

Obesity and CRP, Adiponectin, Leptin, and Lipid Profile in Saudi Arabian Adolescent Females

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Abstract: Overweight and obesity are increasing tremendously in female Saudi Arabian adolescents. Overweight and obesity lead to many medical risks and affects the immune system. In this study, the effects of obesity on the immune system of 100 Saudi female adolescent students were investigated. Using a blood sample from each subject, the following immune related parameters were determined: concentrations of C-reactive protein (CRP), adiponectin and leptin hormones, and the complete lipid profile. Finally, to assess the body weight status of the subjects and to categorize them, the weight, height, and the waist and hip circumferences were measured to calculate the body mass index (BMI), waist-to-hip ratio (WHR), and the waist circumference (WC). Results show highly significant increases for the CRP and leptin and a highly significant decrease of adiponectin with increasing body weight measured by the three methods. As for the lipid profile, both triglycerides and LDL increased while HDL decreased as body weight increased. Cholesterol did not change with changing body weight measured by the three methods. The findings indicate that obesity seriously affects the immune systems of the subjects and confirm the finding of other researchers that obesity is an inflammatory disease, which explains some health complications associated with obesity.

Keywords: CRP, leptin, adiponectin, cholesterol, LDL, HDL, triglycerides, adolescents, BMI, WHR, WC, obesity, inflammation, immunity.

INTRODUCTION

The prevalence of overweight and obesity in children and adolescents is rising worldwide, with most increases observed in countries that are affluent or economically developed. In Saudi Arabia, overweight and obesity are becoming more prevalent and more so in adolescent girls compared to boys [1, 2]. Compared with lean adolescents, overweight and obese ones are more likely to develop diseases such as insulin resistance, glucose intolerance, type 2 diabetes, dyslipidemia, hypertension, hyperuricemia, non-alcoholic fatty liver disease, and higher risk for cardiovascular disease as young adults [3, 4]. This higher prevalence of diseases continues into adulthood and may lead to a shortened life span.

The immune system is sensitive to changes in the body and general health. An unhealthy state, such as overweight and obesity, may weaken the immune system in a general way as evidenced by the fact that obese individuals are more likely than lean ones to contract infections and infectious illnesses [5, 6]. Some studies in animals and humans have supported a link between overweight and obesity and changes in the immune system [5, 7-10] although findings are contradictory [11].

Researchers [12-15] suggest that obesity causes a chronic, systemic low-grade inflammatory state and an increase of inflammatory mediators. C-reactive protein (CRP), a marker for inflammation in the body, is important in innate immunity and is involved in the production of many inflammatory cytokines [16]. Research has shown [17] CRP to increase as inflammation increases and in cases of trauma, infection, and cardiovascular diseases. CRP [13] is secreted by adipocytes and it increases in overweight and obesity as explained by the finding that obesity causes a state an unusual health state and low-grade inflammation. Research on children and adolescents has shown [18-20] higher CRP levels in overweight and obese children.

White (as opposed to brown) fat cells are the primary type of fat cell present in older children and adults and they secrete many cytokines that are inflammatory, such as CRP, and leptin, or anti-inflammatory, such as adiponectin [13]. Adiponectin and leptin are adipocytokines, or adipokines, which are exclusively and specifically secreted by fat cells and are involved in regulating body weight [13] and they have important roles in the immune response [21-23]. Leptin is a satiety signal that decreases food intake in order to regulate weight, while adiponectin leads to increased fat catabolism [24, 25] and has antiatherogenic properties [26].

Fat cells store fat and they contain primarily triglycerides and cholesteryl ester [27]. In overweight and obese individuals, an unhealthy lipid profile, characterized by elevated levels of cholesterol,

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triglycerides (TG), and low density lipoproteins (LDL), and decreased levels of high density lipoproteins (HDL), is prevalent. This profile is also linked to many of the diseases and conditions associated with overweight and obesity [28]. Many immune responses are influenced and regulated by lipids, and fatty acids [4]. Overweight and obesity are a result of increased body fat, and this increase leads to increased influence on the immune system.

