

## Determination of the Effect of the Elimination Diet Applied for Overweight and Obese People with Food Intolerance on Body Composition and Biochemical Parameters

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### ABSTRACT

*Food Intolerance, is a reaction against food, but not immunological manner, and may be confused with real food allergies. In this study, effects of special weight-loss diet together with an elimination diet on body composition and biochemical parameters of overweight and obese people who were diagnosed with food intolerance were investigated. The study group consists of 20 patients in total who were followed-up and treated in Yorktest Turkey*

*Laboratory; who were diagnosed with food intolerance, and whose BMI was  $> 26\text{kg/m}^2$ . Bloodletting for these patients was executed with Lancet from their fingertips, and the blood drawn from these patients was assessed via enzyme-linked immunosorbent assay (ELISA) method, and food reactions of patients were determined for each food. Biochemical parameters of these patients are routine tests, which are necessary for food intolerance tests, and they are analyzed at Yorktest Turkey Laboratory for two times: before and after elimination diet plus special weight-loss diet. It has been determined that, the most common sensivity is obtained against yeast, egg yolk and white, cranberry, cow's milk, chicken, lentils and parsley. Anthropometric measurements and biochemical parameters before and after elimination diet plus special weight-loss diet applied to participants, were significantly improved in statistical manner. Due to positive changes in body composition and biochemical parameters obtained through application of special weight-loss diets together with elimination diet applied to fat and obese people, we think that this diet might be used for medical nutrition treatment of obesity as a treatment option.*

**Key words:** Food intolerance; obesity; IgG; elimination diet; yeast



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## INTRODUCTION

Food Intolerance (FI) is a reaction against food, but not immunological manner, and may be confused with real food allergies [1]. FI is a delayed detrimental reaction. Although any food, beverage, food additive or compound found in food creates symptoms in one or more organs and systems, it is not possible to mention actual food allergy. In case of actual food allergy, gamma immunoglobulin E (IgE) antibodies ingenerate against food; however, IgE antibodies do not arise in FI. The incidence of food intolerance is higher than the incidence of food allergy. Studies have suggested that the gamma immunoglobulin G (IgG) antibodies arising in case of food allergy have a role in obesity [2].

Specific IgG antibodies play an important role to maintain immunological tolerance against food antigens [3]. It is a known fact that chronic inflammation is the biggest threat to human health. Identification of IgG is generally considered as the only and best way for treatment of chronic diseases. Identification of food specific IgG may show up as the only option for determination of the food causing inflammation particularly in case of chronic inflammation induced by food. In recent publications, the significance of IgG as indicator has been emphasized and considerable improvements have been achieved related to the nutrition therapy including elimination of the food causing intolerance [4-7]. All around the world, elimination diets have been performed and chronic inflammation symptoms have improved >80% or totally eliminated in more than 300.000 people for whom IgG was employed as an indicator [8].

Food intolerance has been found to be associated with the following chronic inflammatory diseases: chronic headache, excessive body weight gain, excessive body weight loss, skin problems, autoimmune diseases, fibromyalgia, migraine, stomach and irritable bowel syndrome (IBS), such as gastrointestinal disorders, absorption disorders, rheumatic disorders, shortness of breath, asthma, depression, irritability, type II diabetes, hypertension, metabolic syndrome, hypothyroidism, chronic rhinitis, eczema, acne, swollen eyelids, urinary disorders, Crohn's disease, and cardiac and circulatory problems [9-15]. The aforementioned disease entities/models are of variable importance for the affected individuals, the public health system [16].

In a study, food antigens, C-reactive protein (CRP) and carotid intima-media thickness were evaluated to investigate the correlation between IgG antibodies, food antigens, early atherosclerotic lesions and inflammation in obese children and normal-weight children, and it was found that high CRP, high anti-food IgG antibody concentration and increased intima-media thickness were highly significant in obese children as compared to normal-weight children. The study indicated that obese children have significantly higher IgG antibody values directed against food antigens than normal-weight children. It has been proven that anti-food IgG antibodies are tightly associated with low grade systemic inflammation and with the IMT of the common carotid arteries. Those findings have raised the possibility that anti-food IgG is pathogenetically involved in the development of obesity and

atherosclerosis [17]. Elimination diets are frequently used in food intolerance or allergy, or gastrointestinal disorders and can also set the implementation of lifestyle changes needed to reduce chronic inflammation [18]. In case of food intolerance, after elimination of the food causing intolerance the symptoms may decrease and the person may not show any reaction against that specific food months or even one year after elimination. Nevertheless, it should be always kept in mind that the reaction to that food may occur at any time. If the person continues consuming the intolerance-producing food every day, intolerance may occur again even in a month. Viral infections may lead to re-development of food intolerance. Sometimes, the reactions

induced by FI may immediately disappear through elimination of the food that leads to intolerance, and may not appear again even when the patient starts back her/his normal diet which is more common in children. In general, long term elimination of the intolerance producing food makes the person feel better [7]. The role of FI identification and determination of appropriate elimination diet in improvement of chronic diseases is still a field of research [19]. The present study aimed to analyze the effects of elimination diet supported with a personal weight-loss diet program on body composition and biochemical parameters of overweight and obese people, who were diagnosed with food intolerance.

## **MATERIALS and METHODS**

### **Study Group**

The study involved people that were followed-up and treated with the diagnosis of food intolerance at special office of the YorkTest Turkey Laboratory between February, 2010 and March, 2011. The study was initiated with 50 people with a BMI  $>30 \text{ kg/m}^2$ . Fifteen patients dropped out the study at the end of the first 15 days as they could not carry out the personal weight-loss diet that they should be received along with the elimination diet. Additionally, 10 people and then 11 people gave over the study at the 5<sup>th</sup> week and at the 7<sup>th</sup> week, respectively. As the number of the people dropping the study out was high, 6 overweight people, who were willing to participate (BMI:  $26\text{-}29 \text{ kg/m}^2$ ) were also enrolled in the study. Ultimately, the study group was composed of 20 patients (15 women and 5 men) with a BMI  $>26 \text{ kg/m}^2$ .

