

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

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Medical Studies Department

By

Shaimaa Adel Zaki

M.Sc Pediartics- Ain Shams University Assistant Researcher- National Research Centre Under Supervision of

Dr. Mona Medhat Reda

Professor of Psychiatry
Department of Medical Studies
Institute of Postgraduate Childhood
Studies

Dr. Hala Gouda Alnady

Professor of Child Health Department of Child Health National Research Centre

Dr. Menan Abdelmaksoud Rabie

Assistant professor of psychiatry

Department of psychiatry

Ain Shams university

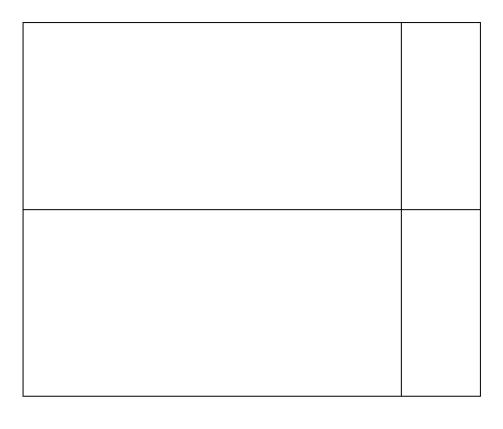
Dr. Rania Fawzy Mahmoud

Researcher of immunogenetics Immunogenetics department National research center

Ain Shams University
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List of abbreviations

Abbreviation	
AAPs	Atypical antipsychotics
ADD	Attention deficit disorder
ADHD	Attention deficit hyperactivity disorder
ADRA2A	Adrenergic alpha 2 A receptor
AKT	Serine threonine kinase
ATX	atmoxetine
BD	Bipolar disorder
BDNF	Brain derived neurotrophic factor
CBT	cognitive behavioral therapy
CD	Conduct disorder
СТ	Cat scan
DA	Dopamine
DAT1	The dopamine active transporter 1 gene
DBH	Dopa- B_ hydroxylase
DMAP	urinary dimethyl alkylphosphate
DRD4,5	Dopamine receptor D4,5
DSM-II	Diagnostic and Statistical Manual of Mental
	Disorders II
DSM-III	Diagnostic and Statistical Manual of Mental
	Disorders III
DSM-IV-TR	diagnostic and statistical manual of mental
ED14	disorders- text revision IV
ERK	extracellular signal related kinase
FDA	Food and Drug Administration
5-HT	5-hydroxytryptamine
ICD9	International Classification of Diseases 9
ID	Intellectual disability
IFC	inferior frontal cortex
LD	learning disabilities
LTP	long-term potentiation
MAPK	mitogen-activated protein kinase
MDD	Major Depressive Disorder
MeCP2	methylated CpG binding protein
MPH	Methyl phenydate
MRI	Magnetic resonance imaging
NAc	nucleus accumbens
NE	norepinephrine

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