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CLINICAL & BASIC RESEARCH

Impaired Fasting Glucose in Omani Adults with no Family History of Type 2 Diabetes

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معدل إنتشار مرحلة ما قبل السكري في العمانيين الذين ليس لديهم تاريخ عائلي للنوع الثاني من داء السكري

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ABSTRACT: ***Objectives:*** The aim of this study was to estimate the prevalence of impaired fasting glucose (IFG) among Omani adults with no family history (FH) of diabetes and to investigate the factors behind the risk of developing type 2 diabetes (T2D), while excluding a FH of diabetes. ***Methods:*** A total of 1,182 Omani adults, aged ≥40 years, visited the Family Medicine & Community Health Clinic at Sultan Qaboos University Hospital, Oman, on days other than the Diabetes Clinic days, from July 2010 to July 2011. The subjects were interviewed and asked if they had T2D or a FH of T2D. ***Results:*** Only 191 (16%) reported no personal history of T2D or FH of the disease. Of these, anthropometric and biochemical data was complete in 159 subjects. Of these a total of 42 (26%) had IFG according to the American Diabetes Association criteria. Body mass index, fasting insulin, haemoglobin A_{1C} and blood pressure (BP), were significantly higher among individuals with IFG (P <0.01, P <0.05, P <0.01 and P <0.01, respectively). In addition, fasting insulin, BP and serum lipid profile were correlated with obesity indices (P <0.05). Obesity indices were strongly associated with the risk of IFG among Omanis, with waist circumference being the strongest predictor. ***Conclusion:*** Despite claiming no FH of diabetes, a large number of Omani adults in this study had a high risk of developing diabetes. This is possibly due to environmental factors and endogamy. The high prevalence of obesity combined with genetically susceptible individuals is a warning that diabetes could be a future epidemic in Oman.

Keywords: Prediabetic State; Diabetes Mellitus, Type 2; Obesity; Prevalence; Oman.

الملخص: الهدف: تهدف هذه الدراسة إلى تقديرمعدل إنتشار مرحلة ما قبل السكري في مجموعة من الأشخاص العمانيين الذين ليس لديهم تاريخ عائلي للنوع الثاني من داء السكري، وكذلك التحقيق في عوامل الاختطار وراء الإصابة بالنوع الثاني من السكري مع إستبعاد وجود تاريخ عائلي للسكري. **الطريقة**: تمت دراسة عدد 1,182 شخصاً عمانياً، أعمارهم 40≤ سنة، من المراجعين لعيادة طب الأسرة و المجتمع في مستشفى جامعة السلطان قابوس،عمان،في غير أيام عيادة السكري خلال الفترة من يوليو 2010 إلى يوليو 2011. تمت مقابلة الأشخاص والاستفسار عن إصابتهم بالسكري أو وجود تاريخ عائلي للنوع الثاني من مرض السكري. **النتائج**: إدعى عدد 191 (16%) شخصاً فقط أنهم غير مصابين بمرض السكري أو عدم وجود تاريخ عائلي لديهم لمرض السكري. أكمل 159 شخصا فقط من هذه المجموعة القياسات الجسمية والبيوكيميائية. تم تصنيف مجموع 42 (26%) شخصا كمصابين باعتلال ما قبل السكري حسب معايير الجمعية الأمريكية لمرض السكري. كانت قياسات منسب كتلة الجسم، مستوى الإنسولين وقت الصوم، هيموغلوبين A_{1C}، وضغط الدم مرتفعة بين الأشخاص الذين هم في مرحلة ما قبل ظهور السكري. و قد لوحظ أرتباط نسبة الانسولين وقت الصيام، وضغط الدم ونسبة الدهون في الدم مع مؤشرات السمنة. معدلات السمنة كانت مرتبطة بشدة بعامل الإصابة بمرحلة ما قبل السكري عند العمانيين، مع اعتبار كفاف الخصر منبئ قوي للإصابة. **الخلاصة**: على رغم الإدعاء من عدم وجود تاريخ عائلي للاصابة بالسكري تم الإستنتاج أن عدد كبير من الأشخاص العمانيين في هذه الدراسة كان لديهم عوامل اختطار عالية للإصابة بمرض السكري. وهذا يعتبر نتيجة لعوامل بيئية وزواج الأقارب. أرتفاع معدل السمنة بالإشتراك مع الاستعداد الوراثي للشخص يعتبر تحذير من أن مرض السكري يمكن أن يتوطن في المستقبل في عمان.

