Dietary Intake and Physical Activity in Relation to Insulin Resistance in Young Overweight Saudi Females: An Exploratory Pilot Study

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ABSTRACT: Insulin resistance is a major contributor to the development of several chronic metabolic diseases, including type 2 diabetes mellitus, and is an increasing health concern in Saudi Arabia. Diet and physical activity have been postulated to affect insulin resistance; however, their effects on development of insulin resistance in young overweight Saudi females have not been explored. Therefore, the aim of the study is to investigate whether diet and physical inactivity increases the risk of insulin resistance in young overweight Saudi females. In a cross-sectional study, 42 overweight female Saudi students, aged between 20 and 30 years, were recruited from King Abdul-Aziz University. A questionnaire was used to collect demographics, anthropometric measurements, physical activity, and food frequency data. Blood biomarkers (lipid profile, fasting glucose, and fasting insulin) were measured. Insulin resistance was assessed using homeostasis model assessment 2 (HOMA2)-insulin resistance (IR) scores. A significant difference in median body mass index (BMI) was observed between the HOMA2-IR normal and HOMA2-IR raised index groups (P=0.04). In terms of dietary habits, the insulin resistant group had a higher intake of canned beverages compared with the normal group (P=0.03). No significant differences were found between the groups in terms of waist circumference, hip circumference, waist-to-hip ratio, or body fat percentage. The lipid profile also did not significantly differ between the two groups. This study demonstrates significant differences in HOMA2-IR-defined insulin resistance according to subjects' BMI and canned beverage intake. A larger study is needed to confirm these associations.

Keywords: insulin resistant, HOMA-IR, dietary patterns, overweight, physical activity

INTRODUCTION

Insulin resistance is the central feature of several metabolic syndrome abnormalities, including glucose intolerance, dyslipidaemia, and hypertension (Grundy, 2016).

In Saudi Arabia, there is an increasing prevalence of insulin resistance-related disorders such as type II diabetes mellitus (Mira et al., 2002; Bahijri et al., 2010). According to the IDF Diabetes Atlas (2017), Saudi Arabia has one of the highest prevalence of diabetes mellitus in the world. Given the rapid rise in the rate of diabetes, early identification of insulin resistance could be utilized as a primary preventative strategy to reduce progression to clinical disease (Green et al., 2012).

Risk factors of insulin resistance include genetics and lifestyle factors, such as diet and physical inactivity (Roberts et al., 2013; Satija et al., 2016; Hu et al., 2001; Hanson et al., 2001). Consumption of fruit and vegetables (Spence et al., 2010), certain types of fish (Ikeda et al., 2018) and low-fat dairy products (Tremblay and Gilbert, 2009) has been inversely associated with insulin resistance, while intake of sugar-sweetened beverages and energy-dense foods, such as fast foods (Ma et al., 2016; Pereira et al., 2005) and saturated fatty acids (Koska et al., 2016), has been positively associated with insulin resistance. Moreover, it was reported that a sedentary lifestyle increases the chance of developing insulin resistance while physical activity helps decrease the risk of diabetes (Balkau et al., 2008). Although insulin resistance may be demonstrated in individuals without an increase in intra-abdominal fat or circulating markers of inflammation (Petersen et al., 2007), excess visceral adipose tissue

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is strongly correlated with insulin resistance (Bacha et al., 2006).

In laboratory and clinical studies, several methods are used to assess insulin resistance. The euglycemic hyperinsulinemic clamp (IS clamp) is considered the gold standard. However, use of the IS clamp is time-consuming, invasive and expensive, and requires an experienced clinician. The most common method to assess insulin resistance, especially among health care workers in Saudi Arabia, is the homeostasis model assessment (HOMA)insulin resistance (IR) (Bahijri et al., 2010). The main advantages of the HOMA-IR model are that it is cheap to carry out and only requires one blood draw from a fasting participant, which can be taken with minimal technical expertise. Although the HOMA-IR is widely used, its cutoff for insulin resistance has not been conclusively determined and may vary between people of different ethnicities (Hedblad et al., 2000; Añez et al., 2015; Esteghamati et al., 2010; Ghasemi et al., 2015; Tang et al., 2015; Timóteo et al., 2014; Tohidi et al., 2018; Yin et al., 2013).

In Saudi Arabia, few studies have used the HOMA as an indicator for insulin resistance; these studies have used cut-off thresholds derived from other study populations, such as Spain (Bahijri et al., 2010; Bahijri et al., 2018; Abdelkarem et al., 2016). In the current study, we categorized participants by HOMA index using the Iranian cut-off (Ghasemi et al., 2015) since, of the available population studies, Iranian dietary patterns are most similar to those of Saudi Arabia.

Factors that contribute to the development of insulin resistance and type 2 diabetes include being overweight or obese (Memish et al., 2014), poor dietary habits (Moradi-Lakeh et al., 2017), and low physical activity (Al-Hazzaa, 2018) are increasing among Saudi women. However, only a limited number of studies examine the dietary habits and physical activity levels in relation to the insulin resistance among Saudi women. Therefore, in this study we aimed to investigate whether diet and physical inactivity increase the risk of insulin resistance in young overweight Saudi women using the HOMA index as an indicator for insulin resistance. Prediction of insulin resistance risk in women of childbearing age is essential to prevent health issues including diabetes/gestational diabetes and to plan for early preventive strategies. We hypothesized that anthropometrics, dietary intake, and physical activity differ between normal and insulin resistance women.

