Original Article

# Study of beta-cell function (by HOMA model) in metabolic syndrome

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## ABSTRACT

**Introduction:** The clustering of cardiovascular risk factors is termed the metabolic syndrome (MS), which strongly predict risk of diabetes and cardiovascular disease. Many studies implicate insulin resistance (IR) in the development of diabetes, but ignore the contribution of beta-cell dysfunction. Hence, we studied beta-cell function, as assessed by HOMA model, in subjects with MS. **Materials and Methods:** We studied 50 subjects with MS diagnosed by IDF criteria and 24 healthy age- and sex-matched controls. Clinical evaluation included anthropometry, body fat analysis by bioimpedance, biochemical, and insulin measurement. IR and secretion were calculated by HOMA model. **Results:** Subjects with MS had more IR (HOMA-IR) than controls (3.35 ± 3.14 vs. 1.76 ± 0.53, $P = 0.029$) and secreted less insulin (HOMA-S) than controls (66.80 ± 69.66 vs. 144.27 ± 101.61, $P = 0.0003$), although plasma insulin levels were comparable in both groups (10.7 ± 10.2 vs. 8.2 ± 2.38, $P = 0.44$). HOMA-IR and HOMA-S were related with number of metabolic abnormalities. HOMA-IR was positively associated with body mass index, waist hip ratio, body fat mass, and percent body fat. HOMA-S was negatively associated with waist hip ratio, fasting plasma glucose and total cholesterol and positively with basal metabolic rate. Percent body fat was an independent predictor of HOMA-IR and waist hip ratio of HOMA-S in multiple regression analysis. **Conclusions:** Subjects with MS have increased IR and decreased insulin secretion compared with healthy controls. Lifestyle measures have been shown to improve IR, insulin secretion, and various components and effects of MS. Hence, there is an urgent need for public health measures to prevent ongoing epidemic of diabetes and cardiovascular disease.

**Key words:** Beta-cell function, insulin resistance, insulin secretion, metabolic syndrome

## Introduction

The clustering of cardiovascular risk factors, which include central adiposity, hypertension, hyperglycemia, and high triglycerides with low high-density lipoprotein (HDL) cholesterol levels, is termed the metabolic syndrome (MS). MS is known to strongly predict long-term risk of diabetes and cardiovascular disease (CVD).[1] Obesity can be said to be the predominant driving force behind the MS.[2] In obese persons, excess adipose tissue releases nonesterified fatty acids that predispose to ectopic fat accumulation in liver, muscle, and visceral adipose tissue stores.[3] Adipose tissue products are reported to affect systemic metabolism. Among these are adiponectin, leptin, inflammatory cytokines, plasminogen activator inhibitor-1, resistin, and angiotensinogen.[4] With obesity, the outputs of all of these products are higher except for adiponectin, which is abnormally low. Many studies implicate all of these changes to insulin resistance (IR) and relate them to development of diabetes.[3,4] Type 2 diabetes mellitus (T2DM) is characterized by decreased beta-cell function on the background of increased IR.[5] Hence, only putting emphasis on IR ignores the contribution of beta-cell dysfunction. Nonoxidative metabolic products of fatty acid spillover have been implicated in lipotoxicity and beta-cell dysfunction.[6] Beta-cell function has been not been well

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studied in MS.[7] Hence, we studied beta-cell function, assessed by HOMA model,[8] in subjects with MS.

## Materials and Methods

This study was carried at the Department of Endocrinology at a tertiary care centre (Army Hospital, Research and Referral). Subjects with age ≥30 years and ≤50 years (while excluding all postmenopausal women) were screened for the presence of MS according to International Diabetes Federation (IDF) criteria[9] as follows: central obesity (waist circumference: male > 90 cm, female > 80 cm) plus any two: raised triglycerides (>150 mg/dl), reduced HDL cholesterol (<40 mg/dl in men or <50 mg/dl in women), raised blood pressure (systolic ≥ 130 mmHg or diastolic ≥ 85 mmHg), or raised fasting plasma glucose (fasting plasma glucose ≥ 100 mg/dl). Age- and sex-matched healthy subjects were screened for absence of MS. Only those cases with waist circumference not fitting the above criteria, and absence of at least three of four parameters were included as controls.

