Global Journal of Health Science; Vol. 9, No. 1; 2017
ISSN 1916-9736 E-ISSN 1916-9744
Published by Canadian Center of Science and Education

# Metabolic, Anthropometric, and Type 2 Diabetes Mellitus Related Risk Factors in Normal and Pre-Diabetic Adults

Mohamed H. Al-Thani[1], Heba S. Nasser [1], Suzan Sayegh[1], Alexandra Haddad[1] & Eman Sadoun[2]

[1] Public Health Division, Ministry of Public Health, Doha, Qatar

[2] Clinical Research Division, Ministry of Public Health, Doha, Qatar

Correspondence: Mohamed H. Al-Thani, Ph.D., Director of Public Health, Public Health Department, Ministry of Public Health, P.O. Box 42, Doha, Qatar. E-mail: malthani@moph.gov.qa

Received: March 17, 2016 Accepted: May 26, 2016 Online Published: June 15, 2016

doi:10.5539/gjhs.v9n1p224 URL: http://dx.doi.org/10.5539/gjhs.v9n1p224

**Abstract**

Type 2 diabetes mellitus (T2DM) is a major global health problem. The present study examines the relationship between the metabolic, anthropometric and Finnish risk score (FINDRISC) among normal and pre-diabetic adults. Subjects (n = 1319, aged above 18 years) from the Qatari population were classified into two groups based on their hemoglobin $A_{1c}$ ($HbA_{1c}$) measurements (non-diabetic $A_{1c}<5.6\%$ and pre-diabetic $5.6\% \leq A_{1c} \leq 6.4\%$) were examined for their anthropometric (height, weight and waist circumference), metabolic [fat, fat free mass (FFM), muscle mass (MM), total body water (TBW), bone mass, degree of obesity, basal metabolic rate (BMR), body mass index (BMI), metabolic age, visceral fat rating, systolic blood pressure (SBP), diastolic blood pressure (DBP), total cholesterol (Total-C), low-density lipoprotein (LDL), high-density lipoprotein (HDL), triglycerides (TG), fasting / random plasma glucose (FPG/RPG), $HbA_{1c}$ and vitamin D (VitD)] and FINDRISC. Means and frequencies were determined in aggregate and by subgroups for all variables and correlations between categorical variables were tested to estimate the association between the anthropometric and metabolic risk factors with the FINDRISC. A percentage of 74.8% (n = 987) of the study population aged below 45 years old and their overall BMI was 28.8±5.2 kg/m$^2$ (overweight). Pre-diabetic subgroup have shown a statistically higher FINDRISC compared to their non-diabetic counterparts (11.2±4.1 vs. 9.8±4, $p<0.001$). The FINDRISC was significantly and directly correlated with the BMI, $HbA_{1c}$ and FPG. However, $HbA_{1c}$ was correlated directly with BMI, SBP, DBP, FPG/RPG and indirectly with the levels of HDL. This study demonstrates an apparent relationship between the $HbA_{1c}$ and FINDRISC score. Pursuing further research on this association may permit using $HbA_{1c}$ with the FINDRISC in predicting the risk of T2DM to be a better tool rather than using the current FPG/RPG, OGTT methods.

**Keywords:** Type II Diabetes Mellitus, pre-diabetes, metabolic risk factors

## 1. Introduction

Type 2 diabetes mellitus (T2DM) is now a global health concern and is one of the main chronic diseases influencing people despite their geographic location and socio-economic status. T2DM has been rising dramatically over the last few decades and is currently identified as a leading cause of mortality worldwide (Wild, Rolic, & Green, 2004). The association between T2DM and increased mortality and morbidity is due to its presence as a key predisposing factor for cardiovascular disease, stroke and other life-threatening complications. Factors contributing to the progression of T2DM are mainly related to genes and the environment. However, T2DM prevalence is found to increase with age and other behaviors including and related to obesity, sedentary lifestyle, physical inactivity, and unhealthy dietary habits (Mokdad et al., 2001; Venkataraman et al., 2004). As incidence of T2DM continue to rise globally, it is critical to maintain early prediction for the risk of the disease in order to avoid or control it.

T2DM is mainly caused by impaired glucose intolerance (IGT) which is a result of insulin resistance and islet β-cell exhaustion (Stumvoll, Goldstein, & Haeften, 2005). It is a metabolic disorder with heterogeneous etiologies mostly characterized by chronic hyperglycemia and disturbances of carbohydrate, fat and protein metabolism. Other factors leading to T2DM include hypertension, hyperglycemia, obesity and dyslipidemia (Dandona, Aljada, & Bandyopadhyay, 2004). Dyslipidemia triggers increased levels of triglyceride (TG) and fatty acids either from diet or from accelerated lipolysis, the exposure of muscle cells to fatty acids causes an

impairment of insulin-mediated glucose uptake and consequently contributes to insulin resistance (Dimopoulos et al., 2006; Bilam et al., 2009). In other words, insulin resistance, accompanied by impaired fasting plasma glucose (IFG) levels, lead to an increase in the levels of low density lipoprotein (LDL) and TG, and a decrease in levels of high density lipoprotein (HDL) cholesterol in the body (Sorrentino, 2005).

