

# Association of Adiponectin and IgE levels with occurrence of asthma in Babylon Province

**Ruwaida Wahab Salman AL-Jebory**

*Assist Professor. Suhayr Aesa Al- Qaysi - Assist professor. Oday Jasim AL-Salihi, Babylon University, College Of Medicine , Biochemistry Dept., Hilla, Iraq*

## Abstract

**Background :** Asthma is a health condition that affects breathing in people. Breathing comfortably is not something most of us need to think about. But if you have asthma, there are times when you have difficulty breathing. The main objectives in the current study are: Determine the role of adiponectin, a hormone that acts as an anti-inflammatory, In addition determine the concentration of immunoglobulin E (IgE) In asthma patients.

**Method:** This study included two groups: Group 1: 80 Patients with asthma were divided into two parts (40 samples obese and 40 samples non-obese). Group 2: 80 Group of healthy subjects with no history or clinical evidence of asthma. samples were divided into two parts (40 samples were obese and 40 were not obese). Asthma was diagnosed through a pulmonary function test (PFT) and then invited to a private clinic and measured nitric oxide gas (FeNO) to confirm the diagnosis. Enzyme-linked immunosorbent assay (ELISA) technique was used to determine the concentration of adiponectin and IgE in serum.

**Results:** The present study demonstrated the increase of adiponectin level with asthmatic patients compared with the control group and showed significant difference between non-obese patients group and non-obese healthy group.

**Conclusion:** Adiponectin related with obesity play crucial role in asthma development and inverse relationship between IgE and adiponectin.

**Key words:** *Adiponectin, IgE, ELISA, BIM, FeNO*

## Introduction

Asthma is a disease of reversible airflow obstruction, bronchial hyper responsiveness, and underlying airway inflammation<sup>1,2</sup>. In other words, it is a disease appear by effect allergic factors and non-allergic factors and result in bronchial obstruction and inflammation so it is multifactorial disease<sup>3</sup>. This disease ranked 16th among the leading causes of years lived with disability and 28<sup>th</sup> among the leading causes of burden of disease<sup>4</sup>. There are many reasons for asthma, but almost certainly reflect variable contributions of genetic and environmental factors in different regions<sup>5</sup>. So Immunoglobulin E (IgE) is the antibody responsible for allergic reactions and is important to the pathogenesis of allergic diseases and the evolution and persistence of inflammation. IgE attaches to cell surfaces and a specific high-affinity receptor<sup>6</sup>. Furthermore, there are some substance like adiponectin,

it also seems influenced to the asthma. Adiponectin is a 244 amino-acid-long polypeptide. The adiponectin has four distinct regions<sup>7,8</sup>. There are many studies on the effect of levels adiponectin on asthma. However, human data remain inconclusive and not known if may be the adiponectin affects asthma control by treatment. Shore in their study of weak mice suggests that exogenous dose from the adiponectin relieve allergen-induced airway hyperreactivity and eosinophilic influx in both sexes<sup>9</sup>. Aims of this study that estimate role adiponectin in pathogenesis of asthma, estimate role IgE in pathogenesis of asthma and to find possible association between IgE with adiponectin.

## Method

at the laboratory of Biochemistry Department. This study was complete some process in a College

of Medicine, University of Babylon at the laboratory of Biochemistry Department. The subjects in this prospective case-control research, include a total of 160 subjects 80 asthmatic subjects (42 females with 38 males) and 80 clinically healthy control subjects (37 females with 43 males). All samples were collected during the period from the August - 2018 till February -2019. Patients were collected from Marjan Hospital and invited to a specialized clinic for examination by fractional exhaled nitric oxide (FeNO) device. Asthmatic subjects were categorized to (40 obese asthmatic subjects with BMI mean SE 34.5 0.52 and 40 non-obese asthmatic subjects with BMI mean 25.40.45), Also control group subdivided into two groups (40 obese control subjects with BMI mean 33.7 0.56 and 40 non-obese control subjects 25.050.45). with BMI mean.

**Inclusion Criteria:** The inclusion criteria for the selection of the study subjects include, Asthmatic patients group (depending on pulmologist decision according to the GINA guideline). All person accept to participate in the current study. Age of all subjects was ≥18 years old.

