the fourth most common psychiatric disorders, with estimated annual fees in excess of 10 billion dollars in the US alone. For example, in the National Comorbidity Survey replication (NCS-R) of U.S. adults with OCD, OCD was associated with substantial comorbidity, not only with anxiety (75.8%) and mood disorders (63.3%), but also with impulse-control (55.9%) and substance use disorders (38.6%). Co-morbidity between mood and anxiety disorders is often associated with greater illness severity (Goes, 2011). In the case of OCD, comorbid depression may be particularly associated with reduced quality of life (Hou, 2010). Rates of major depressive disorder ranging from 40% to 80% have been reported. Recurrent depression in children and adults with OCD has been related to variables including older age, an early age at onset of OCD, more severe obsessive-compulsive symptoms, lower levels of perceived competence and family history of recurrent MDD (Storch, 2012; Hong, 2004; and Péri, 2010). Many epidemiological studies of OCD have taken a cross-sectional approach and longitudinal studies are rare. Additionally, obsessive-compulsive disorder has a profound influence on patients best of lifestyles which are same to the influence mentioned in schizophrenia and depressive disorders and larger than for examples in heroin dependence and hemodialysis patients according (Macy, 2013). The risk was reduced when adjusting for other psychiatric comorbidities, nevertheless remained at a notably excessive level, pointing to important interplay effects of comorbidities for the disease. The most frequent comorbid psychiatric disorders among OCD sufferers are major depressive disorders with over 25%, obsessive-compulsive personality disorders and various types of Anxiety disorders according (Brakoulias, 2017). Finally, it was stated that for adolescent and younger adults' prior obsessive-compulsive disorders is a threat factor for creating bipolar disorders, bulimia nervosa, and Anxiety disorders.

**2.5. The Brain Areas Involved in OCD:** - most of both structural and functional imaging studies have shown differences in PFC, basal ganglia, ACC, and/or thalamus between patients with OCD and healthy comparison subjects according (Friedlander L et al. 2006). A recent meta-analysis reviewed functional imaging studies in OCD and found that the OFC (orbital gyrus) and head of the caudate were the only brain areas that significantly and consistently demonstrated increased tracer uptake in OCD patients relative to comparison subjects. We will discuss the OFC, basal ganglia, ACC, and thalamus in this review, but will focus on the OFC and basal ganglia because these brain areas are most consistently associated with OCD in imaging studies. Additionally, corroborated by the disrupting connections between the OFC, ACC, thalamus, and basal ganglia by means of a cingulotomy, anterior capsulotomy, or sub caudate tractotomy results in a symptomatic improvement in most OCD patients according (Aouizerate B, et al, 2004). Some studies have examined the development of symptoms of OCD after brain injury. Damage to the basal ganglia (especially the caudate), the OFC, and the ACC are associated with the acquisition of OCD symptoms following brain injury. Dysfunction of the basal ganglia secondary to a streptococcal infection or encephalitis lethargica has also been associated with the development of OCD symptoms. the association between lesions in the mesial frontal region (including the ACC) and collecting behavior resembling OCD according (Anderson SW. et al. 2005). Another demonstrated that repetitive motor activity in patients with dementia is uniquely associated with right ACC hypometabolism the observed that repetitive motor activity is associated with right caudate and OFC atrophy in patients with frontotemporal dementia (Rosen HJ et, al. 2005).

## **2.6. The Functions of the Brain Areas Involved in OCD: -**

**The Orbitofrontal Cortex (OFC):** - The OFC is a large brain region, which encompasses both rostral and ventromedial areas. Because, it receives multimodal inputs from the temporal association cortex, amygdala and hypothalamus as well as limbic components of the basal ganglia, it has been viewed as the highest integration center for emotional processing. The suggested proof of neurobiological abnormalities in OCD suggests that the orbitofrontal-striatal model can also no longer be enough to completely so the explain the brain foundation of the disorders according (Piras et al. 2013). From a neurobiological perspective, the "multiple-region pathogenesis" of OCD may want to be considered as supportive of the thinking that OCD emerges from

disordered macro and microstructure as properly as function, of large-scale neural structures (Menziés et al. 2008). The OFC appears to play a predominant position in motivational factors of decision making. Among the more posterior cortical area, the left inferior parietal cortex and parieto-occipital junction are concerned in cognitive responsibilities associated to visual imagery.

**The Basal Ganglia:** -Basically, the role of the basal ganglia is to integrate the various inputs arriving from the cortex and to use this information for selecting certain motor and/or cognitive programs. The point of entry of information to the basal ganglia is through striatum, which receives converging information from the limbic and associative cortices. It then sends projections to the output structures, i.e. the globus pallidus pars internalis (GPI) and the substantia nigra pars reticulata (SNr), through two pathways: one direct and the other indirect. The indirect pathway successively involves the globus pallidus pars externalis and the subthalamic nucleus. In addition, the cortex sends direct inputs to the STN and the connections between the Gpe and Gpi. These two pathways seem to play opposite roles in controlling cortical activity. Activation of the direct loop facilitates the triggering of programs at the cortical level through a double inhibition. The cells in direct pathway particularly specific excitatory D1, and cells in the indirect pathway specific inhibitory D2, dopaminergic receptors. Thus reinforcement, coded by means of an increase in dopamine, can bias the gating of the basal ganglia towards future activation of the rewarded conduct (i.e. facilitate learning). Reilly and Frank, 2006, Suggest that the OFC exerts top-down manage of the basal ganglia through representing reinforcement magnitudes to the basal ganglia.

**Anterior cingulate cortex:** - An abnormality in ACC is supposed to contribute to the pathogenesis of OCD thru impaired connectivity with different cortical and subcortical brain regions. Some research observed a reduced connectivity of dorsal striatum and medial dorsal thalamus to rostral and dorsal ACC in OCD patients (Radua, 2009). The left ACCs extent had been reduced in OCD sufferers in contrast to HCs, and the connectivity of proper ACC with bilateral OFC, and striatum were significantly decreased in sufferers with OCD (Szeszko, 2015). Additionally, the connectivity between left ACC and proper OFC have been discovered to be impaired in OCD group. The smaller ACC and striatal volumes and decreased correlations of ACC with OFC and striatum would possibly recommend injury and hypo-functionality in ACC and striatum in OCD patients. The increased extent in OFC may additionally point out the hyper-activity in this pathway of the circuit. These findings converge with prior research that implicate functional and structural abnormalities in the ACC, OFC and striatum in the pathophysiology of OCD (Szeszk and Ardekan, 2005). However, the negative correlation between the period of OCD and proper ACC volume would possibly display that extent discount in ACC is associated to the longer period of the illness (ventakasu, 2012).

**Dorsolateral prefrontal cortex:** - It is the most important cortex part for cognitive functions in human beings. The involvement of the DLPC in working memory was initially demonstrated in primate studies. The DLPC also plays a role in adaptation to changes in the environment. Lesions of DLPC disturb the subject's ability to process temporal information and impair the successful performance of goal-directed behaviours. The function neuroimaging and structural MRI research supply proof that brain areas concerned in OCD pathophysiology are no longer restrained to orbitofrontal-striatal circuits. The DLPFC has been viewed as an essential region, equal to the ACC in great cognitive features such as interest and executive function which would possibly be impaired in OCD. A volume alternate in DLPFC implicates injury in the CSTC. Some research confirmed decreased volume in the DLPFC whereas different research stated no difference in volume or increase volume.

**Thalamus:** -The thalamus acts as a relay Centre that transmits and methods neuronal records from the basal ganglia to cortical areas. Within the thalamus, the Medio -dorsal and ventral anterior thalamic nuclei are of extremely good activity in OCD due to the fact each nuclei send excessive projections to the ACC and OFC according (Herrero, 2009). Hence, volume modifications determined in cortical areas might also be associated to volume modifications in the thalamus. However, the finding indicated that the affiliation of left thalamus with proper OFC was once impaired in OCD patients. Proceedings learn about pronounced that thalamic and OFC volumes are inversely and specifically associated in OCD patients (Ozdemir, 2007 and Aouizerate, 2009). This finding may also allow the institution of a causal relationship between thalamic and ACC volume changes, consequently, is contradictory to preceding recommendations which argue that thalamo-frontal modifications in OCD might also be constrained to the thalamo-orbitofrontal pathways (Rotge, Dilharreguy and Aouizerate, 2009). Because the thalamus send glutamatergic projections to the cerebral cortex, a discount in ACC volume should be associated to neurodegeneration caused by using glutamate neurotoxicity, as described for different neuropsychiatric disorders, and supported by means of the glutamatergic hyperactivity described in OCD (Rotge, 2009). On the other hand, the indirect loop blocks the activation of thalamic relay by increasing the activity of the Gpi, a GABA-ergic inhibitory structure. Dopamine of nigral origin seems to facilitate the direct pathway through D1 receptors and plays an inhibitory role on the indirect pathway through D2 receptors. The pathological activation of segregated closed loop circuits involving cortex-basal ganglia-thalamus-cortex pathways would produce reverberating activity and result in a persistent discharge of the innate programs

characteristic of OCD. The clinical manifestations of neuronal disorders of the basal ganglia can be viewed as a disruption of information processing at the cortical level due the loss of the focusing action of subcortical inputs.

