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Original Article

High prevalence of metabolic obesity in India: The ICMR-INDIAB national study (ICMR-INDIAB-23)

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***Background & objectives*: While obesity usually produces cardio-metabolic dysfunction, some obese individuals are metabolically healthy, and conversely, some nonobese individuals have significant metabolic dysfunction. This study aims to assess the national prevalence of various obesity subtypes and their association with type 2 diabetes (T2D), coronary artery disease (CAD), and chronic kidney disease (CKD) in the Indian Council of Medical Research-India Diabetes (ICMR-INDIAB) study.**

***Methods*: The ICMR-INDIAB study is a nationally representative cross-sectional survey of 1,13,043 individuals aged ≥20 yr from urban and rural areas across 31 Indian States and Union Territories. In**

every fifth individual (n=19,370), venous blood glucose and lipids were measured. A body mass index (BMI) ≥25 kg/m^2 was defined as being obese, and metabolic obesity was diagnosed if two risk factors, out of the following: high waist circumference, high blood pressure, elevated blood glucose, raised serum triglycerides, or low HDL cholesterol, were present. Four subgroups were identified: Metabolically Healthy Non-Obese (MHNO), Metabolically Healthy Obese (MHO), Metabolically Obese Non-Obese (MONO), and Metabolically Obese Obese (MOO).

***Results*: The prevalence of various obesity subtypes was as follows: MONO: 43.3 per cent [95% confidence interval (CI): 42.6-44%], MOO: 28.3 per cent (27.7-28.9%), MHNO: 26.6 per cent (26-27.2%), and MHO: 1.8 per cent (1.6-2%). MONO was more prevalent in rural areas [Rural *vs.* Urban: MONO: 46 per cent (45-46.9%) *vs.* 39.6 per cent (37.8-41.3%), $P<0.001$]. MOO showed the highest risk for T2D and CAD, while MONO showed the highest risk of CKD, especially among females.**

***Interpretation & conclusions*: Individuals with MONO have a distinct phenotype with adverse metabolic consequences, highlighting the need to shift from body weight-focused approaches to broader strategies to identify and tackle non-communicable diseases (NCDs) in India.**

Key words Asian Indians - coronary artery disease - chronic kidney disease - diabetes - dyslipidaemia - hypertension - obesity - South Asians

Obesity, characterised by an elevated body mass index (BMI), has numerous adverse metabolic consequences on overall health. Worldwide, it is estimated that there are five million deaths every year from noncommunicable diseases (NCDs) attributable to high BMI, type 2 diabetes (T2D), cardiovascular disease (CVD), cancer, neurological disorders, chronic respiratory diseases, and chronic kidney disease (CKD)[1]. As per the World Health Organization (WHO)[2] there are 2.5 billion overweight adults and 890 million with obesity worldwide (representing 43% and 16%, respectively, of the global adult population). Generalised obesity is defined based on BMI cut points, while abdominal obesity is defined based on waist circumference (WC) or various waist-related indices like waist to hip ratio (WHR) or waist to height ratio (WHtR). Compared to other ethnicities, Asian Indians have a distinct susceptibility to develop T2D and other obesity-related metabolic disorders at a lower BMI. The 'Asian Indian Phenotype', marked by high levels of abdominal fat, insulin resistance, and dyslipidaemia with low HDL cholesterol and high serum triglycerides even with normal BMI, is believed to be a primary factor underlying this heightened risk[3-5]. The substantial burden of obesity in India was confirmed by a recent publication from the Indian Council of Medical Research (ICMR)-India Diabetes (INDIAB) study[6], which documented that there are an alarming 254 million and 351 million adults with generalised and abdominal obesity, respectively, in India[6].

