

## INTRODUCTION

The Surgeon General identifies stigma surrounding mental illness and its treatment as a potent barrier to help-seeking (*U.S. Department of Health and Human Services, 1999*). A review of population-based studies highlighted the extent to which stigma surrounds mental illness (*Angermeyer and Dietrich, 2006*); however, this review also demonstrates that stigma associated with many mental illnesses has been understudied, since most stigma research has focused on depression, schizophrenia, and bipolar disorder.

Furthermore, stigma exists at various levels; persons with mental illness not only encounter public stigma, expressed as prejudice and discrimination, but may also suffer from self-stigma, through acceptance of the prejudices that surround them (*Ruesch et al., 2005*), and their families or intimates may experience courtesy stigma based on kinship or affiliation with the stigmatized person (*Goffman, 1963*).

A recent study on stigma associated with child mental health conditions identified substantial stigma concerns among participating adults from a nationally representative sample (*Pescosolido, 2007*).

Moreover, when responding to vignettes depicting several stigmatizing conditions including attention deficit hyperactivity disorder (ADHD), depression, “normal troubles”

and physical illness, a gradient of rejection of these groups was reported such that individuals with ADHD and depression was rejected the most and those with “normal troubles” and physical illness were rejected the least (*Martin et al., 2007*).

These stigmatizing reactions were higher toward adolescents than children. When presented with similar vignettes, children and adolescents (ages 8–18) participating in a national survey were more likely to make negative attributions about peers with ADHD and depression than peers with asthma, particularly with respect to the likelihood of antisocial behavior and violence (*Walker et al., 2008*).

## **RATIONALE OF THE STUDY**

**I**t is well established that most of mental illnesses is associated with stigma.

Therefore, this study was designed to assess stigma in ADHD, its degree and its relation to the severity of the symptoms.

## **HYPOTHESIS**

**T**he study hypothesis that most of patients diagnosed with ADHD and their families suffer from stigma.

Also there is relation between severity of symptoms and increased stigma in patients diagnosed with ADHD and their families.

## **AIM OF THE WORK**

- 1- To assess the stigma in patients diagnosed with ADHD and their family.
- 2- To study the relation between the degree of the stigma and the severity of symptoms.
- 3- And to determine which symptoms may lead to more stigma.

# **ATTENTION DEFICIT HYPERACTIVITY DISORDER**

## **Description**

ADHD is a developmentally inappropriate level of attention, hyperactivity, and impulsivity started since early childhood (*Barkley, 2009*). Not all core features needed to be present. Three subtypes: mainly inattentive type, mainly hyperactive-impulsive type and combined type according dsm 5 (*American Psychiatric Association, 2013*). Problems must start before 12 years old and persist for at least 6 months, child must be maladaptive with behavior inconsistent with developmental level in two or more different settings (*e.g.*, school and home), with clinically significant impairment in social, academic functioning or, occupational.

## **Epidemiology**

### **Incidence and prevalence**

ADHD is the most common psychiatric disorder of the childhood, the prevalence ranges from 5-12% in school-aged children with male to female ratio around 3:1 in children and adolescents, though it is believed that females are under-diagnosed. Approximately 8-10% of males and 3-4% of females, under the age of 18 have ADHD (*Sadek, 2014*).

ADHD's onset occurs around 3 years of age in both sexes (*Neuman et al., 2005*).

About 80% of children with ADHD will continue into adolescence and 60% will maintain their core symptoms into adulthood (*Sadek, 2014*).

ADHD symptoms usually decrease with age and the rate of their persistence is only 15% by age of 25 (*Faraone et al., 2006*).

Girls with ADHD have lower rates of hyperactivity, impulsivity, and inattention but have more level of intellectual problems.

### **Etiology:**

ADHD is best understood as a multifactorial disorder in which genetic and environmental factors play a complex role. Researchers are looking at possible environmental factors, and are studying how brain injuries, nutrition, and environmental factors might contribute to ADHD (*Gilliam et al., 2011*).

### **A-Genetic Factors:**

Studies of twins show that ADHD often runs in families, family-genetic studies indicate that ADHD aggregates in families, with a 5-8 fold increased risk in first-degree relatives and a 2-3 fold increased risk in second-degree relatives, while

twin studies found evidence for heritability of 0.75-0.91 (*Faraone et al., 2006*).

Many polymorphisms in candidate genes, mainly in dopamine, serotonin, and nor-adrenergic signaling pathways have been found to be negatively or positively associated with ADHD, all of them with small effect (*Thapar et al., 2007*).

