

Serum leptin in children with attention deficit hyperactivity disorder

Mona M. Reda^a and Eman El-Hadidy^b

^aMedical Department, Institute of Childhood Postgraduate Studies, Ain Shams University and ^bClinical Pathology Department, Faculty of Medicine, Ain Shams University, Cairo, Egypt

Correspondence to: Mona M. Reda, MD, Medical Department, Childhood Institute of Postgraduate Studies, Ain Shams University, Cairo, Egypt
Tel/fax: +2 02 22591561;
e-mail: monaredapsych@gmail.com

Received 12 September 2010

Accepted 14 November 2010

Middle East Current Psychiatry 2011, 18:1–5

Introduction

Attention deficit hyperactivity disorder (ADHD) is currently defined as a cognitive/behavioral developmental disorder. There are three subtypes of ADHD, namely (i) predominantly hyperactive/impulsive, (ii) predominantly inattentive, and (iii) combined hyperactive/impulsive and inattentive. Overweight and obesity seem to be more prevalent among patients with mental disorder and children with ADHD symptoms. Obesity has become increasingly common in recent years, and is now considered as an epidemic by the World Health Organization. In humans, there is a high correlation between body mass index (BMI) and circulating leptin; the greater the amount of adipose tissue the higher the level of leptin. The aim of this study was to measure the serum leptin level of a group of ADHD children and adolescents and to find its correlation with cognitive function.

Methods

Thirty patients with ADHD of both sexes, aged between 6 and 18 years, with BMI at or above the 95th percentile for age and sex were recruited along with 15 healthy age-matched and sex-matched individuals who served as controls. Patients and controls were assessed for BMI, intelligence quotient (IQ) testing, and serum leptin level that was measured using enzyme-linked immunosorbent assay technique.

Results

The majority of cases (21 cases) were children below the age of 12 years. There was male predominance in our group, with 56% presenting with hyperactive-impulsive type. Obesity was evident in 47% of patients, with no significant difference between children and adolescents. A significant relationship was found between cases and controls with regard to IQ and serum leptin level. Serum leptin level showed a significant relationship with performance IQ, which was not evident with either verbal IQ or total IQ score.

Conclusion

We conclude that patients with ADHD show decline in cognitive abilities, that at a young age, BMI may not reflect the relationship between serum leptin level and cognitive function. Still, leptin level could be a marker for decline in cognitive abilities manifesting in social disturbance in ADHD cases.

Keywords:

attention deficit hyperactivity disorder, body mass index, leptin, obesity

Middle East Curr Psychiatry 18:1–5
© 2011 Institute of Psychiatry, Ain Shams University
2090-5408

Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is currently defined as a cognitive/behavioral developmental disorder [1], but it is also defined as a complex neuro-developmental condition by some other researchers [2].

There are three subtypes of ADHD, namely (i) predominantly hyperactive/impulsive, (ii) predominantly inattentive, and (iii) combined hyperactive/impulsive and inattentive [3]. Although the overt symptoms of ADHD tend to decline with age, they often persist into adolescence and adulthood. For instance, researchers found that up to 70% of children with ADHD will continue to experience significant impairment in adulthood. Furthermore, it is interesting to note that sex ratios for ADHD

change over time [4]. According to the Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision (DSM-IV-TR) [5], in childhood the male to female ratio is 2:1 to 9:1, depending on the subtype (less discrepancy with the inattentive subtype) and setting (more males are seen in clinical settings). However, research has shown, that by adulthood, the ratio of male to female is much more balanced [6]. Adolescents with ADHD have a high rate of comorbidity, including problems with conduct, mood, anxiety disorders [7,8], and eating disorders [9].