There is a lack of enough information about the effects of body weight on the immune system, especially for Arabian populations. This study investigates the effect of obesity on the immune system and lipid profile in Saudi adolescent females in Jeddah, Saudi Arabia, using the BMI, WHR and WC as methods of weight categorization and assessment. This would help in determining whether immunity is affected by increasing weight in adolescent females and would help in setting guidelines for the ideal weight statuses for optimum health in this group.

SUBJECTS AND METHODS

Experimental Subjects and Categorization

This study was carried on 100 randomly chosen Saudi female adolescent students (12-18 years old) from Middle School Number 70 for girls, Jeddah, Saudi Arabia. The parents of all subjects signed a consent form and the students filled a questionnaire to assess their general health state and if they are taking any medications. None of the subjects had any chronic disease, blood diseases, or allergies. In addition, none of them was on any type of medication.

Blood Collection

Whole blood was collected from each subject into lithium heparin vacutainer tubes, which were used within three hours for the differential complete blood count (CBC). Blood samples were also collected into plain tubes, which were allowed to stand at room temperature for the formation of a blood clot. The blood was then centrifuged at 3,000 rpm for 10 minutes to separate the serum from the clot. Clear serum was carefully aspirated using a micropipette, transferred into micro-centrifuge tubes, and subsequently stored at -20 °C until the time of analysis.

Anthropometric Measurements and Categorizations

Each subject was weighed at the same time of blood collection. A new regular household scale was

used for this purpose. Also the height, waist (at the naval), and hips (at the fullest point) measurements were taken using a measuring tape.

The body mass index (BMI) was used to divide the subjects into four groups, or categories, with a total of 18-33 subjects per each BMI category. A BMI below the 5th percentile was considered underweight, healthy or control weight was any BMI between the 5th percentile to less than the 85th percentile, a BMI above the 85th to less than the 95th percentile classifies the subject as overweight, and finally a subject with a BMI equal to or greater than the 95th percentile is considered obese [29].

The waist-to-hip ratio (WHR) was used to assign the subjects into one of three risk groups for diseases. The groups were low risk (WHR of 0.80 or below), moderate risk (WHR between 0.81 and 0.85), and high risk (WHR above 0.85) [30]. The high risk group is the group of subjects who have the highest amount of visceral fat.

The android (men-like) body shape, which is also known as an apple shape or upper body obesity, is associated with a WHR greater than 0.85 for females. A WHR below 0.8 for females is associated with the gynoid (women-like) body shape, which is commonly known as the pear-shaped or lower body obesity [31].

The waist circumference (WC) was used to distribute the subjects into three risk groups according to reference ranges for each group. The low risk group was for WC below 32.5 inches, a WC between 32.5 and 35 inches was considered moderate risk, while the high risk group was for WC above 35 inches [31].

Determination of Serum Adiponectin Concentrations

Adiponectin concentrations were determined using an *in vitro* diagnostic ELISA kit (Alpco Immunoassays, USA) and the intensity of the developed color was read at 429 nm by an ELISA microplate reader (Elx800, Biotek, USA).

Determination of Serum Leptin Concentrations

Leptin concentrations were determined using an *in vitro* AssayMax Human Leptin ELISA kit (Assaypro, USA) and the results were read at 450nm on an ELISA microplate reader, as above.

Determination of Serum C-Reactive Protein Concentrations

The C-reactive protein (CRP) concentrations were determined using a high sensitivity CRP protein enzyme immunoassay test kit (BioCheck, Inc., USA) according to the manufacturer's instructions and the absorbance was measured at 450 nm on an ELISA microplate reader, as above.

Determination of Lipid Concentrations

Serum cholesterol, triglycerides, HDL-Cholesterol, and LDL-cholesterol were determined using the appropriate Flex reagent cartridge (Dade Behring, USA) for each determination, according to the manufacturer's instructions. The determinations were performed on the Dimension Clinical Chemistry System RXL max.