The leading risk factor for T2DM is the "Pre-diabetes" stage which is identified by hemoglobin $A_{1c}$ ($HbA_{1c}$) levels of a range between 5.6% to 6.4% (American Diabetes Association, 2010). This stage is characterized by the elevated blood glucose level (accumulated over the previous 2-3 months) which is above the normal range but below that of clinical diabetes. This "pre-diabetic" stage is just an indication of the risk of getting the disease and could be altered to the normal stage (<5.6%) if a healthier lifestyle and better control of the sugar intake is adopted.

An International Expert Committee was convoked in 2008 by the European Association for the Study of Diabetes, the American Diabetes Association (ADA), and the International Diabetes Federation to consider the means for diagnosing diabetes in non-pregnant individuals using $HbA_{1c}$ as an alternative, if not superior better, tool (International Expert Committee, 2009). Diabetes has been diagnosed for decades with fasting/random plasma glucose (FPG/RPG) assessment or, much less frequently, with an oral glucose tolerance test (OGTT). The measurement of $HbA_{1c}$ equals the assessment of hundreds (virtually thousands) of FPG and also captures postprandial glucose peaks; therefore, it is a more robust measurement than FPG and/or 2hr OGTT plasma glucose (Bonora & Tuomilehto, 2011).

A well-known screening tool to predict the risk of developing T2DM is the Finnish risk score (FINDRISC) questionnaire (www.diabetes.fi/files/502/eRiskitestilomake.pdf) which gives an estimate number (0-26 points) that identifies a 72% (Saaristo et al., 2005) risk of developing T2DM within the next 10 years (Lindstorm & Tuomilehto, 2003). It includes eight questions about age, body mass index (BMI), waist circumference, physical activity, dietary consumption of fruits, vegetables and berries, as well as history of antihypertensive medications, history of high blood glucose, and family history of diabetes. The total test score provides a measure of the probable development of T2DM among participants, where a score of 15 and above indicates a high risk. Therefore, both $HbA_{1c}$ and FINDRISC can identify the biomarker features that may assist in the early prediction of T2DM and assist in future control of the disease. Eventually, interweaving $HbA_{1c}$ in the FINDRISC will improve the power of the conventional tool and provide a more accurate predictor of developing the disease. This cross-sectional study was conducted consequently to prove the same.

## 2. Methods

### *2.1 Study Design and Population*

This cross-sectional study was conducted to explore the relationship between metabolic, anthropometric, and T2DM-related risk factor profiles in normal and pre-diabetic stages in a Qatari population and their link to the early prediction of the disease as well as the profile of vitamin D serum levels in study subjects. Screening campaigns were held in different regions in Qatar for a period of six months; three researchers were trained and certified to work on participant data. Consequently, we finalized the statistical evaluation of the metabolic, anthropometric and T2DM related risk factors from 1,319 participants excluding diabetic patients. No definite sample size was planned as it was a cross-sectional study. The inclusion criteria were Qatari residents (both males and females), above 18 years of age, and with no history of diabetes. Participants excluded were those already diagnosed with diabetes, gestational diabetes, or those whose $HbA_{1c}$ levels were higher than 6.4%, in addition to pregnant and nursing women (due to hormone imbalance). Those classified as diabetics had their $HbA_{1c}$ measured twice to confirm. The primary selection criteria were the $HbA_{1c}$ levels and those were limited to individuals with less than 6.5%. Accordingly, participants were classified based on these $HbA_{1c}$ measurements into two groups: the non-diabetics ($A_{1c}$<5.6%) and pre-diabetics (5.6%≤$A_{1c}$≤ 6.4%). $HbA_{1c}$ was measured by Quo-lab analyzer. This study was approved by Hamad Medical Corporation/Weill Cornell Medical College in Qatar Joint Institutional Review Board and registered with ClinicalTrials.gov, number: NCT02098980.

### *2.2 Anthropometric and Metabolic Measures*

Anthropometric measurements, including height, weight and waist circumference, were measured. The waist circumference was measured by using a measuring tape at the midpoint between the last floating rib and the top of the iliac crest in the midaxillary line. Other weight and metabolic rate-related measurements were obtained using a Tanita body composition analyzer model BC-420MA, (TANITA BC-420MA, Tanita Corporation, Japan). These were mainly BMI ($kg/m^2$), fat percentage (%), fat mass (kg), fat free mass (FFM; kg), muscle mass (MM; kg), total body water (TBW; %), bone mass (kg), degree of obesity, basal metabolic rate (BMR; Kcal), metabolic age (years) and visceral fat rating (%). These factors were assessed according to the manufacturer's protocol

(Tanita Corporation). Blood was taken from the finger tip from each subject after a 12hr overnight fast to measure their lipid profile including total cholesterol (Total-C; mmol/L), HDL (mmol/L), LDL (mmol/L), and TG (mmol/L) using the CardioCheck P-A machine and the Lipid panel strips, while the serum glucose (FPG / RPG; mmol/L) was measured by the same machine but using the Glucose panel strips. Systolic and Diastolic blood pressure (SBP, DBP; mmhg) were also measured. Vitamin D (VitD) serum levels were assessed using ARCHITECT machine.