Obesity has become increasingly common in recent years, and is now considered as an epidemic by the World Health Organization [10]. The World Health Organization uses

Body Mass Index (BMI) to classify weight according to the formula, weight/height (kilogram/meter square), such that a BMI between 24.99 and 29.99 is 'overweight', and a BMI over 30 is considered 'obese'. A BMI over 25 not only gives rise to a greater risk of developing a variety of potentially life-threatening diseases, including various types of cancers, diabetes, and cardiovascular diseases in people, but also gives rise to a large strain on healthcare resources in many countries [10]. According to Statistics Canada [11], overweight and obesity in Canadian children and adolescents are on the rise. In 1979, 12% of children and adolescents aged between 2 and 17 years were overweight, whereas 3% were obese, but by 2004, these numbers had risen to 18% and 8%, respectively. Overall, the national rate of overweight and obesity is 26%.

In humans, there is a high correlation between BMI and circulating leptin; the greater the amount of adipose tissue the higher the level of leptin. In addition, the adipocyte size seems to be another major determinant of leptin mRNA expression [12].

Leptin, a hormone produced by adipocytes, provides signals to specific regions of the hypothalamus to control energy homeostasis [13]. However, the past decade of research has not only showed that leptin receptors are widely expressed in the central nervous system, but has also identified numerous additional functions for this hormone in the brain [14]. Moreover, a number of studies identified a role for leptin in cognitive processes, which involves activation of leptin receptors in limbic structures, such as the hippocampus [15]. Indeed, leptin influences hippocampal-dependent learning and memory, and more recently, leptin has been shown to have antidepressant properties [16].

The researchers also found that the cerebellum was activated by leptin. The cerebellum is involved in the coordination of physical motion. A weak cerebellum contributes to anxiety and learning disabilities, such as dyslexia [17]. Thus, researchers have also begun to identify a link between ADHD and overweight and obesity [18–20].

In this study, we aimed to measure serum leptin level in a group of children and adolescents with ADHD and to find its correlation with intelligence quotient (IQ).

Materials and methods

Thirty children and adolescents with ADHD fulfilling the DSM-IV-TR diagnostic criteria for the diagnosis of ADHD for research [5] were recruited for the study from outpatient clinics of Special Needs Center of Postgraduate Childhood Studies, Ain-Shams University. Fifteen healthy age-matched and sex-matched children and adolescents served as controls. Inclusion criteria for the patient group included (i) children meeting the DSM-IV-TR diagnostic criteria for the diagnosis of ADHD for research [5], (ii) children aged between 6 and 18 years, and (iii) children who have a BMI at or above the 95th percentile for age and sex, and others with a normal centile

range. Exclusion criteria for both groups included (i) children of mothers with a history of diabetes or toxemia during pregnancy. (ii) Patients with any of the following conditions: traumatic brain injury during delivery or a history of brain surgery; medical illnesses that significantly impair neuro-cognitive function (e.g. severe diabetes, severe malnutrition, or kidney and liver failure); sensory-motor handicaps; a history of alcohol or drug dependence; on steroid or other hormonal therapy; and mentally handicapped. (iii) Patients or mothers with endocrinal diseases (Cushing's syndrome or hypothyroidism).

An informed consent was obtained from the parents of the patients before participation in the study. A full medical history of all the children were noted including age, sex, consanguinity, developmental history, and socioeconomic status leveled according to the El Bouhy criteria [21].

In addition, all patients underwent a full clinical examination followed by anthropometric measures including

1. Weight assessment (kilogram): this was assessed using a weighing balance with weights ranging from 1–140 kg, while the patient stood with light clothes and was bare footed; weight was recorded to the nearest 0.5 kg.
2. Standing height assessment (centimeter): all patients were assessed barefoot, the measurement was taken while the patient was standing against a firm wall with a fixed stadiometer, the heels stretched upward to full extent, the back as straight as possible, and the head in a horizontal Frankfurt position (imaginary line passing by the middle of the ear tragus and lower edge of the eye globe). Measurements were taken to the nearest 0.5 cm.
3. BMI calculation: BMI was calculated from the previous weight and height measurements using the equation: body weight (kilogram)/standing height (meter square).
4. According to the Official Center for Disease Control growth charts [22] created by the National Center for Health Statistics BMI values of cases and controls were plotted against Standard Egyptian Percentile Curves for BMI of patients.