**2.7. Pathogenesis and neurobiology of obsessive-compulsive disorders:** The most common types of obsessions are related to contamination, pathological, doubts, somatic dysfunctions, want for symmetry, aggressions, and hypersexual drive. The classical varieties of compulsions include checking, washing, counting, need to ask, precision and hoarding. In obsessive-compulsive disorders, senseless repetitive rituals (such as counting, washing etc). The current accounts of pathogenesis identify genetic and environmental elements as properly as neurobiological alterations as key factors. The following part will quickly describe components of genetics and environmental factors before offering a greater larger description of the prevailing neurobiological model, considering historical proof on useful as properly as structural brain alterations. The pathophysiology of OCD has been extensively conceptualized inside the cortico-striato-thalamo-cortical (CSTC) model according (Milad MR, Rauch SL et al. 2012). To this model, tracts from frontal areas challenge to the striatum and then, tour through direct and indirect pathways to the thalamus and undertaking decrease back to the frontal regions. This model has been corroborated by using various reviews of structural and practical adjustments found in magnetic resonance imaging (MRI) studies. Volumetric differences inside the orbitofrontal cortex (OFC), anterior cingulate cortex (ACC) and thalamus have been said in patients according (At maca M, Yildirim H, et al. 2007, Rotge J-Y, Guehl D, ET al.2009). Furthermore, early proof from purposeful imaging research indicated an expanded metabolism and hyperactivity in several brain areas in OCD suffers in the course of assignment performance, which include the basal ganglia according (Friedlander L, Desrocher M ET al.2006, Menzies L, Chamberlain SR et al. 2008) orbitofrontal cortex and anterior cingulate cortex on the opposite, a diminished activation in dorsolateral prefrontal cortex according (Nakao T, Okada K et al. 2014) and parietal cortex.. Emerging proof suggests a broader cortical dysfunction, involving structural and practical modifications of the anterior insula, lateral and medial temporal lobe regions according (Stern ER, Fitzgerald KD et al. 2012). Furthermore, latest multimodal meta-analytic proof highlights the relevance of the cerebellum and the parietal cortex for the OCD pathophysiology.

**2.8. Neurobiological Model of obsessive-compulsive disorders:** -A current meta-analysis demonstrated the biological independency of OCD from different anxiety disorders through displaying that individuals with OCD had expanded bilateral Gray matter volumes in the lenticular/ caudate nuclei, whilst sufferers with different anxiety disorders had reduced Gray matter volumes in the left lenticular nucleus according (Radua J, van den Heuvel, 2010). Furthermore, proposed that OCD is mediated by means of an imbalance between the direct (excitatory, OFC striatum-Globus pallidus-thalamus-cortical) and indirect (inhibitory, DLPFC-striatum-Globus pallidus sub thalamic nucleus-cortical) pathways inside this circuit, which causes brain lock in the caudate nucleus and a mutual hyper activation between the OFC and thalamus. In OCD patients, the authors recommend the existence of an imbalance between direct and indirect pathways in prefer of the direct excitatory pathways. As a result, stimuli draw interest to themselves ensuing in the lack of ability to change to other types of conduct whilst additionally resulting in performing behaviors in a ritualistic manner. This mechanism is supposed to be responsible for the manufacturing of findings of hyperactive circuitry normally suggested in neuroimaging studies. the framework of CSTC circuitry involvement is nevertheless regarded the major basis of neurobiological model of OCD, according (Milad and Rauch et al. 2012). Additionally, the role of a range of areas outside the classical CSTC circuits related to disorders mechanisms and argues for a great deal wanted extension of the classical framework. Finally, Milad and Rauch 2012 recommended making it clear the role of amygdala for pathophysiological model of OCD. Thorsen, 2018 Recognized FMRI- as properly as PET-based research assessing variation in responses between emotionally valence and impartial stimulation. Furthermore, activation in OFC, ACC and pre cuneus was once associated to symptoms severity whilst comorbidity with mood and anxiety disorders used to be associated to greater activation in amygdala, putamen, as properly as insula and decreased activation in left amygdala and proper ventro-medial prefrontal cortex (VMPFC). The current style appears to facilitate research efforts aiming at elucidating the position of brain regions outdoor the classical CSTC circuits. This is for instance mirrored in a current suggestion of a neurobiological model of for compulsive conduct put ahead through (Heuvel, 2016). Genetic and Environmental Factor.

### **2.9. Genetic and Environmental Factor:**

Studies into the role of genes in obsessive-compulsive disorders began with family and twin studies. These have produced variable results but generally when the findings are interpreted together, they indicate that OCD runs in families. One possible reason for the higher rates of OCD in family members is that parents pass on genes to their offspring that increase the risk of developing OCD. Relatives of adults with OCD had been about two times greater in all likelihood to be affected than the controls, while family of teens and children with OCD have been about ten times extra possibly to have OCD as well. However, that familial affiliation could additionally have been caused by cultural or environmental factors according (David L. Pauls, "The Genetics of OCD: A Review," Dialogues in Clinical Neuroscience (2010). However, in a retrospective learning about environmental risk factors that involves the development of this disorders, some studies determined sometimes

extended labor and edema at some stages in pregnancy have been correlated to OCD, according (Salem Vasconcelo et al. 2007). suggesting that the surrounding performs some role in identifying its manifestation. As a result, twin studies have been carried out to show that the symptoms of OCD are heritable and consequently genetically related according ((Daniel S. van Grootheest et al. 2005).). Early onset OCD, manifesting in childhood or adolescence, is a subtype of OCD etiologically wonderful from adult onset OCD. This early onset disorder is stated to be genetically related to tic problems and Tourette syndrome, as one study has determined that patients with early onset OCD have a great rate of Tourette's and other tic disorders (Salem & Marcos, 2007). Consequently, it is feasible that there are distinctive genetic mechanisms at the back of two kinds of OCD; therefore, it can also be essential to manage for age of onset when inspecting candidate genes. Which examine whether specific genes increase the risk of developing OCD, have also shown variable results, but promising findings include genes that play a role in the neurotransmitter systems. Consequently, genes may play a role in OCD through their influence on the serotonin, dopamine, and glutamate system. In pharmacological a meta-analysis of genetic association studies certainly reported an affiliation of such polymorphisms with obsessive-compulsive disorders as well as a gender precise polymorphism involving catecholamine modulation with style huge results for dopamine-and glutamate-related polymorphisms according (Taylor, 2013).

## **2.10. Structural neuroimaging of OCD:**

**Region of interest:** - The usage of computed tomography or MRI, the proof from functional neuroimaging research has led many researches to look at brain structures of areas that confirmed abnormal activation, such as the OFC, caudate, and thalamus. The structural imaging finding in some researchers employed area of interest (ROI) techniques till voxel-based morphometry (VBM) strategies have been developed. (Zarei, Mataix-Cols & Heyman, 2011). Most ROI studies suggested volume reduction in the areas such as the OFC, caudate, thalamus, amygdala, and ACC (Rotge, 2009). Performed a meta-analysis research and discovered a decreased extent of the left ACC and bilateral OFC and an expand of bilateral thalamic volumes. The observed that patients with OCD had significantly decreased bilateral OFC and amygdala volumes in contrast with health manipulate topic and lacked the regular hemispheric asymmetry of the hippocampus-amygdala complex. Voxel-based morphometry (VBM) analysis allows the investigation of gray matter volume (GMV) and white matter volume (WMV) in the whole brain. Diffusion tensor imaging (DTI), on the other hand, is available to measure the in vivo water molecule diffusion within the WM fibers, which renders more exquisite details on microstructure changes in WM. Fractional anisotropy (FA) and mean diffusivity (MD) are the two most widely used diffusion indices to investigate the pathology of OCD.