### **Employed Analysis Methods**

#### **Anthropometric Measurements**

The overweight and obese people diagnosed with food intolerance had their body weight, body composition, (percentages of fat, muscle and water) and body mass index measured with the Tanita BC-418 MA Body Composition Analyzer at 15-days intervals during 3 months while they were with light clothes and without shoes, and the obtained values were recorded in the follow-up form.

Height of the patients was measured when the feet were abreast and head was in the Frankfort plane (orbitale-tragion horizontal line), and the obtained values were recorded in the follow-up form [20].

#### **Detection of Food Intolerance**

Lancet was used to collect blood samples from the fingertips of each participant, and the samples were sent out to England. The blood samples were analyzed at the YorkTest Laboratories in England using the ELISA method. As a result of the analyses, reactions of the participants were identified for each food. The foods that the participants showed reactions were scaled in the result report between +1 (the lowest reaction) and +4 (the highest reaction), and the reports were mailed to participants via e-mail.

#### **Diet**

Besides elimination of the intolerance producing food shown in Table 1 from the diets of each female and male participant, each patient followed a personal weight-loss diet program determined in accordance with their height, body weight, physical activity, dietary habits, socio-economic status and cultural conditions. Anthropometric measurements were performed at every interview during three

months with little changes when necessary. Each patient was informed of the energy and macronutrient ratio of the suggested diet and each food that s/he had sensitivity according to the YorkTest analyses. As there are a great number of intolerance-producing foods, only the foods causing intolerance at a level of +2, +3 and +4 are indicated in the Table 1.

**Table 1:** Personal Weight-Loss Diet Program and Intolerance- Inducing (+2, +3 and + 4) Food by Each Female and Male Patient

Case	Gender (M/F)	BMI (kg/m <sup>2</sup> )	Energy (Kcal)	KH (%)	Protein (%)	Fat (%)	Food causing intolerance (+2,+3,+4)
Case-1	M	33,9	1612	57	14	29	Gluten, yeast
Case-2	M	45,1	2270	54	19	27	Yeast
Case-3	M	37,6	1800	58	13	29	Lamb, cow's milk, yeast
Case-4	M	33,0	1506	56	17	27	Corn, rye, yeast
Case-5	M	36,5	1780	55	16	29	Gluten, lentils, cashews, yeast
Case-6	F	37,5	1495	58	14	28	Egg white, yeast
Case-7	F	34,6	1310	57	15	29	Cow's milk, yeast, soybean
Case-8	F	29,9	1100	57	14	29	Wheat, cow's milk, yeast, soy beans, Lentils
Case-9	F	26,0	995	55	17	28	Wheat, cow's milk, egg white, yeast
Case-10	F	49,1	2480	56	15	29	Wheat, egg yolks, cashew
Case-11	F	32,6	1220	55	20	25	Wheat, sunflower seeds, yeast
Case-12	F	34,8	1407	59	14	28	Wheat, chicken, cow's milk
Case-13	F	26,4	1000	59	13	28	Cow's milk, yeast
Case-14	F	38,8	1796	60	14	26	Lamb, cranberry, apple, yeast
Case-15	F	35,5	1290	50	20	30	Gluten, wheat, cow's milk, lentil, soybean, yeast
Case-16	F	28,9	1080	52	19	29	Gluten, wheat, beef, corn, nuts, yeast
Case-17	F	27,9	1080	58	14	28	Wheat, lentils, celery, pepper, yeast
Case-18	F	30,3	1200	55	20	25	Gluten, beef, chicken, yeast
Case-19	F	34,5	1190	55	16	29	Wheat, egg white, cashew, sesame seeds, yeast
Case-20	F	26,0	1020	56	16	28	Rye, corn, yeast

**M: Male, F: Female**

### Determination of Food Consumption Frequency

The "adult semiquantitative food frequency questionnaire" developed specifically for PURE (Prospective Urban Rural Epidemiological Study)-Turkey with proven validity and reliability was applied for the overweight and obese participants for two times- at the beginning of the study and at the end of the personal weight-loss diet programs conducted along with elimination diet, and the obtained data was recorded in the questionnaire form [21].

The findings of the questionnaires were evaluated using the Nutrition Information System in order to calculate energy, water, protein, fat, carbohydrate, fiber, minerals, alcohol, vitamin A, vitamin D, vitamin E, vitamin K, vitamin B1, vitamin B2, niacin, pantatonic acid, vitamin B6, biotin, folic acid, vitamin B12, vitamin C, sodium, potassium, calcium, magnesium, phosphorus, iron, zinc, iodine, glucose, fructose, galactose, monosaccharides, sucrose, maltose, lactose, disaccharides, starch, polysaccharides, isoleucine, leucine, lysine, methionine, cysteine, phenylalanine, tyrosine, threonine, tryptophan, valine, arginine, histidine, butyric acid, saturated

fatty acids, monounsaturated fatty acids, polyunsaturated fatty acids, and cholesterol, and the results were compared with the results obtained after completion of the elimination diet combined with a personal weight-loss diet program.

### Biochemical Parameters

The biochemical parameters in the blood samples collected from the overweight and obese people with food intolerance both before and after the elimination diet plus personal weight-loss diet were measured at a private laboratory (Biruni Laboratory, Istanbul), and the biochemical parameters including blood glucose (BG), HbA1c, fasting insulin, Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), total cholesterol (TC), low density lipoprotein (LDL)-cholesterol (LDL-C), high density lipoprotein (HDL)-cholesterol (HDL-C), triglycerides (TG), aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), thyroid stimulating hormone (TSH), free thyroxine (F-T4), free triiodothyronine (F-T3), calcium, hemoglobin (Hgb) and hematocrit (Hct) were obtained. Energy and nutrient values of the foods consumed by the participants were compared using the data obtained from the Nutrition Frequency Questionnaires applied before and after the diets.

Insulin resistance was estimated using the homeostasis model assessment for insulin resistance (HOMA-IR) [22]:  $\text{HOMA-IR index} = (\text{fasting insulin (mU/L)} \times \text{fasting glucose (mmol/L)}) / 22.5$ .

Plasma TC, LDL cholesterol, HDL, TGs, fasting glucose, calcium and GGT, ALT, AST were measured with a Dimension EXL with LM Integrated Chemistry System Analyzer (Siemens, Newark, Delaware) [23]. Values for LDL cholesterol were estimated according to the formula of Friedewald et al [24]. The intra-assay and interassay coefficients of variation were 1.3% and 0.8% for TC, 0.5% and 1% for TGs, 2.3% and 2.4% for HDL cholesterol, 1.5% and 1.6% and 1% for glucose, respectively.