مفتاح الكلمات: مرحلة ما قبل السكري، مرض السكري، النوع الثاني، السمنة، معدل، عمان.

Advances in Knowledge

- *In spite of having no family history of diabetes, a sizable sector of Omani adults are at a high risk of developing diabetes due to environmental factors and endogamy.*

Application to Patient Care

- *All adult Omanis should be screened routinely for impaired fasting glucose at all health facilities.*
- *Obesity management clinics should be set up in all healthcare centres in Oman.*

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Type 2 diabetes (T2D) has a strong genetic component and its heritability has been estimated at approximately 25%.[1] To date, more than 70 common T2D susceptibility gene variants and *loci* have been identified; however, all these common gene variants only explain ~10–15% of the heritability of T2D, suggesting that other rare variants have yet to be discovered.[2,3]

T2D is also known to cluster amongst relatives whose households share similar environmental, dietary and cultural lifestyles.[4] Recent studies have further demonstrated the impact of obesity and environmental factors on the risk of developing T2D.[5,6] The InterAct Project study showed that the interaction between obesity and a family history (FH) of diabetes increases the risk of T2D by more than 20-fold compared with lean subjects without a FH of T2D.[6] As body mass index (BMI) is influenced by both environmental and genetic factors, this interaction could be attributed to gene-environment and gene-gene interactions.[5]

The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, working under the sponsorship of the American Diabetes Association (ADA), defined prediabetics in 1997 and 2003 as an intermediate group of individuals whose glucose levels are elevated above normal values but are not high enough to be classified as having T2D.[7–9] These individuals were defined as having impaired fasting glucose (IFG), i.e. fasting plasma glucose levels of 5.6–6.9 mmol/L, or impaired glucose tolerance (IGT) with values of 7.8–11.0 mmol/L in the two-hour oral glucose tolerance test. The World Health Organization (WHO), on the other hand, defines the cut-off for IFG at 6.1 mmol/L rather than the 5.6 mmol/L value defined by the ADA.[10] Therefore, the exact number of individuals with IFG differs according to the definition used.

Over a 10-year period, IFG carries a 50% risk of progression to T2D. In a recent study, the progession to T2D was reported to occur in under three years.[11] The prediabetic state is also associated with abdominal obesity, insulin resistance, dyslipidaemia and hypertension.

One in nine adults in the Middle East and North Africa have diabetes and more than half of them are unaware of their condition.[12] In addition, 6.7% of the population in this region have IGT; this number is expected to double by 2030.[12] Following the oil boom of the past four decades, major socioeconomic changes have taken place in Oman and other Arabian Gulf countries. These include rapid urbanisation and Westernisation, with increased levels of inactivity and changes in food habits. Four countries in this region (Kuwait, Saudi Arabia, Qatar and Bahrain) are among the 10 countries in the world with the highest T2D prevalence for adults aged 20–79 years.[12] In addition to the lifestyle, activity and dietary changes brought about by rapid economic development, these populations have a high genetic susceptibility due to the frequency of consanguineous marriages and endogamy.[13,14]

In Oman, according to the National Health Surveys of 1991 and 2000, the prevalence of diabetes mellitus rose from 8.3% in 1991 to 11.6% in 2000 among adults aged 20 years or older.[15] The survey also showed that 7.1% of men and 5.1% of women had IFG.[16] Other studies, using the ADA criteria, have shown that abnormal glucose metabolism is common in Omanis, with 35% of adults having prediabetes.[17,18] The Ministry of Health (MOH) in Oman has accordingly established a nationwide screening programme to detect prediabetes among those aged ≥40 years.[19]

This study aimed to determine the prevalence of IFG among a group of Omani adults claiming not to have a FH of diabetes. The risk factors of developing T2D among these Omani adults were also investigated.

Methods

This hospital-based cross-sectional study recruited subjects from a primary healthcare clinic, the Family Medicine & Community Health Clinic (FAMCO) at Sultan Qaboos University Hospital (SQUH) in Muscat, Oman. FAMCO offers a range of services, including: general appointments; dermatology consultations; walk-in appointments; Diabetes Clinic days; antenatal, postnatal and baby care; birth spacing advice; immunisations, and general counselling.

A total of 1,182 Omani adults aged ≥40 years visited FAMCO with general appointments on days other than the Diabetes Clinic days from July 2010 to July 2011. All of the subjects were interviewed and asked whether they had T2D and/or a FH of T2D in their first-degree relatives. Pregnant women were not included in the study. Inclusion and exclusion criteria are explained in Figure 1.