**Exclusion Criteria:** Any person suffered from the following were excluded from this study: Patient with diabetes. Heart diseases, hypertension, malignancy, and any chronic disease. Patient with any other chronic disease (metabolic and endocrine) Smokers. Pregnancy women. Patient with chronic obstructive pulmonary disease (COPD) disease and emphysema. Another lung disease.

**Body Mass Index (BMI):**

Body mass index (BMI) is the ratio of a person’s weight to height in square meters; it commonly used to classify weight as healthy person or unhealthy person. BMI calculated by below equation <sup>10</sup> as follow:

$$BMI(Kg/m^2) = \frac{Weight(Kg)}{(Height)^2 m^2}$$

**Table 1: Demographic characteristics of the study groups.**

Variable	Study groups	No.	Means ±SE	P- value
Age (years)	Asthmatic patients	80	34.06 ±1.28	0.74
	Control	80	33.45 ±1.27	
BMI	Asthmatic patients	80	30.77 ±0.61	0.02
	Control	80	28.66 ±0.65	
Waist/ Hip ratio	Asthmatic patients	80	0.89 ±0.01	0.94
	Control	80	0.89 ±0.008	

P value 0.05 was significant.

**Waist to Hip Ratio:**

A tap measure was used to check the distance around the smallest part of the waist (this is circumference of the waist. Then measure the distance round the largest section of hips this is circumference of the hip). According to the World Health Organization (WHO) was calculated the waist to hip ratio by dividing the waist circumference on the hip circumference <sup>11</sup>. Waist to hip ratio = waist/cmhip/cm

**Determination of human Serum Adiponectin and IgE Concentration by enzyme-linked immunosorbent assay (ELISA) kit**

The adiponectin and IgE levels were measured in plasma using commercially available enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturers order (Elabscience company, Calbiotech company) respectively. The adiponectin levels ranged from 25 to 250 ng/ml, while IgE levels ranged for male IgE 250 ng/ml and for female IgE >175 ng/ml. Low and high quality controls were estimate in parallel in each test <sup>12,13,14</sup>.

**Results**

**Demographic Characteristics of the Study Subject.**

The results of the current comparative study of the patient and control groups were statistically calculated using the T-test to determine the difference in mean between control groups and asthma groups as well as the correlation between different parameter for all patients. The total number 160 adults in this study were divided on 4 groups the cases 80 (40 non obese and 40 obese) and 80 control group (40 non-obese and 40 obese). The results of demographic data in Table 1.

BMI (body mass index)

**Age:**

The results were expressed as means and standard error (SE). There no significant difference ( $p > 0.05$ ) in age (as means) between control and asthmatic patients, means  $\pm$ SE for the case is (34.06  $\pm$ 1.28) and for control (33.45  $\pm$ 1.27) as appear in Table 1. When we matching the age this is play a role to eliminate differences in results that may be due to the big variation in age <sup>15</sup>.

Body mass index (BMI):

There significant difference ( $p < 0.05$ ) in Body mass index (BMI) (as means) between control groups and asthmatic patients, mean  $\pm$ SE for the case are (30.77 $\pm$ 0.61) and for control (28.66 $\pm$ 0.65) as shown in table 1-1. Even though we were already taken in both groups obese and non-obese. So these results support some of the studies indicated a significant difference in the overlap between asthma and obesity, with suggesting that the association of obesity with both incidence asthma and prevalence it in women more than in men <sup>16</sup>.So, the difference in BMI between patients and controls in this study accepted with the other studies <sup>17,18</sup>.

Waist to Hip Ratio:

There no significant difference ( $p > 0.05$ ) in waist/hip ratio (as means) between control and asthmatic

patients, means  $\pm$ SE for case is (0.89  $\pm$ 0.01) and for control (0.89  $\pm$ 0.008), Table 1-1. Although there are many studies showed that BMI was associated with asthma as risk factor <sup>19</sup> but, waist/hip ratio shown independent factor than BMI <sup>11</sup>

Gender:

Other factor effect on the asthma it is gender.

Figure 1 Gender distribution of control compared with the patient group

The Figure above indicates a slight difference between male and female, and some studies can be adopted like Zein and Erzurum that shown asthma is severing and more prevalent in women especially with multiple gestation or with early menarche, offer a role for sex hormones on asthma disease <sup>20</sup>.

