# INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) is a mental disorder of the neurodevelopmental type **(Sroubek et al., 2013)**. It is characterized by problems paying attention, excessive activity, or difficulty controlling behavior which is not appropriate for a person's age **(National Institute of Mental Health, 2016)**. The symptoms appear before a person is 12 years old, are present for more than six months, and cause problems in at least two settings (such as school, home, or recreational activities). Symptoms are grouped into three categories; inattention, hyperactivity and impulsivity **(Dulcan et al., 2011)**.

It's prevalence in children aged 18 and under found an overall pooled estimate of 7.2% **(Thomas et al., 2015)**. Rates are similar between countries and depend mostly on how it is diagnosed **(Tsuang et al., 2011)**. ADHD is diagnosed approximately three times more often in boys than in girls, although the disorder is often overlooked in girls due to their symptoms differing from those of boys **(Emond et al., 2009)**. About 30–50% of people diagnosed in childhood continue to have symptoms into adulthood **(Ginsberg et al., 2014)**.

According to one meta-analysis, the most two prevalent sleep disorders among children are confusional arousals and sleep walking. An estimated 17.3% of kids between 3 and 13 years old experience confusional arousals. About 17% of

children sleep walk, with the disorder being more common among boys than girls. The peak ages of sleep walking are from 8 to 12 years old **(Carter et al., 2014)**. A different systematic review offers a high range of prevalence rates of sleep bruxism for children. Between 15.29 and 38.6% of preschoolers grind their teeth at least one night a week. All but one of the included studies reports decreasing bruxism prevalence as age increased as well as a higher prevalence among boys than girls **(Machado et al., 2014)**.

Another study noted 7-16% of young adults suffer from delayed sleep phase disorder. This disorder reaches peak prevalence when people are in their 20's. Also, 7-36% has difficulty initiating sleep **(Gradisar et al., 2011)**.

Sleep disorders and ADHD commonly co-exist **(Corkum et al., 2011)**. Sleep problems, particularly difficulty initiating and maintaining sleep, are frequently reported in children and adolescents with attention-deficit/ hyperactivity disorder (ADHD) in mental health practice settings. In fact, the relationship between sleep problems and ADHD is hardly straightforward. For example, manifestations of primary sleep disorders such as obstructive sleep apnea may “mimic” ADHD symptomatology in some patients, comorbid sleep problems may exacerbate ADHD symptoms in others, psychotropic medications used to treat ADHD or comorbid psychiatric conditions associated with ADHD (e.g., mood disorders, anxiety) may themselves result in sleep problems **(Owens, 2009).**

In addition, clear evidence from experimental laboratory–based studies and clinical observations indicates that insufficient and poor-quality sleep result in daytime sleepiness and behavioral/mood dysregulation and impact upon neurocognitive functions in children. Thus, from a clinical standpoint, sleep difficulties in children with ADHD not only have a potentially direct negative impact on the nature and severity of daytime ADHD symptoms but also present a considerable challenge for the clinician attempting to develop effective treatment strategies **(Owens, 2009).**

In children with ADHD, insomnia is the most common sleep disorder. Problems with sleep initiation are common among individuals with ADHD; those children often will be deep sleepers and have significant difficulty getting up in the morning **(Brown, 2008)**.

Primary sleep disorders may affect attention and behavior, and the symptoms of ADHD may affect sleep. It is thus recommended that children with ADHD be regularly assessed for sleep problems. Obstructive sleep apnea can also cause ADHD type symptoms **(Lal et al., 2012)**.

Obstructive sleep apnea affects 0.7: 3.0% of children and can produce excessive daytime sleepiness, behavioral problems, learning disabilities, right-sided heart failure, growth retardation, or failure to thrive **(Chervin et al., 2000)**.

Recent conservative estimates suggest that more than 80% of children with obstructive sleep apnea syndrome have yet to be diagnosed, as they frequently have distinct signs and symptoms that are less widely recognized **(Chervin et al., 2000)**.

Several studies have shown that obstructive sleep apnea sufferers consistently show deficits in the cognitive domains of attention, episodic and verbal memory, and executive functions **(Gagnon et al., 2014).**

According to recent meta-analysis which evaluated cognitive effects of Obstructive Sleep Apnea through objective neuropsychological measures: The greatest deficits were found in the areas of psychomotor speed and executive function, while memory functions, motor control, construction, attention, and speed of processing abilities were affected to a lesser extent **(Bawaden et al., 2011).**

# OBJECTIVES

***This study was conducted to:***

1- Find out the rate of sleep problems among ADHD patients.

2- Study factors correlating sleep problems among study group.

3- Explore factors that might affect sleep disturbance if present.

4- Do further assessment for sleep apnea in those patients who might have sleep problems.

Chapter 1

# ATTENTION DEFICIT HYPERACTIVITY DISORDER

## Introduction:

Attention deficit hyperactivity disorder (ADHD) is one of the most prevalent psychiatric disorders in children which greatly impairs social and cognitive functions in affected individuals.

