



# ***A Study Of Circulating Brain Derived Neurotrophic Factor In A Sample Of Egyptian Attention Deficit Hyperactive Children.***

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# List of abbreviations

Abbreviation	
<b>AAPs</b>	Atypical antipsychotics
<b>ADD</b>	Attention deficit disorder
<b>ADHD</b>	Attention deficit hyperactivity disorder
<b>ADRA2A</b>	Adrenergic alpha 2 A receptor
<b>AKT</b>	Serine threonine kinase
<b>ATX</b>	atmoxetine
<b>BD</b>	Bipolar disorder
<b>BDNF</b>	Brain derived neurotrophic factor
<b>CBT</b>	cognitive behavioral therapy
<b>CD</b>	Conduct disorder
<b>CT</b>	Cat scan
<b>DA</b>	Dopamine
<b>DAT1</b>	The dopamine active transporter 1 gene
<b>DBH</b>	Dopa- B_ hydroxylase
<b>DMAP</b>	urinary dimethyl alkylphosphate
<b>DRD4,5</b>	Dopamine receptor D4,5
<b>DSM-II</b>	Diagnostic and Statistical Manual of Mental Disorders II
<b>DSM-III</b>	Diagnostic and Statistical Manual of Mental Disorders III
<b>DSM-IV-TR</b>	diagnostic and statistical manual of mental disorders- text revision IV
<b>ERK</b>	extracellular signal related kinase
<b>FDA</b>	Food and Drug Administration
<b>5-HT</b>	5-hydroxytryptamine
<b>ICD9</b>	International Classification of Diseases 9
<b>ID</b>	Intellectual disability
<b>IFC</b>	inferior frontal cortex
<b>LD</b>	learning disabilities
<b>LTP</b>	long-term potentiation
<b>MAPK</b>	mitogen-activated protein kinase
<b>MDD</b>	Major Depressive Disorder
<b>MeCP2</b>	methylated CpG binding protein
<b>MPH</b>	Methyl phenydate
<b>MRI</b>	Magnetic resonance imaging
<b>NAc</b>	nucleus accumbens
<b>NE</b>	norepinephrine

<b>NGF1</b>	nerve growth factor 1
<b>NICE</b>	National Institute for Health and Clinical Excellence
<b>NIMH</b>	National Institute of Mental Health
<b>NDMA</b>	N-methyl-d-aspartate
<b>NTFs</b>	neurotrophins
<b>NTRK2</b>	Neurotrophic tyrosine kinase, receptor, type 2.
<b>ODD</b>	Oppositional defiant disorder
<b>OFC</b>	orbitofrontal cortex
<b>PCB</b>	Polychlorinated biphenyls
<b>PET</b>	positron emission tomography
<b>PFC</b>	prefrontal cortex
<b>PI3K</b>	phosphatidylinositol 3-kinase
<b>PLC</b>	phospholipase C
<b>PTSD</b>	post-traumatic stress disorder
<b>RTT</b>	Rett syndrome
<b>SLC6A2</b>	Solute carrier family 6 (neurotransmitter transporter, dopamine), member 2
<b>SLC6A3</b>	Solute carrier family 6 (neurotransmitter transporter, dopamine), member 3
<b>SLC6A4</b>	Solute carrier family 6 (neurotransmitter transporter, dopamine), member 4
<b>SNAP25</b>	Synaptosomal associated protein 25
<b>SNPs</b>	single-nucleotide polymorphisms
<b>SPECT</b>	Single-photon emission computed tomography
<b>SSRIs</b>	Selective serotonin reuptake inhibitor
<b>SUDs</b>	substance use disorders
<b>SZ</b>	Schizophrenia
<b>TrkB</b>	tropomyosin-related kinase B
<b>(V66M)</b>	Valine to methionine mutation at position 66
<b>VTA</b>	Ventral tegmental area.



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## **Aim of the work**

The aims of this study were; to investigate the possible relation between BDNF level and ADHD so it could be used as a potential marker for this disorder, to investigate the relation between severity of inattention and BDNF, to investigate the effect of pharmacotherapy on level of BDNF.

## **Introduction**

Attention-deficit hyperactivity disorder (ADHD) has been identified as an important psychiatric condition in terms of its prevalence (around 5% worldwide) and its impact on quality of life for patients and their families (**Cho et al., 2010**). Also ADHD is the most commonly diagnosed behavioral disorder of childhood (**American Academy of Pediatrics, 2000**).

Attention-deficit/hyperactivity disorder (ADHD) is characterized by hyperactivity, diminished sustained attention and higher levels of impulsivity in a child or adolescent than expected for someone of that age and developmental level (**Sadock and Sadock, 2007**). These core behavioral symptoms must be pervasive across situations, persistent for more than 6 months and observed before the age of 7 years, as defined by the diagnostic and statistical manual of mental disorders (DSM-IV-TR) issued by (**the American Psychiatric Association, 2000**).

These behavioral manifestations contribute to diminished academic, occupational and social functioning, and have neurobiological bases (**De La Fuente A, 2013**). 30 to

50% of those individuals diagnosed in childhood continue to have symptoms into adulthood. As they mature (**Bálint et al, 2008**).

The etiology of ADHD is now viewed to be pathophysiologically and clinically heterogeneous entity, hypotheses on the etiology of ADHD have evolved from simple one-cause theories to multi-factorial processes that reflect the confluence of many types of risk factors, including genetic, neurochemical, environmental and psychosocial factors (**Biederman and Faraone, 2005**).

Genetic research on ADHD started with the finding that hyperactivity tends to aggregate in families since then, family studies have shown that ADHD shows familial clustering both within and across generations. Increased rates of ADHD among the parents and siblings of ADHD children have been observed (**Franke et al., 2012**).

Evidence from various sources suggests primary involvement of the dopaminergic system. Molecular genetic studies also indicate a linkage of genetic polymorphisms in the dopaminergic system, such as dopamine D4 and D5 receptors, and dopamine transporter (DAT), to ADHD (**Bobb et al., 2005**).