A total of 50 drug naive subjects with MS (25 males and 25 females) and 24 controls (12 males and 12 females) were included in this study. All underwent clinical examination. Subjects with hepatic disease, renal disease, other endocrine diseases, alcoholism, infectious diseases, or receiving any medications, were excluded from the study.

Body mass index (BMI) was calculated by weight in kilograms divided by square of height in meters. Fasting blood samples were drawn for the estimation of fasting plasma glucose, renal and hepatic parameters, glycated hemoglobin (A1C), lipid profile, and fibrinogen. One aliquot were frozen at −80°C for measurement of plasma insulin. Urine spot samples were collected for measurement of urine microalbumin. The study was approved by the ethics committee of Army Hospital (Research and Referral), Delhi Cantt, and all subjects gave written informed consent.

Body fat measurement was done using InBODY composition analyser-biospacer manufactured by M/S Biodex Medical Systems Inc., New York. It measured waist hip ratio (WHR), body fat mass (BFM), percent body fat (PBF), and basal metabolic rate (BMR). Biochemical estimations were carried out using automated analyzer (Beckman Coulter, Synchrone CX-9 PRO, fully automated biochemistry analyzer, USA) and commercial kits (DiaSys Diagnostic Systems, Germany). The normal range for different biochemical parameters are as follows: fasting plasma glucose (70–100 mg/dl), serum creatinine (0.6–1.6 mg/dl), total cholesterol (<240 mg/dl), serum triglycerides (TG, <150 mg/dl), HDL cholesterol (>40 mg/dl for males and >50 mg/dl for females), and low-density lipoprotein (LDL) cholesterol (calculated) (<160 mg/dl). A1C was measured by HPLC method using commercial kit ClinRep®, Recipe Chemicals and Instruments, Germany, which was calibrated to value level of DCCT. Intraassay and interassay precision was 1–2% and 3%, respectively. Plasma insulin levels were measured by immuno-radiometric-assay using Immunotech, Czech Republic, commercial kits, with measurement range 0.5–300 μIU/ml and normal value 2.1–22 μIU/ml. It had sensitivity of 0.5 μIU/ml. Intraassay and interassay coefficient of variations were 4.3% and 3.4%, respectively. The HOMA model was used to calculate IR and insulin secretion. The formulae are as follows:

$$\text{Insulin resistance} = \frac{FI \times G}{22.5} \qquad \text{Insulin secretion} = \frac{20 \times FI}{G - 3.5}$$

where FI = fasting insulin μIU /ml, and G = fasting glucose (mmol/l).

Statistical analysis was carried out using EPI2003. Data were presented as mean ± SD or number (%) unless specified. All parametric data were analyzed by Student's *t* test. If Barlett's chi-square test for equality of population variances was <0.05, then Kruskal–Wallis test was applied. All nonparametric data were analyzed by chi-square test. Multiple regression analysis was done to ascertain association between various parameters. A *P* value of <0.05 was considered statistically significant.

## Results

This study was carried out in 50 cases of MS and 24 normal healthy controls. Basal characteristics of cases and controls are depicted in Table 1. BMI, body fat mass, and PBF were significantly higher in cases than controls. However, cases had significantly lower basal metabolic rate than controls. There were 34 (68%) cases with T2DM and 14 (32%) cases with impaired glucose tolerance (IGT). All controls had normal glucose tolerance. Hypertension was present in 26 cases (52%) among cases and none among controls. Among cases, TG, total cholesterol, and LDL were significantly higher and HDL was significantly lower than controls. However, 14 controls (58%) also had low HDL. Most of the cases (28, 56%) had four features of MS followed by all features (16, 32%) and 6 (12%) cases had three features of MS [Table 1].

### Beta-cell function

Subjects with MS had more IR (HOMA-IR) than controls (3.35 ± 3.14 vs. 1.76 ± 0.53, $P = 0.029$) and secreted less insulin (HOMA-S) than controls (66.80 ± 69.66 vs. 144.27 ± 101.61, $P = 0.0003$), although plasma insulin levels