MATERIALS AND METHODS

Study design and subjects

Utilizing a cross-sectional study design, 42 female participants aged 20 to 30 years old were recruited from the female campus (including both medical and non-medical students) at King Abdulaziz University, Jeddah, Saudi Arabia using direct advertising through the distribution of pamphlets. Participants with chronic conditions such as hypertension, endocrine disorders, diabetes, liver diseases, kidney diseases, respiratory disorders, and any type of cancer were excluded. Inclusion criteria included overweight Saudi adult females aged 20~30 years old. The aim and objectives of the study were explained to each participant and all gave informed written consent. Participants were asked to fill out a questionnaire consisting of five parts: demographics, anthropometrics, food intake frequency, laboratory measurements, and physical activity. Demographic information including name, date of birth, gender, and nationality were collected through a face-toface interview. Ethical approvals were obtained from the Unit of the Biomedical Ethics Research Committee at King Abdulaziz University (Reference No. 400-19).

Anthropometric measurements

Anthropometric measurements, including height, weight, waist circumference (WC), hip circumference (HC), waist-hip ratio (WHR), body mass index (BMI), and fat percentage were performed. Height, weight, WC, HC, and WHR was measured according to standard procedures (Lohman et al., 1988). Participants were weighed in light clothing and without shoes, and weight was recorded in kilograms (kg) using a calibrated digital scale to the nearest 0.1 kg. Height was measured to the nearest 0.1 cm according to the Frankfurt plane position. BMI was computed as a fraction of weight to height squared; the BMI cut-off for adults was considered. WC was measured at the narrowest point between the lowest costal border and the iliac crest and HC was measured at the widest part of the hip. WHR was calculated WC divided by HC. Fat percentage was measured via Bioelectrical Impedance Analysis. In this study, we considered the ethnicity-specific cut-off for Arabs and Europids: normal (BMI 18.5 \sim 24.9 kg/m²); overweight (BMI 25.0~29.9 kg/m²), and obese (BMI greater than 30 kg/m^2) (World Health Organization, 2000). In addition, we considered the optimal BMI, WC, and WHR cut-off values for predicting metabolic syndrome in Saudi women (28 kg/m², 87 cm, and 0.81, respectively) (Al-Rubean et al., 2017).

Dietary intake assessment

A self-administered food-frequency questionnaire (FFQ) to assess dietary intake was used (Alissa and Alama, 2015). For each food item or food group, subjects were asked the frequency of consumption (daily/weekly/ monthly) and the number of different portion sizes they had consumed over the past year. The estimation of macronutrients content (energy, carbohydrate, fat, protein, dietary fiber, cholesterol, saturated fatty acids, monoun-

saturated fatty acids, and polyunsaturated fatty acids) was performed by Prof. Alissa according to the analysis described (Alissa and Alama, 2015). Analysis of food group intake was performed according to Alkhaldy using Microsoft Excel (Alkhaldy, 2014). Food intake was grouped into food groups (cereals, breads, fruits, vegetables, legumes, nuts and seeds, meat and meat products, poultry, fish, egg, dairy products, fats and oils, canned beverages, and sweet).

Laboratory measurements

Blood samples were collected in the morning after an overnight fast of at least $8 \sim 12$ h. All blood samples were processed within 2 h. Serum and plasma extracted from samples were stored at -80° C for analysis of lipid profiles, fasting blood glucose, and fasting insulin level. Fasting glucose was assessed by an automated enzymatic method (Dade Behring Ltd., Milton Keynes, UK). Plasma insulin was measured via an electro-chemi-luminescence immunoassay. Insulin resistance was calculated using the HOMA models (HOMA-2) using fasting glucose and fasting insulin [fasting glucose: 3~25 mmol/L and fasting insulin: 2.88~43.16 LU/mL (20~300 pmol/ L)] by the HOMA calculator available from http://www. dtu.ox.ac.uk/homacalculator. Moreover, HOMA of b-cell function (HOMA-B) and HOMA of insulin sensitivity were also considered as they are reported to provide useful insights into glucose metabolism (Song et al., 2007; Caumo et al., 2006). Blood lipid profiles, including highdensity lipoprotein (HDL), low-density lipoprotein (LDL), total cholesterol, and triglycerides, were analysed using automated enzymatic methods (Dade Behring Ltd.). All measurements were performed at the Biochemistry Laboratory of the King Abdulaziz University Hospital.

Physical activity assessment

A self-reported physical activity questionnaire that included nine questions about weekly activities such as transport, household, fitness, and sports was used based on that previously reported by Al-Hazzaa and Al-Ahmidi (2003). The questionnaire had 4 categories depending on the participants' answers. The total score was measured as follows: very active (\geq 40 point), active (39~30 point), moderately active (29~20 point), and inactive (<20 point) (Al-Hazzaa and Al-Ahmidi, 2003).

Statistical analysis

Statistical analysis of all data was performed using Statistical Package for Social Sciences (SPSS) software, version 21.0 for Windows (SPSS Inc., Chicago, IL, USA). A normality test was run for all data to determine if data sets can be modelled by a normal distribution. Descriptive statistics were presented as medians with inter-quartile range (IQR). Comparisons between groups were performed using the Mann-Whitney test for non-normally distributed data and by paired two-sample *t*-tests for normally distributed data. Statistical significance was set a level of P<0.05.

RESULTS

Subjects characteristics

Forty-two female participants aged $20 \sim 30$ years old were recruited. The subjects' characteristics are presented in Table 1. Median BMI was 27.0 kg/m², which is within the cut-off range for being classified as overweight. The median WC, HC, and WHR were within the normal female range.