FINDRISC sheet was filled for each participant by the researcher; from the data obtained on height and weight the BMI was calculated (weight in kg ÷ height in m square). The data on waist circumference was obtained from earlier tests. The rest of the FINDRISC questions were self-reported from the participants' knowledge including information on: age, fruits and vegetables intake, 30 minutes daily exercise, blood pressure medication, previous high blood glucose and family history of diabetes.

*2.3 Statistical Analyses*

All statistical analyses were conducted using the IBM Statistical Package for Social Sciences (SPSS version 21.0, IBM Corp., Armonk, NY, USA). Means and frequencies were determined in aggregate and by subgroup analysis of the classified $HbA_{1c}$ categories (normal:<5.6% and pre-diabetes: 5.6% - 6.4%). Continuous variables are given as mean±SDs and an independent sample t-test was used to examine the association between the aforementioned groups. The α error was set at 0.05 and 2-sided P values of ≤ 0.05 were reported as significant. Associations between categorical variables were tested through chi-square $\chi^2$ test which aimed to analyze the difference between these two groups. Pearson correlation coefficients ($r$) were calculated to evaluate the cross-correlation between the cardio-metabolic risk factors and T2DM risk scores.

## 3. Results

As shown in Table 1, means and frequencies of the different characteristics where identified in the entire study subjects and stratified by their $HbA_{1c}$ status (non-diabetic:<5.6% and pre-diabetic: 5.6%–6.4%). The study results showed that there was a predominance in males (male to female ratio 2.3:1). The majority of the study population was below the age of 45 years old (74.8%), overweight and with waist circumference between 94 - 102cm in males and over 88cm in females. The majority of the study participants reported the absence of physical activities of over 30 minutes/day, and daily consumption of fruits and vegetables, and the presence of first degree relatives with T2DM. Only 7–10% of the study population had a previous history of high-blood pressure and high-blood glucose. There was a statistically significant difference ($p$=0.001) between the non-diabetic and pre-diabetic subgroups for their FINDRISC.

Table 1. Characteristics of the study population[1]

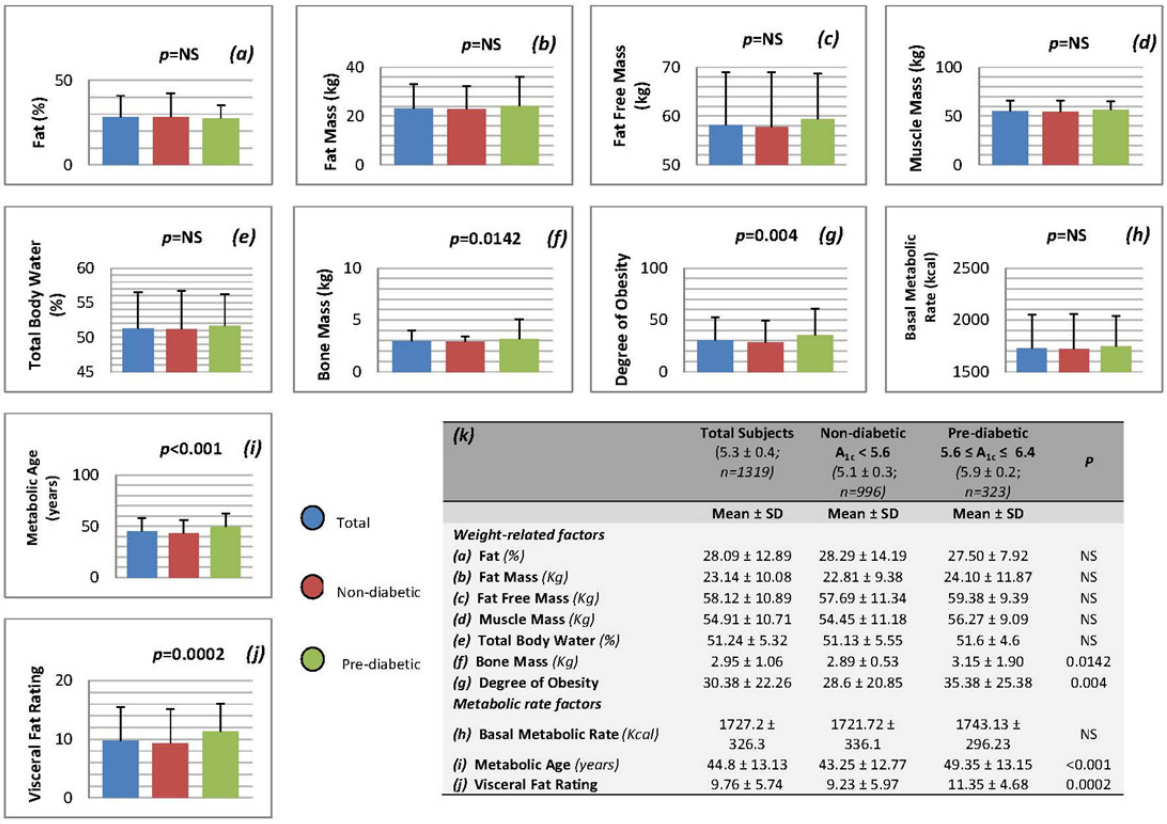
| | **Total Subjects** *(n=1319)* | **Non-diabetic** $A_{1c}$**<5.6** *(n=996)* | **Pre-diabetic** $5.6 \leq A_{1c} \leq 6.4$ *(n=323)* | **P** $(\chi^2)$ |
|---|---|---|---|---|
| | ***n* (%)** | ***n* (%)** | ***n* (%)** | |
| **Sex** | | | | <0.001 |
| Male | 917 (69.52) | 640 (69.8) | 277 (30.2) | |
| Female | 402 (30.48) | 356 (88.5) | 46(11.4) | |
| **Age** *(years)* | | | | <0.001 |
| < 45 | 987 (74.83) | 787 (79.7) | 200 (20.3) | |
| 45-54 | 240 (18.20) | 157 (65.4) | 83 (34.6) | |
| 55-64 | 90 (6.82) | 50 (55.6) | 40 (40.4) | |
| > 65 | 2 (0.15) | 2 (100.0) | 0 (0.0) | |
| **BMI** *(kg/m²)* | | | | <0.001 |
| < 25 | 307 (23.28) | 263 (85.6) | 44 (14.3) | |
| 25-30 | 570 (43.21) | 432 (75.8) | 138 (24.2) | |
| > 30 | 411 (31.16) | 282 (68.6) | 129 (31.4) | |
| **Waist Circumference** (*cm)* | | | | |