Laboratory measurement of the serum level of leptin by enzyme-linked immunosorbent assay

The patient and control groups were fasting for at least 8 h. Blood sampling was done under complete aseptic condition. All specimens were fixed in an ice bag. Serum leptin level was assayed using enzyme-linked immunosorbent assay kits supplied by Diagnostic Systems Laboratories, Inc. (Webster, Texas, USA). A solid phase enzyme-linked immunosorbent assay was carried out with the above-mentioned kits. All the samples were incubated in the microtiter wells that had been coated with antihuman leptin antibody. After incubation and washing, the wells were treated with another antihuman leptin detection antibody labeled with the enzyme, horseradish peroxidase. After a second incubation and washing step, the wells were incubated with the substrate, tetramethylbenzidine. An acidic stop solution

Table 1 Comparison between cases and control groups with regard to age, sex, obesity, and socioeconomic leveling distribution

Sample variable	Cases	Control	<i>P</i> value
Age (years)			
Children (6–12)	21	11	0.502
Adolescents (13–18)	9	4	
Sex			
Male	26	8	0.014*
Female	4	7	
Obesity			
Nonobese	16	10	0.393
Obese	14	5	
Socioeconomic level			
High	2	5	0.053
Middle	16	7	
Low	12	3	

**P* < 0.05.

was then added and the degree of enzymatic turnover of the substrate was determined by dual wavelength absorbance measurement at 450 and 620 nm. The absorbance measured is directly proportional to the concentration of leptin. Standards were used to plot a standard curve of absorbance versus leptin concentration in all patients, in whom the leptin concentration in the study samples can be calculated.

An intelligence quotient testing

Cognitive ability was assessed using Wechsler Intelligence Scale [23] for Children, which is the most widely used test. It contains 13 tests that combine to form six verbal IQs and five performance IQs (PIQs). These two scales make up the total IQ (TIQ) [24]. All statistical analyses were carried out using SPSS (statistical package of social science) version 10 (Chicago, Illinois, USA).

Results

Thirty ADHD cases were recruited in this study. Table 1 shows that there was no significant difference between cases and controls with regard to age, obesity, socio-demographic status, but there was a significant difference with regard to sex in the form of male predominance among cases.

Table 2 Comparison of sex, obesity, and attention deficit hyperactivity disorder subtypes in sample children and adolescent sample

Sample variable	Children (6–12 years)	Adolescents (13–18 years)	<i>P</i> value
Sex			
Male	18	8	0.815
Female	3	1	
Obesity			
Obese	13	1	0.351
Nonobese	13	3	
Types of ADHD			
Predominantly inattentive	2	3	0.166
Predominantly hyperactive–impulsive	14	3	
Combined	5	3	

ADHD, attention deficit hyperactivity disorder.

Table 3 Comparison between intelligence quotient, serum leptin, and body mass index in cases and controls

Sample variable	Cases (mean ± SD)	Control (mean ± SD)	<i>P</i> value
IQ	79.57 ± 13.26	105.13 ± 8.91	0.000**
Leptin	11.32 ± 14.35	20.87 ± 13.30	0.037*
BMI	21.50 ± 5.74	20.48 ± 6.01	0.583

BMI, body mass index; IQ, intelligence quotient; SD, standard deviation.

P* < 0.05.*P* < 0.001.

In our sample, five cases (16.6%) were diagnosed as predominantly inattentive type, whereas 17 cases (56.6%) were diagnosed as predominantly hyperactive–impulsive type (ADHD), and remaining eight cases (26.7%) were diagnosed as combined type.

With regard to obesity, 14 (47%) cases were obese, whereas the remaining 16 (53%) cases were nonobese.