Participants were subdivided into normal and insulin resistance groups based on HOMA index measurements (HOMA2-IR) per the optimal insulin resistance cut off level determined in Iranian populations of 1.41 (Ghasemi et al., 2015). Using the HOMA2-IR index, 78.5% (n=33) of participants were within the optimal insulin resistance level (<1.41) and classified as normal, while 21% (n=9) had a high level of insulin resistance (>1.41) and were classified as insulin resistance. Table 2 presents differences in subject characteristics between the normal

| Table 1. Characteristics of study participants (n= | 42 | <u>'</u>) |
|--|----|------------|
|--|----|------------|

| Parameter | Median | IQR (Q1 \sim Q3) |
|----------------------------|----------|--------------------|
| Demographic | | |
| Age (yrs) | 21 | 20~22 |
| Marital status | | |
| Single | 39 (93%) | - |
| Married | 3 (7%) | _ |
| Arthrometric | | |
| Height (cm) | 158.0 | 152.7~162.3 |
| Weight (kg) | 67.6 | 64.1~74.4 |
| BMI (kg/m²) | 27.0 | 25.6~29.0 |
| WC (cm) | 78.0 | 76.0~81.6 |
| HC (cm) | 107.5 | 104.1~114.5 |
| WHR | 0.7 | 0.7~0.8 |
| Fat (%) | 29.9 | 26.7~34.2 |
| Blood profile | | |
| FG (mmol/L) | 4.5 | 4.1~4.8 |
| FSI (pmol/L) | 51.5 | 41.1~73.6 |
| HOMA2-IR | 0.9 | 0.7~1.4 |
| HOMA2-B | 120.7 | 101.8~161.8 |
| HOMA2-S (%) | 108.2 | 73.0~136.4 |
| Total cholesterol (mmol/L) | 4.1 | 3.7~4.9 |
| TG (mmol/L) | 0.7 | 0.5~0.9 |
| LDL-C (mmol/L) | 2.3 | 2.0~2.9 |
| HDL-C (mmol/L) | 1.4 | 1.2~1.6 |

IQR, inter-quartile range; BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist-hip ratio; Fat %, fat percentage; FG, fasting glucose; FSI, fasting serum insulin; HOMA2-IR, homeostasis model assessment 2-insulin resistance; TG, triglycerides; LDL-C, low-density lipoprotein-cholesterol; HDL-C, high-density lipoprotein-cholesterol.

| Parameter | | mal group f <1.41, n=33) | Insulin r (cut-o | <i>P</i> -value | | |
|--|--------|-----------------------------|---------------------|-----------------|--------|--|
| | Median | IQR (Q1~Q3) | Median | IQR (Q1~Q3) | _ | |
| Age (yrs) ¹⁾ | 21.0 | 20.0~22.0 | 20.0 | 20.0~21.0 | 0.09 | |
| Arthrometric | | | | | | |
| Height (cm) ¹⁾ | 159.0 | 152.6~163.0 | 155.5 | 154.0~159.0 | 0.4 | |
| Weight (kg) ¹⁾ | 67.0 | 64.3~74.0 | 72.0 | 64.0~75.0 | 0.4 | |
| $BMI (kg/m^2)^{1}$ | 26.9 | 25.5~28.1 | 29.5 | 26.6~30.0 | 0.049 | |
| WC (cm) ¹⁾ | 77.0 | 76.0~80.0 | 80.5 | 78.5~86.0 | 0.09 | |
| HC (cm) ¹⁾ | 107.0 | 104.0~111.5 | 115.0 | 107~116.5 | 0.15 | |
| WHR ¹⁾ | 0.7 | 0.7~0.8 | 0.8 | 0.7~0.8 | 0.29 | |
| Fat % ²⁾ | 29.1 | 26.5~32.6 | 34.2 | 29.7~36.0 | 0.10 | |
| Blood profile | | | | | | |
| FG (mmol/L) ¹⁾ | 4.5 | 4.1~4.8 | 4.3 | 4.2~4.5 | 0.61 | |
| FSI (pmol/L) ¹⁾ | 48.6 | 38.8~59.1 | 132,5 | 122,5~142,2 | <0.001 | |
| HOMA2-IR ¹⁾ | 0.8 | 0.7~1.1 | 2.2 | 2,1~2,5 | <0.001 | |
| HOMA2-B ¹⁾ | 112.7 | 95.2~130.6 | 222.5 | 205.5~247.6 | <0.001 | |
| HOMA2-S (%) ¹⁾ | 120.6 | 93.8~144.6 | 44.9 | 40.7~46.6 | <0.001 | |
| Total cholesterol (mmol/L) ²⁾ | 4.2 | 3.7~5.0 | 4.1 | 3.5~4.2 | 0.36 | |
| TG (mmol/L) ¹⁾ | 0.6 | 0.5~0.8 | 0.8 | 0.7~0.9 | 0.09 | |
| LDL-C (mmol/L) ²⁾ | 2.4 | 2.0~2.9 | 2.2 | 1.5~2.3 | 0.12 | |
| HDL-C (mmol/L) ¹⁾ | 1.4 | 1.2~1.6 | 1.4 | 1.3~1.5 | 0.79 | |

Table 2. Characteristics of study participants according to homeostasis model assessment 2 (HOMA2)-insulin resistance (IR) cut-off in the normal and insulin resistance groups

Data are presented as median (Q1 \sim Q3).

¹⁾*t*-tests.

²⁾Mann-Whitney test.