Measurement of HbA1c was done by cationexchange high-pressure liquid chromatography (HPLC) on a D-10 system (Biruni Laboratory, Istanbul) using reagents according to the manufacturer's instructions. This method quantifies HbA1c, which is defined as Hb A with glucose attached to the N-terminal valine beta chain, as a percentage of HbA1c:  $(\text{HbA1c area} / \text{total HbA area of the chromatogram}) \times 100$ . When HbS, HbC, or 'Variant-Window' was detected in a sample on HPLC D-10, a 'corrected HbA1c value' was automatically obtained by exclusion of the area of the variant from the total area of the chromatogram. Correction is necessary because retention times of glycated HbS and glycated HbC are similar to that of the major HbA fraction on the chromatogram, so these fractions coelute with HbA [25].

Hemoglobin and Hematocrit were measured [26, 27].

Free thyroxine (F-T4), free triiodothyronine (F-T3), and thyroid stimulating hormone (TSH) and insulin were measured on cobas e 601 module (Electrochemiluminescence Technology, Roche Diagnostics), while the remaining analytes on cobas c 501 clinical chemistry module (Photometric Technology), according to the instructions of the manufacturer. TSH was considered normal if it was between 0.4 and 4.0 mIU/L [28]. The reference intervals for FT3 and FT4 were 2.70–5.20 pg/mL and 0.80–1.90 ng/dL, respectively

These biochemical parameters are routine tests for the patients that are followed-up and treated at the YorkTest Turkey Laboratory. These tests were analyzed at a private laboratory (Biruni Laboratory, Istanbul) and financed by the participants.

### Statistical Method

The Windows SPSS 17.0 Statistics Package Program was employed for statistical analysis of study data. ANOVA test was used for repeated measurements (body composition measurements). The Chi-square and Paired T tests were used for evaluation of data. The P value calculated in the analyses was compared using the 0.05 type 1 error.  $p < 0.05$  was considered statistically significant.

### RESULTS

According to the results of the food intolerance tests, the participants showed highest sensitivity to yeast, egg white and yolk, cranberry, cow's milk, chicken, lentils and parsley. It was identified that there was statistically significant improvement in the anthropometric measurements and biochemical parameters after the elimination diet combined with personal weight-loss diet.

As shown in Table 2, men were detected to be sensitive to vegetables such as rye, corn, millet, beef, turkey meat, salmon / trout fish, peas, string beans, carrots, potatoes, spinach, celery, cabbage; fruits such as and peach, lime, pineapple, currants and nuts, seeds, cola nuts, tea and chillies while sensitivity of women to these nutrients was only 6.7%- 26%. While cranberry sensitivity was 60% among men, women did not show sensitivity to cranberry; this difference was considered statistically significant ( $p < 0.01$ ). Similarly, men's sensitivity to sesame seeds was 40% and women did not show sensitivity to sesame; thus, there was a significant difference between males and females regarding sesame sensitivity ( $p < 0.05$ ). Yeast was the nutrient that both sexes showed the highest sensitivity which was 86.7% in females and 100% in males.

**Table 2-** Food Intolerance Test Results of the Study Group \*  
(One person may have intolerance to more than one food.)

Food hypersensitivity	Female		Male		p
	N	%	N	%	
Gliadin (gluten)	2	13,3	2	40,0	0,197
Wheat	2	13,3	1	20,0	0,718
Rye	1	6,7	0	0,0	0,554
Corn	4	26,7	0	0,0	0,197
Millet	1	6,7	0	0,0	0,554
Egg White	5	33,3	3	60,0	0,292
Egg yolk	4	26,7	3	60,0	0,176
Cow's milk	5	33,3	2	40,0	0,787
Veal	1	6,7	0	0,0	0,554
Lamb	2	13,3	1	20,0	0,718
Chicken	1	6,7	2	40,0	0,071
Turkey	1	6,7	0	0,0	0,554
Fowl	2	13,3	2	40,0	0,197
Salmon / trout	1	6,7	0	0,0	0,554
Flounder / sole	0	0,0	1	20,0	0,076
Molluscs (mussels / squid / oyster)	1	6,7	1	20,0	0,389
Lentil	2	13,3	2	40,0	0,197
Soy bean	4	26,7	1	20,0	0,766
Pea	1	6,7	0	0,0	0,554
String bean	2	13,3	0	0,0	0,389

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Spinach	1	6,7	0	0,0	0,554
Mix mustard (cauliflowers / cabbage / broccoli / Brussels sprouts)	1	6,7	0	0,0	0,554
Onion	1	6,7	0	0,0	0,554
Potato	1	6,7	0	0,0	0,554
Carrot	3	20,0	0	0,0	0,278
Celery	1	6,7	0	0,0	0,554
Stuffing / green pepper	1	6,7	1	20,0	0,389
Parsley	0	0,0	2	40,0	0,010
Garlic	2	13,3	1	20,0	0,718
Mixed mint (mint / basil / sage / thyme)	0	0,0	1	20,0	0,076
Cherry	1	6,7	1	20,0	0,389
Cranberry	0	0,0	3	60,0	<b>0,001*</b>
Apple	0	0,0	1	20,0	0,076
Peach	1	6,7	0	0,0	0,554
Lime	1	6,7	0	0,0	0,554
Pineapple	1	6,7	0	0,0	0,554
Blackberry	0	0,0	1	20,0	0,076
Currant	2	13,3	0	0,0	0,389
Cashew	4	26,7	2	40,0	0,573
Almond	2	13,3	1	20,0	0,718
Hazelnut	4	26,7	0	0,0	0,197
Peanut	2	13,3	0	0,0	0,389
Brazil nuts	1	6,7	0	0,0	0,554
Seeds	2	13,3	0	0,0	0,389
Sesame seeds	0	0,0	2	40,0	<b>0,010**</b>
Chili pepper	1	6,7	0	0,0	0,554
Black pepper	3	20,0	2	40,0	0,371
Colanut	3	20,0	0	0,0	0,278
Tea	1	6,7	0	0,0	0,554
Yeast	13	86,7	5	100,0	0,389