Fifty percent of cases were on medications (13% treated with psychostimulant and antiepileptic, 27% treated with antidepressant and psychostimulant, and 10% treated with antiepileptic and complementary alternative medication) and the remaining 50% were medication free.

Our study group was divided according to the age of the children (6–12 years) and adolescents (12–18 years). Children predominance was found as 21 children compared with nine adolescents were in the study. Comparing children with the adolescent group with regard to sex, obesity, and subtype of ADHD, no significant differences were observed (Table 2).

Comparison of IQ, leptin level, and BMI in cases and controls showed a significant difference in IQ and leptin levels, and no significance in BMI (Table 3).

Using Person's correlation coefficient (*p*) between different parameters we could not detect a significant correlation among serum leptin, BMI, TIQ, and age or sex in our group of patients with ADHD. Analysis of the relationship between BMI and IQ showed that there is no significant correlation. Further analysis showed that there is a significant correlation between serum leptin and PIQ (*P* = 0.049). In contrast, there is no significant correlation between serum leptin and TIQ or verbal IQ.

Discussion

ADHD is a chronic early-onset syndrome of developmentally inappropriate levels of inattention, hyperactivity, and impulsivity. Male to female ratio among the studied cases with ADHD all over the world by the American Psychiatric Association [25] was 4:1. However, some investigators [26] found that the male to female ratio was 3:1. Moreover, other researchers [27] explained that the sex difference is due to the fact that girls were usually more frequently presented in an attentive form of the disorder than the hyperactivity form; therefore, they were underdetected and remained as silent cases. This is consistent with our study in which male cases showed a predominant presentation of 26 cases compared with four cases in female patients.

This could explain the frequent presentation of the hyperactive type in our group of 56.7% patients, while the inattentive type was present in 16.6% and combined type in 26.7%.

Overweight and obesity seem to be more prevalent among people with mental disorder and children with ADHD symptoms [28]. Recently, researchers have also begun to identify a link between ADHD and overweight and obesity [18–20]. In a large population study of 1429 high school students in China, it was found that there was a significant relationship between ADHD and obesity, but not between ADHD and being overweight [29]. In our study, the obese patients were 46.7%, whereas the obese controls were 33.3%, and this inconsistency with the earlier study could be explained by correlating to the socioeconomic status of our sample, as the majority of cases were from low and middle socioeconomic classes of the society, in which income plays a major role in determining the nature of food content.

Earlier studies gave no conclusive results with regard to the relationship between medication of ADHD and overweight and obesity. Other researchers [19] found that there were no significant differences between those patients with ADHD who were on medication, and those who were not with regard to their weight. In addition, other researchers [30] found that children with ADHD who were medicated were significantly less likely to be overweight than those who were not. Other investigators [31] reported that children and adolescents with ADHD are more likely to be overweight when not medicated; Moreover children with ADHD who are medicated are more likely to be underweight. In our study, we found that there is no significant difference between patients and controls with regard to obesity.

The literature has clearly documented that increased severity of ADHD symptoms is related to poorer academic performance even in normally intelligent children [32]. This was consistent with our results, in which highly significant difference was observed between cases and controls in IQ ($*P = 0.000$). This is still in agreement with the researchers who reported that patients with ADHD persisting symptoms typically show subtle cognitive and behavioral impairments that, nonetheless, are often associated with significant educational, occupational, interpersonal, emotional, and even legal difficulties.

Leptin, is a hormone produced by adipocytes, which provides signals to specific regions of the hypothalamus to control energy homeostasis, regulates the development and function of feeding circuits, and influences hippocampal-dependent learning and memory as well. Moreover, leptin-induced alterations in neuronal excitability have been implicated in the regulation of food intake, reward behavior, and anticonvulsant effects [33]. Measuring leptin in our sample showed a positive correlation between BMI and serum leptin hormone level, which was consistent with the study carried out by Flier [34] who reported that obesity is associated with hyperleptinemia.