**Table 1: Basic characteristics of cases and controls**

| Parameters | Cases ($n$ = 50) | Controls ($n$ = 24) | $P$ |
|---|---|---|---|
| Age | 43.4 ± 5.3 | 41.9 ± 4.0 | 0.21 |
| WHR | | | |
| Male | 1.18 ± 0.1 | 0.84 ± 0.04 | <0.0001 |
| Female | 1.21 ± 0.11 | 0.74 ± 0.03 | <0.0001 |
| BMI | 28.1 ± 2.1 | 22.5 ± 2.3 | <0.0001 |
| Body fat mass | 28.9 ± 11.5 | 12.9 ± 4.1 | <0.0001 |
| Body Fat (%) | 34.3 ± 7.0 | 18.4 ± 4.3 | <0.0001 |
| Basal metabolic rate | 1435 ± 134 | 1740 ± 119 | <0.0001 |
| Hypertension | 26 (52) | – | |
| Glycemic status | | | |
| Fasting PG | 136 ± 37 | 87 ± 6 | <0.0001 |
| Post-glucose PG | 207 ± 51 | 111 ± 22 | <0.0001 |
| A1C | 7.9 ± 0.9 | 5.0 ± 0.3 | <0.0001 |
| Insulin | 10.7 ± 10.2 | 8.2 ± 2.38 | |
| Insulin (median) | 10.24 | 7.30 | 0.44 |
| DM/IGT (%) | 34 (68)/16 (32) | – | |
| Lipid profile | | | |
| Triglycerides | 200 ± 67 (47.94) | 92 ± 30 | <0.0001 |
| HDL | 36 ± 6 (42.84) | 45 ± 13 (14.58) | <0.0001 |
| Total cholesterol | 221 ± 40 | 161 ± 33 | <0.0001 |
| LDL | 145 ± 47 | 109 ± 19 | <0.0001 |
| VLDL | 40 ± 14 | 20 ± 17 | <0.0001 |
| Urine microalbumin | 6.94 ± 1.62 | 6.86 ± 1.65 | 0.85 |
| Metabolic features | | | |
| None | – | 10 (42) | |
| One | – | 13 (58) | |
| Three | 6 (12) | – | |
| Four | 28 (56) | – | |
| Five | 16 (32) | – | |

WHR: Waist hip ratio, BMI: Body mass index, DM: Diabetes mellitus, IGT: Impaired glucose tolerance, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, Figures in parentheses are in percentage

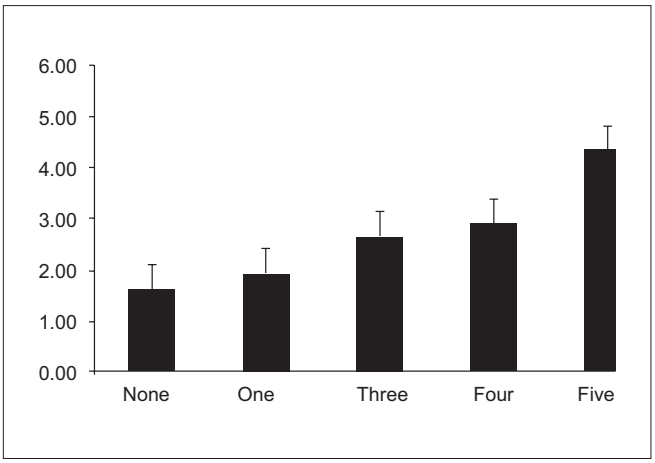


**Figure 1:** HOMA-IR according to the number of metabolic abnormalities

**Table 2: Univariate regression analysis of HOMA-IR among all subjects**

| Parameters | Beta coefficient | $r^2$ value | $P$ |
|---|---|---|---|
| Age | 0.031 | 0.02 | 0.232 |
| Sex | 0.150 | 0.0 | 0.812 |
| BMI | 0.157 | 0.08 | 0.013 |
| Body fat mass | 0.071 | 0.10 | 0.004 |
| Percent body fat | 0.109 | 0.15 | 0.005 |
| Basal metabolic rate | −0.004 | 0.07 | 0.02 |
| WHR | 4.381 | 0.09 | 0.007 |
| Fasting PG | 0.002 | 0.0 | 0.846 |
| Hypertension | 1.646 | 0.08 | 0.012 |
| Triglycerides | 0.007 | 0.04 | 0.109 |
| HDL | −0.035 | 0.02 | 0.293 |
| Total cholesterol | 0.005 | 0.01 | 0.45 |
| LDL | 0.002 | 0.0 | 0.831 |