**2.11.FUNCTIONAL NEUROIMAGING OF OCD:** - Functional imaging studies are most useful to clarify the neural substrate underlying the pathophysiology of OCD. The usage of neuropsychological and useful neuroimaging methods, the neutral correlates of the brain useful deficit, anatomical change, and metabolic abnormalities with working memory dysfunction underlying OCD continue to be unknow. With the help of magnetic resonance (MR) base techniques, several structural and functional neuroimaging research (Nakao , 2009; Tang, 2016) on OCD have suggested that an impairment of the cortico-striato-thalamo-cortical (CSTC) circuits is associated with the principal pathophysiology of OCD. The CSTC circuits have large connective to several cortical and subcortical regions, and thus, they dysregulation of the CSTC circuits is associated with impaired executive performance, lack of ability to inhibit cognitive and behavior, and more advantageous error monitoring techniques in patients with OCD, according (Hou, 2014). In addition, the imbalances in neuronal metabolites and neurotransmitters within the CSTC circuit were proposed to be the cause for the onset of OCD and have been implicated as pathophysiological mechanisms of OCD (Zhu., 2015). Especially, proton magnetic resonance spectroscopy (1H-MRS) is a good candidate for enabling the quantification of metabolites in tissues in vivo. The MRS technique has been commonly used to study the neurobiology of OCD (Zhu, 2015). To our knowledge, however, no one has applied the combined neuroimaging study of functional, morphometric, and metabolic brain abnormalities in conjunction with recognition deficits during working memory tasks in patients with OCD using functional magnetic resonance imaging (fMRI), the diffeomorphic anatomical registration through exponentiated Lie algebra (DARTEL) algorithm-based voxel-based morphometry (VBM), and 1H-MRS, which are the vital steps in the identification of neural mechanisms associated with neurocognitive dysfunction in OCD. the combined MR-based techniques including event related MRI, DARTEL-based VBM, and localized 1H-MRS were used to assess the association of neurofunctional, morphometric and metabolic abnormalities, and to further reveal their correlations with clinical symptom severity and neurocognitive dysfunction under working memory tasks.

**2.12. Deep learning algorithm in diagnosis obsessive-compulsive disorders:**Deep learning classifiers have recently become an attractive choice for mental illness prediction. Deep learning classifiers can learn the features with optimal discriminating power directly from the raw data by using the different studies in neuroimage. We collect in this data previous study in Magnetic resonance imaging (MRI) methods have been used to detect cerebral anatomical distinction between obsessive-compulsive disorder (OCD) patients and healthy controls (HC). Machine learning approach allows for the possibility of discriminating patients on the

individual level. However, few studies had used this automatic technique based on multiple modalities to identify potential biomarkers of OCD. High-resolution structural MRI and diffusion tensor imaging (DTI) data were acquired from 48 OCD patients and 45 well-matched HC. Gray matter volume (GMV), white matter volume (WMV), fractional anisotropy (FA), and mean diffusivity (MD) were extracted as four features were examined using support vector machine (SVM). Ten brain regions of each feature contributed most to the classification were also estimated. Used different algorithms, the classifier achieved accuracies of 72.08, 61.29, 80.65, and 77.42% for GMV, WMV, FA, and MD, respectively. The most discriminative Gray matter regions that contributed to the classification were mainly distributed in the orbitofronto-striatal “affective” circuit, the dorsolateral, prefronto-striatal “executive” circuit, and the cerebellum. For WMV feature and the two feature sets of DTIs, the shared regions contributed the most to the discrimination mainly included the uncinate fasciculus, the cingulum in the hippocampus, corticospinal tract, as well as cerebellar peduncle. Based on whole brain volumetry and DTI images, SVM algorithm revealed high accuracies for distinguishing OCD patients from healthy subjects at the individual level. The MRI approaches provide a perspective to investigate the neuropathological changes of OCD and allow researchers to identify better biological markers of this disease. To date numerous neuroimaging studies had used the between—group comparison—types of analyses to investigate subtle differences between OCD patients and healthy controls (HC). Reported volumetric abnormalities lied in multiple neural structures. Volume reduction in the medial orbitofrontal, anterior cingulate and temporolimbic cortices, and tissue expansion in the striatum and thalamus was among the most widely accepted pathological model of OCD which assumes brain abnormalities in the “affective circuit” according (Frydman I, et al.2016; Piras F, et al.2015). Additionally, volume changes of the cortical source of the dorsolateral (DL) prefronto-striatal “executive” circuit (dorsomedial, DL, ventrolateral and frontopolar prefrontal cortices), and of reciprocally connected regions (temporo-parieto-occipital associative areas) are consistently described in OCD patients (Piras F, et al.2015). Reported WM integrality abnormalities also involved extensive brain areas, such as the corpus callosum (CC), cingulum bundles, corticospinal tract, superior longitudinal fasciculus (SLF), uncinate fasciculus (UNC), and cerebellum (Frydman I, et al.2016). The MRI-related machine learning technique offers a systematic approach in developing sophisticated, automatic, and objective classification frameworks for analyzing high-dimensional data and provides promise for improving the sensitivity and/or specificity of detection and diagnosis of disease (Bryan RN, 2016; Rathore S, 2017). The analysis based on multivariate pattern is more sensitive to identify subtle differences in the brain structure than group-level statistics. The SVM algorithm establishes model by discriminating the different categories (such as patients and controls) and further applying new data to test its generalizability (Noble WS, 2006; Li F, Huang X, et al. 2014).

**Regions contributed most for classification:-**For GMV feature, the most informative regions for classification between OCD patients and HC included right anterior cingulate gyrus (ACG), right angular gyrus, right inferior parietal, bilateral paracentral lobule, left inferior frontal gyrus, and bilateral cerebellum regions. For the WMV feature and two feature sets of DTI, regions contributed the most to the discrimination were relatively consistent, mainly included the UNC, the cingulum in the hippocampus, corticospinal tract, as well as cerebellar peduncle. Additionally, right external capsule, left fornix, and stria terminalis, left anterior corona radiata, bilateral cerebral peduncle, pontine crossing tract and bilateral cerebral peduncle are among the informative regions for classification.

**Image Acquisition:-**MRI scanning was carried out by a skilled radiological technician at a Philips Achieva 3.0T scanner (Philips Healthcare, Best, the Netherlands). First, acquisitions included a conventional normal T1- and T2-weighted sequences to rule out obvious structural abnormalities such as cerebrovascular diseases.

3D T1-weighted volumetric structural MRI scan sequence was acquired using a fast spoiled gradient recalled acquisition (FSPGR) with the following parameters: TR/TE = 7.38/3.4 MS, matrix size = 256 × 256, FOV = 250 × 250 mm, number of slices: 230, flip angle = 90°, scan time = 6 min 53 s.

DTI images were acquired using an echo-planar imaging (EPI) sequence in 50 axial planes and was collected along 33 independent orientations through the whole brain using the following parameters: TR/TE = 6,800 MS/80 MS, slice thickness = 3 mm, FOV = 230 mm<sup>2</sup> × 230 mm<sup>2</sup>, matrix size = 116 × 112, voxel size = 1.98 mm × 2.05 mm × 3 mm, b value = 1000 s/mm<sup>2</sup>, flip angle = 90°.

**Image preprocessing:-** Structural MRI images were pre-processed using the VBM8 toolbox (<http://dbm.neuro.uni-jena.de/vbm>) with the Diffeomorphic Anatomical Registration using the Exponentiated Lie algebra (DARTEL) toolbox (Ashburner J, et al, 2007) implemented in the statistic parametric mapping software package (SPM8, <http://www.fil.ion.ucl.ac.uk/spm>) running on MATLAB 2012a (MathWorks, Natick, MA, USA). This procedure comprises creating a study-specific template and segmenting each individual image using the template aiming to maximize accuracy and sensitivity (Krakauer K, et al, 2017). Then the GM, WM, and cerebrospinal fluid (CSF) were automatically segmented. After Jacobian modulation, the GM images and WM images were smoothed with 8-mm full width at half maximum (FWHM) Gaussian kernel for further SVM analysis. The diffusion MRI data were processed by the FMRIB Software Library (FSL, Version 5.0; available

from <http://fsl.fmrib.ox.ac.uk/fsl>). Tract-based spatial statistics (TBSS) was used to perform voxel-wise processing of diffusivity measures. First, the skull was stripped using Brain Extraction Tool (BET) of FSL.