IQR, inter-quartile range; BMI, body mass index; WC, waist circumference; HC, hip circumference; WHR, waist-hip ratio; Fat %, fat percentage; FG, fasting glucose; FSI, fasting serum insulin; HOMA2-IR, homeostasis model assessment 2-insulin resistance; TG, triglycerides; LDL-C, low-density lipoprotein-cholesterol; HDL-C, high-density lipoprotein-cholesterol.

and insulin resistance groups. HOMA2-IR, HOMA-B, and HOMA-S significantly differed between groups (P < 0.001). In terms of the anthropometric measurements, the median BMI for the normal group (26.9 kg/m²) was less than the proposed values (28.0 kg/m²) for identifying risk of metabolic syndrome in Saudi women; the median value for the insulin resistance group (29.5 kg/m²) was above this proposed cut-off value, and significantly differed from that of the normal group (P=0.04). No sig-

nificant differences were found between the groups in terms of WC, HC, WHR, or body fat percentage. In addition, no correlation with the HOMA index was found for WHR (R^2 =0.0006) or the fat percentage (R^2 =0.02853) (data not shown).

Dietary intake assessment

No differences in median estimated energy, carbohydrate, fat, protein, dietary fiber, cholesterol, saturated fatty ac-

| Table 3. Estimated energy and macronutrient intake from | food-frequency questionnaire in the normal and insulin resistance groups |
|---|--|
|---|--|

| Nutrients | Overall (n=42) | | | ormal group .ff <1.41, n=33) | Insulin (cut- | <i>P</i> -value | |
|----------------------------------|----------------|-----------------|---------|---------------------------------|------------------|-----------------|------|
| | Median | IQR (Q1~Q3) | Median | IQR (Q1~Q3) | Median | IQR (Q1~Q3) | _ |
| Energy (kcal/d) ¹⁾ | 2,270.4 | 1,884.9~2,568.9 | 2,304.4 | 1,890.6~2,538.6 | 2,236.4 | 1,883.0~2,584.2 | 0.83 |
| Carbohydrate (g/d) ¹⁾ | 286.9 | 227.2~324.5 | 277.8 | 226.6~322.6 | 296.0 | 238.9~339.7 | 0.84 |
| Fat (g/d) ¹⁾ | 96.5 | 76.7~107.5 | 97.8 | 68.5~110.6 | 84.7 | 78.3~97.3 | 0.51 |
| Protein (g/d) ¹⁾ | 82.6 | 62.2~94.6 | 85.7 | 63.6~94.8 | 81.4 | 61.7~94.1 | 0.94 |
| Dietary fibre $(g/d)^{2}$ | 18.3 | 16.1~22.1 | 18.5 | 16.2~22.5 | 18.3 | 15.5~19.5 | 0.41 |
| Cholesterol (mg/d) ¹⁾ | 285.8 | 218.8~359.2 | 288.2 | 217.3~359.6 | 283.3 | 264.1~311.0 | 0.87 |
| SFA (g/d) ²⁾ | 31.8 | 26.1~36.9 | 34.2 | 25.1~37.3 | 30.6 | 27.6~34.2 | 0.43 |
| MUFA (g/)d ²⁾ | 32.8 | 26.0~37.4 | 33.6 | 22.5~37.5 | 29.1 | 27.7~37.3 | 0.81 |
| PUFA (g/d) ²⁾ | 17.3 | 14.4~21.5 | 17.5 | 14.4~24.4 | 17.1 | 15.1~21.2 | 0.76 |

¹⁾t-tests.

²⁾Mann-Whitney test.

IQR, inter-quartile range; SFA, saturated fatty acids; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.

ids, monounsaturated fatty acids, or polyunsaturated fatty acids intake were reported between the normal and insulin resistance groups (Table 3).

Further, there was no difference in median food intake between the normal and insulin resistance groups, with the exception of consumption of canned beverages, which was higher in the insulin resistance group (14.0 serving/ week, IQR 2.3~14.0) than the normal group (2.3 seving/week, IQR $0.3 \sim 4.0$) (*P*=0.03) (Table 4).

Physical activity assessment

The data from the physical activity questionnaire classified 79% of the participants as inactive, 19% as moderately active, and 2% as active; no subjects (0%) were classed as very active. There were no significant differences in physical activity levels between the two groups (Table 5).

DISCUSSION

This study was conducted to test the hypothesis that di-

etary intake and physical inactivity may increase the risk of insulin resistance in overweight Saudi adult females. Although numerous factors have been associated with insulin resistance, our study only reported an association with BMI and consumption of canned beverages.

Based on the anthropometric data measured in this study, we found participants in the insulin resistance group had a significantly higher BMI than those in the normal group (P=0.04). Although the median BMI for both groups was within the overweight range, the median BMI for the HOMA-IR "normal" group was in the bottom half of the range (26.9 kg/m^2) and the median BMI for the insulin resistance group was close to the overweight-obese boundary (29.5 kg/m²). In addition, the median BMI for the HOMA-IR normal group was less than the proposed value for identifying the risk of metabolic syndrome (28.0 kg/m²), whereas the median BMI of the insulin resistance group was above the proposed value. The results of our study support the work by Geloneze et al. (2009), who reported a parallel increase in HOMA indexes and BMI. The mechanism behind this