BMI: Body mass index, WHR: Waist hip ratio, HDL: High-density lipoprotein, LDL: Low-density lipoprotein

were comparable in both groups (10.7 ± 10.2 vs. 8.2 ± 2.38 μIU/ml, $P = 0.44$). Subjects with IGT demonstrated more IR (6.29 ± 2.51 vs. 1.76 ± 0.53, $P < 0.00001$) and had higher insulin (15.6 ± 9.2 vs. 8.2 ± 2.38, $P < 0.00001$) than controls, but had similar HOMA-S (123.67 ± 69.66 vs. 144.27 ± 101.61, $P = 0.48$). Subjects with T2DM had comparable insulin levels (8.44 ± 9.96 vs. 8.2 ± 2.38, $P = 0.137$) and higher HOMA-IR (2.94 ± 3.36 vs. 1.76 ± 0.53, $P = 0.048$), but had significantly lower HOMA-S (40.04 ± 50.40 vs. 144.27 ± 101.61, $P < 0.00001$).

IR increased with increasing number of metabolic abnormalities [Figure 1]. There was no difference in HOMA-IR between sexes (2.76 ± 2.48 vs. 2.91 ± 2.93, $P = 0.81$). In univariate regression analysis, HOMA-IR showed strong positive association with BMI, body fat mass, and PBF, and negatively with basal metabolic rate. Among various parameters of MS, HOMA-IR was positively associated with WHR and hypertension. There was no association between HOMA-IR and FPG, and lipid parameters [Table 2].

Multiple regression analysis was done in stepwise manner in two parts: first, among metabolic parameters and second, among other parameters. Parameters with the highest significance value were regressed with other parameters. During multiple regression analysis among metabolic parameters, WHR maintained significance till hypertension was added [Table 3]. Only PBF remained positively associated with HOMA-IR when adjusted for anthropometric parameters, e.g., BMI, BFM, and BMR in multiple regression analysis [Table 4].

Insulin secretion measured by HOMA-S, decreased with increasing number of metabolic abnormalities [Figure 2]. There was no difference in HOMA-S between sexes (86.68 ± 17.13 vs. 97.17 ± 101.0, $P = 0.61$). In univariate regression analysis, HOMA-S was negatively associated with BMI and positively with basal metabolic rate. Among various parameters of MS, HOMA-S was negatively associated with WHR and fasting plasma

glucose. There was no association between HOMA-S and hypertension and lipid parameters [Table 5].

During multiple regression analysis, among metabolic parameters, fasting plasma glucose maintained strongly negative association after adjustment with hypertension, TG, HDL and WHR. WHR was also negatively associated with HOMA-S in multiple regression analysis [Table 6]. BMI lost its statistical significance on adjustment with anthropometric parameters, and none of the parameters showed association with HOMA-S in multiple regression analysis [Table 7].

## Discussion

MS is known to strongly predict long-term risk of diabetes and CVD[1] and have also been reported to experience increased morbidity and mortality.[10] It is becoming increasingly common in the United States and worldwide and is emerging as the dominant risk factor in Asia.[11] Although multiple influences contribute to the MS, the syndrome appears to be relatively uncommon in the absence of some excess body fat. As obesity increases, so does the prevalence of the MS.[12] In obese persons, excess adipose tissue releases varieties of factors including nonesterified fatty acids that predispose to ectopic fat accumulation in liver, muscle, and visceral adipose tissue

**Table 3: Multivariate regression analysis of HOMA-IR with metabolic parameters among all subjects**

| Parameters | Beta coefficient | *P* |
|---|---|---|
| **WHR*** | 4.381 | 0.007 |
| **WHR** + FPG | 5.424 | 0.003 |
| **WHR** + FPG + Triglycerides | 4.990 | 0.013 |
| **WHR** + FPG + Triglycerides + HDL | 4.881 | 0.011 |
| **WHR** + FPG + Triglycerides + HDL + Hypertension | 3.595 | 0.092 |