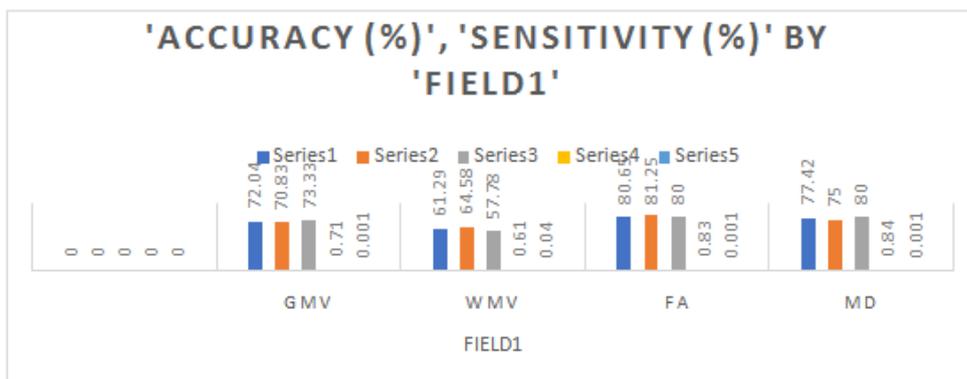
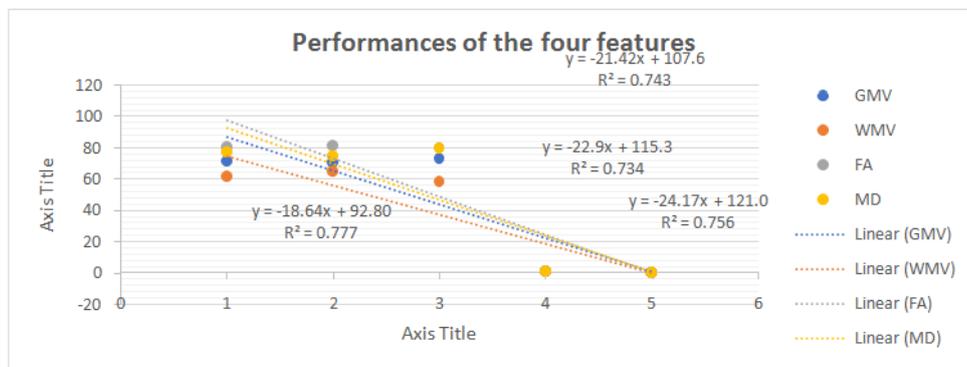
**Support vector machine** a margin-based statistical technique to classify individuals into two or more groups by establishing a hyperplane based on specific cases called support vectors. SVM was applied by using the Pattern Recognition for Neuroimaging Toolbox (PRoNT) according (Schrouff,et.al.2013) (<http://www.mnl.cs.ucl.ac.uk/pronto>) to estimate potential WM areas contribute most in calcifying OCD. Briefly, the main steps of the SVM method include: (i) feature extraction and feature selection, (ii) selecting discriminative regions, (iii) training the SVM classifier model using the training data, and (iv) evaluating the performance of the SVM model using the evaluation data (Dyrba M,at.al.2015; Amarreh I,2014 ). In neuroimaging studies, the number of features is much more than the number of subjects, which induces the “curse of dimensionality” in machine learning studies (De Martino F, et. al;.2007; Fort G, et.al.2005).Thus, this feature vector encoded the pattern of GMV, WMV, FA, or MD values. By comparison, feature selection involves the selection of a subset of features which facilitates learning as well as in this study, feature selection consisted of identifying brain regions that are expected to differ between groups (Orri G,et.al.2012; Noble WS,2006).

In the classification of the two groups, for the whole brain GMV, the accuracy was 72.04% (permutation p = 0.001) with a sensitivity of 70.83% and a specificity of 73.33%. For the whole brain WMV, the accuracy was 61.29% (permutation p = 0.040) with a sensitivity of 64.58% and a specificity of 57.78%. Results for the FA and MD values were more promising. A classification accuracy of 80.65 to 77.42% were achieved for the two feature sets of DTI (permutation p = 0.001), respectively. Sensitivity of the two parameters was 81.25 and 75%, respectively, and specificity of both FA and MD values were 80% (Table 1). No significant results were found between the four features and duration.

**Table 1** SVM classification performances of the four features:

	Accuracy (%)	Sensitivity (%)	specificity (%)	AUC	Permutation p-values
<b>GMV</b>	72.04	70.83	73.33	0.71	0.001
<b>WMV</b>	61.29	64.58	57.78	0.61	0.040
<b>FA</b>	80.65	81.25	80.00	0.83	0.001
<b>MD</b>	77.42	75.00	80.00	0.84	0.001

GMV, Gray matter volume; WMV, volume; FA, fractional anisotropy; MD, mean diffusivity; AUC, area under the ROC curve.



**2.13. schizophrenia:**the comorbidity between OCD and schizophrenia will be mentioned in more detail in the following this study. Contrary to earlier reports,

the risk of developing schizophrenia is now not extensively increased in major OCD. Roughly one quarter of patients with schizophrenia, however, will experience OC symptoms during their lifetime, and about 12% meet the standards for OCD (e.g. Braga et al.2013). the immoderate occurrence of OC symptoms in schizophrenia may also be an indication of shared neurobiological characteristics. one necessary criterion for differentiating between psychotic symptoms and OC symptoms is that OC sufferers recognize that their intrusive ideas or impulses are a product of their own wondering mind. Another criterion is that OC sufferers have – at least from time to time – some perception into the irrationality of their obsessions and rituals. However, as stated above, whilst most OC sufferers have great or very properly perception into the senselessness of their obsessions and compulsions, a minority of patients possess low insight. Finally, resistance in opposition to psychotic symptoms such as delusions and hallucinations is usually not very strong, whilst sufferers with OC symptoms generally attempt to distance themselves through a certain degree of resistance in opposition to their obsessions and compulsions. Moreover, in a current study in accordance (Zink et al. 2014), about 11 % of patients with at risk mental states for psychosis had OC symptoms and 5 % full filled the criteria for OCD.

**2.14. Pathogenic Concepts on Schizophrenia and OCD:** - Schizophrenia is perceived as a common remaining clinical manifestation of several wonderful and heterogenous neurobiological processes. The alterations trade brain connectivity and the interplay of neurotransmitter system, most importantly serotonergic, dopaminergic, and amino acid neurotransmissions (Howes and Lewis, 2012; Kantrowitz and Javitt 2013). The interaction of genetic and environmental factors is of imperative importance in genetic properties alter early neural improvement and elicit long-lasting effects due to persisting plastic methods of pre-and perinatal improvement in accordance (Kapur, 2009). Current concepts of OCD define a critical dysfunction with the frontocorticalstriato-thalamo-cortical circuitry connecting the orbitofrontal cortex, the anterior cingulate cortex and basal ganglia thalamus and caudate nucleus, according to (Pogarel, et al.2011). The concepts of schizophrenia and OCD share common pathogenic mechanisms. This corresponds to the clinical phenomenology of patients presenting with both psychosis and OCS. The complex psychopathology of their co-occurrence might be elucidated if nosological knowledge and studies perspectives of schizophrenia and OCD are combined. It is further necessary to define greatest homogeneous subgroups within the broad heterogeneity of comorbid patients.

**2.15.Neuroimaging detecting of OCS in Schizophrenia:** - The research literature on schizophrenia and OCD neuroanatomic circuits suggest more parallels than distinctions between the two disorders. The basal ganglia defects, the frontal lobes, the thalamus, and the cerebellum have been suggesting in both schizophrenia and OCD. Recent findings have in fact suggested similar anomalies in both schizophrenia and OCD (Kang, 2008). Similarities between OCD and schizophrenia often appear when one finds sensory input gating or filtering to playing a role in either disorders. Few researches investigated structural and functional disorders as possible, as distinct neurobiological underpinnings of a schizo-obsessive disorder. Although numerous clinical studies have explored the medical features and epidemiological dimensions of OCD and schizophrenia knowledge about neuroimaging is scarce. A research conducted in OCS patients with substantially reduced volume of the left hippocampus, frontal lobes and anterior horn of the lateral and third ventricles (Aoyama, 2000;). In schizo-obsessive patients but not in patients with schizophrenia, the same study also found an inverse association between length of the disorder and frontal lobe scale. The structural magnetic resonance imaging (MRI) studies, the effects of OCS on brain activity was recorded in one functional MRI study; schizo-obsessive patients demonstrated a negative association between left dorsolateral prefrontal cortex activity and OCD intensity. Another recent functional MRI research used inhibition response and working memory paradigms to elucidate the effects of second-generation antipsychotic drugs on OCS related neural systems in schizophrenia and altered brain activity (especially increased OFC activity during response inhibition) apathogenic mechanism for the development of antipsychotic OCS in schizophrenia may be linked to antipsychotic treatment (Schirmbeck, et al. 2014) .