| | | | | |
|---|---|---|---|---|
| ***Men*** | | | | NS |
| < 94 | 214 (16.22) | 158 (73.8) | 56 (26.2) | |
| 94-102 | 369 (27.98) | 265 (71.8) | 104 (28.2) | |
| > 102 | 311 (23.58) | 204 (65.6) | 107 (34.4) | |
| ***Women*** | | | | *0.027* |
| < 80 | 89 (6.75) | 85 (95.5) | 4 (4.5) | |
| 80-88 | 118 (8.95) | 108 (91.5) | 10 (8.5) | |
| > 88 | 178(13.50) | 152 (85.4) | 26 (14.6) | |
| **Physical Activity** *(30mins/day)* | | | | *NS* |
| Yes | 437 (33.13) | 343 (78.5) | 94 (21.5) | |
| No | 806 (61.11) | 621(77.05) | 185 (22.9) | |
| **Vegetables/Fruits Consumption** | | | | *NS* |
| Daily | 684 (51.86) | 528 (75.1) | 156 (22.8) | |
| Occasional | 559 (42.38) | 436 (77.2) | 123 (22.0) | |
| **Blood Pressure Medication History** | | | | 0.010 |
| Yes | 127 (9.63) | 87 (68.5) | 40 (31.4) | |
| No | 1116 (84.61) | 877 (78.6) | 239 (21.41) | |
| **High Blood Glucose History** | | | | *NS* |
| Yes | 100 (7.58) | 71 (71) | 29 (29) | |
| No | 1143 (86.66) | 893 (78.13) | 250 (21.9) | |
| **Family History of DM** | | | | *NS* |
| ***1st degree:*** parents, brother, sister, child | 680 (51.55) | 517 (76.03) | 163 (23.9) | |
| ***2nd degree:*** close relatives other than parents | 182 (13.80) | 147 (80.77) | 35 (19.23) | |
| No | 381 (28.89) | 300 (78.74) | 81 (21.3) | |
| **Finnish T2DM Risk Score**[1] | | | | <0.001 |
| < 7 *(Low)* | 205 (15.54) | 166 (80.97) | 39 (19.02) | |
| 7-11 *(Slightly elevated)* | 591 (44.81) | 472 (79.86) | 119 (20.14) | |
| 12-14 *(Moderate)* | 315 (23.88) | 230 (73.02) | 85 (26.9) | |
| 15-20 *(High)* | 148 (11.22) | 93 (62.8) | 55 (37.2) | |
| > 20 *(Very high)* | 13(0.99) | 7 (53.8) | 6 (46.2) | |

[1]: The difference of the number of the individual category from the total number is due presence of some missing measures.