*Beta coefficient and *P* value for parameters are given in bold

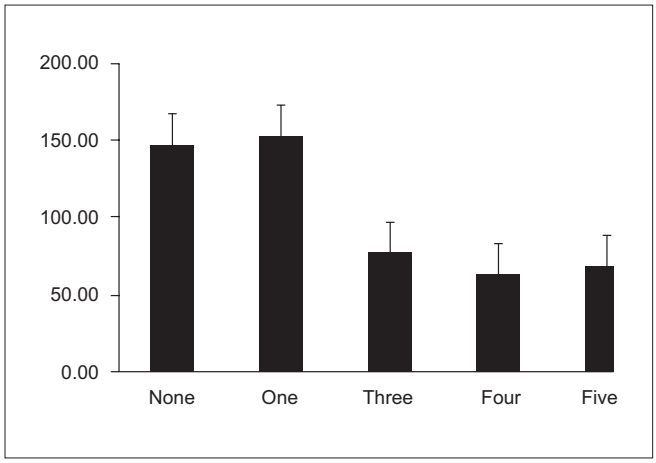


**Figure 2:** HOMA-S according to the number of metabolic abnormalities

**Table 4: Multivariate regression analysis of HOMA-IR with anthropometric parameters among all subjects**

| Parameters | Beta coefficient | *P* |
|---|---|---|
| **Percent Body Fat** | 0.109 | 0.005 |
| **Percent Body Fat** + BMR | 0.112 | 0.011 |
| **Percent Body Fat** + BMR + BMI | 0.207 | 0.013 |
| **Percent Body Fat** + BMR + BMI + BFM | 0.202 | 0.038 |

*Beta coefficient and *P* value for parameters are given in bold

**Table 5: Univariate regression analysis of HOMA-S among all subjects**

| Parameters | Beta coefficient | $r^2$ value | *P* |
|---|---|---|---|
| Age | −2.101 | 0.01 | 0.318 |
| Sex | 10.482 | 0.0 | 0.614 |
| BMI | −4.557 | 0.06 | 0.03 |
| Body fat mass | −0.971 | 0.02 | 0.251 |
| Percent body fat | −2.033 | 0.05 | 0.055 |
| Basal metabolic rate | 0.105 | 0.05 | 0.049 |
| WHR | −182.323 | 0.15 | 0.0005 |
| Fasting PG | −1.182 | 0.26 | <0.00001 |
| Hypertension | −26.927 | 0.02 | 0.223 |
| Triglycerides | −0.205 | 0.03 | 0.129 |
| HDL | 1.694 | 0.03 | 0.119 |
| Total cholesterol | 0.699 | 0.14 | 0.001 |
| LDL | −0.823 | 0.16 | 0.0004 |

BMI: Body mass index, WHR: Waist hip ratio, HDL: High-density lipoprotein, LDL: Low-density lipoprotein

**Table 6: Multivariate regression analysis of HOMA-S with metabolic parameters among all subjects**

| Parameters | Beta coefficient | *P* |
|---|---|---|
| **FPG** | −1.182 | <0.00001 |
| **FPG** + HT | −1.329 | <0.00001 |
| **FPG** + HT + Triglycerides | −1.312 | 0.00001 |
| **FPG** + HT + Triglycerides + HDL | −1.296 | 0.00002 |
| **FPG** + HT + Triglycerides + HDL + WHR | −1.164 | 0.00009 |
| FPG + HT + Triglycerides + HDL + **WHR** | −141.62 | −0.017 |

*Beta coefficient and *P* value for parameters are given in bold

**Table 7: Multivariate regression analysis of HOMA-S with anthropometric parameters among all subjects**

| Parameters | Beta coefficient | *P* |
|---|---|---|
| **BMI** | -4.557 | 0.03 |
| **BMI** + BFM | -0.923 | 0.018 |
| **BMI** + BFM + Percent Body Fat | -8.053 | 0.11 |
| **BMI** + BFM + Percent Body Fat + BMR | -8.404 | 0.10 |

*Beta coefficient and *P* value for parameters are given in bold

stores.[13] Ectopic fat links closely to risk factors and adversely affects beta-cell function through lipotoxicity.[6]

In this study, we evaluated 50 subjects with MS (25 males and 25 females) and 24 controls (12 males and 12 females). Different definitions have been proposed for MS,[10] and we have used IDF criteria as it provides ethnic specific criteria for central obesity. All cases had significantly higher WHR, BMI, body fat mass, and PBF than controls in both sexes, which is similar to Asian Indian obesity phenotype.[14] However, cases had significantly lower basal metabolic rate than controls. Contrary to this, one study reported higher BMR in morbidly obese subjects with MS.[15]