Earlier study by Jequier [35] reported that sex is an important factor determining plasma leptin, with women having markedly higher leptin concentrations than men

for any given degree of fat mass. This contradicted with our results, as there was no significant correlation between sex and serum leptin that could be attributed to the large male predominance in our sample.

Significant correlation between serum leptin level and PIQ was detected in our sample. Earlier studies hypothesized that leptin insufficiency in the brain either alone, or in concert with hyperglycemia and/or hyperinsulinemia is a significant risk factor for impaired cognitive function in the aging population [17]. In addition, a chronic low-grade decrease in central leptin levels in humans, a consequence of impaired blood–brain barrier transport, may inflict hippocampal damage, volume loss, and atrophy, decrease general cognition performance, and memory impairment [36].

We conclude that patients with ADHD show decline in cognitive abilities and that at a young age, BMI may not reflect the relationship between leptin and IQ. Leptin is important for the development of neurocognition in humans, but as our study was conducted on small number of cases and as there was limitation of funding researches on larger scale, we were not able to assess the exact correlation between various cognitive functions and serum leptin in ADHD cases of children and adolescents.

The authors have no conflict of interest to declare.

References

- 1 Sagvolden T, Johansen EB, Aase H, Russell VA. A dynamic developmental theory of Attention-Deficit/Hyperactivity Disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behav Brain Sci* 2005; 28:397–419; discussion 419–468.
- 2 Stefanatos GA, Baron IS. Attention-Deficit/Hyperactivity Disorder: a neuropsychological perspective towards DSM-V. *Neuropsychol Rev* 2007; 17:5–38.
- 3 Biederman J, Faraone SV, Spencer T, Wilens T, Mick E, Lapey KA. Gender differences in a sample of adults with Attention Deficit Hyperactivity Disorder. *Psychiatry Res* 1994; 53:13–29.
- 4 Biederman J, Mick E, Faraone SV. Age-dependent decline of symptoms of Attention Deficit Hyperactivity disorder: impact of remission definition and symptom type. *Am J Psychiatry* 2000; 157:816–818.
- 5 American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. Washington DC, USA: American Psychiatric Publishing, Inc; 2000.
- 6 Biederman J, Faraone SV, Spencer T, Wilens T, Norman D, Lapey KA, et al. Patterns of psychiatric comorbidity, cognition and psychosocial functioning in adults with Attention Deficit Hyperactivity Disorder. *Am J Psychiatry* 1993; 150:1792–1798.
- 7 Barkley RA. *Attention Deficit Hyperactivity Disorder: a handbook for diagnosis and treatment*. New York: Guilford Press; 1990.
- 8 Biederman J, Faraone S, Milberger S, Guite J, Mick E, Chen L, et al. A prospective 4-year follow-up study of attention-deficit hyperactivity and related disorders. *Arch Gen Psychiatry* 1996; 53:437–446.
- 9 Mikami AY, Hinshaw SP. Resilient adolescent adjustment among girls: buffers of childhood peer rejection and Attention-Deficit/Hyperactivity Disorder. *J Abnormal Child Psychol* 2006; 34:825–839.
- 10 World Health Organization (WHO). Global strategy on diet, physical activity and health. 2004; Available at: http://apps.who.int/gb/ebwha/pdf_files/WHA57/A57_9-en.pdf. (Accessed August 2008).
- 11 Shields M. *Measured obesity: overweight Canadian children and adolescents*. Ottawa: Statistics Canada; 2005.
- 12 Banks WA. The many lives of leptin. *Peptides* 2004; 25:331–338.
- 13 Zlokovic BV, Jovanovic S, Miao W, Samara S, Verma S, Farrell CL. Differential regulation of leptin transport by the choroid plexus and blood-brain barrier and high affinity transport systems for entry into hypothalamus and across the blood-cerebrospinal fluid barrier. *Endocrinology* 2000; 141:1434–1441.
- 14 Valeric A, Dossena M, Bertolotti P, Boroni F, Sarnico I, Faraco G, et al. Leptin is induced in the ischemic cerebral cortex and exerts neuroprotection through NF- κ B/c-Rel-Dependent transcription. *Stroke* 2009; 40:610–617.