Out of the 1,182 subjects, 191 (16%) reported not having T2D or a FH of the disease. These subjects were then asked to fast overnight and report the following morning for glucose measurements. According to their fasting glucose results, they were classified into three categories using the ADA criteria: normoglycaemic (<5.6 mmol/L), IFG (5.6–6.9 mmol/L) or diabetic (≥7.0 mmol/L). Participants were also classified as having IFG according to the 2006 WHO diagnostic criteria (6.1–6.9 mmol/L). Of the 191 test subjects, 32 were excluded either because they were found to

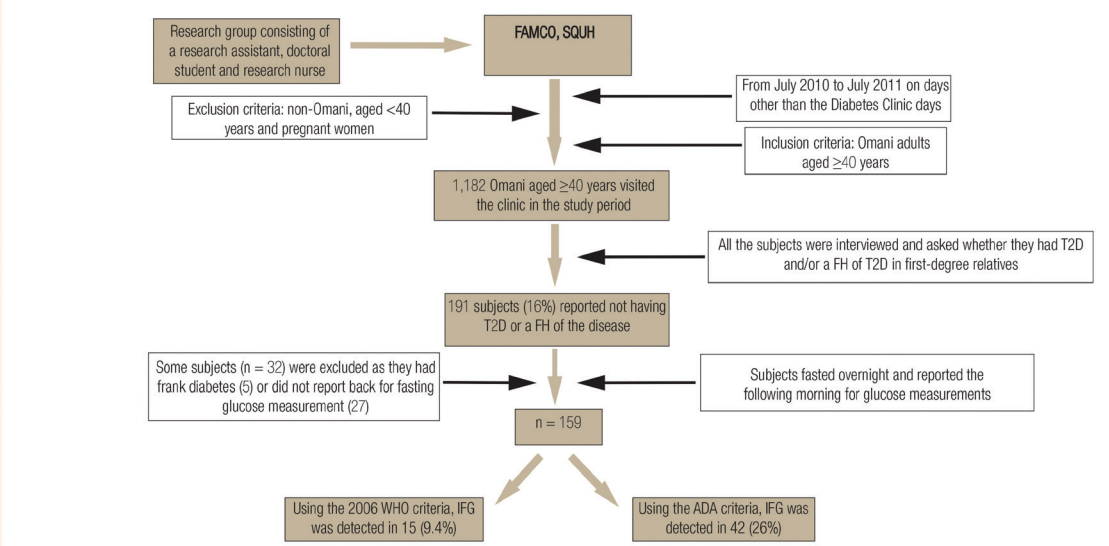

Figure 1: The setting, design and population of the current hospital-based cross-sectional study.
FAMCO = Family Medicine & Community Health Clinic; SQUH = Sultan Qaboos University Hospital; T2D = type 2 diabetes; FH = family history; WHO = World Health Organization; IFG = impaired fasting glucose; ADA = American Diabetes Association.

have frank diabetes (5) or because they did not report back for fasting glucose measurement (27). After these exclusions, complete data were obtained from the remaining 159 subjects.

Demographic (age and sex), anthropometric (height, weight, waist and hip circumference) and biochemical information was collected from all of these subjects. Their obesity status was also determined using the international classification of BMI (weight in Kg/height in m^2).[20] The subjects were classified as being normal if they had a BMI of 18.50–24.99 Kg/m^2, overweight with a BMI of 25.00–29.99 Kg/m^2 and obese with a BMI of ≥30.00 Kg/m^2. Height and weight were measured using standard methods with the subjects wearing indoor clothes without shoes.

Waist circumference (WC) was measured in cm at a midway level between the lower rib margin and iliac crest using a non-stretchable measuring tape. Hip circumference was measured in cm around the widest portion of the buttocks using a non-stretchable measuring tape with the tape parallel to the floor. A WC of ≥94 cm for males and ≥80 cm for females was considered to be a risk factor for insulin resistance.[21]

The waist-to-hip ratio (WHR) was also calculated for each subject. The WHR was considered a health risk for males according to the following ranges: low risk was considered for a WHR of 0.95 or below, moderate risk at 0.96 to 1.0 and high risk for a WHR of over 1.0. Female WHR was considered a health risk according to the following ranges: low risk for a WHR of 0.80 or below, moderate risk at 0.81 to 0.85 and high risk for those with a WHR of over 0.85.[22]

Blood pressure (BP) was also measured in mmHg for all of the subjects by nurses using standard methods. The biochemical investigations included measurements of fasting glucose levels, fasting insulin, glycated haemoglobin (HbA_{1C}), serum creatinine, serum lipid profile (total cholesterol, low density lipoprotein [LDL] cholesterol, high density lipoprotein [HDL] cholesterol and triglycerides) and apolipoprotein B.