Statistical Methods

The statistical program SPSS-V12 was employed to obtain the descriptive and analytical statistics. The Mean (\bar{x}), standard deviation (\pm SD), standard error of the mean (\pm SE), and range (minimum-maximum) were calculated for all parameters, using the SPSS-V12 statistical program.

After testing the normal distribution and the homogeneity of the populations, it was found that although the sample (all subjects) follows the normal distribution, since it is a large sample (exceeds 50), some of the samples are not homogeneous. Therefore, the ANOVA one-way test was used to test the significance in the correlations between body weight measurements (BMI, WHR and WC) and each parameter measured for each subject. The Post hoc tests were used for the multi-comparisons. The Dunnett test was used for the homogeneous parameters, while the Tamhane's T2 test was used for the non-homogeneous parameters. The resulting P values demonstrate significance or lack thereof as follows: $P > 0.05$ = Not significant (NS), $P \leq 0.05$ = Significant (S), $P < 0.01$ = Highly significant (HS). Finally, data were graphically represented using the SPSS program.

RESULTS

Subjects and BMI Categories

One hundred adolescent female students, also used in a previous study (Mahassni and Sebaa 2013), with an age range of 12-18 years (mean \pm SD = 15.20 \pm

1.29 years), had a BMI range of 29.02 kg/m² (mean \pm SD = 24.92 \pm 7.06). At a confidence level of 99%, the mean of the BMI belongs to the confidence interval (23.07, 26.78) with a SD of 7.06, and a small standard error of 0.71.

The subjects were divided into four groups according to their BMI, with each group having 18-33 subjects. The Four groups were: underweight subjects (frequency, percent: 18, 18%), healthy (control) subjects (33, 33%), overweight subjects (21, 21%), and obese subjects (28, 28%). The minimum BMI for the underweight category was 13.68, while the maximum was 23.14. For the healthy subjects (control group) the range of the BMI was 16.80 to 24.95. For the overweight subjects the BMI range was 25.20 to 29.80. Finally, the BMI for the obese subjects ranged from 30.17 to 42.70.

Subjects and WHR Categories

The subjects had WHRs that ranged from 0.20 to 1.62 (mean \pm SD = 0.84 \pm 0.05) with a very small standard error of 0.01 and a short confidence interval (0.82, 0.85). The WHR was divided into three groups as follows: subjects with low risk (frequency, percent: 35, 35%), subjects with moderate risk (21, 21%), and subjects with high risk (44, 44%).

Subjects and WC Categories

The WC for the subjects ranged from 22-47 inches (mean \pm SD = 32.45 \pm 5.95) with a standard error of 0.60 and a confidence interval of (30.88, 34.01). The WC was divided into three groups as follows: subjects with low risk (frequency, percent: 42, 42%), subjects with moderate risk (21, 21%), and subjects with high risk (37, 37%).

The Relationships between Weight Measures and Measured Parameters

The ANOVA one-way test was used to determine the relationship between concentration means for the categories of each parameter and the weight measures (BMI, WHR, and WC). The results (Tables 1-3) show that CRP, leptin, triglycerides, LDL, and HDL each was highly significantly related to the BMI, WHR, and WC. As for adiponectin it was highly significantly related to the BMI, but significantly related to WHR and WC. As for Cholesterol, it was not related to any of the used measures of obesity.

Table 1: Descriptive Statistics and Test of Significance for the Parameters and BMI