[2]: values were generated from $\chi^2$.

The metabolic- and weight-related factors are represented in Figure 1. Fat mass, FFM, MM and TBW were non-significantly higher in the pre-diabetic stage compared to the non-diabetic stage, while the bone mass and degree of obesity were statistically significantly higher. The mean value of the metabolic age for the entire study (44.8±13.1 years) was 18% higher than the actual age of the group (37.9±10.2 years) which describes the declined relationship between the body composition and BMR measurements in the total subjects (data not shown). The metabolic age was also significantly higher in the pre-diabetic subjects compared to their non-diabetic counterparts (49.4±13.2 vs. 43.3±12.8 years, $p<0.001$; Figure 1-i). The visceral fat rating in the entire population is 9.8±5.7 which is below the ideal range of 13%, indicating an overall healthy population. However, there was a significantly higher visceral fat rating in the pre-diabetic subgroup compared to the non-diabetic one.




| (k) | Total Subjects (5.3 ± 0.4; n=1319) | Non-diabetic $A_{1c}$ < 5.6 (5.1 ± 0.3; n=996) | Pre-diabetic 5.6 ≤ $A_{1c}$ ≤ 6.4 (5.9 ± 0.2; n=323) | p |
|---|---|---|---|---|
| | Mean ± SD | Mean ± SD | Mean ± SD | |
| *Weight-related factors* | | | | |
| *(a)* Fat *(%)* | 28.09 ± 12.89 | 28.29 ± 14.19 | 27.50 ± 7.92 | NS |
| *(b)* Fat Mass *(Kg)* | 23.14 ± 10.08 | 22.81 ± 9.38 | 24.10 ± 11.87 | NS |
| *(c)* Fat Free Mass *(Kg)* | 58.12 ± 10.89 | 57.69 ± 11.34 | 59.38 ± 9.39 | NS |
| *(d)* Muscle Mass *(Kg)* | 54.91 ± 10.71 | 54.45 ± 11.18 | 56.27 ± 9.09 | NS |
| *(e)* Total Body Water *(%)* | 51.24 ± 5.32 | 51.13 ± 5.55 | 51.6 ± 4.6 | NS |
| *(f)* Bone Mass *(Kg)* | 2.95 ± 1.06 | 2.89 ± 0.53 | 3.15 ± 1.90 | 0.0142 |
| *(g)* Degree of Obesity | 30.38 ± 22.26 | 28.6 ± 20.85 | 35.38 ± 25.38 | 0.004 |
| *Metabolic rate factors* | | | | |
| *(h)* Basal Metabolic Rate *(Kcal)* | 1727.2 ± 326.3 | 1721.72 ± 336.1 | 1743.13 ± 296.23 | NS |
| *(i)* Metabolic Age *(years)* | 44.8 ± 13.13 | 43.25 ± 12.77 | 49.35 ± 13.15 | <0.001 |
| *(j)* Visceral Fat Rating | 9.76 ± 5.74 | 9.23 ± 5.97 | 11.35 ± 4.68 | 0.0002 |

Figure 1. Weight & Metabolic related risk factors; bars represent mean±SD of the total subjects, non-diabetic and pre-diabetic. Values are reflected in insert (*k*)

Table 2. Cardiometabolic risk factors of the study subjects

| | **Total Subjects** (5.3±0.4; *n=1319*) | **Non-diabetic** **$A_{1c}$<5.6** (5.1±0.3; *n=996*) | **Pre-diabetic** **5.6 ≤ $A_{1c}$ ≤ 6.4** (5.9±0.2; *n=323*) | **P** |
|---|---|---|---|---|
| | **Mean±SD** | **Mean±SD** | **Mean±SD** | |
| *Finnish T2DM risk score* | 10.1±4.05 | 9.75±3.97 | 11.24±4.11 | <0.001 |
| *Cardiometabolic Risk Factors* | | | | |
| Age *(years)* | 37.89±10.15 | 36.5±9.93 | 41.95±9.77 | <0.001 |
| BMI *(kg/m$^2$)* | 28.78±5.19 | 28.12±4.65 | 30.29±5.98 | <0.001 |
| SBP *(mmhg)* | 130.16±16.82 | 128.74±16.78 | 133.34± 16.51 | 0.0013 |
| DBP *(mmhg)* | 81.75±11.67 | 81.47±12.05 | 82.36±10.78 | NS |
| Total Cholesterol *(mmol/L)* | 4.60±1.10 | 4.37±1.04 | 5.00±1.09 | <0.001 |
| Triglycerides *(mmol/L)* | 1.65±1.01 | 1.69± 1.09 | 1.59±0.89 | NS |
| HDL *(mmol/L)* | 1.17±0.60 | 1.24±0.72 | 1.06±0.27 | 0.0045 |
| LDL *(mmol/L)* | 2.92±1.00 | 2.69±0.96 | 3.29±0.95 | <0.001 |
| Fasting Glucose *(mmol/L)* | 5.03±0.93 | 4.96±0.94 | 5.15±0.89 | NS |
| Random Glucose *(mmol/L)* | 5.14±1.02 | 4.98±0.86 | 5.69±1.31 | <0.001 |
| Vitamin D *(nmol/L)* | 44.19±17.79 | 48.42±28.34 | 43.73±16.41 | NS |

[1]: values were generated from t-test.