The Statistical Package for the Social Sciences (SPSS) software, Version 20.0 (IBM, Corp., Chicago, Illinois, USA), was used for the statistical analysis of the data. The anthropometric and biochemical parameters were tested for normal distribution using the one-sample Kolmogorov-Smirnov test. The Student's t-test was used to test the significance of the difference in the mean values for the parameters with a normal distribution, while the Mann-Whitney U test was used for the variables with a skewed distribution. In addition, bivariate correlation and simple linear regression analyses were carried out to evaluate the influence of obesity indices (BMI, WC and WHR) on diabetes-related parameters.

All of the subjects were informed about the project and provided written consent. This study was approved by the Ethics & Research Committee of the College of Medicine & Health Sciences at Sultan Qaboos University, Oman.

Results

According to the ADA criteria,[7] 42 (26%) of the 159 participants had IFG (males = 19, females = 23). However, according to the 2006 WHO diagnostic

criteria, only 15 (9.4 %) had IFG.

Table 1 shows the demographic, anthropometric and biochemical characteristics of the IFG and normoglycaemic groups. Age, weight, BMI, BP, fasting glucose, fasting insulin and HbA_{1C} levels were significantly different between the IFG and normoglycaemic subjects. There was approximately a five-year age difference between the two groups (P <0.01). The mean BMI among IFG subjects was in the obese range, while the mean for normoglycaemics was in the overweight range (P <0.01). The prevalence of overweight and obese subjects among the IFG and normoglycaemic subjects were 88% and 74%, respectively.

With regards to WC, 37% and 91% of IFG males and females, respectively, had an elevated WC, while 41% and 88% of normoglycaemic males and females, respectively, had an elevated WC.[20] In addition, the mean value of the participants' WHRs indicated moderate risk among males with IFG (0.96 ± 0.05 cm) but low risk among normoglycaemic males (0.95 ± 0.04 cm). However, it indicated high risk in both IFG (0.94 ± 0.1 cm) and normoglycaemic (0.92 ± 0.1 cm) females.

There was no significant difference in the serum lipids concentrations among IFG and normoglycaemic subjects. However, total cholesterol and LDL cholesterol mean values were borderline high, while HDL cholesterol was moderately low among both groups [Table 1].

Tables 2 and 3 show the results of the Pearson correlation and simple linear regression analyses, respectively. Increased fasting insulin, systolic and diastolic BP and serum lipids were found to correlate significantly with an increase in obesity indices. Approximately 48% (r^2 = 0.48) and 33% (r^2 = 0.33) of the variance of the fasting insulin and 11% (r^2 = 0.11) and 4% (r^2 = 0.04) of the variance of the diastolic BP among IFG and normoglycaemic subjects, respectively, could be accounted for by an increase in WC. In addition, 15% (r^2 = 0.15) and 8% (r^2 = 0.08) of the variance of the systolic BP among IFG and normoglycaemic subjects, respectively, could be accounted for by an increase in WHR. For the lipids profile, approximately 18% (r^2 = 0.18) of the variance of the total cholesterol and 14% (r^2= 0.14) of the variance of the triglycerides among IFG subjects could be accounted for by an increase in WHR. Both the Pearson correlation and simple linear regression analyses revealed that the increase in WC had a stronger influence on the parameters related to diabetes than the increase in BMI.

Table 1: Demographic, anthropometric and biochemical characteristics of the impaired fasting glucose and normoglycaemic groups of Omani participants with no family history of type 2 diabetes (N = 159)