Recent meta-analysis confirms the relevance of a number of these genes. The associations found so far, only account for a small percentage of the genetic component of ADHD, which clearly suggests the need for broader (hypothesis-generating) genetic analysis. Abnormalities in the dopamine D4 and D5 receptor genes are associated with ADHD. Dopamine transporter genes (DAT1) lead to low levels of dopamine in patients with ADHD (*Williams et al., 2012*).

Researchers are looking at several genes that may make children more likely to develop this disorder, knowing the genes involved may one day help researchers prevent the occurrence of the disorder (*Faraone et al., 2010*).

Researchers from the National Human Genome Research Institute in Bethesda, Maryland, have further characterized a genetic risk for attention-deficit/hyperactivity disorder (ADHD). Their study focused on gene variants within a previously characterized ADHD linkage region called *ADGRL3* (*Martinez et al., 2016*).



**B-Neurobehavioral Concepts:**

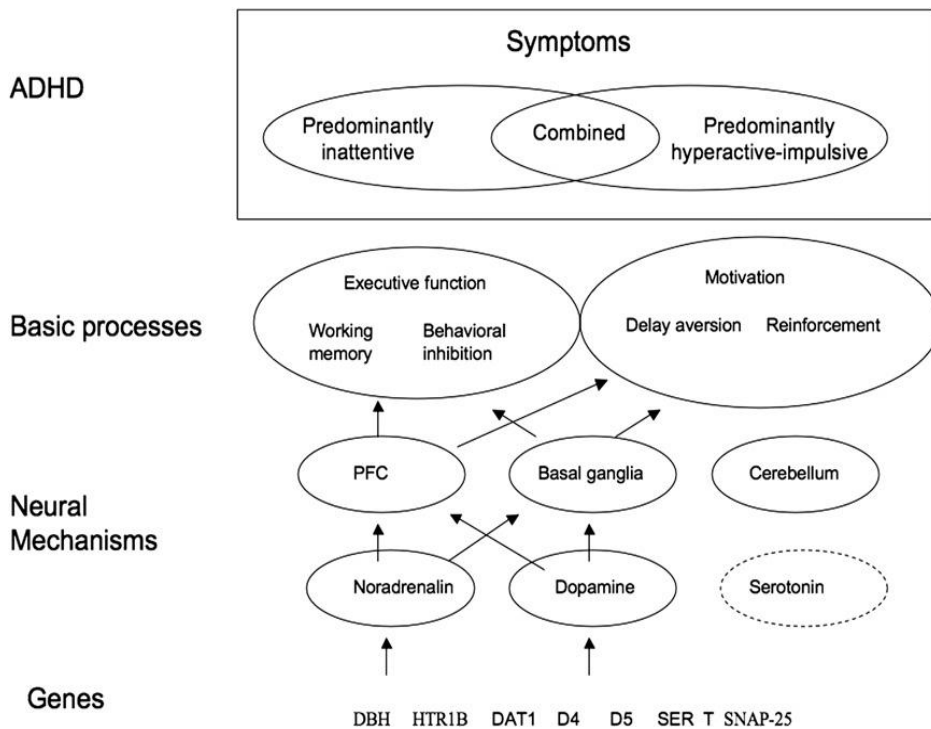
There are several neurophysiological models of ADHD, one of such focuses on the role of executive dysfunction as apparent in deficient inhibitory control, working memory and planning. These deficits are thought to be due to hyperactivity in the frontodorsal striatal circuit and associated neocortical dopaminergic branches due to decreased extracellular dopamine concentrations and upregulated post synaptic D1 receptors, stimulus evoked release of dopamine was showed to be decreased in these models which is consistent with the impairment of dopamine transmission. There is also considerable evidence suggesting that the noradrenergic system is poorly controlled due to hypo function of the alpha 2 auto receptors. And so new evidence suggests that ADHD behavior may be the result of imbalance between increased noradrenergic and decreased dopaminergic regulation of neural circuits in the prefrontal cortex (*Russell et al., 2005*).

Another model focuses on problems in time reproduction and timing of motor behaviors which may be due to problems in frontocerebellar circuits which denotes that children with ADHD tend to perform more slowly, more variably, and less accurately, and frequently have problems with timing of motor output when performing motor tasks (*Rommelse et al., 2007*).

Other views assume that the existence of neuropsychological heterogeneity in ADHD has an important

implication for management and potentially impacts both diagnostic strategies and treatment options (*Sonuga-Barke, 2005*).