IR and insulin secretion were calculated with HOMA method that has been validated against insulin clamp studies.[16] Subjects with MS exhibited more IR and secreted less insulin than controls, although plasma insulin levels were comparable in both groups. This further support the hypothesis thata decrease in beta-cell function on the background of increased IR is the main determinant of progression to T2DM.[17-19] Similar to our study, Ajjan *et al*.[20] reported significantly higher HOMA-IR in 95 South Asian individuals with MS compared with controls. But another study from India did not find HOMA-IR as a core component of MS.[21] IR increased with increasing number of metabolic abnormalities. In univariate regression analysis, HOMA-IR was positively associated with BMI, body fat mass, and PBF, and negatively with basal metabolic rate, which was similar to reported by Snehlata *et al*.[22] in Indian young teenagers. Among various parameters of MS, HOMA-IR was positively associated with WHR and hypertension.

Insulin sensitivity is affected by age, genetic factors, life style, medications, and body fat distribution.[17] Waist circumference and waist hip ratio have been considered as the best surrogate marker of IR in epidemiological and clinical studies.[23,24] There was no association between HOMA-IR and FPG, and lipid parameters. Snehlata *et al*.[22] also found no correlation of HOMA-IR with lipid parameters. On the contrary, others found significant positive correlation between HOMA-IR and triglycerides, and fasting plasma glucose,[24,25] and inverse correlation between HOMA-IR and HDL cholesterol.[24,26] Only PBF remained positively associated with HOMA-IR when adjusted for anthropometric parameters, e.g., BMI, body fat mass, and BMR in multiple regression analysis.

Insulin secretion measured by HOMA-S decreased with increasing number of metabolic abnormalities. In univariate regression analysis, HOMA-S was negatively associated with BMI, and positively with basal metabolic rate. Among various parameters of MS, HOMA-S was negatively associated with WHR and fasting plasma glucose. There was no association between HOMA-S and hypertension and lipid parameters (TG and HDL). WHR was also negatively associated with HOMA-S in multiple regression analysis. Similarly, LDL levels showed strong negative association with HOMA-S. However, LDL cholesterol was not associated with IR in multivariate analysis. Hence in Indian subjects with T2DM, atherogenic dyslipidemia reflects underlying IR with insulin secretory defects. Moreover, in subjects with MS, increasing LDL cholesterol may indicate declining insulin secretory defects. Baez-Duarte *et al*.[6] studied 190 subjects with MS in Mexican population. They also found significantly higher HOMA-IR and decreased HOMA-S in cases compared with controls. Surprisingly their cases had similar HOMA-IR as in our study (3.35 ± 3.14 vs. 3.1 ± 1.9), but cases in the present study had much lower HOMA-S (66.80 ± 69.66) than their study (115.2 ± 62.3). They also reported significant negative correlation between HOMA-S and BMI, HDL, waist circumference and positive correlation between HOMA-IR and BMI. Similar to our study, they also found inverse correlation between HOMA-S with increasing numbers of parameters of MS. This indicates that Indian subjects, although having similar IR, secrete less insulin, due to beta-cell dysfunction, as compared with Mexican population. Similar observation has also been in Indians compared with Chinese and Creoles living in Mauritius, and in Brazilians who were at risk for diabetes.[27,28] The importance of HOMA-S, as an indicator of beta-cell function, is the detection of subjects with high risk of development of T2D as decreased beta-cell function is evident when fasting plasma glucose concentration is still well within the normal range.[17]

The primary limitation of this study is its cross-sectional design and the inherent possibility that genetic and/or lifestyle factors may have influenced the results of our group comparisons. However, in an effort to minimize the influence of lifestyle behaviors, we studied subjects of similar age who were nonsmokers, who were not currently taking medication that could influence insulin levels, and who did not differ in habitual physical activity.

## Conclusion

Subjects with MS have increased IR and decreased insulin secretion compared with healthy controls. IR is positively associated BMI, WHR, body fat mass, and PBF. Insulin secretion is negatively associated with WHR, fasting plasma glucose, total cholesterol, and positively with BMR. Lifestyle measures have been shown to improve IR, insulin secretion, and various components and effects of MS,[1,18,29-31] Hence

there is an urgent need for public health measures to prevent ongoing epidemic of diabetes and CVD.

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