\*p&lt;0.05

\*\*p&lt;0.01

While gluten and wheat sensitivity was 13.3% among women, wheat sensitivity was 20% and gluten sensitivity was 40% among men. Sensitivity to egg white and cow's milk was 33.3% among women, it was 60% and 40% among men, respectively. Additionally, men were 40% sensitive to foods of animal origin like chicken and fowl, but women were found to be less sensitive to such foods. When sensitivity to plant-derived foods including lentils and soy bean was evaluated, sensitivity to lentils was 13.3% among women while 40% among men; and sensitivity to soy bean was 26.7% and 20% for women and men, respectively. Men were identified to be more sensitive to nutrients, such as paper, parsley, and mint. The changes in the body weight (kg), BMI ( $\text{kg}/\text{m}^2$ ), fat (kg), muscle (kg) levels of women were statistically significant ( $p<0.05$ ). It has been specified in Table 3 that although body weight, BMI and fat percentage of women decreased, the muscle and water percentages did not show any statistically significant change during 3 months of

follow-up ( $p>0.05$ ). When the measurements of men were assessed, there was statistically significant changes in body weight (from 121 kg to 105 kg), BMI (from  $37.2 \text{ kg/m}^2$  to 32.4) and fat (from 46 kg to 30 kg) levels ( $p<0.01$ ). It was indicated that although body weight, BMI and fat percentage in body of men decreased, the muscle and water percentages did not show any statistically significant change during 3 months of follow-up ( $p>0.05$ ).

**Table 3.** Antropometric Measurements of the Women and Men participated in the Study

	1. Measurement	2. Measurement	3. Measurement	4. Measurement	5. Measurement	6. Measurement	P
Body weight (kg)	89.0±17.5	85.6±16.6	83.6±16.2	82.0±16.1	80.4±16.3	78.1±15.6	<b>0.000**</b>
	121.5±19.1	118.5±18.7	115.0±17.6	111.8±15.0	108.7±14.5	105.7±13.0	<b>0.000**</b>
BMI (kg/m <sup>2</sup> )	32.7±6.2	31.5±5.9	30.8±5.8	30.2±5.8	29.2±5.8	28.7±5.6	<b>0.000**</b>
	37.2±4.8	36.3±4.7	35.2±4.4	34.3±3.8	33.3±3.6	32.4±3.3	<b>0.000**</b>
Fat (kg)	38.1±12.3	35.4±12.0	33.3±11.2	32.2±10.9	30.5±11.3	28.2±10.8	<b>0.000**</b>
	46.1±10.3	40.0±9.0	38.0±8.6	36.2±7.8	33.9±6.7	30.4±6.0	<b>0.000**</b>
Muscle (kg)	50.9±5.8	50.2±5.3	50.2±5.5	49.8±5.7	49.9±5.6	50.0±5.4	<b>0.041*</b>
	75.3±13.0	78.5±10.8	77.1±10.3	75.8±9.2	74.8±9.0	75.3±9.2	0.271
Water (kg)	37.3±4.2	36.7±3.9	36.8±4.0	37.3±4.7	36.8±4.1	36.5±3.9	0.060
	55.2±9.5	57.5±7.9	56.4±7.4	55.5±6.7	54.8±6.6	55.1±6.7	0.271

\* $p<0.05$ (Males) \*\* $p<0.01$  (Females)

#BMI: Body mass index.

As indicated in Table 4, there was a statistically significant difference between levels of energy, water, protein, protein%, oil, carbohydrate, fiber, glucose, fructose, galactose, monosaccharides, sucrose, maltose, lactose, disaccharides, starch, polysaccharides, isoleucine, leucine, lysine, methionine, cysteine, phenylalanine, tyrosine, threonine, tryptophan, valine, arginine, histidine, butyric acid, saturated fatty acid, MUFA, PUFA, cholesterol consumed by women after the elimination diet as compared to the levels consumed before the diet ( $p<0.01$ ).

**Table 4.** Comparison of the Mean and Standard Deviation Values of Pre- and Post-Diet Macronutrient Consumption of Women and Men Participated in the study

Nutrients	Female		p	Male		p
	Before Diet	After Diet		Before Diet	After Diet	
	$\bar{x} \pm S$	$\bar{x} \pm S$		$\bar{x} \pm S$	$\bar{x} \pm S$	
Energy (kcal)	3429±1000	1027±194	<b>0.001**</b>	6179±2139	1614±258	<b>0.043*</b>
Water (g)	3404±799	1367±519	<b>0.001**</b>	5745±2296	1589±254	<b>0.043*</b>
Fiber (g)	63.8±22.5	22.3±7.5	<b>0.001**</b>	104.1±44.5	28.5±9.9	0.080
Protein (g)	143.5±45.6	58.8±14.8	<b>0.001**</b>	220.6±86.2	112.7±42.0	0.080
Protein %	17.1±2.0	24.3±6.4	<b>0.001**</b>	14.8±1.9	28.6±10.4	<b>0.043*</b>
Isoleucine (g)	7.0±2.3	2.6±0.7	<b>0.001**</b>	10.6±3.4	5.2±2.1	<b>0.043*</b>