Characteristic	IFG group (n = 42)	Normo-glycaemic group (n = 117)	*P* value
	Mean ± SD	Mean ± SD	
Age in years (range)	52 ± 10	46 (35–79)*	<0.01
Weight in Kg	77 ± 15	72 ± 13	<0.05
Height in cm	156 ± 11	157 ± 8	0.478
BMI in Kg/m^2	32 ± 8	29 ± 5	<0.01
WC in cm	96 ± 11	92 ± 14	0.099
Hip circumference in cm (range)	99 ± 9	97 (76–191)*	0.223
WHR	0.97 ± 0.1	0.94 ± 0.1	0.076
Systolic BP in mmHg	138 ± 20	127 ± 20	<0.01
Diastolic BP in mmHg	81 ± 10	77 ± 9	<0.01
Fasting glucose in mmol/L (range)	5.8 (5.6–6.7)*	4.9 ± 0.4	<0.001
Fasting insulin in mIU/L	9.9 ± 5.4	7.4 ± 3.9	<0.05
HbA_{1C} in %	5.9 ± 0.5	5.7 ± 0.4	<0.01
Serum creatinine in µmol/L	69 ± 20	63 ± 16	0.071
Total cholesterol in mmol/L	5.5 ± 1.0	5.4 ± 0.9	0.575
LDL cholesterol in mmol/L	3.5 ± 1.1	3.5 ± 0.9	0.803
HDL cholesterol in mmol/L	1.2 ± 0.3	1.3 ± 0.3	0.339
Triglycerides in mmol/L (range)	1.6 ± 0.8	1.2 (0.3–4.0)*	0.167
Apolipoprotein B in g/L	1.0 ± 0.2	1.0 ± 0.3	0.621

IFG = impaired fasting glucose; SD = standard deviation; BMI = body mass index; WC = waist circumference; WHR = wasit-to-hip ratio; BP = blood pressure; HbA_{1C} = glycated haemoglobin; LDL = low-density lipoprotein; HDL = high-density lipoprotein.

**The median (range of minimum–maximum) is displayed if the variable does not follow a normal distribution pattern.*

Discussion

This study was conducted on a random hospital cohort of 159 Omani adults who reported no personal or FH of T2D and who were not excluded due to incomplete data or other exclusion criteria. Using the ADA criteria, 26% of those who were unaware of a personal or FH of diabetes had IFG. However, using the WHO criteria, IFG was found in 9.4%. This is considered a high percentage among this unique sample of subjects

Table 2: Pearson correlation (r) of measured biochemical paramenters with body mass index, waist circumference and waist-to-hip ratio among the impaired fasting glucose and normoglycaemic groups of Omani patients (N = 159)

Biochemical parameters	Pearson correlation (r)					
	BMI		WC		WHR	
	IFG	NGG	IFG	NGG	IFG	NGG
Fasting insulin in mIU/L	0.223	0.501**	0.691**	0.576**	0.440**	0.227*
Systolic BP in mmHg	0.113	0.193*	0.189	0.249**	0.387*	0.282**
Diastolic BP in mmHg	0.177	0.143	0.331*	0.210*	0.195	0.288**
Total cholesterol in mmol/L	0.048	0.104	0.254	0.188*	0.421**	0.118
HDL cholesterol in mmol/L	0.251	-0.119	0.008	-0.227*	-0.161	-0.186
Triglyceride in mmol/L	0.054	0.133	0.228	0.187	0.370*	0.235*
Apolipoprotein B in g/L	-0.132	0.127	0.070	0.277**	0.242	0.299**

BMI = body mass index; WC = waist circumference; WHR = waist-to-hip ratio; IFG = impaired fasting glucose group; NGG = normoglycaemic group; BP = blood pressure; HDL = high-density lipoprotein.

**Significant at the P <0.05 level (two-tailed); **Significant at the P <0.01 level (two-tailed).*

with no FH of diabetes. The risk factors for diabetes, such as age and BMI, were found to be significantly higher among subjects with IFG compared to the normoglycaemic group. Furthermore, diabetes-associated conditions, such as an increase in fasting insulin, HbA_{1C} and BP, were significantly higher among subjects with IFG compared to normoglycaemics. Obesity indices had some influence on the measured parameters among both IFG and normoglycaemic subjects. An increase in obesity indices seems to correlate with an increase in fasting insulin, BP and serum lipids.

The findings of this study are in agreement with previous Omani studies, where the isolated IFG prevalence among Omani adults was reported at 30% using the ADA criteria.[18] However, it was reported at 6.1% (in subjects ≥20 years) and 8.3% (in subjects ≥30 years) using the WHO criteria.[15,23] The Oman World Health Survey (OWHS), also using the WHO criteria, estimated the prevalence of prediabetes among Omani adults at 4.4% in those aged ≥18 years, 8.4% in those aged 45–54 years and 11.2% in those aged 55–64 years.[22]

Using the WHO criteria, IFG among Emiratis (8.8%) was very similar to the Omani figures.[25] However, using the ADA criteria, it was reported at 1.3% among Qataris and 3.1% among Saudis.[26,27] These values are considered low in comparison with the prevalence of diabetes among their populations. Furthermore, using the ADA criteria, IFG was found to be 13.8% among Kuwaitis.[28] In Spain, IFG was reported at 2.8% and 3.4% (using the WHO criteria), while in the USA it was reported at 26% (using the ADA criteria).[29–31]