Biochemical Result:

**Mean differences of adiponectin level according to studied groups:**

Mean differences of biochemical results including adiponectin was revealed in Table 2.The significant difference ( $p < 0.05$ ) in adiponectin (as means) between control groups and asthmatic patients, means  $\pm$ SE for the case is (140.50 $\pm$ 8.46) and for control (114.27  $\pm$ 7.48). The results were shown in the Table 1-2 and the concentration of adiponectin for asthmatic patients was higher than the control group.

**Table 2 Mean difference SE for Adiponectin level.**

Variable	Study Group	No.	MeanSE	P-value
Adiponectin	Asthmatic patients	80	140.50 $\pm$ 8.46	0.02
	Control	80	114.27 $\pm$ 7.48	

P-value 0.05 was significant.

Adiponectin is a protein hormone and is 30 kDa in size; In adults it is excrete and synthesized solely by adipose cells, the levels of adiponectin in plasma are inversely proportional to percentage of fat. A little levels of adiponectin shown in Obese individuals <sup>21</sup>.With higher adiponectin levels in animal models leads to minus airway responsiveness and inflammation as a challenge for allergens <sup>22</sup>. Furthermore, this hormone prevents the

proliferation of cultured vascular smooth muscle cells <sup>23</sup>. Sood *et al.* showed that higher serum adiponectin concentrations were independently associated with adverse clinical outcomes of asthma (such as asthma-related symptoms, medications and disease activity) among men but not women <sup>24</sup>.

Yet, poor muscle function and physical disability are linked with higher levels of adiponectin in circulating blood, which suggests a possible related between risk of falls and adiponectin <sup>25</sup>.

Mean differences of IgE level according to asthmatic patient group:

The IgE level was estimated in asthmatic patient. In aid to Categories patient as allergic and non-allergic dependent on reference level that obtained from kit{(male>250 and female>175)} this allergic group} IgE consider as a perfect factor to determine the inflammatory status of patients <sup>13,14</sup>. Present results indicated.

**Table 3 Mean difference of IgE level of asthmatic patient.**

Variable	Study group	NO.	MeanSE	P-value
IgE	Non-allergic	43	80.089.22	0.000
	Allergic	37	601.00 43.46	

Some studies have reported the increased levels of IgE in asthmatic <sup>26,27</sup>. Thus, it is in conformity with the well-known fact that IgE plays a major role in the pathophysiology of asthma and another allergic disorder <sup>28</sup>.

**Correlation between serum adiponectin and IgE in asthmatic patients:**

The result in table 4 appeared no significant difference between the adiponectin and IgE statistically but there is an inverse relationship between them.

**Table 4 the correlation between adiponectin and IgE in asthmatic patients.**

variable		Adiponectin	IgE
adiponectin	Pearson correlation	1	-0.183
	Sig.(2-tailed)	0.107	0.107
	No.	80	80
IgE	Pearson correlation	-0.183	1
	Sig.(2-tailed)	0.107	0.107
	NO.	80	80

Serum adiponectin in (obese and non-obese) for patient and control Group.

Table 5 revealed that mean SE of adiponectin in asthmatic obese are (119.2510.70) and control obese are (106.578,21) respectively so no significant difference. While significant differences between patient non-obese and control non-obese P-value <0.05 another compares between patient obese and non-obese and appeared no significant difference also.

**Table 5: The relationship between obese and non-obese for adiponectin**

Type	No.	meanSE	P-value
Patient obese	40	119.2510.70	0.21
Control obese	40	106.578.21	
Patient non- obese	40	171.16±11.21	0.01
Control non-obese	40	108.73±7.96	
Patient obese	40	119.32±10.70	0.36
Patient non- obese	40	154.09±11.85	

## Discussion

The reduction in adiponectin in obese persons possible participate to rise smooth muscle mass in asthmatic persons<sup>29</sup>. But in this study the level of adiponectin in asthmatic patient was high so the most common risk factor for falls is muscle weakness<sup>30</sup>. The change of the pattern in asthma therapy perhaps include weight regulation and may be change pharmacotherapeutic systems shall require special interest to that underserved populations face in the treatment of asthma. These obstructions include limited access to care and medications, cultural changes in understanding and accepting treatment options, and decreased health literacy<sup>31,32</sup>. To manage these complexities, innovative way to therapy are needed. There is actual evidence that obesity and asthma are linked together<sup>33</sup>.