- 15 Farr SA, Banks WA, Morley JE. Effects of leptin on memory processing. *Peptides* 2006; 27:1420–1425.
- 16 Lu XY, Kim CS, Frazer A, Zhang W. Leptin: a potential novel antidepressant. *Proc Natl Acad Sci USA* 2006; 103:1593–1598.
- 17 Paulus K, Schulz C, Lehnert H. Central nervous effects of leptin and insulin on hippocampal leptin and insulin receptor expression following a learning task in Wistar rats. *Neuropsychobiology* 2005; 51:100–106.
- 18 Cortese S, Isnard P, Frelut ML, Michel G, Quantin L, Guedeney A, *et al.* Association between symptoms of Attention-Deficit/Hyperactivity Disorder and bulimic behaviors in a clinical sample of severely obese adolescents. *Int J Obes* 2007; 31:340–346.
- 19 Holtkamp K, Konrad K, Muller B, Heussen N, Herpertz S, Herpertz Dahlmann B, *et al.* Overweight and obesity in children with Attention-Deficit/Hyperactivity Disorder. *Int J Obes Relat Metab Disord* 2004; 28:685–689.
- 20 Hubel R, Jass J, Marcus A, Laessle RG. Overweight and basal metabolic rate in boys with Attention-Deficit/Hyperactivity Disorder. *Eat Weight Disord* 2006; 11:139–146.
- 21 El Bouhy FS. Tool for socioeconomic levels. In: Aly SI, editor. *Educational year book*, vol 1: Egyptian Association for Graduate College of Education; 1988. pp. 434–443.
- 22 Kuczmarski RJ, Ogden CL, Grummer Strawn LM, Flegal KM, Guo SS, Wei R, *et al.* CDC growth charts: United States. *Adv Data* 2000; 248:1–27.
- 23 Wechsler D. *The Wechsler intelligence scale for children, 3rd ed.* San Antonio, TX: The Psychological Corporation; 1991.
- 24 Malika LK. *Wechsler intelligence scale for children (Arabic translation)*. Cairo, Egypt: Dar El Nahda El Arabia; 1996.
- 25 American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders DSM-IV*. 4th ed. Washington DC, USA: American Psychiatric Association; 1994.
- 26 Mannuzza S, Klein RG, Moulton JL III. Persistence of Attention-Deficit/Hyperactivity Disorder into adulthood: what have we learned from the prospective follow-up studies? *J Atten Disord* 2003; 7:93–100.
- 27 Faraone SV, Biederman J, Mick E, Williamson S, Wilens T, Spencer T, *et al.* Family study of girls with Attention Deficit Hyperactivity disorder. *Am J Psychiatry* 2000; 157:1077–1083.
- 28 Baumeister H, Härter M. Mental disorders in patients with obesity in comparison with healthy probands. *Int J Obes* 2007; 31:1155–1164.
- 29 Lam LT, Yang L. Overweight/obesity and Attention Deficit and Hyperactivity disorder tendency among adolescents in China. *Int J Obes* 2007; 31:584–590.
- 30 Brown RT, Amler RW, Freeman WS, Perrin JM, Stein MT, Feldman HM, *et al.* Treatment of Attention-Deficit/Hyperactivity disorder: overview of the evidence. *Pediatrics* 2005; 115:e749–e757.
- 31 Taylor DM, McAskill R. Atypical antipsychotics and weight gain: a systematic review. *Acta Psychiatr Scand* 2000; 101:416–432.
- 32 Hechtman L, Abikoff H, Klein RG, Weiss G, Respit C, Kouri J, *et al.* Academic achievement and emotional status of children with ADHD treated with long-term methylphenidate and multimodal psychosocial treatment. *J Am Acad Child Adolesc Psychiatry* 2004; 43:812–819.
- 33 Bouret SG, Simerly RB. Minireview: leptin and development of hypothalamic feeding circuits. *Endocrinology* 2004; 145:2621–2626.
- 34 Flier JS. Obesity wars: molecular progress confronts an expanding epidemic. *Cell* 2004; 116:337–350.
- 35 Jequier E. Leptin signaling, adiposity and energy balance. *Ann NY Acad Sci* 2002; 967:379–388.
- 36 Banks WA, Farr SA, Morley JE. The effects of high fat diets on the blood-brain barrier transport of leptin: failure or adaptation? *Physiol Behav* 2006; 88:244–248.