Table 4. Estimation of dietary habits (serving/week) in the normal (n=33) and insulin resistant (n=9) groups, as assessed by food-frequency questionnaire

| Food items | Overall (n=42) d items | | | Normal group (cut-off <1.41, n=33) | | Insulin resistance group (cut-off >1.41, n=9) | | |
|--------------------------------|---------------------------|--------------------|--------|---------------------------------------|--------|--|------|--|
| | Median | IQR (Q1~Q3) | Median | IQR (Q1~Q3) | Median | IQR (Q1~Q3) | | |
| Cereals | 4.8 | 2.9 (10.9~8.0) | 4.9 | 8.4 (3.2~11.6) | 3.2 | 5.4 (2.6~8.0) | 0.20 | |
| Breads | 11.3 | 6.375 (21.5~15.1) | 11.3 | 15 (6.6~21.6) | 9.6 | 8.6 (6.3~14.9) | 0.66 | |
| Fruits | 12.0 | 6.7 (26.6~19.9) | 13.5 | 20.2 (6.6~26.8) | 10.3 | 7.2 (9.8~17.0) | 0.45 | |
| Vegetables | 21.3 | 11.55 (36.6~25.1) | 22.2 | 26.1 (10.7~36.8) | 18.5 | 11.1 (15.3~26.4) | 0.79 | |
| Legumes | 2.0 | 0.9 (4.3~3.4) | 2.3 | 3.4 (1.2~4.6) | 1.2 | 1.4 (0.6~2.0) | 0.19 | |
| Nuts and seeds | 1.2 | 0.6 (4.5~3.9) | 1.2 | 4.0 (0.6~4.6) | 2.9 | 3.1 (0.9~4.0) | 0.34 | |
| Meat and meat products | 2.3 | 0.9 (4.8~3.9) | 2.3 | 3.4 (0.9~4.3) | 2.3 | 2.9 (2.0~4.9) | 0.53 | |
| Poultry | 2.0 | 2.0 (7.0~5.0) | 2.0 | 5.0 (2.0~7.0) | 2.0 | 5.0 (2.0~7.0) | 0.72 | |
| Fish | 0.6 | 0.6 (2.3~1.7) | 0.6 | 1.7 (0.6~2.3) | 0.6 | 1.7 (0.6~2.3) | 0.94 | |
| Egg | 2.3 | 2.075 (6.5~4.4) | 2.3 | 5.3 (2.0~7.3) | 2.3 | 1.7 (2.3~4.0) | 0.96 | |
| Dairy products | 26.8 | 16.075 (39.2~23.2) | 28.9 | 24.7 (16.3~41) | 24.6 | 20.7 (10.5~31.2) | 0.40 | |
| Fats and oils | 12.3 | 6.7 (21.2~14.5) | 13.0 | 14.7 (6.6~21.3) | 11.3 | 8.1 (7.9~16) | 0.97 | |
| Fresh juices | 0.3 | 0.3 (2.0~1.7) | 0.3 | 1.7 (0.3~2.0) | 2.0 | 1.7 (0.3~2.0) | 0.24 | |
| Canned beverages ¹⁾ | 2.3 | 0.375 (7.3~6.9) | 2.3 | 3.7 (0.3~4.0) | 14.0 | 11.7 (2.3~14.0) | 0.03 | |
| Sweet ²⁾ | 5.8 | 2.375 (9.8~7.4) | 4.9 | 7 (2.3~9.3) | 7.0 | 18.7 (4.6~23.3) | 0.22 | |

¹⁾Canned juices and soda.

²⁾Cream caramel, custard, chocolate, and toffee.

IQR, inter-quartile range.

Table 5. Physical activity in the homeostasis model assessment 2-insulin resistance in the normal and insulin resistance groups

| Physical activity | Overall | Overall (n=42) | | Normal group (cut-off <1.41, n=33) | | Insulin resistance group (Cut-off >1.41, n=9) | |
|--|---------|----------------|---------|---------------------------------------|--------|--|-------|
| | n | % | n | % | n | % | |
| Very active (>40 score) | 0 | 0 | 0 | 0 | 0 | 0 | |
| Active (30 \sim 39 score) | 1 | 2 | 1 | 3 | 0 | 0 | 0.083 |
| Moderate active (20 \sim 29 score) Inactive (<20 score) | 8 33 | 19 79 | 8 24 | 24 72 | 0 9 | 0 100 | |

could be relate to an increased amount of non-esterified fatty acids present in circulation (Heptulla et al., 2001) and inflammatory mediators triggered by adipose tissues (Hajer et al., 2008). The remaining anthropometric indicators of being overweight, such as WC, HC, WHR, and body fat percentage, were not significantly correlated with the HOMA2-IR index. Several studies have reported a positive relationship between WHR, WC, and fat percentage with insulin resistance (Wahrenberg et al., 2005; Patel and Abate, 2013; Keswell et al., 2016); however, we could not detect any difference in our study, which could be influenced by the indirect measurements we used. Carey et al. (1996) suggested the relationship between insulin resistance and central abdominal fat in non-obese women cannot be found using indirect traditional anthropometry measurements and that these measurements may be no better than estimates of total fat for predicting insulin sensitivity and central obesity. However, results from direct measurements of regional fat and insulin sensitivity [such as by dual-energy X-ray absorptiometry (DEXA) and an euglycemic clamp or oral glucose tolerance tests, respectively] are significantly correlated with insulin resistance in overweight and normal women (Carey et al., 1996). Furthermore, although the median fat percentage of the insulin resistance group was higher than that of the normal group (29.1% vs. 34.2%), it should be noted that fat percentage reflects whole-body fat and does not specifically measure central obesity, which is linked to insulin resistance and risk of metabolic syndrome.

The HOMA2-IR cut-off used in our study was determined based on an Iranian prospective study which included nine years of follow-up of reporting incidences of diabetes; this study classified 1.41 and 1.7 HOMA2-IR cut-off values as optimal and having insulin resistance, respectively. However, there is a need to determine the cut-off points for diagnosing insulin resistance in the Saudi population according BMI.