Table 3. Cross-correlation analysis among the cardiometabolic risk factors as well as T2DM risk scores [1]

| | BMI | Finnish T2DM Risk Score | SBP | DBP | $HbA_{1c}$ | Total Cholesterol | Triglycerides | HDL | LDL | Fasting Glucose |
|---|---|---|---|---|---|---|---|---|---|---|
| Finnish T2DM Risk Score | .560* | | | | | | | | | |
| SBP | .100* | .056 | | | | | | | | |
| DBP | .082* | .048 | .601* | | | | | | | |
| $HbA_{1c}$ | .235* | .192* | .202* | .122* | | | | | | |
| Total Cholesterol | .091 | .075 | .025 | -.018 | .269* | | | | | |
| Triglycerides | .084 | .133* | .178* | .247* | -.003 | .212* | | | | |
| HDL | -.15* | -.055 | -.176* | -.11* | -.191* | .167* | -.127* | | | |
| LDL | .148* | .043 | .064 | -.007 | .304* | .880* | -.019 | .040 | | |
| Random Glucose | .004 | .072 | .053 | .031 | .208* | -.072 | .067 | -.809 | .675 | |
| Fasting Glucose | .046 | .032 | .065 | -.020 | .154* | -.106 | .239* | -.090 | -.108 | |
| Vitamin D | -.159 | .029 | .006 | .010 | .016 | -.044 | -.124 | .055 | -.017 | 0.047 |

[1]: values represents Pearson correlation coefficient (r)

*: Correlation is significant at<0.05 level (2-tailed).

The cardiometabolic risk factors were also assessed in the entire study and in the non-diabetic and pre-diabetic subgroups (see Table 2). In general, the pre-diabetic subjects have shown a statistically higher FINDRISC compared to their non-diabetic counterparts (11.2±4.1 vs. 9.8±4, $p$<0.001). The mean levels of cardiometabolic factors in the study population fell within the normal clinical range. Age, BMI, SBP and Total-C were all significantly higher in the pre-diabetic subgroups compared to the non-diabetic one. There was no significant difference between the non-diabetic and pre-diabetic subgroups for the DBP and TG. However, HDL was significantly lower in pre-diabetic subjects and LDL was significantly higher compared to the non-diabetic subjects. Both fasting and random plasma glucose where higher; as expected; in the pre-diabetic subjects compared to the non-diabetic subgroup. VitD serum levels, measured as 25(OH)D in the entire group was on the deficient range (44.2±17.8 nmol/l), there was a slight but non-significant further drop in the pre-diabetic subjects compared to the non-diabetic subgroup.

A cross-correlation analysis among the cardiometabolic risk factors as well as the FINDRISC is shown in Table 3. The risk of T2DM identified by FINDRISC was significantly and directly correlated with the BMI, levels of $HbA_{1c}$ and TG. However, $HbA_{1c}$ was correlated directly with BMI, SBP, DBP, Total-C, LDL, FPG/RPG and indirectly with the levels of HDL. $HbA_{1c}$ tended to increase dramatically with increasing FINDRISC scores (Costa et al., 2013). The above findings indicate that introducing $HbA_{1c}$ into the FINDRISC can be a more precise prediction of T2DM than utilizing the FINDRISC alone.

VitD serum levels were indirectly associated with the BMI and cholesterol levels (Total-C, TG, LDL), whereas directly correlated with the risk of T2DM (FINDRISC), blood pressure, $HbA_{1c}$ and HDL. Although these associations were identified to be insignificant (perhaps due to the small sample size and the large inter-individual variation in vitamin D serum level), they substantiate a possible role of VitD deficiency in the emergence of pre-diabetic stage.

## 4. Discussion

The present study examined the association between the anthropometric, metabolic and FINDRISC factors in 1319 Qatari – subjects (local and expat, excluding diabetics, nursing and pregnant women) which represent

2.169 million Qataris. There was an overall strong relation between the aforementioned factors and the prevalence of T2DM risk factors where 24% ($n$ = 323) of the population sample were identified as pre-diabetic subjects in their middle age (42±9.8 years). This is considered to be a high percentage in accordance to their age due to their sedentary lifestyle and food habits, where 61% (n = 806) were not doing 30 minutes per day of physical activity and 42% ($n$ = 559) were occasionally consuming fruits and vegetables (as shown in Table 1). There are even high genetic factors involved as 52% (n = 680) were reported to have first-degree relatives with T2DM. Eventually, Qatar is among the countries with the highest percentage prevalence of diabetes (Christos et al., 2014). Our findings are similar to previous studies that showed the relation between the anthropometric factors along with FINDRISC diabetes scores in obese populations (Stijn et al., 2015).