Parameter	BMI	Concentration			SD	P
		Mean	Min	Max		
CRP (mg/l)	Underweight	17.74	0.05	61.31	17.74	0.000 ^{HS}
	Healthy	7.74	0.15	95.77	21.16	
	Overweight	49.30	0.56	121.73	38.40	
	Obese	94.34	0.76	238.54	65.00	
Adiponectin (ng/ml)	Underweight	4.45	1.10	7.20	1.64	0.003 ^{HS}
	Healthy	4.14	0.80	9.20	1.67	
	Overweight	3.75	2.20	5.70	1.02	
	Obese	2.87	1.50	5.00	0.98	
Leptin (ng/ml)	Underweight	0.67	0	2.70	0.89	0.000 ^{HS}
	Healthy	1.93	0	8.20	1.98	
	Overweight	4.39	1.80	7.60	1.54	
	Obese	5.77	0.50	13.60	3.29	
Cholesterol (mmol/l)	Underweight	4.38	3.44	5.63	0.62	0.595 ^{NS}
	Healthy	4.13	3.26	5.34	0.58	
	Overweight	4.18	3.07	5.80	0.66	
	Obese	4.26	2.71	5.61	0.71	
TG (mmol/l)	Underweight	0.86	0.37	2.47	0.65	0.000 ^{HS}
	Healthy	1.62	0.21	3.70	0.96	
	Overweight	1.86	0.54	3.42	0.85	
	Obese	4.54	3.00	7.80	1.06	
LDL (mmol/l)	Underweight	2.60	1.83	3.54	0.50	0.000 ^{HS}
	Healthy	2.57	1.65	3.30	0.47	
	Overweight	2.63	1.52	3.79	0.59	
	Obese	4.46	3.25	6.00	0.81	
HDL (mmol/l)	Underweight	1.78	1.38	2.31	0.25	0.000 ^{HS}
	Healthy	1.43	0.84	2.70	0.33	
	Overweight	1.37	1.06	1.69	0.19	
	Obese	0.47	0.02	0.94	0.26	

The ANOVA one way test was used for the significance test.

HS: highly significant ($P < 0.01$).

NS: Not significant ($P > 0.05$).

Max: Maximum, Min: Minimum.

Table 2: Descriptive Statistics and Test of Significance for the Parameters and WHR

Parameter	WHR	Concentration			SD	P
		Mean	Min	Max		
CRP (mg/l)	Low Risk	8.41	0.06	95.77	20.66	0.000 ^{HS}
	Moderate Risk	34.64	0	161.93	49.35	
	High Risk	78.14	0.16	238.54	60.18	
Adiponectin (ng/ml)	Low Risk	4.33	0.80	7.20	1.47	0.01 ^{HS}
	Moderate Risk	3.41	2.10	9.20	1.55	
	High Risk	3.33	1.50	5.70	1.18	

(Table 2). Continued.

Parameter	WHR	Concentration			SD	P
		Mean	Min	Max		
Leptin (ng/ml)	Low Risk	1.09	0	5.20	1.37	0.000 ^{HS}
	Moderate Risk	3.58	0.02	8.20	2.16	
	High Risk	5.57	0.05	13.60	2.92	
Cholesterol (mmol/l)	Low Risk	4.17	3.26	5.63	0.59	0.742 ^{NS}
	Moderate Risk	4.29	3.16	5.80	0.72	
	High Risk	4.24	2.71	5.61	0.66	
TG (mmol/l)	Low Risk	1.20	0.12	3.70	1.01	0.000 ^{HS}
	Moderate Risk	2.09	0.45	5.26	1.27	
	High Risk	3.72	0.27	7.80	1.57	
LDL (mmol/l)	Low Risk	2.53	1.65	3.54	0.49	0.000 ^{HS}
	Moderate Risk	2.99	1.52	5.80	0.93	
	High Risk	3.86	1.73	6.00	1.10	
HDL (mmol/l)	Low Risk	1.55	0.48	2.31	0.35	0.000 ^{HS}
	Moderate Risk	1.26	0.19	2.70	0.47	
	High Risk	0.79	0.02	1.69	0.51	

The ANOVA one way test was used for the significance test.

HS: Highly significant ($P < 0.01$).

NS: Not significant ($P > 0.05$).

Max: Maximum, Min: Minimum.