### **III. Methodology**

**3.1.1Study design:** The aim of this chapter will initiate a methodological framework which allows to distinguish since 2009. We will examine the number of patients, patient's ages, method used, and distinctive performances of the patients effects the obsessive-compulsive disorders and schizophrenia by a metanalysis in machine learning algorithm to determine in the brain regions and the prevalence occurs in the patients. Hence, this study also used documentary analysis since there was some information that the researcher gathered based on the existing cases and the aim of our studies to investigate structural and functional abnormalities differences between schizophrenia and obsessive-compulsive disorder.

**3.1.2 Research methodology and data collection:**The qualitative methods will allow this study to collect information from the selected element of the population. Questionnaire and interview forms of qualitative methods shall be employed which will be distributed among the obsessive-compulsive disorders and schizophrenia patients. Before conducting the interview, an invitation message shall be sent to selected

participants seeking their permission to conduct an interview with them on the subject matter. More so, the questionnaire will be subjected to professional review to know the validity and reliability of its structure. The methods used in this study were the direct method and the documentary analysis. In the direct method, the researcher will be made such as Participants and online research, Statistical Analysis, Demographic and clinical characteristic.

**4.1. PRESENTATION, ANALYSIS, AND INTERPRETATION OF DATA:**

The Participants This qualitative method of this research will allow collecting in formations elements of the populations. we will use previous questionnaire and interview forms of qualitative methods will be employed which distributed among the obsessive-compulsive and schizophrenia patients. Inclusion criteria included studies comprising subjects diagnosed of OCD and schizophrenia according to DSM-IV criteria, which were assessed for outcome measures related to this comorbidity. Diagnosis of OCD and schizophrenia duration of illness were determined by two experienced clinical psychiatrists before the MR imaging examination, by using a structured clinical interview listed in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV).

**Demographic and clinical characteristic:**

Of the study We conducted previous study a systematic assessment of age-of-onset of schizophrenic and obsessive-compulsive symptoms among patients who were consecutively admitted to Trait.

**Demographic and clinical characteristics of the study participants**

Variable	Schizophrenia with OCD (N=133)	Schizophrenia without OCD (N=113)	Statistic (d. f.=244)
Gender (M/F)	97/36	81/32	$\chi^2=0.05$ ; $p=0.83a$
Age, mean (S.D.), y	30.1 (8.7)	29.6 (9.3)	$t=0.51$ ; $p=0.61$
No. hospitalizations	2.9 (2.6)	2.5 (2.7)	$t=1.25$ ; $p=0.21$
Duration, mean (S.D.), y	10.3 (7.9)	6.4 (8.0)	$t=3.70$ ; $p=0.00$
Age at interview, y	30.1 (8.7)	29.6 (9.3)	$t=0.51$ ; $p=0.61$
Education, y	11.7 (2.0)	12.0(2.0)	$t=1.10$ ; $p=0.27$
Married	28	22	$\chi^2=0.09$ , $p=0.76$
Single/divorced	105	91	
Full/partial	38	37	$\chi^2=0.49$ , $p=0.48$
Unemployed	95	76	
SAPS	7.9 (3.6)	7.3(3.9)	$t=1.25$ , $p=0.21$
SANS	11.3(4.2)	11.5(4.8)	$t=0.34$ , $p=0.73$
CGI	4.1 (0.8)	4.1 (0.9)	$t=0.10$ ; $p=0.93$
Y-BOCS obsessions	10.6(4.6)	0.1 (0.5)	$t=24.52$ ; $p=0.00$
Y-BOCS compulsions	9.5 (4.7)	0.2(0.8)	$t=22.72$ ; $p=0.00$
Y-BOCS total	20.0(8.0)	0.1 (0.7)	$t=28.43$ ; $p=0.00$

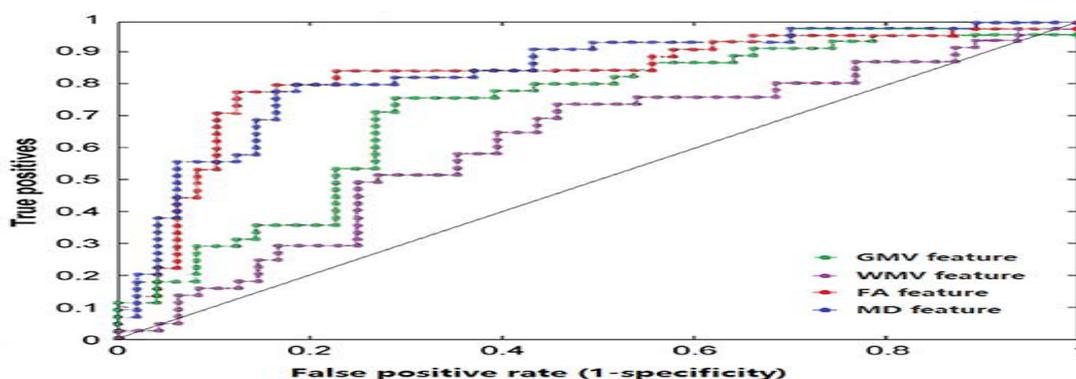
Abbreviations: OCD = obsessive-compulsive disorder, SAPS = Schedule for the Assessment of Positive Symptoms, SANS = Schedule for the Assessment of Negative Symptoms, CGI =

**The demographic and clinical characteristics**

This significant difference between groups was found in gender and age, in the 48 OCD patients had an average duration of illness of  $45.42 \pm 41.02$  months, the total Y-BOCS score was  $25.50 \pm 3.56$ , the Y-BOCS obsession score was  $12.90 \pm 2.40$ , the Y-BOCS compulsion score was  $12.58 \pm 3.07$ . The total HDRS and HAMA scores were  $8.10 \pm 3.71$  and  $9.29 \pm 2.89$ , respectively.

	OCD patients (n = 48)	HC (n = 45)	t/F/ $\chi^2$	p-Value
Age, years	32.29 ± 12.62	30.62 ± 9.02	4.733	0.464
Gender (male/female)	27/21	24/21	0.080	0.778
Duration (month)	45.42 ± 41.02	–	–	–
Y-BOCS total score	25.50 ± 3.56	–	–	–
Y-BOCS obsession score	12.90 ± 2.40	–	–	–
Y-BOCS compulsion score	12.58 ± 3.07	–	–	–
HDRS score	8.10 ± 3.71	–	–	–
HAMA score	9.29 ± 2.89	–	–	–

OCD, obsessive-compulsive disorder; HC, Healthy Controls; Y-BOCS, Yale-Brown Obsessive-Compulsive Scale; HDRS, Hamilton Depression Rating Scale; HAMA, Hamilton Anxiety Scale.



**4.2. Neuropsychological profile of OCS in schizophrenia:** The neuropsychological in OCD recommend abnormalities brain circuits specifically the fronto-striato-pallido-thalamo-frontal loop-in these patients. The frontal subcortical circuitry presents a unifying framework for appreciation procedures that manage cognition, decision making, the planning of complicated behavioral strategies, and neuropsychiatric symptoms (Bonelli, et al. 2007). In the schizophrenia patients with antipsychotic-associated OCD had higher deficits in visuospatial appreciation and visual memory, executive functioning and cognitive flexibility in contrast to patients with schizophrenia with no distinction in performance on different measure of executive function in accordance (Cunill, 2013). This reduces cognitive deficits to be grouped between executive versus emotional deficits. The former is coordinated especially through the dorsolateral prefrontal cortex and the latter by the ventral prefrontal cortex. Both these brain areas (and the function sub-served by way of them as well) are influenced by means of interactions with the anterior cingulate cortex. Such a view, althoughsimplistic, is beneficial as a preliminary heuristic to apprehend the complicated neural circuitry abnormalities recommended through these neuropsychological deficits.

**4.3. Neurophysiological profile of OCS in schizophrenia:** The neurophysiological abnormalities in Schizo-obsessive patients may be used to probe neurophysiological correlates of the cognitive, emotional and behavioral disturbances found in neuropsychiatric entities such as schizo-OCD. We also performed these measurements in patients with OCD without psychotic features, as well as in patients with schizophrenia without OC symptoms. Schizo-obsessive compulsive patients showed a distinct ERP pattern, with abnormally increased target activation and reduced P300 amplitudes. Like the control subjects, schizo-OCD patients showed larger amplitudes in the non-target condition than in the target condition. The study suggested a wonderful ERPs pattern, with abnormally increased goals activation and decreased p300 amplitudes in a schizo-obsessive group in comparison to a schizophrenia group and OCD group in accordance (Pallanti et al.2009). whilst the neurobiological OCD and schizophrenia has been considerably studies in the previous decade, there lack of managed study in patients with OC schizophrenia of the brain activation during cognitive tasks in schizophrenia and schizo-obsessive participants have elicited similar reduction in activation in the right DLPFC and right caudate, as well as decreased functional connectivity during performance of the N-back task compared to healthy controls; these patterns seem to be unrelated to severity of symptoms (Bleich, 2014).