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Leucine (g)	11.1±3.6	4.0±1.1	<b>0.001**</b>	16.9±5.6	7.9±3.1	<b>0.043*</b>
Lysine (g)	9.2±3.1	3.8±1.2	<b>0.001**</b>	13.5±3.5	8.2±3.5	0.080
Methionine (g)	2.9±1.0	1.15±0.4	<b>0.001**</b>	4.2±1.3	2.3±1.1	<b>0.043*</b>
Cysteine (g)	1.7±0.6	0.7±0.2	<b>0.001**</b>	3.1±1.1	1.4±0.5	<b>0.043*</b>
Phenylalanine (g)	6.3±2.0	2.3±0.5	<b>0.001**</b>	10.2±3.5	4.3±1.6	<b>0.043*</b>
Tyrosine (g)	5.1±1.6	1.9±0.5	<b>0.001**</b>	7.7±2.4	3.7±1.4	<b>0.043*</b>
Threonine (g)	5.6±1.9	2.2±0.6	<b>0.001**</b>	8.6±2.7	4.4±1.7	<b>0.043*</b>
Valine (g)	1.7±0.5	0.6±0.2	<b>0.001**</b>	2.6±1.0	1.1±0.4	<b>0.043*</b>
Arginine (g)	7.9±2.6	2.9±0.8	<b>0.001**</b>	12.2±3.8	5.4±2.0	<b>0.043*</b>
Histidine (g)	7.7±2.5	3.1±0.9	<b>0.001**</b>	14.6±3.5	5.9±2.5	<b>0.043*</b>
	3.6±1.2	1.4±0.5	<b>0.001**</b>	5.7±1.5	2.7±1.2	<b>0.043*</b>
Fat (g)	140±52	40±16	<b>0.001**</b>	243±93	57±21	<b>0.043*</b>
Fat%	35.8±4.6	34.3±10.0	0.755	35.8±6.3	32.0±10.8	0.686
Saturated fat (g)	51.4±17.6	11.3±5.1	<b>0.001**</b>	66.6±26.7	17.7±8.3	<b>0.043*</b>
MUFAs (g)	48.4±18.9	15.7±6.9	<b>0.001**</b>	99.5±44.1	20.4±8.5	<b>0.043*</b>
PUFAs (g)	33.8±13.6	9.1±4.9	<b>0.001**</b>	60.0±21.6	14.09±4.4	<b>0.043*</b>
Cholesterol(mg)	504±267	246±182	<b>0.012*</b>	649±327	248±142	<b>0.043*</b>
Butyric acid (g)	1.4±0.5	0.2±0.2	<b>0.001**</b>	1.4±0.8	0.5±0.2	0.080
Carbohydrate (g)	384±107	101±25	<b>0.001**</b>	722±305	148±48	<b>0.043*</b>
Carbohydrate %	46.2±5.7	41.5±8.8	0.139	48.4±5.6	37.6±7.6	0.138
Glucose (g)	43.5±19.7	15.7±4.7	<b>0.001**</b>	76.2±20.9	17.8±3.3	<b>0.043*</b>
Fructose (g)	47.3±19.1	18.6±5.2	<b>0.001**</b>	77.4±22.4	24.0±5.4	<b>0.043*</b>
Galactose (g)	3.3±2.2	0.4±0.6	<b>0.001**</b>	1.9±0.8	0.0±0.0	<b>0.043*</b>
Monosaccharides (g)	94.2±39.8	34.7±8.4	<b>0.001**</b>	155.5±43.6	41.8±8.1	<b>0.043*</b>
Sucrose (g)	80.3±27.7	20.0±10.2	<b>0.001**</b>	161.3±99.9	23.6±11.4	<b>0.043*</b>
Maltose (g)	3.2±2.8	0.0±0.0	<b>0.001**</b>	0.3±0.3	0.0±0.0	0.080
Lactose (g)	21.2±6.9	5.6±5.3	<b>0.001**</b>	22.6±11.3	18.2±1.4	0.345
Disaccharide (g)	103.8±31.2	25.7±13.2	<b>0.001**</b>	183.0±105.7	41.9±10.7	<b>0.043*</b>
Starch (g)	166.3±67.0	34.8±23.1	<b>0.001**</b>	354.0±281.9	58.5±42.0	<b>0.043*</b>
Polysaccharides(g)	167.6±66.9	35.0±23.1	<b>0.001**</b>	355.0±281.9	58.5±42.0	<b>0.043*</b>

#MUFA: monounsaturated fatty acid; PUFA: polyunsaturated fatty acid \***p<0.05** \*\***p<0.01**

Similarly, the changes in consumption levels of energy, water, protein%, oil, carbohydrate, glucose, fructose, galactose, monosaccharides, sucrose, disaccharides, starch, polysaccharides, isoleucine, leucine, methionine, cysteine, phenylalanine, tyrosine, threonine, tryptophan, valine, arginine, histidine, saturated fatty acids, MUFA, PUFA, and cholesterol by men after elimination diet were found statistically significant ( $p<0.01$ ).

When the biochemical parameters obtained before and after the elimination diet were compared, the decrease in glucose, HbA1c, insulin, HOMA-IR, total cholesterol, LDL cholesterol, triglycerides, AST, GGT and ALT levels of women after the diet was found statistically significant as shown in Table 5 ( $p<0.01$ ). Also, hemoglobin levels of women showed a statistically significantly increase after the diet in comparison with the levels obtained before the diet ( $p<0.01$ ).

**Table 5.** The Mean and Standard Deviation Values of the Biochemical Parameters Obtained from the Study Group Before and After Elimination Diet

	Female			Male		
	Before Diet $\bar{X} \pm S$	After Diet $\bar{X} \pm S$	p	Before Diet $\bar{X} \pm S$	After Diet $\bar{X} \pm S$	p
Glucose (mg / dl)	99.1±13.0	91.3±8.5	<b>0.008**</b>	101.8±12.8	89.2±9.7	<b>0.043*</b>
HbA1c (%)	5.6±0.5	5.4±0.4	<b>0.008**</b>	5.6±0.8	5.0±0.6	0.068
Insulin (µ / ml)	14.3±6.7	8.8±3.3	<b>0.001**</b>	19.3±7.1	11.7±3.6	<b>0.043*</b>
HOMA-IR	3.1±1.5	2.1±0.7	<b>0.001**</b>	5.2±2.3	2.6±0.7	<b>0.043*</b>
Total cholesterol (mg / dl)	215.3±45.5	187.3±29.7	<b>0.001**</b>	189.2±21.4	175.0±15.0	<b>0.043*</b>
HDL-cholesterol (mg / dl)	53.3±12.6	54.73±11.21	0.864	43.8±4.5	47.20±6.6	0.066
LDL-cholesterol (mg / dl)	140.4±39.1	110.9±28.6	<b>0.001**</b>	120.4±11.5	111.4±13.7	<b>0.042*</b>
Triglycerides (mg / dl)	121.7±51.7	79.1±21.8	<b>0.001**</b>	117.4±40.9	91.6±16.7	0.104
AST (IU / l)	22.2±4.7	15.73±5.5	<b>0.001**</b>	32.6±13.2	21.0±2.6	<b>0.043*</b>
GGT (IU / L)	19.4±19.1	12.1±11.9	<b>0.003**</b>	29.2±18.4	13.2±3.1	<b>0.042*</b>
ALT (IU / l)	20.1±12.0	13.9±9.6	<b>0.002**</b>	34.0±11.2	14.4±5.4	<b>0.043*</b>
TSH (µ / L)	2.5±3.4	1.3±0.7	0.363	1.7±0.4	1.7±0.3	0.893
Free T3 (pmol / l)	2.9±0.5	2.7±0.8	0.363	3.1±0.9	3.8±1.2	0.500
Free T4 (pmol / l)	11.6±37.3	4.3±6.2	0.211	6.2±6.8	6.4±7.2	0.686
Calcium (mg)	9.1±0.6	9.4±0.5	0.052	9.6±0.4	9.8±0.3	0.102
Hgb (g / dl)	12.5±1.1	13.1±1.1	<b>0.002**</b>	15.1±0.4	15.2±0.3	0.059
Hct (%)	37.6±3.2	38.6±2.6	0.053	44.8±0.4	45.9±1.9	<b>0.042*</b>