There was a statistical relation between the cardiometabolic risk factors and FINDRISC. BMI was the oddest figure with 43% of the sample population has their BMI value laying between 25–30 kg/m$^2$ with the pre-diabetic stage being the highest (30.3±6 kg/m$^2$). Statistics also reflected a strong correlation between the BMI and FINDRISC. As known obesity plays a vital role in the etiology of T2DM from different prospective; (a) activation of innate-immune system, (b) release of cytokines (tumor necrosis factor-α, interleukin-1 β and interleukin-6) blocking major anabolic cascades of insulin, (c) triggering of acute-phase proteins (C-reactive protein, plasminogen activator, inhibitor-1, serum amyloid-A, haptoglobin) which characterize the pre-diabetic stages of T2DM (Badawi et al., 2010). Eventually, the weight-related factors were slightly higher in the pre-diabetic stage compared to their non-diabetic counterparts.

Results have clearly shown that vitamin D values were in the deficient range (44.2±17.8 nmol/l) for the entire study population, however, was not significant. A potential explanation of such insignificancy would be due to the large inter-individual variation between the subjects in each subgroup and the relative small sample size. In the present study, BMI was inversely associated with plasma 25(OH)D which suggests that vitamin D plays an active role in obesity, with low levels stimulating synthesis and release of parathyroid hormone, increasing calcium in adipocytes, and promoting weight gain (McCarty & Thomas, 2003). As studied earlier, vitamin D deficiency has been implicated to the development of metabolic syndrome, obesity and T2DM (Brenner et al., 2011). Recently it has been reported that about 90% of the Qatari population has insufficient levels of vitamin D (Badawi et al., 2012), which contributes to the prevalence of T2DM.

The current cross-sectional study impacted the utility of $HbA_{1c}$ as a useful tool in T2DM prediction. The strong direct correlation of $HbA_{1c}$ with the FINDRISC, BMI, SBP, DBP, Total-C, LDL and FPG/RPG and the indirect correlation with HDL statistically shows that $HbA_{1c}$ can be predictive to all aforementioned. Altogether are markers of T2DM, so $HbA_{1c}$ can be a representative tool and even a better one. Therefore, FINDRISC along with $HbA_{1c}$ factors are considered to be the best prognostication of the T2DM disease.

There were several limitations in the collection of data such as: (a) the self-reporting nature of the data (e.g. physical activity), (b) the absence of 5% of data from the 1,319 participants, (c) the sample size was not representative of the total population – a cross sectional study, (c) the high cost of $HbA_{1c}$ testing compared to the inexpensive FPG, and (d) the inability to rule out the possibility of residual confounding factors (e.g. diet, transportation-related physical activity and sun exposure).

In conclusion, the growing prevalence of T2DM requires the development of a better preventative method to reduce the incidence of the disease, which in turn will help in reducing health costs. It is currently recommended that screening for diabetes and pre-diabetes should be carried out using the FINDRISC, followed by a glucose test (FPG, 2hr OGTT and $HbA_{1c}$). This cross-sectional study showed a direct association between $HbA_{1c}$ along with BMI and FINDRISC, which indicates that interweaving $HbA_{1c}$ into the FINDRISC can be a strong predictive tool. The early prediction of T2DM will help in applying strategies for the future prevention of the disease and reducing the burden of its chronic complications.

**Acknowledgements**

We would like to thank Adham Salem and Herluf Nis Thomsen for their efforts in preparing campaigns and providing us with the required machines and instruments to conduct our study. The authors declare no conflict of interests in this work.

**Competing Interests Statement**

The authors declare that there is no conflict of interests regarding the publication of this paper.

**References**

American Diabetes Association. (2010). Diagnosis and classification of diabetes mellitus. *Diabetes Care, 27*(1), s5-s10. http://dx.doi.org/10.2337/diacare.27.2007.S5

Badawi, A., Klip, A., Haddad, P., EC Cole, D., Bailo, B. G., El-Sohemy, A., & Karmali, M. (2010). Type 2 diabetes mellitus and inflammation: Prospects for biomarkers of risk and nutritional intervention. *Diabetes Metab Syndr Obes, 3*, 173-186. PMID: 21437087. http://dx.doi.org/10.2147/DMSOTT.S9089

Badawi, A., Arora, P., Sadoun, E., Al-Thani, A. A., & Al Thani, M. (2012). *Prevalence of Vitamin D Insufficiency in Qatar: a Systematic Review*. http://dx.doi.org/10.4081/jphr.2012.e36

Bilan, P. J., Samokhvalov, V., Koshkina, A., Schertzer, J. D., Samaan, M. C., & Klip, A. (2009). Direct and macrophage-mediated actions of fatty acids causing insulin resistance in muscle cells. *Archives of physiology and biochemistry*, *115*(4), 176-190. http://dx.doi.org/10.1080/13813450903079314