**4.4. Biological mechanisms leading to OCS in schizophrenia:**The biology of Obsessive-compulsive disorder refers biologically based theories about the mechanism of OCD. Cognitive models generally fall into the category of executive dysfunction or modulatory control (Friedlander, L.et al.2006). Neuroanatomical functional and structural neuroimaging studies implicate the prefrontal cortex (PFC), basal ganglia (BG), insula, and posterior cingulate cortex (PCC). Genetic and neurochemical studies implicate glutamate and monoamine neurotransmitters. this study of neurobiological findings points towards significantly shared structural and functional brain abnormalities, namely, frontal lobe dysfunction and basal ganglia dysfunction in both schizophrenia and OCD. Explaining the neurobiological mechanisms is not only essential for the main psychiatric disorders, however additionally for a higher appreciation of their comorbidities, such as the co-occurrence of psychotic and obsessive-compulsive symptoms. The interplay between genetic and environmental factors (GxEs) has been in the improvement of depression, anxiety disorders and OCD. Although extraordinary number of researches have investigated these mechanisms for the mentioned main disorders, studies focusing on comorbidities, the place syndromes or disorders occur concurrently in character patients, is nevertheless limited. Preliminary effects of research investigating GxEs in the improvement of comorbid OCD in schizophrenia strongly recommend the involvement of genetic and environmental stressors (e.g. pharmacological and/or psychosocial factors) (Schirmbeck and Zink, 2013).

**4.5. Neuroimaging detecting in OCD:**Much of this research, We going to take the previous studies that were determined in the different neuroimaging methods, and this analysis we concentrate on the different research that was determined from neuroimaging methods has looked for feasible modifications considered in the OCD brain patients, especially findings how to affect the brain regions in this condition. The cortical-striato-thalamo-cortical model of OCD neurobiology used to be postulated based on the hyperactivity in the prefrontal cortex (mainly orbitofrontal cortex), anterior cingulate cortex, and caudate nucleus shown in the preliminary studies. Some previous studies concentrate on the CSTC loop; and we also suggest involving large associative networks consisting of parietal cortex region, limbic region and cerebellum. Throughout this narrative analysis, we analyze results from the structural (diffusion tense or imaging to evaluate the integration of white matter, magnetic resonance imaging to evaluate the state of grey and white matter), functional (function MRI, positron emission tomography and functional MRI to evaluate brain function abnormalities, single photon emission computed tomography, incorporating functional and structural imaging study to identify OCD neurobiology. The structural and functional imaging researches in OCD are summarized in table 1.1. the first research suggested brain abnormalities in OCD had been based on qualitative assessment of neuroimage or measurement of whole brain volume and ventricle to-brain ration.

**4.6. Discussion:** Although schizophrenia and obsessive-compulsive disorders are wonderful nomological entities, obsessive-compulsive symptoms and schizophrenia symptoms regularly coexist in a significant percentage of patients.

Author, year	Imaging	N	Control group	Findings	Association with severity	Discussion/re marks
Radua mataix-Cols,2009	Meta-analysis of 14 OCD and 12 anxiety disorder MRI studies	430 OCD and 209 anxiety disorder	737	Inc GM b/l caudate (OCD>HC, anxiety disorder) and right superior parietal lobule (OCD>HC) Dec GM b/l dorsomedial frontal gyrus and anterior cingulate gyrus (OCD and anxiety >HC.		Symptom dimension, medication status did not analyze no separate analysis for pediatric sample.
Rotge et al.2010	Meta-analysis of 10 MRI-VBM studies	343(263 adult)	318(263 adults)	Dec GM density prefrontal region, b/l middle frontal including right supramarginal gyrus, b/l DLPFC and OFC Inc GM density b/l putamen and left inferior frontal gyrus.		Symptom dimension medication status, comorbidity, and symptom severity not analyzed.
Peng et al. 2012	Meta-analysis 15 MRI-VBM and 7DTI studies	VBM-455, DTI-121	VBM-449, DTI - 124	VBM-DEC GM in frontal eye fields, DLPFC, ACC, OFC, Inc GM in left lenticular, caudate nucleus and superior parietal lobule, DTI, Dec FA in cingulum bundle, inferior fronto-occipital fasciculus, superior longitudinal fasciculus, Inc FA left uncinate fasciculus.	Inc GM in b/lenticular nucleus associated with YBOCS severity, Dec FA in right cingulum bundles associated with YBOCS severity.	Symptom dimension, medication status, comorbidity, age not taken into consideration.
Nakamae et al. 2011	DTI Tract-based spatial	30(16 drug-free, 14	30	Dec FA in the anterior body of corpus callosum, trend for		

	statistically	drugs naïve)		lower FA in larger portion of CC, right cingulum, and left anterior limb of internal capsule.		
Van der straten et al. 2017	Mata-analysis of 8 PET and 6 SPECT studies pre- and post-treatment with SSRI clomipramine/ CBT	188	Health controls present	Post successful trt, Dec in activity in caudate and OFC, no change in thalamus PET studies, Dec in glucose metabolism in caudate, OFC and thalamus, SPECT studies, Dec in blood flow in caudate.		
Perani et al.2008	PET (11C) MDL for serotonin receptors 11C Raclopride for dopamine receptors.	9 unmedicated OCD	6for serotonin receptors, 9 for dopamine receptors	Dec serotonin binding potential in b/1 frontal lobes-DLPFC, frontal polar, and medial FC, ACC and parietal and temporal associative cortex	Neg correlation between YBOCS severity and OFC, DLPFC, lateral and medial temporal cortex, and inferior parietal lobule.	
Zhang et al.2017	Resting state FMRI targeting rostral and dorsal ACC	23 unmedicated OCD (off medication > 2 month	23 age and gender matched	Rostral ACC showed inc FC with associative visual cortex and Dec FC with left DLPFC dorsal ACC inc FC with middle temporal gyrus, right caudate nucleus.	FC between dACC and caudate pos correlated with YBOCS severity.	Differential role rostral and dorsal ACC

Healthy control unless otherwise specified. Inc- increased; Dec- decreased; b/1-Bilateral; GM-Gray matter; WM-white matter; VBM-voxel-based morphometry; SBM-surface-based morphometry; POS-positive; Neg-negative; trt-treatment; PFC-prefrontal cortex; DLPFC-dorsolateral prefrontal cortex; ACC- anterior cingulate cortex; PCC-posterior cingulate cortex; OFC-orbitofrontal cortex; rCBF-regional cerebral blood flow; SERT-serotonin transporter; DAT-dopamine transporter; PET-positron emission tomography; SPECT-single photon emission computed tomography; CBT-cognitive behavior therapy; SSRI-selective serotonin reuptake inhibitors; YBOCS-yale-Brown obsessive compulsive scale; OCD-obsessive-compulsive disorder; It left; rt-right. DTI; diffusion tensor imaging. MRI; magnetic resonance imaging. FMRI; functional magnetic resonance imaging.

**4.7. Neuroimaging detecting in schizophrenia:** Research on neuroimaging has established that hallucinations and delusion have neural associations with the left medial temporal lobe cingulate cortex while disorganized thought and behavioral characteristics are correlated with anterior cingulate, dorsal parietal regions and ventral frontal cortex. At the other hand, psychomotor deprivation is shown to be associated with decreased frontal cortex function. The goal of these studies is to provide previous knowledge on the basic concepts of various neuroimaging techniques that are used during schizophrenia diagnosis and treatment with focus on current and possible future clinical use. Established ideas on schizophrenia mechanisms postulate rather a disturbance in dispersed neural pathways than an abnormality in a brain region such as the prefrontal cortex. In addition to the frontal cortex, brain disorders in the basal ganglia, thalamus, in addition to the frontal cortex, brain disorders in the basal ganglia, thalamus and cerebellum have been reported (Keshavan, 2008). Thalamus abnormalities in both structural and functional imaging studies have been identified in patients with schizophrenia with reduced thalamic volumes relative to health controls (Rao,2010).