Table 3: Descriptive Statistics and Test of Significance for the Parameters and WC

Parameter	WC	Concentration			SD	P
		Mean	Min	Max		
CRP (mg/l)	Low Risk	16.01	0	95.77	29.39	0.006 ^{HS}
	Moderate Risk	37.32	0.05	121.73	40.61	
	High Risk	59.82	0.03	238.54	66.38	
Adiponectin (ng/ml)	Low Risk	4.18	0.80	7.20	1.65	0.027 ^S
	Moderate Risk	4.08	2.20	6.30	0.96	
	High Risk	3.29	1.50	9.20	1.41	
Leptin (ng/ml)	Low Risk	1.13	0	5.20	1.41	0.003 ^{HS}
	Moderate Risk	4.11	0	9.70	2.52	
	High Risk	4.62	0.20	13.60	3.10	
Cholesterol (mmol/l)	Low Risk	4.21	3.28	5.63	0.62	0.477 ^{NS}
	Moderate Risk	4.09	3.07	5.80	0.68	
	High Risk	4.30	2.71	5.61	0.64	
TG (mmol/l)	Low Risk	1.19	0.21	5.30	1.23	0.000 ^{HS}
	Moderate Risk	2.63	0.45	6.49	1.68	
	High Risk	3.13	0.72	7.80	1.49	
LDL (mmol/l)	Low Risk	2.59	1.65	4.07	0.59	0.000 ^{HS}
	Moderate Risk	3.02	1.73	5.80	1.11	
	High Risk	3.58	1.52	6.00	1.08	
HDL (mmol/l)	Low Risk	1.55	0.31	2.31	0.44	0.000 ^{HS}
	Moderate Risk	1.17	0.06	1.99	0.45	
	High Risk	0.99	0.02	2.70	0.58	

The ANOVA one way test was used for the significance test.

HS: Highly significant ($P < 0.01$).

S: Significant ($P \leq 0.05$).

NS: Not significant ($P > 0.05$).

Max: Maximum, Min: Minimum.

Multiple Comparisons for CRP and Weight Measures

The Tamhane Post Hoc multiple comparisons test was used to compare the mean serum CRP concentration of each weight measure group to the respective control group. Using BMI (Table 4) there is a highly significant increase in the CRP levels for the overweight and obese groups in comparison to the control (healthy) group, For the WHR (Table 5) and the WC (Table 6) there is a significant increase for the high risk groups compared to the control (low) group. All other groups show no differences from the controls.

Multiple Comparisons for the Lipid Profile and Weight Measures

The Dunnett Post Hoc multiple comparisons test was used for the group comparisons (Table 2) for TG with the respective controls. For the BMI groups (Table 4), the underweight group shows a significant decrease, while the obese group shows a highly significant increase compared to the healthy group. For the WHR (Table 5) and WC (Table 6) both the

moderate and high risk groups show statistically significant increases from the respective controls.

The Tamhane Post Hoc multiple comparisons test was used for LDL group comparisons. The BMI groups (Table 4), the mean LDL level of the obese group is highly significantly higher compared to the control. As for the WHR (Table 5) and WC (Table 6), the high risk groups show highly significant increases from the respective controls. All other groups show no significant differences.

For multiple comparisons for HDL, the Dunnett Post Hoc multiple comparisons test was used for the BMI, while the Tamhane Post Hoc test was used for the WHR and WC. For the BMI groups (Table 4), the mean HDL level of the underweight group is highly significantly higher, while the level for the obese group is highly significantly lower compared to the mean level of the control. The HDL levels of the high risk groups for both WHR (Table 5) and WC (Table 6) and the moderate risk group of the WC are statistically significantly lower than the respective controls. The remaining groups show no differences.

Table 4: Multiple Comparisons between the Mean Concentrations for the Healthy Groups and the other BMI Groups