Table 3: Regression coefficient (B) of measured biochemical parameters with body mass index, waist circumference and waist-to-hip ratio among the impaired fasting glucose and normoglycaemic groups of Omani patients (N = 159)

Biochemical parameters	Regression coefficient					
	BMI		WC		WHR	
	IFG	NGG	IFG	NGG	IFG	NGG
Fasting insulin in mIU/L	0.137	0.373**	0.316**	0.216**	29**	10.2*
Systolic BP in mmHg	0.274	0.753*	0.333	0.496**	97*	71**
Diastolic BP in mmHg	0.218	0.247	0.296*	0.182*	24.8	31.8**
Total cholesterol in mmol/L	0.006	0.018	0.021	0.012*	5.0**	1.3
HDL cholesterol in mmol/L	0.008	-0.007	0.0	-0.005*	-0.540	-0.698
Triglyceride in mmol/L	0.006	0.019	0.017	0.010	3.9*	2.1*
Apolipoprotein B in g/L	-0.005	0.006	0.001	0.005**	0.662	0.861**

BMI = body mass index; WC = waist circumference; WHR = waist-to-hip ratio; IFG = impaired fasting glucose group; NGG = normoglycaemic group; BP = blood pressure; HDL = high-density lipoprotein.

**Significant at the P <0.05 level (two-tailed); **Significant at the P <0.01 level (two-tailed).*

In Omani subjects, 88% of the IFG subjects were either overweight or obese compared to 74% of the normoglycaemic group. A recent study found that 51% of Omani adults and 75% of isolated IFG individuals were either overweight or obese.[18] Among the Qatari population, 86% of diabetics and 75% of normoglycaemics were overweight or obese.[26] These findings are similar to the figures found in the subjects of the current study. Family and twin studies have shown that BMI and abdominal obesity, measured by WC or WHR, is 40–60% heritable.[32,33]

In this study, it was found that an increase in WC had a stronger influence on diabetes-associated conditions than an increase in BMI. Approximately 48% and 33% of the variance of the fasting insulin among IFG and normoglycaemic subjects, respectively, could be accounted for by an increase in WC. These results are similar to an earlier study, which found that all three indices of obesity were strongly and independently associated with the risk of T2D among Omanis, with WC being the strongest predictor.[34] In this study, Omani females were found to have a higher prevalence of elevated WC than Omani males. Furthermore, females with IFG were found to have a higher prevalence of elevated WC than normoglycaemic females. The mean value of WHR indicated a moderate risk among males with IFG, but a low risk among normoglycaemic males. However, it indicated high risk in both IFG and normoglycaemic females. In a previous study, the prevalence of elevated WC among Omanis was found to be 23% and 65% among males and females, respectively.[35]

In a complex disease like diabetes, with a multitude of potential interacting genes and environmental factors, and with no control for socioeconomic status or endogamy, the outcome of the current study is not surprising.[14] This study found a high prevalence of IFG in an Omani cohort of subjects with no FH of diabetes; therefore, the high prevalence is believed to be due primarily to changes in diet and lifestyle. However, it may not be possible to claim this as the precise cause, since Oman has an endogamous society where approximately half of all marriages are consanguineous.[36] Therefore, the Omani subjects in this study may still have had diabetic ancestors of whom they were unaware.[14,37,38] Also, obesity, which is a major predisposing factor for diabetes, has both genetic and environmental elements.

The main limitation of this study was the small sample size. However, this study aimed to separate the influence of genetics from the environmental factors of developing T2D among the Omani population. Previous studies among the Omani population concentrated on finding the prevalence of IFG among the whole population. This study discovered that obesity among the Omani population has a strong effect on developing T2D, even without the presence of a FH of diabetes.

The difficulty in recruiting Omani adults with no FH of diabetes was the main reason for the small sample of subjects in this study as almost everybody has a relative with diabetes mellitus.

Conclusion

In this study, 26% of the Omani population sample reported no FH of diabetes, but had IFG. Obesity indices are strongly associated with the risk of IFG and T2D among Omanis, with WC being the strongest predictor. The high prevalence of central obesity among this Omani population acting on genetically susceptible individuals warns of a future diabetes epidemic in Oman. Health education programmes could make a substantial difference in promoting lifestyle modifications, which are important tools to prevent diabetes and its complications.

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