**Summary of neuroimaging techniques in schizophrenia:**

Imaging technique	Description
Computed tomography (CT)	<ul style="list-style-type: none"> <li>• First imaging technique use to study schizophrenia neuropathology</li> <li>• Involves the use of computers to enhance images formed when X-rays are differentially absorbed by brain tissues.</li> <li>• Strengths: Less costly than other techniques; readily available; fewer contraindications than other techniques.</li> <li>• Limitations: Poorer contrast and resolution in the imaging of brain tissues and soft images; exposure to radiation</li> </ul>
Magnetic resonance imaging (MRI)	<ul style="list-style-type: none"> <li>• Capitalizes on the magnetic properties of hydrogen atoms in the brain • MRI alters the energy state of atoms via magnetic fields, radiofrequency energy, and movements during return to resting state</li> <li>• Has been extensively applied to the study of progressive brain change in schizophrenia</li> <li>• Strengths: Good resolution in the imaging of brain structure; can depict 2–3 dimensional images; does not require x-rays or radioactive tracers; non-invasive</li> <li>• Limitations: Costly; impacted by movement; not indicated for claustrophobic patients and patients with metallic devices.</li> </ul>
Magnetic resonance spectroscopy (MRS)	<ul style="list-style-type: none"> <li>• Creates spectrum peaks that correspond to chemical composition of the brain—chemicals generate unique spectrums in the presence of nuclear magnet</li> <li>• Spectral peaks are often generated through magnetization of metabolite such as N-acetyl aspartate</li> </ul>

	(NAA), glutamate, gamma aminobutyric acid (GABA) <ul style="list-style-type: none"> <li>• Strengths: Able to index in vivo neurochemistry; non-invasive</li> <li>• Limitations: Poorer contrast/resolution relative to MRI; not adaptable to measuring change due to cognitive activity; limited accessibility of hardware.</li> </ul>
Position emission tomography (PET)	<ul style="list-style-type: none"> <li>• First functional neuroimaging technique—detects gamma rays released from the breakdown of radioactive tracers attached to O-labeled water</li> <li>• Produces images by measuring cerebral blood flow using a positron-emitting radio nuclear as its radioactive tracer or fluorodeoxyglucose (FDG) cellular absorption as an index of neural metabolic activity.</li> <li>• Has been used extensively in the study of hypo-and-hyper frontality in schizophrenia and reduced activity in other areas</li> <li>• Strengths: Good temporal and spatial resolution; undisturbed by motion.</li> <li>• Limitations: Costly; invasive set-up; exposure to radioactivity; low time resolution</li> </ul>
Single photon emission CT (SPECT)	<ul style="list-style-type: none"> <li>• Detects gamma rays released from the breakdown of radioactive tracer attached to ethyl cysteamine dimer</li> <li>• Has been applied to the study of hypo-and-hyper frontality</li> <li>• Strengths: Good temporal and spatial resolution; undisturbed by motion</li> <li>• Limitations: Costly; Poorer spatial resolution relative to PET; exposure to radiation; impractical for longitudinal studies.</li> </ul>
Functional MRI (fMRI)	<ul style="list-style-type: none"> <li>• Measures neuronal activity by detecting changes in cerebral blood flow</li> <li>• Its most used display form is the MRI slice with Blood Oxygen-Level Dependent (BOLD) signal; other forms are the Glass brain and 3D Render</li> <li>• Has been used extensively in schizophrenia to index abnormal activities in the frontal and temporal areas</li> <li>• Images both functional and structural characteristics</li> <li>• Strengths: Greater spatial and temporal resolution than PET and SPECT; Non-invasive; Cheap; readily available hardware; adaptable to repeated assessments and longitudinal studies</li> <li>• Limitations: Low time resolution; impacted by movement; poor depiction of structure; complex analysis and interpretation.</li> </ul>
Diffusion tensor imaging (DTI)	<ul style="list-style-type: none"> <li>• An advancement of MRS that detects the diffusion of water through white matter such as axonal projections through myelin sheaths</li> <li>• DTI creates images by capitalizing on the anisotropic properties of water diffusion through white matter</li> <li>• Strengths: Most effective method for studying white matter architecture and neural connectivity</li> <li>• Limitations: Expertise is not readily available.</li> </ul>

#### IV. Conclusions

The master thesis has aimed to determine the main general information regarding to the use of machine and deep learning algorithms diagnosis of obsessive-compulsive disorders: an analysis of Meta study. The thesis was divided into several chapters, introduction, literature overview, methodology, results, diagnosis, treatment, conclusion, and recommendation.

1. In first chapter, introduction, objective of the study including to the specific objective to determine obsessive-compulsive disorders and schizophrenia which affect in the brain human and brings about irrational thoughts, problem statement, overview obsessive-compulsive disorder and schizophrenia.
2. In the second chapter, literature overview of the thesis was reflected, at the beginning the term concepts of course, structure-symptoms relationship in obsessive-compulsive disorder with examine epidemiology and comorbidity, pathogenesis and neurobiology of OCD, genetic and environmental factor that involved OCD, and explained some important articles about abnormalities in the neuroanatomical and functional variations between schizophrenia, and obsessive-compulsive disorders involving Gray matter and white matter abnormalities that connect in the brain regions. finally, was emphasize the schizo-obsessive compulsive; obsessive-compulsive disorder and schizophrenia using different neuroimaging and machine learning algorithm.
3. In the third chapter of my thesis methodology of this research was provided with qualitative method including tables and diagram related OCD and schizophrenia patients.
4. In chapter four, review of neurobiological findings points towards significantly shared structural and functional brain abnormalities, schizophrenia, and OCD. The preliminary evidence also suggests a greater deficit in schizo-obsessive patients than schizophrenia patients without OCS on neuropsychological and neurophysiological, biological mechanism leading to OCS in schizophrenia, neurochemical circuits, neuroimaging measures. Additionally, analysis in some previous study in different neuroimaging used of OCD and schizophrenia patients to summarize the tables further examination in longitudinal studies with bigger samples to delineate the underlying neurobiology.
5. chapter five, it is the diagnosis and management of OCS and schizophrenia patients.
6. Finally, I have done my recommendation, I highlighted some important points which I have seen while I was doing my thesis and I suggested specific opinion that needs to put more efforts regarding obsessive compulsive disorders to provide mental health service and improve the care of patients those suffers schizophrenia.