Parameter	Statistical test	BMI (Cat)	Mean Difference (H-Cat)	SE	P
CRP	Tamhane	Underweight	-2.69	7.00	0.999
		Overweight	-41.55	9.52	0.001 ^{HS}
		Obese	-86.59	14.01	0.000 ^{HS}
Adiponectin	Dunnett	Underweight	-0.30	0.48	0.875
		Overweight	0.39	0.39	0.650
		Obese	1.27	0.37	0.003 ^{HS}
Leptin	Tamhane	Underweight	1.26	0.40	0.019 ^S
		Overweight	-2.46	0.48	0.000 ^{HS}
		Obese	-3.84	0.71	0.000 ^{HS}
TG	Dunnett	Underweight	0.76	0.27	0.017 ^S
		Overweight	-0.24	0.26	0.685
		Obese	-2.92	0.24	0.000 ^{HS}
LDL	Tamhane	Underweight	-0.03	0.14	1.000
		Overweight	-0.06	0.15	0.999
		Obese	-1.89	0.17	0.000 ^{HS}
HDL	Dunnett	Underweight	-0.35	0.08	0.000 ^{HS}
		Overweight	0.06	0.08	0.794
		Obese	0.96	0.07	0.000 ^{HS}

The mean difference is significant against healthy weight subjects at the 0.05 level.

HS: Highly Significant ($P < 0.01$).

S: significant ($P \leq 0.05$).

H: Healthy, Cat: Category.

Table 5: Multiple Comparisons between the Mean Concentrations for the Low Risk Groups and the other WHR Groups

Parameter	Statistical Test	WHR (Cat)	Mean Difference (L - Cat)	SE	P
CRP	Tamhane	Moderate Risk	-26.23	11.39	0.087
		High Risk	-69.73	11.11	0.000 ^{HS}
Adiponectin	Dunnett	Moderate Risk	-0.93	0.40	0.041 ^S
		High Risk	-1.00	0.35	0.009 ^{HS}
Leptin	Tamhane	Moderate Risk	-2.49	0.52	0.014 ^S
		High Risk	-4.48	0.52	0.000 ^{HS}
TG	Dunnett	Moderate Risk	0.89	0.31	0.023 ^S
		High Risk	2.52	0.12	0.000 ^{HS}
LDL	Tamhane	Moderate Risk	-0.46	0.22	0.122
		High Risk	-1.33	0.20	0.000 ^{HS}
HDL	Tamhane	Moderate Risk	0.29	0.12	0.056
		High Risk	0.77	0.10	0.000 ^{HS}

The mean difference is significant against low risk subjects at the 0.05 level.

HS: Highly significant ($P < 0.01$).

S: significant ($P \leq 0.05$).

L: Low risk, Cat: Category.

Table 6: Multiple Comparisons between the WC Low Risk Group and other Groups

Parameter	Statistical test	WC (Cat)	Mean difference (L - Cat)	SE	P
CRP	Tamhane	Moderate Risk	-21.31	11.02	0.175
		High Risk	-43.81	11.92	0.002 ^{HS}
Adiponectin	Dunnett	Moderate Risk	-0.10	0.43	0.962
		High Risk	-0.89	0.36	0.029 ^S
Leptin	Tamhane	Moderate Risk	-2.98	0.60	0.000 ^{HS}
		High Risk	-3.50	0.52	0.000 ^{HS}
TG	Dunnett	Moderate Risk	1.44	0.40	0.001 ^{HS}
		High Risk	1.94	0.33	0.000 ^{HS}
LDL	Tamhane	Moderate Risk	-0.42	0.26	0.313
		High Risk	-0.99	0.19	0.000 ^{HS}
HDL	Tamhane	Moderate Risk	0.35	0.12	0.022 ^S
		High Risk	0.53	0.11	0.000 ^{HS}

The mean difference is significant against low risk subjects at the 0.05 level.

HS: Highly significant ($P < 0.01$).

S: significant ($P \leq 0.05$).

L: Low risk, Cat: Category.

Multiple Comparisons for Adiponectin and Weight Measures

The Dunnett Post Hoc test was used for the multiple comparisons between the BMI, WHR, and WC groups each and the respective control groups (Tables 4-6). Results show that there are highly significant decreases in the adiponectin concentrations means for the obese BMI group and the high risk WHR group, and significant decreases for the high risk WC group

and moderate risk WHR group. The other groups do not show any differences from the controls.