**Ethical Clearance:** Approval by scientific committee of Babylon Medical College (University of Babylon, Iraq) and the Biochemistry Department in the medical college.

**Source of Funding:** The funding body written out in full by self

**Conflict of Interest:** If any then mention it otherwise write it as nil

## References

- 1- Postma DS, Kerstjens HA. Characteristics of airway hyperresponsiveness in asthma and chronic obstructive pulmonary disease. *American Journal of Respiratory and Critical Care Medicine*. 1998 Nov 1;158(supplement\_2):S187-92.
- 2- Cukic V, Lovre V, Dragisic D, Ustamujic A. Asthma and chronic obstructive pulmonary disease (COPD)—differences and similarities. *Materia socio-medica*. 2012;24(2):100.
- 3- Bijanzadeh M, Mahesh P, Ramachandra N. An understanding of the genetic basis of asthma. *Indian Journal of Medical Research*. 2011 Aug 1;134(2):149.
- 4- Ellwood P, Asher MI, Billo NE, Bissell K, Chiang CY, Ellwood EM, El-Sony A, García-Marcos L, Mallol J, Marks GB, Pearce NE. The Global Asthma Network rationale and methods for Phase I global surveillance: prevalence, severity, management and risk factors. *European respiratory journal*. 2017 Jan 1;49(1):1601605.
- 5- Beasley R, of Asthma TI. Worldwide variation in prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and atopic eczema: ISAAC. *The Lancet*. 1998 Apr 25;351(9111):1225-32.
- 6- Kelly BT, Grayson MH. Immunoglobulin E, what is it good for?. *Annals of Allergy, Asthma & Immunology*. 2016 Mar 1;116(3):183-7.
- 7- Shapiro L, Scherer PE. The crystal structure of a complement-1q family protein suggests an evolutionary link to tumor necrosis factor. *Current Biology*. 1998 Mar 12;8(6):335-40.
- 8- Litwack G. *Human biochemistry*. Academic Press; 2017 Nov 9.
- 9- Shore SA. Obesity and asthma: cause for concern. *Current opinion in pharmacology*. 2006 Jun 1;6(3):230-6.
- 10- Nordander C, Willner J, Hansson GÅ, Larsson B, Unge J, Granquist L, Skerfving S. Influence of the subcutaneous fat layer, as measured by ultrasound, skinfold calipers and BMI, on the EMG amplitude. *European journal of applied physiology*. 2003 Aug 1;89(6):514-9.
- 11- World Health Organization. Waist circumference and waist-hip ratio: report of a WHO expert consultation, Geneva, 8-11 December 2008.
- 12- Ewadh MJ, Al-Mamoori AN, Al-Qaysi SE. Association of Adiponectin, Oxidative Stress, and Obesity in Asthmatics children. *Medical Journal of Babylon*. 2017;14(2):267-79.
- 13- Vercelli D. Molecular regulation of the IgE immune response. *Clinical & Experimental Allergy*. 1995 Nov;25:43-5.
- 14- Marsh DG, Neely JD, Breazeale DR, Ghosh B, Freidhoff LR, Ehrlich-Kautzky E, Schou C, Krishnaswamy G, Beaty TH. Linkage analysis of IL4 and other chromosome 5q31. 1 markers and total serum immunoglobulin E concentrations. *Science*. 1994 May 20;264(5162):1152-6.
- 15- Battaglia S, Benfante A, Spatafora M, Scichilone N. Asthma in the elderly: a different disease?. *Breathe*. 2016 Mar 1;12(1):18-28.
- 16- Koper I, Hufnagl K, Ehmann R. Gender aspects and influence of hormones on bronchial asthma—Secondary publication and update. *World Allergy Organization Journal*. 2017 Dec;10(1):46.
- 17- Beuther DA, Sutherland ER. Overweight, obesity, and incident asthma: a meta-analysis of prospective