Bonora, E., & Tuomilehto, J. (2011). The pros and cons of diagnosing diabetes with A1C. *Diabetes care*, *34*(Supplement 2), S184-S190. http://dx.doi.org/10.2337/dc11-s216

Brenner, D., Arora, P., Garcia-Bailo, B., Wolever, T., Morrison, H., El-Sohemy, A., … Badawi, A. (2011). Plasma vitamin D levels and risk of metabolic syndrome in Canadians. *Clin invest med, 34*(6), E377-E384. PMID: 22129928

Christos, P. J., Chemaitelly, H., Abu-Raddad, L. J., Zirie, M. A., Deleu, D., & Mushlin, A. I. (2014). Prevention of type II diabetes mellitus in Qatar: Who is at risk?. *Qatar medical journal*, *2014*(2), 70. http://dx.doi.org/10.5339/qmj.2014.13

Costa, B., Barrio, F., Piñol, J. L., Cabré, J. J., Mundet, X., Sagarra, R., ... Solà-Morales, O. (2013). Shifting from glucose diagnosis to the new HbA1c diagnosis reduces the capability of the Finnish Diabetes Risk Score (FINDRISC) to screen for glucose abnormalities within a real-life primary healthcare preventive strategy. *BMC medicine*, *11*(1), 1. http://dx.doi.org/10.1186/1741-7015-11-45

Dandona, P., Aljada, A., & Bandyopadhyay, A. (2004). Inflammation: The link between insulin resistance, obesity and diabetes. *Trends in immunology*, *25*(1), 4-7. http://dx.doi.org/10.1016/j.it.2003.10.013

Dimopoulos, N., Watson, M., Sakamoto, K., & Hundal, H. S. (2006). Differential effects of palmitate and palmitoleate on insulin action and glucose utilization in rat L6 skeletal muscle cells. *Biochemical Journal*, *399*(3), 473-481. http://dx.doi.org/10.1042/BJ20060244

International Expert Committee. (2009). International Expert Committee report on the role of the A1C assay in the diagnosis of diabetes. *Diabetes care*, *32*(7), 1327-1334. http://dx.doi.org/10.2337/dc09-9033

Lindström, J., & Tuomilehto, J. (2003). The Diabetes Risk Score A practical tool to predict type 2 diabetes risk. *Diabetes care*, *26*(3), 725-731. http://dx.doi.org/10.2337/diacare.26.3.725

McCarty, M. F., & Thomas, C. A. (2003). PTH excess may promote weight gain by impeding catecholamine-induced lipolysis-implications for the impact of calcium, vitamin D, and alcohol on body weight. *Medical hypotheses, 61*(5), 535-542. http://dx.doi.org/10.1016/S0306-9877(03)00227-5

Mokdad, A. H., Bowman, B. A., Ford, E. S., Vinicor, F., Marks, J. S., & Koplan, J. P. (2001). The continuing epidemics of obesity and diabetes in the United States. *Jama, 286*(10), 1195-1200. http://dx.doi.org/10.1001/jama.286.10.1195

Saaristo, T., Peltonen, M., Lindström, J., Saarikoski, L., Sundvall, J., Eriksson, J. G., & Tuomilehto, J. (2005). Cross-sectional evaluation of the Finnish Diabetes Risk Score: a tool to identify undetected type 2 diabetes, abnormal glucose tolerance and metabolic syndrome. *Diabetes and vascular disease research*, *2*(2), 67-72. http://dx.doi.org/10.3132/dvdr.2005.011

Sorrentino, M. J. (2005). Implications of the metabolic syndrome: the new epidemic. *The American journal of cardiology, 96*(4), 3-7. http://dx.doi.org/10.1016/j.amjcard.2005.05.008

Stijn Meijnikman, A., Verrijken, A., Mertens, I. E., De Block, C. F., Van Gaal, L. (2015). *The Findrisc score helps in assessing diabetes status in an overweight or obese population* (2015; THR-565).

Stumvoll, M., Goldstein, B. J., & van Haeften, T. W. (2005). Type 2 diabetes: Principles of pathogenesis and therapy. *The Lancet*, *365*(9467), 1333-1346. http://dx.doi.org/10.1016/S0140-6736(05)61032-X

Venkataraman, R., Nanda, N. C., Baweja, G., Parikh, N., & Bhatia, V. (2004). Prevalence of diabetes mellitus and related conditions in Asian Indians living in the United States. *Am J Cardiol.* http://dx.doi.org/10.1016/j.amjcard.2004.06.048

Wild, S., Roglic, G., Green, A., Sicree, R., & King, H. (2004). Global prevalence of diabetes estimates for the year 2000 and projections for 2030. *Diabetes care*, *27*(5), 1047-1053.

http://dx.doi.org/10.2337/diacare.27.5.1047.