The second significant factor identified in our study was related to dietary intake. Participants in the insulin resistance group consumed significantly more canned beverages than those in the normal group. Longitudinal studies have reported that consumption of sugar-sweetened fruit juice, but not 100% fruit juice, is linked with diabetes incidence. The reason 100% fruit juice is not linked to diabetes incidence could be due to its other beneficial components, such as dietary fiber, vitamins, and phytochemicals, which could offset any harm (Imamura et al., 2015).

The FFQ used in this study was not exhaustive and thus not all food items that may be commonly consumed by younger adults could be included (such as fast food meals). During a cross-sectional study, Hsieh et al. (2014) observed that higher insulin resistance was associated with greater access to fast food restaurants. As the FFQ used in this study did not include fast food restaurant meals, we cannot comment on this correlation.

In the current study, we reported that physical activity levels were low in both groups, and that most normal participants (72%) and all insulin resistance participants were scored as inactive according to the Physical Activity Questionnaire, validated by King Saud University. This may be contributed to by a lack of public walking areas for females, and weather in the Jeddah region. Previous studies have reported an inverse association between risk of insulin resistance as measured by HOMA2-index and physical activity levels (Jiménez-Pavón et al., 2013; Byrkjeland et al., 2014). In addition, a study conducted on overweight adolescents reported a significant increase in the risk of insulin resistance in relation to sedentary behaviors (i.e., watching television for at least three hours daily) and increased visceral fats (which are linked to WC) (Velásquez-Rodríguez et al., 2014). The results of our study did not detect a significant association between physical activity and HOMA2-IR; however, it is noted that all individuals in the insulin resistance group had a score in the lowest category for physical activity (score of <20; inactive); therefore, a larger sample size may be required to gain the statistical power needed to examine this factor in more detail. Studies in Saudi Arabia have reported that females are much more inactive than males, which could be due to social and cultural factors. For example, females have fewer opportunities than males to be involved in outdoor leisure activities. Additionally, there are fewer fitness centers available for females and those that are available are often more expensive (Al-Eisa and Al-Sobayel, 2012). Furthermore, in Saudi Arabia may people rely of cars rather than walking for short-distance and trips (Al-Hazzaa, 2004).

In this study, we did not detect any significant differences in lipid profiles between the two groups, which could be due to the small number of participants limiting the statistical power. Studies have reported that plasma triglyceride, high density lipoprotein cholesterol, and total cholesterol are independently associated with insulin resistance and are predictors of cardiovascular diseases (McLaughlin et al., 2003; Jeppesen et al., 1998). This is the first pilot study specifically investigating young overweight Saudi females that assesses the relationship of nutritional status and physical activity with insulin resistance. Furthermore, the combination of nutritional assessments and blood biomarkers makes this study unique. Although this study had a limited sample size, the data generated could inform the design and sample size calculation of larger follow-up studies. Further studies need to consider using direct measurement (such as DEXA) in combination with the indirect anthropometric measurements to measure the visceral and subcutaneous fat.

Although these techniques are more time-consuming and technically difficult, they should be considered for the benefits the results will provide for establishing the data for people in Saudi Arabia. In addition, elevated plasma free fatty acids have been previously associated with insulin resistance (Kashyap et al., 2003), therefore measurement of the plasma free fatty acid concentration as well as inflammatory biomarkers should be considered for future studies.

This study showed that BMI and a high intake of canned beverages are associated with HOMA2-IR in young overweight Saudi females. These factors could contribute to the increase in prevalence of insulin resistance and chronic diseases in the Saudi population. Further prospective studies of larger sample sizes, focusing on Saudi lifestyle factors, are needed to establish a HOMA2-IR cut-off specific to the Saudi population. Such knowledge and awareness could help reduce the prevalence of insulin resistance.

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AUTHOR DISCLOSURE STATEMENT

The authors declare no conflict of interest.

REFERENCES

- Abdelkarem HM, El-Sherif MA, Gomaa SB. Vitamin D status and insulin resistance among young obese Saudi females. Saudi Med J. 2016. 37:561-566.
- Al-Eisa ES, Al-Sobayel HI. Physical activity and health beliefs among Saudi women. J Nutr Metab. 2012. 2012:642187.
- Al-Hazzaa H, Al-Ahmidi M. A self-reported questionnaire for the assessment of physical activity in youth 15~25 years: development, reliability and construct validity. Arab J Food Nutr. 2003. 4:279-291.
- Al-Hazzaa HM. Physical inactivity in Saudi Arabia revisited: a systematic review of inactivity prevalence and perceived barriers to active living. Int J Health Sci. 2018. 12:50-64.
- Al-Hazzaa HM. The public health burden of physical inactivity in Saudi Arabia. J Family Community Med. 2004. 11:45-51.
- Alissa EM, Alama NA. Nutritional intake and cardiovascular risk factors in Saudi subjects with different degrees of atherosclerosis: a case control study. J Nutri Med Diet Care. 2015. 1:008.
- Alkhaldy A. Inter-individual variability of polyphenol metabolism and colonic health. Dissertation. University of Glasgow, Glasgow, Scotland. 2014.