\*p&lt;0.05 \*\*p&lt;0.01

#TC: Total cholesterol, HDL: High density lipoprotein, LDL: Low density lipoprotein, VLDL: Very low density lipoprotein, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, GGT: Gama glutamil transferase, Hgb: Hemoglobin, Hct: Hemotocrit, TSH: Thyreoid stimule hormone, T3: Triiodotironin, T4: Thyroxine.

Like the case of women, the decrease in glucose, HbA1c, insulin, HOMA-IR, total cholesterol, LDL cholesterol, triglycerides, AST, GGT and ALT levels of men after the diet was found statistically significant (p<0.05). The Hct % level of men showed a statistically significant increase after the diet when compared with the levels before the diet (p<0.05).

## DISCUSSION

Lactose intolerance occurs in one person out of every 3 in Turkey [<sup>29</sup>]. The results of this study seem to support that finding [<sup>30</sup>]. In a study conducted on patients with rheumatoid arthritis, 27% of the patients were identified to have intolerance to various foods, particularly cow's milk, meat and wheat gluten. The reason why males were observed to be more intolerant to the foods of animal origin than females may correlate with the consumption frequency and volume of these foods [<sup>31</sup>]. Soy sensitivity is quite common in children and adults in the USA. In this study, soy bean sensitivity was found higher in women with a rate of 26.7% than in men (20%). The higher rate of soy sensitivity in women may be associated with the fact that women consume more soy products, because as the immune system is under soy

bombardment due to excessive consumption, the person may become sensitive to soy products [<sup>32</sup>]. Yeast intolerance is present in 80 million people in the United States with higher incidence in women (70%) [<sup>32</sup>]. In our study, conversely, all male participants (100%) showed sensitivity to yeast. We are aware that as the size of study group is small, we can not generalize these findings to whole Turkish population, and we wish to specify that there should be further larger studies on the issue. Yeasts are one-celled organisms that live on the foods with simple carbohydrate such as sugar, bread, biscuits and cake along with fermented foods such as alcohol and vinegar. Overuse of antibiotics and changes in diet are the main reasons of yeast intolerance [<sup>32,33</sup>]. In our study group, yeast was the nutrient that both sexes showed the highest sensitivity which was 86.7% in females and 100% in males. As noted above, we believe that the unconscious use of antibiotics is a major reason for widespread yeast sensitivity in Turkish population. Moreover, considering that as dietary habit is a significant risk factor for yeast sensitivity, we may correlate this difference with the nature of Turkish diet which mainly bases on grain and excessive consumption of fermented foods and sugar. In a study, 84% and 83% of the patients showed high intolerance to processed cheese and yeast, respectively [<sup>34</sup>].

Clinicians and researchers indicated in the last 60 years that tissue insulin resistance may play a role in development of chronic diseases. Metabolic syndrome characterized by excessive accumulation of abdominal fat, high triglycerides, low HDL and hypertension is defined as a "cardiovascular risk factors beam" of metabolic origin accompanied with increased risk for type II diabetes and cardiovascular disease. Accordingly, diseases related to insulin resistance are a major health problem in Western societies [<sup>35-39</sup>]. Identification of food specific IgG has been emphasized as a significant factor in chronic inflammatory diseases, recently. IgG is also claimed to play a role in development of obesity [<sup>40,41</sup>]. In a previous study carried out to analyze the correlation between early atherosclerotic lesions in obese and normal-weight children, good antigens, C-reactive protein (CRP) and carotid artery intima media thickness (IMT) were measured and IgG antibodies were evaluated. The results of that study proved that obese children had significantly higher anti-food IgG antibodies as compared to normal-weight children. It was also revealed that anti-food IgG antibodies are significantly correlated with low degree systemic inflammation and carotid artery intima media thickness. These findings have increased the possibility that IgG antibodies play a role in development of pathogenetic obesity and atherosclerosis [<sup>42</sup>].

According to comparison of the biochemical findings obtained before and after the elimination diet combined with personal weight-loss diet, there was an improvement in biochemical findings in general and the decrease of blood glucose, HbA1C, insulin, HOMA-IR, total cholesterol, LDL cholesterol, triglycerides, AST, GGT and ALT levels was statistically significant. Likewise, there was an improvement in biochemical findings in general and the decrease of blood glucose, HbA1C, insulin, HOMA-IR, total cholesterol, LDL cholesterol, triglycerides, AST, GGT and ALT levels after the elimination diet combined with personal weight-loss diet was statistically significant. It is conceivable that the decrease in the biochemical parameters may be associated with weight loss and improvement of anti-food IgG antibody concentration as a result of elimination of intolerance-inducing food from the diet. Additionally, we think that decrease in body fat and improvement in biochemical parameters may have resulted from implementation of personal weight-loss diet together with elimination diet. In still another study, researchers investigated the effect of anti-food IgG antibodies in obesity and revealed that

intestinal microflora, infection, obesity and anti-food IgG antibodies may be correlated. There is another study revealing similar results [43,44,45]. However, there is a study, in which no correlation was found between anti-food IgG antibodies and obesity [46].