Multiple Comparisons for Leptin and Weight Measures

The Tamhane Post Hoc test was used for the multiple comparisons between the BMI, WHR, and WC groups and their respective controls for mean leptin levels (Tables 4-6). The results show that the mean

leptin concentration of the underweight BMI decrease significantly, while all other groups show significant increases (highly significantly for the overweight and obese BMI groups, high risk WHR and WC groups, and moderate WC group; and significantly for the moderate WC group).

DISCUSSION

The method that is most widely used and approved, by the Centers for Disease Control and Prevention and the American Academy of Pediatrics, for weight assessment in children and adolescents (ages 2 through 19 years) is the BMI-for-age percentiles, which are age- and sex-specific [29, 31, 32]. Other methods include the waist-to-hip ratio (WHR), and the waist circumference (WC). Only the WHR and WC indicate body shape and the location of fat and thus could differentiate between lower body obesity (pear or gynoid shape) and central or abdominal obesity (apple or android shape). Central obesity is associated with a higher amount of visceral, as opposed to subcutaneous, fat, which predisposes to diseases, health risks and an unfavorable lipid profile [31, 33-35].

The results show a highly significant increase of the CRP levels for the overweight and obese BMI, and the high risk WHR and WC groups compared to the respective controls. Studies by other scientists and in other populations have the same findings [3, 16-18, 36-38]. The increased levels of CRP in these overweight and obese adolescent girls confirms the findings of other researchers, in both adults and children, of a state of higher inflammation and a resultant increased risk for cardiovascular disease as adolescents get older since high CRP levels are strongly correlated to cardiovascular disease [14, 17, 37, 38].

The present results show a significant decrease in the adiponectin levels for the obese BMI, moderate and high risk WHR and high risk WC groups. In contrast, leptin levels are significantly higher in both overweight and obese BMI categories, and moderate and high risk WHR and WC groups, while the level in the underweight category is significantly lower than the control. Other researchers have also observed rising leptin levels [17, 39, 40] and decreasing adiponectin levels [17, 26, 40] in association with increased obesity in children. These levels have been suggested to be linked to increased obesity in both children and adults.

The study subjects' serum cholesterol concentrations did not show any association with obesity measured by the three measures of body

weight. This result is in agreement with previous results [41]. Compared to the control, serum TG levels are significantly higher in the obese BMI group and in both the moderate and high risk WHR and WC groups, while the level is significantly lower in the underweight WHR. LDL cholesterol levels increase for the obese BMI, and the high risk WHR and WC groups compared with the control group. In contrast, HDL levels are significantly lower for obese BMI, high risk WHR group, and both moderate and high risk WC groups, each compared to its respective control. On the other hand, the underweight BMI shows a higher HDL level compared to the control. This lipid profile indicates that obesity adversely affects the lipid profile, which leads to higher risk for cardiovascular disease adverse effect on the immune system. These results are in agreement with other researchers [14, 17, 38].

The observed unhealthy lipid profile in the study subjects along with the decreased adiponectin levels have also been observed by other researchers. Bacha and co-workers [26] have similar findings of decreased HDL in obese adolescents and a positive correlation with adiponectin. Weiss and coworkers [17, 39] have observed increased intramyocellular lipid accumulation in obese adolescents with low adiponectin levels and Zou and coworkers [40] have observed that (low) adiponectin levels in adolescents were positively associated with HDL-cholesterol levels.

Findings in the present study show that even at the young age of these healthy adolescents the markers for adverse effects of overweight and obesity on health and the immune system are present. The higher levels of CRP, a marker for inflammation, lower levels of adiponectin, an anti-inflammatory cytokine, both leading to a state of inflammation, along with an unfavorable lipid profile all lead to a higher risk of developing cardiovascular and other diseases in adulthood. In addition, the studied parameters affect immunity, thus explaining the findings, by other researchers, of reduced immunity and increased infections in overweight and obese adolescents. Therefore, it is recommended that more focus should be placed on the maintenance of healthy weight and weight loss for obese individuals through a healthy diet.

More research is needed to clarify the clinical implications of the changes in the immune system that are induced by overweight and obesity in adolescents in conjunction with studies on adolescent Saudi males for any sex-related differences.

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