- Al-Rubean K, Youssef AM, Al Farsi Y, Al-Sharqawi AH, Bawazeer N, Al Otaibi MT, et al. Anthropometric cutoff values for predicting metabolic syndrome in a Saudi community: from the SAUDI-DM study. Ann Saudi Med. 2017. 37:21-30.
- Añez R, Morillo J, Rojas M, Torres Y, Apruzzese V, Martínez MS, et al. Homeostasis model assessment (HOMA-IR) cut-off point for insulin resistance in adults from Maracaibo municipality-Zulia State, Venezuela. Avan Biomed. 2015. 4:9-18.
- Bacha F, Saad R, Gungor N, Arslanian SA. Are obesity-related metabolic risk factors modulated by the degree of insulin resistance in adolescents?. Diabetes Care. 2006. 29:1599-1604.
- Bahijri SM, Ajabnoor G, Hegazy GA, Alsheikh L, Moumena MZ, Bashanfar BM, et al. Supplementation with oligonol, prevents weight gain and improves lipid profile in overweight and obese saudi females. Curr Nutr Food Sci. 2018. 14:164-170.
- Bahijri SM, Alissa EM, Akbar DH, Ghabrah TM. Estimation of insulin resistance in non-diabetic normotensive Saudi adults by QUICKI, HOMA-IR and modified QUICKI: a comparative study. Ann Saudi Med. 2010. 30:257-264.
- Balkau B, Mhamdi L, Oppert JM, Nolan J, Golay A, Porcellati F, et al. Physical activity and insulin sensitivity: the RISC study. Diabetes. 2008. 57:2613-2618.
- Byrkjeland R, Edvardsen E, Njerve IU, Arnesen H, Seljeflot I, Solheim S. Insulin levels and HOMA index are associated with exercise capacity in patients with type 2 diabetes and coronary artery disease. Diabetol Metab Syndr. 2014. 6:36.
- Carey DG, Jenkins AB, Campbell LV, Freund J, Chisholm DJ. Abdominal fat and insulin resistance in normal and overweight women: direct measurements reveal a strong relationship in subjects at both low and high risk of NIDDM. Diabetes. 1996. 45:633-638.
- Caumo A, Perseghin G, Brunani A, Luzi L. New insights on the simultaneous assessment of insulin sensitivity and β -cell function with the HOMA2 method. Diabetes Care. 2006. 29:2733-2734.
- Esteghamati A, Ashraf H, Khalilzadeh O, Zandieh A, Nakhjavani M, Rashidi A, et al. Optimal cut-off of homeostasis model assessment of insulin resistance (HOMA-IR) for the diagnosis of metabolic syndrome: third national surveillance of risk factors of non-communicable diseases in Iran (SuRFNCD-2007). Nutr Metab. 2010. 7:26.
- Geloneze B, Vasques AC, Stabe CF, Pareja JC, Rosado LE, Queiroz EC, et al. HOMA1-IR and HOMA2-IR indexes in identifying insulin resistance and metabolic syndrome: Brazilian Metabolic Syndrome Study (BRAMS). Arq Bras Endocrinol Metabol. 2009. 53:281-287.
- Ghasemi A, Tohidi M, Derakhshan A, Hasheminia M, Azizi F, Hadaegh F. Cut-off points of homeostasis model assessment of insulin resistance, beta-cell function, and fasting serum insulin to identify future type 2 diabetes: Tehran Lipid and Glucose Study. Acta Diabetol. 2015. 52:905-915.
- Green LW, Brancati FL, Albright A; Primary Prevention of Diabetes Working Group. Primary prevention of type 2 diabetes: integrative public health and primary care opportunities, challenges and strategies. Fam Pract. 2012. 29:i13-i23.
- Grundy SM. Metabolic syndrome update. Trends Cardiovasc Med. 2016. 26:364-373.
- Hajer GR, van Haeften TW, Visseren FL. Adipose tissue dysfunction in obesity, diabetes, and vascular diseases. Eur Heart J. 2008. 29:2959-2971.
- Hanson RL, Imperatore G, Narayan KM, Roumain J, Fagot-Campagna A, Pettitt DJ, et al. Family and genetic studies of indices of insulin sensitivity and insulin secretion in Pima Indians. Diabetes Metab Res Rev. 2001. 17:296-303.
- Hedblad B, Nilsson P, Janzon L, Berglund G. Relation between insulin resistance and carotid intima-media thickness and stenosis in non-diabetic subjects. Results from a cross-sectional

study in Malmö, Sweden. Diabet Med. 2000. 17:299-307.