Recent publications present examples of that elimination diet therapy using anti-food IgG as an indicator may be helpful for treatment of related diseases [4-7]. All around the world, elimination diets have been performed and chronic inflammation symptoms were improved >80% or totally eliminated in more than 300.000 people for whom IgG was employed as an indicator [8]. There is a study, in which elimination diet was applied for children with constipation that did not respond to laxative therapy, and thus, all children, who participated in the research, were treated successfully and their bowel functions returned to normal. There are also other studies reporting successful results with elimination diet for people with constipation [47-49].

As a result of the evaluation of the pre-diet and post-diet consumption frequency results of the women and men receiving elimination diet combined with personal weight-loss diet program for 3 months, we believe that the decrease in daily energy intake (from 3428 kcal to 1206 kcal; from 6179 kcal to 1613 kcal, respectively) and in body fat ratio may be associated with the improvement of biochemical parameters. In our study group, while the muscle and water percentage of the body were kept at the same level, weight and body fat of overweight and obese participants decreased significantly at the end of the elimination diet combined with a personal weight-loss diet program for three months. We have encountered a study in the literature in which participants lost weight as eliminating the intolerance-producing foods from their diet for 3 months elimination diet like in our study [32]. In case of elimination diet plus personal weight-loss diets for treatment of obesity, energy intake should be kept above 1200 kcal for women and 1600-1700 kcal for men; energy derived from fat should not be very high and from carbohydrate should not be very low; and proteins should be kept at recommended levels.

## CONCLUSION

The results of this study suggest that food elimination based on IgG antibodies may be effective in reducing weight. Due to the positive changes in body composition and biochemical parameters of overweight and obese people achieved with elimination diet combined with personal weight-loss diet programs, we consider such a diet as a potential treatment option for medical nutrition therapy of obesity. Substitution of nutritionally important foods and professional guidance are necessary for the successful treatment of food intolerance and is worthy of further clinical research.

## REFERENCES

- 1- Sherry K, Hubbard LD. Medical nutrition therapy for food allergy and food intolerance. Krause's Food and Nutrition Therapy. L. Kathleen Mahan, Sylvia Escott-Stump: .Seattle, Washington, Saunders Elsevier, 2008.
- 2- Gerth van Wijk R, van Cauwenberge PB, Johansson SG. Revised terminology for allergies and related conditions. *Ned Tijdschr Tandheelkd.* 2003, 110: 328-331.
- 3- Gocki J, Bartuzi Z. Role of immunoglobulin G antibodies in diagnosis of food allergy. *Postepy Dermatol Alergol.* 2016, 33(4): 253-6.
- 4- Atkinson W, Sheldon TA, Shaath N, Whorwell PJ, Food elimination based on IgG antibodies in irritable Bowel Syndrome: a randomised controlled trial. *Gut.* 2004,53: 1459-1464.

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- 5- Schmidh MI, Duncan BB. Diabetes: An Inflammatory Metabolic Condition. *Clinical Chemistry and Laboratory Medicine*. 2003, 41: 1120-1130.
- 6- Aljada A, Mohanty P, Ghanim H, Abdo T, Tripathy D, Chaudhuri A, et al. Increase in intranuclear nuclear factor B and decrease in inhibitor B in mononuclear cells after a mixed meal: evidence for a proinflammatory effect. *American Journal of Clinical Nutrition*. 2004, 79: 682-690.
- 7- Brostoff J, Gamlin L. Food Allergies and Food Intolerance: The Complete Guide to Their Identification and Treatment. United States, Healing Arts Press, 2000.
- 8- Rueff D, Weber B, Lieners C, Amazzllag W. Immuno-nutrition. *Immunité*. 2007, 13: 978-1073.
- 9- Schiepers OJ, Wichers MC, Maes M. Cytokines and major depression. *Prog Neuropsychopharmacol Biol Psychiatry*. 2005, 29: 201-217.
- 10- Hotamisligil GS. Mechanisms of TNF-alpha-induced insulin resistance. *Exp Clin Endocrinol Diabetes*. 1999, 107: 119-125.
- 11- Bentz S, Hausmann M, Paul S, Falk W, Obermeier F, Schölmerich J, et al. Clinical relevance of IgG antibodies against food antigen in Chron's disease- a double blind cross-over diet intervention study. *Presented at the 15th annual United European Gastroenterology Week Paris*. 2007.
- 12- Kalliomaki MA. Food allergy and irritable bowel syndrome. *Curr Opin Gastroenterol*. 2005, 21: 708-711.
- 13- Drisko J, Bischoff B, Hall M, McCallum R. Treating irritable bowel syndrome with a food elimination diet followed by food challenge and prebiotics. *J Am Coll Nutr*. 2006, 25: 514-522.
- 14- Garg R, Tripathy D, Dandona P. Insulin resistance as a proinflammatory state: mechanisms, mediators, and therapeutic interventions. *Current Drug Targets*. 2003, 4: 487-492.
- 15- Alpay K, Ertaş M, Kanca D, Lieners C. Ig G antikorlarına dayalı kısıtlanmış diyetin migren atakları üzerine etkisinin kontrollü, çift kör randomize çalışma ile araştırılması. *Cephalalgia*. 2010, Doi: 10.1177/033110241036140.
- 16- Kleine-Tebbe J, Wabmann-Otto A, Mönnikes H. Food allergy and intolerance: Distinction, Definitions and Delimitation. *Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz*. 2016, 59(6): 705-22.
- 17- Wilders-Truschning MH, Mangge C, Lieners C, Mayer W. IgG Antibodies Against Food Antigens are Correlated with Inflammation and Intima Media Thickness in Obese Juveniles. *Exp Clin Endocrinol Diabetes*. 2008, 116: 241-5.
- 18- Drummond J, Ford D, Daniel S, Meyerink T. Vulvodinia and Irritable Bowel Syndrome treated with an elimination diet: a case report. *Integr Med (Encinitas)*. 2016, 15(4): 42-7.
- 19- Gaby AR. The Role of Hidden Food Allergy/Intolerance in Chronic Disease. *Alternative Medicine Review*. 1998, 3: 90-100.
- 20- Pekcan G. Malnütrisyon. Hastaların antropometrik yönden değerlendirilmesi ve izlenmesi Enteral – Parenteral Beslenme. Derleyenler: S. Başoğlu. N. Karaağaoğlu. Hizmetiçi Eğitim Semineri, Türkiye Diyetisyenler Derneği Yayını, Ankara, 1996.
- 21- <http://www.metsend.org/pure.php>
- 22- Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta cell function from fasting glucose and insulin concentration in man. *Diabetologia* 1985;28:412-9.
- 23- Clinical and Laboratory Standards Institute/NCCLS. Procedures for the Handling and Processing of Blood Specimens; Approved Guideline. Third Edition. 2004.
- 24- Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of the preparative centrifuge. *Clin Chem* 1972;18:499-502.
- 25- Bouzid K, Bahlous A, Ferjani W, Kalai E, Ducrocq R, Ben Mami F, et al. Advantage of HbA1c assay by HPLC D-10 versus cobas integra 400 in a population carrier for HbS and HbC. *Clin Lab*. 2012; 58: 8218.
- 26- National Committee for Clinical Laboratory Standards. Procedure for determining packed cell volume by the micro hematocrit method (NCCLS document H7-A). Villanova, PA, 1985.