- epidemiologic studies. American journal of respiratory and critical care medicine. 2007 Apr 1;175(7):661-6.
- 18- Schatz M, Zeiger RS, Yang SJ, Chen W, Sajjan S, Allen-Ramey F, Camargo Jr CA. Prospective study on the relationship of obesity to asthma impairment and risk. The Journal of Allergy and Clinical Immunology: In Practice. 2015 Jul 1;3(4):560-5.
- 19- Aune D, Sen A, Schlesinger S, Norat T, Janszky I, Romundstad P, Tonstad S, Riboli E, Vatten LJ. Body mass index, abdominal fatness, fat mass and the risk of atrial fibrillation: a systematic review and dose-response meta-analysis of prospective studies.
- 20- Zein JG, Erzurum SC. Asthma is different in women. Current allergy and asthma reports. 2015 Jun 1;15(6):28.
- 21- Parida S, Siddharth S, Sharma D. Adiponectin, obesity, and cancer: Clash of the bigwigs in health and disease. International journal of molecular sciences. 2019 Jan;20(10):2519.
- 22- Shore SA, Terry RD, Flynt L, Xu A, Hug C. Adiponectin attenuates allergen-induced airway inflammation and hyperresponsiveness in mice. Journal of Allergy and Clinical Immunology. 2006 Aug 1;118(2):389-95.
- 23- Ding M, Xie Y, Wagner RJ, Jin Y, Carrao AC, Liu LS, Guzman AK, Powell RJ, Hwa J, Rzucidlo EM, Martin KA. Adiponectin induces vascular smooth muscle cell differentiation via repression of mammalian target of rapamycin complex 1 and FoxO4. Arteriosclerosis, thrombosis, and vascular biology. 2011 Jun;31(6):1403-10.
- 24- Sood A, Dominic E, Qualls C, Steffes MW, Thyagarajan B, Smith LJ, Lewis CE, Jacobs Jr DR. Serum adiponectin is associated with adverse outcomes of asthma in men but not in women. Frontiers in pharmacology. 2011 Oct 5;2:55.
- 25- Huang C, Momma H, Niu K, Chujo M, Otomo A, Cui Y, Nagatomi R. High serum adiponectin levels predict incident falls among middle-aged and older adults: a prospective cohort study. Age and ageing. 2016 Mar 23;45(3):366-71.
- 26- Sandeep T, Roopakala MS, Silvia CR, Chandrashekara S, Rao M. Evaluation of serum immunoglobulin E levels in bronchial asthma. Lung India: official organ of Indian Chest Society. 2010 Jul;27(3):138.
- 27- Sharma S, Kathuria PC, Gupta CK, Nordling K, Ghosh B, Singh AB. Total serum immunoglobulin E levels in a case-control study in asthmatic/allergic patients, their family members, and healthy subjects from India. Clinical & Experimental Allergy. 2006 Aug;36(8):1019-27.
- 28- Lama M, Chatterjee M, Chaudhuri TK. Total serum immunoglobulin e in children with asthma. Indian Journal of Clinical Biochemistry. 2013 Apr 1;28(2):197-200.
- 29- Sood A, Cui X, Qualls C, Beckett WS, Gross MD, Steffes MW, Smith LJ, Jacobs DR. Association between asthma and serum adiponectin concentration in women. Thorax. 2008 Oct 1;63(10):877-82.
- 30- Alan IS, Alan B. Side Effects of Glucocorticoids. Pharmacokinetics and Adverse Effects of Drugs-Mechanisms and Risks Factors. 2018 May 23.
- 31- Pachter LM, Weller SC, Baer RD, Garcia de Alba Garcia JE, Trotter RT, Glazer M, Klein R. Variation in asthma beliefs and practices among mainland Puerto Ricans, Mexican-Americans, Mexicans, and Guatemalans. Journal of asthma. 2002 Jan 1;39(2):119-34.
- 32- McLean DE, Bowen S, Drezner K, Rowe A, Sherman P, Schroeder S, Redlener K, Redlener I. Asthma among homeless children: undercounting and undertreating the underserved. Archives of pediatrics & adolescent medicine. 2004 Mar 1;158(3):244-9.
- 33- Mohanan S, Tapp H, McWilliams A, Dulin M. Obesity and asthma: pathophysiology and implications for diagnosis and management in primary care. Experimental Biology and Medicine. 2014 Nov;239(11):1531-40.