- Heptulla R, Smitten A, Teague B, Tamborlane WV, Ma YZ, Caprio S. Temporal patterns of circulating leptin levels in lean and obese adolescents: relationships to insulin, growth hormone, and free fatty acids rhythmicity. J Clin Endocrinol Metab. 2001. 86:90-96.
- Hsieh S, Klassen AC, Curriero FC, Caulfield LE, Cheskin LJ, Davis JN, et al. Fast-food restaurants, park access, and insulin resistance among Hispanic youth. Am J Prev Med. 2014. 46:378-387.
- Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, et al. Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. N Engl J Med. 2001. 345:790-797.
- IDF Diabetes Atlas. 8th ed. 2017. [cited 2019 Jan 2]. Available from: https://diabetesatlas.org/en/resources/
- Ikeda K, Sato T, Nakayama T, Tanaka D, Nagashima K, Mano F, et al. Dietary habits associated with reduced insulin resistance: The Nagahama study. Diabetes Res Clin Pract. 2018. 141:26-34.
- Imamura F, O'Connor L, Ye Z, Mursu J, Hayashino Y, Bhupathiraju SN, et al. Consumption of sugar sweetened beverages, artificially sweetened beverages, and fruit juice and incidence of type 2 diabetes: systematic review, meta-analysis, and estimation of population attributable fraction. BMJ. 2015. 351:h3576.
- Jeppesen J, Facchini FS, Reaven GM. Individuals with high total cholesterol/HDL cholesterol ratios are insulin resistant. J Intern Med. 1998. 243:293-298.
- Jiménez-Pavón D, Sesé MA, Huybrechts I, Cuenca-García M, Palacios G, Ruiz JR, et al. Dietary and lifestyle quality indices with/without physical activity and markers of insulin resistance in European adolescents: the HELENA study. Br J Nutr. 2013. 110:1919-1925.
- Kashyap S, Belfort R, Gastaldelli A, Pratipanawatr T, Berria R, Pratipanawatr W, et al. A sustained increase in plasma free fatty acids impairs insulin secretion in nondiabetic subjects genetically predisposed to develop type 2 diabetes. Diabetes. 2003. 52:2461-2474.
- Keswell D, Tootla M, Goedecke JH. Associations between body fat distribution, insulin resistance and dyslipidaemia in black and white South African women. Cardiovasc J Afr. 2016. 27: 177-183.
- Koska J, Ozias MK, Deer J, Kurtz J, Salbe AD, Harman SM, et al. A human model of dietary saturated fatty acid induced insulin resistance. Metabolism. 2016. 65:1621-1628.
- Lohman TG, Roche AF, Martorell R. Anthropometric standardization reference manual. Human Kinetics Books, Champaign, IL, USA. 1988. p 3, 39, 125.
- Ma J, Jacques PF, Meigs JB, Fox CS, Rogers GT, Smith CE, et al. Sugar-sweetened beverage but not diet soda consumption is positively associated with progression of insulin resistance and prediabetes. J Nutr. 2016. 146:2544-2550.
- McLaughlin T, Abbasi F, Cheal K, Chu J, Lamendola C, Reaven G. Use of metabolic markers to identify overweight individuals who are insulin resistant. Ann Intern Med. 2003. 139:802-809.
- Memish ZA, El Bcheraoui C, Tuffaha M, Robinson M, Daoud F, Jaber S, et al. Obesity and associated factors—Kingdom of Saudi Arabia, 2013. Prev Chronic Dis. 2014. 11:E174.
- Mira SA, Akbar DH, Hashim IA, Salamah SH, Zawawi TH. The insulin resistance syndrome among type II diabetics. Saudi Med J. 2002. 23:1045-1048.

- Moradi-Lakeh M, El Bcheraoui C, Afshin A, Daoud F, AlMazroa MA, Al Saeedi M, et al. Diet in Saudi Arabia: findings from a nationally representative survey. Public Health Nutr. 2017. 20:1075-1081.
- Patel P, Abate N. Body fat distribution and insulin resistance. Nutrients. 2013. 5:2019-2027.
- Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR Jr, et al. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. Lancet. 2005. 365:36-42.
- Petersen KF, Dufour S, Savage DB, Bilz S, Solomon G, Yonemitsu S, et al. The role of skeletal muscle insulin resistance in the pathogenesis of the metabolic syndrome. Proc Natl Acad Sci USA. 2007. 104:12587-12594.
- Roberts CK, Hevener AL, Barnard RJ. Metabolic syndrome and insulin resistance: underlying causes and modification by exercise training. Compr Physiol. 2013. 3:1-58.
- Satija A, Bhupathiraju SN, Rimm EB, Spiegelman D, Chiuve SE, Borgi L, et al. Plant-based dietary patterns and incidence of type 2 diabetes in US men and women: results from three prospective cohort studies. PLoS Med. 2016. 13:e1002039.
- Song Y, Manson JE, Tinker L, Howard BV, Kuller LH, Nathan L, et al. Insulin sensitivity and insulin secretion determined by homeostasis model assessment and risk of diabetes in a multiethnic cohort of women: the Women's Health Initiative Observational Study. Diabetes Care. 2007. 30:1747-1752.
- Spence M, McKinley MC, Hunter SJ. CVD, diabetes and cancer: diet, insulin resistance and diabetes: the right (pro)portions. Proc Nutr Soc. 2010. 69:61-69.
- Tang Q, Li X, Song P, Xu L. Optimal cut-off values for the homeostasis model assessment of insulin resistance (HOMA-IR) and pre-diabetes screening: developments in research and prospects for the future. Drug Discov Ther. 2015. 9:380-385.
- Timóteo AT, Miranda F, Carmo MM, Ferreira RC. Optimal cutoff value for homeostasis model assessment (HOMA) index of insulin-resistance in a population of patients admitted electively in a Portuguese cardiology ward. Acta Med Port. 2014. 27: 473-479.
- Tohidi M, Baghbani-Oskouei A, Ahanchi NS, Azizi F, Hadaegh F. Fasting plasma glucose is a stronger predictor of diabetes than triglyceride-glucose index, triglycerides/high-density lipoprotein cholesterol, and homeostasis model assessment of insulin resistance: Tehran Lipid and Glucose Study. Acta Diabetol. 2018. 55:1067-1074.
- Tremblay A, Gilbert JA. Milk products, insulin resistance syndrome and type 2 diabetes. J Am Coll Nutr. 2009.28:91S-102S.
- Velásquez-Rodríguez CM, Velásquez-Villa M, Gómez-Ocampo L, Bermúdez-Cardona J. Abdominal obesity and low physical activity are associated with insulin resistance in overweight adolescents: a cross-sectional study. BMC Pediatrics. 2014. 14:258.
- Wahrenberg H, Hertel K, Leijonhufvud BM, Persson LG, Toft E, Arner P. Use of waist circumference to predict insulin resistance: retrospective study. BMJ. 2005. 330:1363-1364.
- World Health Organization. Obesity: preventing and managing the global epidemic: report of a WHO consultation. WHO Technical Report Series 894. 2000. p 9.
- Yin J, Li M, Xu L, Wang Y, Cheng H, Zhao X, et al. Insulin resistance determined by homeostasis model assessment (HOMA) and associations with metabolic syndrome among Chinese children and teenagers. Diabetol Metab Syndr. 2013. 5:71.