## Reference:

- [1]. Abramovitch, A., Abramowitz, J. S., & Mittelman, A. (2013). The neuropsychology of adult obsessive-compulsive disorder: a meta-analysis.
- [2]. Abramovitch, A., Dar, R., Mittelman, A., & Wilhelm, S. (2015). Comorbidity Between Attention Deficit/Hyperactivity Disorder and Obsessive-Compulsive Disorder Across the Lifespan: A Systematic and Critical Review.
- [3]. Abramowitz, J. S., Taylor, S., & McKay, D. (2009). Obsessive-compulsive disorder
- [4]. Alexander-Bloch, A., Raznahan, A., Bullmore, E., & Giedd, J. (2013) The convergence of maturational change and structural covariance in human cortical networks.
- [5]. Hoexter, M. Q. (2012). Obsessive-compulsive symptom dimensions correlate to specific gray matter volumes in treatment-naive patients.
- [6]. Anholt, G. E., Aderka, I. M., van Balkom, A. J., Smit, J. H., Schruers, K., van der Wee, N. J. van Oppen, P. (2014). Age of onset in obsessive-compulsive disorder: admixture analysis with a large sample.
- [7]. Bassett, D. S., Brown, J. A., Deshpande, V., Carlson, J. M., & Grafton, S. T. (2011). Conserved and variable architecture of human white matter connectivity. *Neuroimaging*, 54(2), 1262-1279.
- [8]. Benedetti, F., Giacosa, C., Radaelli, D., Poletti, S., Pozzi, E., Dallaspezia, S., Smeraldi, E. (2013). Widespread changes of white matter microstructure in obsessive-compulsive disorder: effect of drug status.
- [9]. Carmona, S., Bassas, N., Rovira, M., Gispert, J. D., Soliva, J. C., Prado, M., Vilarroya, O. (2007). Pediatric OCD structural brain deficits in conflict monitoring circuits: a (VBM) study.
- [10]. Del Casale, A., Kotzalidis, G. D., Rapinesi, C., Serata, D., Ambrosi, E., Simonetti, A., Girardi, P. (2011). Functional neuroimaging in obsessive-compulsive disorder
- [11]. Fan, Q., Palaniyappan, L., Tan, L., Wang, J., Wang, X., Li, C., Liddle, P. F. (2013). Surface anatomical profile of the cerebral cortex in obsessive-compulsive disorder: a study of cortical thickness, folding and surface area.
- [12]. Huey, E. D., Zahn, R., Krueger, F., Moll, J., Kapogiannis, D., Wassermann, E. M., & Grafman, J. (2008). A psychological and neuroanatomical model of obsessive-compulsive disorder.
- [13]. Jayarajan, R. N., Venkatasubramanian, G., Viswanath, B., Janardhan Reddy, Y. C., Srinath, S., Vasudev, M. K., & Chandrashekar, C. R. (2012). White matter abnormalities in children and adolescents with obsessive-compulsive disorder: a diffusion tensor imaging study
- [14]. Koch, K., Reess, T. J., Rus, O. G., Zimmer, C., & Zaudig, M. (2014). Diffusion tensor imaging (DTI) studies in patients with obsessive-compulsive disorder (OCD): a review.
- [15]. Lochner, C., Fouche, J. P., du Plessis, S., Spottiswoode, B., Seedat, S., Fineberg, N., Stein, D. J. (2012). Evidence for fractional anisotropy and mean diffusivity white matter abnormalities in the internal capsule and cingulum in patients with obsessive-compulsive disorder
- [16]. Matsumoto, R., Ito, H., Takahashi, H., Ando, T., Fujimura, Y., Nakayama, K., Suhara, T. (2010). Reduced gray matter volume of dorsal cingulate cortex in patients with obsessive-compulsive disorder: a voxel-based morphometric study.
- [17]. Pauls, D. L., Abramovitch, A., Rauch, S. L., & Geller, D. A. (2014). Obsessive-compulsive disorder: an integrative genetic and neurobiological perspective.
- [18]. Radua, J., & Mataix-Cols, D. (2009). Voxel-wise meta-analysis of grey matter changes in obsessive-compulsive disorder.
- [19]. Ruscio, A. M., Stein, D. J., Chiu, W. T., & Kessler, R. C. (2010). The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication.
- [20]. Sinopoli, V. M., Burton, C. L., Kronenberg, S., & Arnold, P. D. (2017). A review of the role of serotonin system genes in obsessive-compulsive disorder.
- [21]. Tang, W., Huang, X., Li, B., Jiang, X., Li, F., Xu, J., Gong, Q. (2015). Structural brain abnormalities correlate with clinical features in patients with drug-naive OCD: A DARTEL-enhanced voxel-based morphometry study.
- [22]. Taylor, S. (2011). Etiology of obsessions and compulsions: a meta-analysis and narrative review of twin studies.
- [23]. Taylor, S. (2013). Molecular genetics of obsessive-compulsive disorder: a comprehensive meta-analysis of genetic association studies.
- [24]. Bhattacharyya S, Chakraborty K (2007) Glutamatergic dysfunction—newer targets for anti-obsessional drugs.
- [25]. Bleich-Cohen M, Hendler T, Weizman R, Faragion S, Weizman A, Poyurovsky M (2014) Working memory dysfunction in schizophrenia patients with obsessive-compulsive symptoms: an fMRI study.
- [26]. loch MH, Landeros-Weisenberger A, Kelmendi B, Coric V, Bracken MB, Leckman JF (2006) A systematic review: antipsychotic augmentation with treatment refractory obsessive-compulsive disorder.
- [27]. Cunill R, Huerta-Ramos E, Castells X (2013) The effect of obsessive-compulsive symptomatology on executive functions in schizophrenia: a systematic review and meta-analysis. *Psychiatry Res*.
- [28]. Dickel DE, Veenstra-VanderWeele J, Cox NJ, Wu X, Fischer DJ, Van Etten-Lee M, Himle JA, Leventhal BL, Cook EH Jr, Hanna GL (2006) Association testing of the positional and functional candidate gene SLC1A1/EAAC1 in early-onset obsessive-compulsive disorder. *Arch Gen Psychiatry* 63(7):778–785.
- [29]. Keshavan MS, Tandon R, Boutros NN, Nasrallah HA (2008) Schizophrenia, “just the facts”: what we know in 2008 Part 3: neurobiology.
- [30]. Koch K, Reess TJ, Rus OG, Zimmer C, Zaudig M (2014) Diffusion tensor imaging (DTI) studies in patients with obsessive-compulsive disorder (OCD): a review. *J Psychiatry Res*
- [31]. Meyer-Lindenberg A (2010) From maps to mechanisms through neuroimaging of schizophrenia.
- [32]. Rotge JY, Langbour N, Guehl D, Bioulac B, Jaafari N, Allard M, Aouizerate B, Burbaud P (2010) Gray matter alterations in obsessive-compulsive disorder: an anatomic likelihood estimation meta-analysis.
- [33]. Monzani B, Rijdsdijk F, Harris J et al (2014) The structure of genetic and environmental risk factors for dimensional representations of DSM-5 obsessive-compulsive spectrum disorders.
- [34]. Ryu S, Won H-H, Oh S et al (2013) Genome-wide linkage scan of quantitative traits representing symptom dimensions in multiplex schizophrenia families.
- [35]. Schirmbeck F, Nieratschker V, Frank J et al (2012) Polymorphisms in the glutamate transporter gene SLC1A1 and obsessive-compulsive symptoms induced by second-generation antipsychotic agents.
- [36]. Galderisi S, Quarantelli M, Volpe U, et al. Patterns of structural MRI abnormalities in deficit and nondeficit schizophrenia. *Schizophr Bull*. 2008;34(2):393–401.
- [37]. Bora E, Fornito A, Radua J, et al. Neuroanatomical abnormalities in schizophrenia: a multimodal voxelwise meta-analysis and meta-regression analysis. *Schizophr Res*. 2011.
- [38]. Vemuri P. Diagnostic neuroimaging across diseases. *Neuroimage* (2012) 61:457–63. doi: 10.1016/j.neuroimage.2011.11.002

- [39]. Noble WS. What is a support vector machine? *Nat Biotechnol.* (2006) 12:1565–7. doi: 10.1038/nbt1206-1565
- [40]. Li F, Huang X, Tang W, Yang Y, Li B, Kemp GJ, et al. Multivariate pattern analysis of DTI reveals differential white matter in individuals with obsessive-compulsive disorder. *Hum Brain Mapp.* (2014) 35:2643–51. doi: 10.1002/hbm.22357
- [41]. Ingalhalikar M, Parker D, Bloy L, Roberts TP, Verma R. Diffusion based abnormality markers of pathology: toward learned diagnostic prediction of ASD. *Neuroimage* (2011) 57:918–27. doi: 10.1016/j.neuroimage.2011.05.023
- [42]. O'Dwyer L, Lamberton F, Bokde AL, Ewers M, Faluyi YO, Tanner C, et al. Using support vector machines with multiple indices of diffusion for automated classification of mild cognitive impairment. *PLoS ONE* (2012) 7: e32441. doi: 10.1371/journal.pone.0032444
- [43]. Besga A, Termenon M, Grana M, Echeveste J, Perez JM, Gonzalez-Pinto A. Discovering Alzheimer's disease and bipolar disorder white matter effects building computer aided diagnostic systems on brain diffusion tensor imaging features. *Neurosci Lett.* (2012) 520:71–6. doi: 10.1016/j.neulet.2012.05.033
- [44]. Schnyer DM, Clasen PC, Gonzalez C, Beevers CG. Evaluating the diagnostic utility of applying a machine learning algorithm to diffusion tensor MRI measures in individuals with major depressive disorder. *Psychiatry Res.* (2017) 264:1–9. doi: 10.1016/j.psychres.2017.03.003
- [45]. Rozycki M, Satterthwaite TD, Koutsouleris N, Erus G, Doshi J, Wolf DH, et al. Multisite machine learning analysis provides a robust structural imaging signature of schizophrenia detectable across diverse patient populations and within individuals. *Schizophr Bull.* (2017) 44:1035–44. doi: 10.1093/schbul/sbx137
- [46]. Sacchet MD, Prasad G, Foland-Ross LC, Thompson PM, Gotlib IH. Support vector machine classification of major depressive disorder using diffusion-weighted neuroimaging and graph theory. *Front Psychiatry* (2015) 6:21. doi: 10.3389/fpsy.2015.00021

Nasra Bashir Mohamed. “The Use of Machine and Deep Learning Algorithms in The Diagnosis of Obsessive-Compulsive Disorders: An Analysis of Meta study..” *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 19(6), 2020, pp. 42-57.