Another important area of research which have gained attention in the past few years is the executive and attentional contributions to theory of mind deficit in ADHD. Theory of mind progressively emerges during childhood starting with the ability to understand emotions of others in a particular situation (first order theory of mind), followed with the understanding that people have specific beliefs about the beliefs and thoughts of other people (second order theory of mind) and finally the development of higher order theory of mind competences involving more complex social situations (advanced theory of mind) (*Premack and Woodruff, 1978*).



**Fig. (1):** Illustration of relation between factors affecting development of ADHD (Fliers et al., 2010).

**Neuroimaging findings in ADHD.** A network of distributed brain regions is thought to be involved in attention, cognition and behavioural self-regulation (Posner and Petersen, 1990). Indeed, structural neuroimaging studies in ADHD research suggest that patients have widespread anatomical differences from controls; smaller volumes in the dorsolateral prefrontal cortex, the caudate nucleus, the corpus callosum and the cerebellum have been reported (Seidman et al., 2005). Functional neuroimaging studies predominantly using positron emission tomography (PET) and functional MRI (fMRI) support the involvement of frontostriatal abnormalities

(particularly in the dorsal anterior cingulate cortex, the lateral prefrontal cortex and the striatum) in ADHD (*Bush et al., 2005*). The study of neurobiological endophenotypes in ADHD has led to a better understanding of the relationship between structural and functional abnormalities in ADHD. Dopamine deficits are thought to have a role in the anatomical and functional differences observed in dopamine-related brain areas, including the caudate nucleus, the globus pallidus, the corpus callosum and the cerebellum vermis (*Valera et al., 2007*).

Volumetric and anatomical differences in brain areas are integral to comprehensive models of ADHD pathophysiology, and they could theoretically be used to inform neuroimaging biomarkers of ADHD. Such biomarkers could eventually become part of a comprehensive clinical evaluation for ADHD. At present, however, both structural and functional neuroimaging data on ADHD are inconclusive, owing in part to the use of different imaging technologies across studies and to a lack of adolescent and adult data (*Seidman et al., 2005*). In addition, most imaging studies of ADHD are underpowered, using samples of fewer than 20 subjects per group.

### **C-Environmental Factors:**

Studies suggest a potential link between tobacco smoking and alcohol use during pregnancy with ADHD in children (*Millichap, 2008*).

In addition, preschoolers who are exposed to high levels of lead, which can sometimes be found in plumbing fixtures or paint in old buildings, have a higher risk of developing ADHD (*Froehlich et al., 2009*).

Meanwhile there is currently no research showing that artificial food coloring causes ADHD, however a small number of children with ADHD may be sensitive to food dyes, artificial flavors, preservatives, or other food additives (*Nigg et al., 2012*).

### **Comorbidities with ADHD:**

ADHD is a clinically heterogeneous condition, in which symptom overlap and comorbidity with other conditions is the rule rather than the exception (*Yoshimasu et al., 2012*).

- Oppositional defiant disorder: 35%
- Conduct disorders: 26%
- Delinquency or criminal behavior: 25%
- Depressive disorder: 18%
- Anxiety disorders: 26%
- Emotional problems: 50%
- Problems with social skills: 50% or more

- Learning disabilities: 6% to 20%; when a broad definition of academic underachievement is used, the rates show a wide variability of 10% to 90%
- Drug abuse and dependence
- Tourette syndrome

## **Diagnosis**

### **Summary approach**

- Any child 6 to 12 years of age who presents with inattention, hyperactivity, impulsivity, academic underachievement, or behavior problems should be evaluated for ADHD.

### **The following are the most used diagnostic criteria for ADHD:**

#### **1- The International Statistical Classification of Diseases and Related Health Problems, 10th revision (ICD-10)**

#### **Criteria for diagnosis of hyperkinetic disorders:**

**Inattention:** At least six of the following symptoms of inattention have persisted for at least 6 months, to a degree that is maladaptive and inconsistent with the developmental level of the child:

1. Often fails to give close attention to details or makes careless errors in schoolwork, work, or other activities.
2. Often fails to sustain attention in tasks or play activities
3. Often appears not to listen to what is being said to him or her
4. Often fails to follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (not because to oppositional behavior or failure to understand instructions).
5. Is often impaired in organizing tasks and activities.
6. Often avoids, strongly dislikes tasks, or is reluctant to engage in tasks such as schoolwork or homework that require sustained mental effort.
7. Often loses things necessary for tasks and activities (e.g., toys, school assignments, pencils, books, or tools).
8. Is often easily distracted by external stimuli.
9. Is often forgetful in the course of daily activities.

**Hyperactivity:** At least three of the following symptoms of hyperactivity have persisted for at least 6 months to degree that is maladaptive and inconsistent with the developmental level of the child:

1. Often fidgets with hands or feet or squirms in seat.