It is characterized by a childhood-onset of hyperactivity, inattention and impulsivity that starts in early childhood and often persists into adulthood ***(Barkley, 2009)***

The current psychiatric disease classification system, DSM-5 classifies ADHD into three subtypes: a mainly inattentive, a mainly hyperactive–impulsive and a combined subtype ***(American Psychiatric Association, 2013).***

Historically, a series of names including minimal brain damage syndrome, minimal brain dysfunction, hyperkinetic reaction of childhood, and attention deficit disorder have been used to describe the disorder now known as attention-deficit/hyperactivity disorder. Since 1980, the term "attention" has been incorporated into the name of the disorder (e.g., attention deficit disorder with/without hyperactivity or

attention- deficit/hyperactivity disorder) ***(American Psychiatric Association,1994).***

## Prevalence of ADHD:

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).***

## Diagnosis:

Concerns have been raised that group of symptoms do not constitute a diagnosis and that core symptoms might not constitute a diagnostic entity for ADHD ***(Furman et al., 2004)***

Following the "symptoms do not constitute a diagnosis" logic leads us in two immediate directions:

**First**, several authors have considered other hypotheses regarding the core symptoms of inattention, distractibility, and

hyperactivity, including the possibilities that this behavior may represent:

1. One end of a normal distribution of school-aged behavior (especially for boys, who are overrepresented in every study)
2. An expression of endogenous temperament
3. Differences in rates of developmental maturation
4. Rigid or unreasonable parental, societal, or educational expectations for school-aged children **(Carey, 2002).**

**Second**, we need to examine the possibility that for some or many children, the core symptoms of ADHD are neither a normal variant nor a defined disease state, these symptoms might represent expressions of internalized conflict or unmet emotional or educational needs that differ from child to child. In this scenario, each child should have a full medical, educational, and psychological or psychiatric evaluation. ***(Furman et al., 2004)***

The American Academy of Child and Adolescent Psychiatry recommended an initial evaluation for children with symptoms suggestive of ADHD: **(Taylor, 2004**)

1. An interview with the parents (to include the child's and the family's history)
2. Use of standardized rating scales

3. School information, including the results of academic testing
4. A family diagnostic interview
5. A (psychiatric) child diagnostic interview then:

   (1) Complete physical examination

   (2) Referral for additional testing as needed.

Thus in this approach, multiple diagnoses are actively considered, including psychiatric and affective disorders, educational and learning disorders, and or bereavement to domestic violence, abuse, and substance abuse **(Taylor, 2004).**

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

1. The International Statistical Classification of Diseases and Related Health Problems, 10threvision (ICD-10) **(World Health Organization, 1994).**
2. Diagnostic and Statistical Manual of Mental Disorders, 5th Edition (DSM-5) **(American Psychiatric Association, 2013)**

ADHD was moved within the manual and can now be found in the "Neurodevelopmental Disorders" chapter to reflect brain developmental correlates with ADHD.

## 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)**.

Children with ADHD are more likely to experience one or more psychiatric comorbidities by the age of 19 as autistic spectrum disorders, motor coordination problems, tic disorders, sleep disorders, specific learning disorders such as dyslexia, and child-psychiatric disorders such as depression, anxiety, oppositional defiant and conduct disorders **(Yoshimasu et al., 2012)**.

## 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).**

***B- Neurobehavioral Concepts:***

There are several neurophysiological models of ADHD, one of such models 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 mesocortical dopaminergic branches due to decreased extracellular dopamine concentrations and up regulated 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 **(Sagvolden 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., 2010)**.

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 & Woodruff, 1978).**

Theory of mind represents complex aspects of social cognition that relies on the ability to attribute mental states (desires, beliefs, feelings, thoughts and intentions) to one self and others and predict and understand people's behavior based on their mental states **(Premack&Woodruff, 1978).**

Socio-emotional problems in ADHD are strongly associated with educational and interpersonal problems and increased risk of developing other psychiatric disorders which emphasize the need to clarify the nature of their theory of mind deficits **(Daly et al., 2007).** Investigations of theory of mind in patients with ADHD have shown inconsistent findings, also studies about this aspect remain scarce and controversial. For example, several studies have found performances in various theory of mind tasks in children with ADHD as similar as other typically developing children and despite the absence of theory of mind dysfunctions in these study, executive dysfunctions were clear in children with ADHD **(Yang et al., 2009)**.

These findings suggest that executive dysfunctions don't necessary lead to theory of mind deficits **(McWhinnie, 2009).**

Conversely other studies have shown both theory of mind and executive function deficits in children with symptoms of inattention **(Alison et al., 2015).**

### *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).**

Chapter 2

# SLEEP AND ITS DISORDERS IN CHILDREN AND ADOLESCENTS

Sleep is an alternating pattern of neural activity that arises from multiple brain regions, neurotransmitter systems and modulatory hormones ***(Wulff et al., 2009)***.

## Normal sleep architecture:

The progression of sleep stages across the night is called 'sleep architecture' and typically consists of repetitive but slightly changing 'sleep cycles', each lasting for 90-120 minutes. Sleep in adults lasts approximately 8 hours. Collectively, sleep has been classified into rapid eye movement (REM) and non-REM (NREM) sleep stages. Upon the initiation of sleep an individual will pass down slowly through stages 1–4 NREM sleep and then rapidly ascend these stages into REM sleep, which is accompanied by considerable muscle paralysis of the body (motor atonia). Adults experience approximately four to five NREM/REM sleep cycles every night with period lengths between 80 and 110 min ***(Hirshkowitz 2004)***.

NREM stages 3 and 4 predominate during the first third of the night and REM sleep predominate in the last half of the night. The NREM/REM cycle is repeated three to six times each night and with each cycle; the amount of slow-wave sleep