- 26- National Committee for Clinical Laboratory Standards. Procedure for determining packed cell volume by the micro hematocrit method (NCCLS document H7-A). Villanova, PA, 1985.
- 27- National Committee for Clinical Laboratory Standards. Reference procedure for the quantitative determination of hemoglobin in blood (NCCLS document H15-A). Villanova, PA, 1984.
- 28- G. Brabant, P. Beck-Peccoz, B. Jarzab et al., "Is there a need to redefine the upper normal limit of TSH?" *European Journal of Endocrinology*, vol. 154, no. 5, pp. 633–637, 2006.
- 29- Baysal A. Beslenme. Yenilenmiş 12. Baskı. Ankara, Hatiboğlu Yayınevi, 2009.
- 30- Mamone G, Picariello G, Addeo F, Ferranti P. Proteomic analysis in allergy and intolerance to wheat products. *Expert Rev Proteomics*. 2011, 8: 95-115.
- 31- Liden M, Kristjansson G, Valtysdottir S, Venge R, Hallgren R. Self-reported food intolerance and mucosal reactivity after rectal food protein challenge in patients with rheumatoid arthritis. *Scand J Rheumatol*. 2010, 39: 292-8.
- 32- Trickett S. *Coping with Candida*. Sheldon Press, 2007.
- 33- Belkaid Y, Hand TW. Role of the microbiota in immunity and inflammation. *Cell*. 2014, 157(1): 121-41.
- 34- Bentz S, Hausmann M, Piberger H, Kellermeier S, Paul S., Held L. Clinical Relevance of IgG Antibodies against Food Antigens in Crohn's Disease: A Double-Blind Cross-Over Diet Intervention Study. *Digestion*. 2010, 81: 252–264.
- 35- Kershaw EE, Flier JS. Adipoz tissue as an endocrine organ. *The Journal of Clinical Endocrinology and Metabolism*. 2004, 89: 2548-2556.
- 36- Ingelsson E. Insulin resistance and risk of congestive heart failure. *JAMA*. 2005, 294: 334-341.
- 37- Srikanth S, Deedwania P. Comprehensive risk reduction of cardiovascular risk factors in the diabetic patient: an integrated approach. *Cardiology Clinics*. 2005, 23: 193-210.
- 38- Sowers JR, Frohlich ED. Insulin and insulin resistance: impact on blood pressure and cardiovascular disease. *Med Clin North Am*. 2004, 88: 63-82.
- 39- Yaffe K, Kanaya A, Lindquist K, Simonsick EM, Harris T, Shorr RI, et al. The metabolic syndrome, inflammation, and risk of cognitive decline. *JAMA*. 2004, 292: 2237-2242.
- 40- Rueff D, Weber B, Lieners C, Amazzlag W. Immuno-nutrition. *Immunité*. 2007, 13: 978-1073.
- 41- Gonzalez-Quintela A, Alende R, Gude F, Campos J, Rey J, Mejjide LM, et al. Serum levels of immunoglobulins (IgG, IgA, IgM) in a general adult population and their relationship with alcohol consumption, smoking and common metabolic abnormalities. *Clin Exp Immunol*. 2008,15:42-50.
- 42- Wilders-Truschnig MH, Mangge C, Lieners C, Mayer W. IgG Antibodies Against Food Antigens are Correlated with Inflammation and Intima Media Thickness in Obese Juveniles. *Exp Clin Endocrinol Diabetes*. 2008, 116: 241-5.
- 43- Nadal I, Santacruz A, Marcos A, Warnberg J, Gargorri M, Moreno LA, et al. Shifts in clostridia, bacteridia, bacteroides and immunoglobulin-coating fecal bacteria associated with body weight loss in obese adolescents. *Int J Obes (Lond)*. 2009, 33: 758-67.
- 44- Jaworowska A, Bazylak G. Chlamydomydia pneumonia antibodies may be independently associated with increased BMI and percentage of body fat among women. *Int J Obes (Lond)*. 2011, 35: 1225-1232.
- 45- Bargo F, Verduci E, Riva A, Lassandro C, Riva E, Morace G, et al. Relative abundance in bacterial and fungal gut microbes in obese children: a case control study. *Child Obes*. 2016, 23.
- 46- Zuniga-Torres MG, Martinez-Corrillo BE, Pardo-Morales RV, Warnberg J, Marcos A, Benitez-Arciniega AD, et al. Are immunoglobulin concentrations associated with the body composition of adolescents? *Hum Immunol*. 2009, 70: 891-4.
- 47- Carrocci A, Di Prima L, Lacono G, Florena AM, D Arpa F, Sciume C, et al. Multiple food hypersensitivity as a cause of refractory chronic constipation in adults. *Scand J Gastroenterol*. 2006, 41:498-504.
- 48- Drisko J, Bischoff B, Hall M, McCallum R. Treating Irritable bowel syndrome with a food elimination diet followed by food challenge and probiotics. *J Am Coll Nutr*. 2006, 25: 514-22.
- 49- Iacono G, Bonventre S, Scalia C, Maresi E, Di Prima L, Soresi M, et al. Food intolerance and chronic constipation: manometry and histology study. *Eur J Gastroenterol Hepatol*. 2006, 18: 143-50.

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