الملخص العربي

دراسة أكاديمية لقياس هرمون الليبتين في الدم لدى الأطفال المصابين بفرط الحركة ونقص الانتباه. منى رضا *، إيمان الحديدى ** *معهد دراسات الطفولة- جامعة عين شمس **قسم الباثولوجي الاكلينيكي- كلية طب- جامعة عين شمس

يعد مرض اضطراب فرط الحركة ونقص الانتباه من أمراض العصر واسعة الانتشار ، الأمر الذي جعله محط اهتمام صحي عالمي واسع النطاق ، خاصة في ظل تزايد بعض المؤشرات الإحصائية الدولية التي تشير إلى ارتفاع عدد الإصابة بهذا المرض. ومن واقع الإحصاءات ، يتبين أنه يصاب من ثلاثة إلى خمسة بالمئة من طلاب المدارس بهذه الحالة ، وأن الذكور أكثر إصابة من الإناث . من ناحية أخرى ، وجدت الدراسات أن هناك علاقة بين ارتفاع نسبة الإصابة بالسمنة والإصابة بمرض اضطراب فرط الحركة ونقص الانتباه .

تفترض الدراسة وجود دور لنقص " هرمون الليبتين في الإصابة بمرض اضطراب فرط الحركة ونقص الانتباه ، وذلك من حيث تأثيره على المخ والوظيفة المعرفية.

تهدف الدراسة تقدير العلاقة بين هرمون الليبتين والإصابة بمرض اضطراب فرط الحركة ونقص الانتباه تأثير هذه العلاقة والوظيفة المعرفية للأطفال المصابين بمرض اضطراب فرط الحركة ونقص الانتباه .

تمت الدراسة على 30 مصاب بمرض اضطراب فرط الحركة ونقص الانتباه ، وقد تم مقارنة هؤلاء المرضى بـ 15 حالة ضابطة من نفس المرحلة العمرية ونفس الجنس وجد أن 26 ذكر و 4 إناث في العينة مصابون بمرض اضطراب فرط الحركة ونقص الانتباه. و ان نسبة الاطفال دون 12 سنة هي الاكثر في الدراسة و ان اغلبية الحالات يعانون من فرط الحركة بشكل اساسي.

في هذا العمل وجد ان هناك فارق كبير بين الحالات المرضية والحالات الضابطة من حيث مستوى الذكاء وأيضاً وجد أن هناك فرق كبير بين الطرفين فيما يتعلق بمستوى هرمون الليبتين في الدم. بينما لا يوجد فارق كبير بين الحالات المرضية والحالات الضابطة فيما يتعلق بمعدل كتلة الجسم. ولكن وجد أن هناك علاقة ايجابية كبيرة بين معدل كتلة الجسم وهرمون الليبتين في الدم في كل من الحالات المرضية والحالات الضابطة ، بينما لم يلاحظ وجود علاقة بين مستوى هرمون الليبتين في الدم ومعدل الذكاء ، كما لوحظ عدم وجود علاقة بين معدل كتلة الجسم ومعدل الذكاء