Metabolic dysfunction is often linked with obesity, but some individuals with obesity have no cardiometabolic risk factors. A recent Lancet Commission on Clinical Obesity has distinguished between individuals with excess body fat who have evidence of obesity-associated illness (termed 'clinical obesity') and those who do not (termed 'preclinical obesity')[7]. Conversely, some normal-weight or lean individuals may have significant cardiometabolic risks. In the 1980s, Ruderman[8,9] introduced the concept of Metabolically Unhealthy Normal Weight (MUHNW) or Metabolically Obese Normal Weight (MONW) to describe individuals who are not obese based on BMI but show traits like hyperinsulinemia, insulin resistance, high triglycerides, and increased risk of coronary artery disease (CAD) and T2D. Another group of individuals who do not exhibit high-risk metabolic profiles yet meet conventional BMI criteria for obesity are classified as 'metabolically healthy obese (MHO)'[10,11]. As cardiometabolic risk varies in each of these subtypes, it is important to assess their prevalence to plan preventive or treatment strategies. This is particularly relevant in the Asian Indian context, where individuals tend to develop obesity-related comorbidities even in the non-obese ranges of BMI, leading to a delay of medical interventions when screening is based solely on BMI. There is no national data on the various obesity subtypes in India. We used data from the large, nationally representative, ICMR-INDIAB study to report on the prevalence of various obesity subtypes and evaluate the risk of each subtype for T2D, CAD, and CKD among adults in India.

Materials & Methods

This cross-sectional survey was undertaken by the department of Epidemiology and Research Operations and Diabetes Complications, Madras Diabetes Research Foundation, Chennai, Tamil Nadu, India. The study was approved by the Institutional Ethics Committee of the coordinating centres and individual States. Written informed consent was obtained from all study participants. The study was registered with the Clinical Trials Registry of India (CTRI/2019/03/018095).

Sampling and study population: Adults aged ≥20 yr were recruited from the ICMR-INDIAB study[12-19], a cross-sectional, population-based survey in India. The study methodology, including sampling strategies, sample size, and phases, have been described previously[12] and provided in the supplementary material. Of the total 1,19,022 individuals from 31 States studied, 1,13,043 individuals participated in the study, yielding a response rate of 95 per cent.

Assessments: Data on medical history, family history of diabetes, physical activity, and socioeconomic status were collected using a standardised and structured questionnaire in all participants. Self-reported data included alcohol and smoking (current or in the prior six months). Physical activity was measured using a validated Physical Activity Questionnaire[18]. Individuals were classified into two categories based on their physical activity level (PAL), which was determined by dividing their total energy expenditure for 24 h by their basal metabolic rate: 1.4-1.69 for inactive individuals and 1.7-2.4 for active individuals. Dietary information was obtained using the MDRF-Food Frequency Questionnaire (M-FFQ)[20], a validated, interviewer-administered tool. This meal-based questionnaire lists 222 common food items from urban and rural areas. Daily intake of calories, macronutrients, dietary fibre, and fatty acids was calculated using 'EpiNu' Software (Version 2.0). The nutrient densities expressed as the percentage of energy derived from carbohydrates, proteins, and fats were estimated and used in the analysis.

Standardised methods were used to determine blood pressure (BP) and anthropometric measurements, including weight, height, and waist circumference[21]. Height (in centimetres) was measured using a stadiometer (SECA Model 214, Seca Gmbh Co, Hamburg, Germany). Body weight (in kilograms) was measured using an electronic weighing scale (SECA Model 807, Seca Gmbh Co, Hamburg, Germany) placed on a flat horizontal surface. BMI was calculated using the formula: weight (kg)/height $(m)^2$. Waist circumference (centimetres) was measured using a non-stretchable measuring tape at the smallest horizontal girth between the costal margins and the iliac crest at the end of expiration. Using an electronic sphygmomanometer (Omron HEM-7101; Omron Corporation, Tokyo, Japan), BP was recorded to the nearest 1 mm Hg. The final reading was recorded as the average of two measurements taken five minutes apart. Inter-observer and intra-observer coefficients of variation between the field technicians were documented and were less than 5 per cent. Equipment with the same specifications was used during the investigations as a quality control measure.

A One Touch Ultra glucose meter (LifeScan Johnson & Johnson, Milpitas, California) was used to assess each individual's capillary blood glucose (CBG) after confirming an overnight fast of 8-12 h. Participants were administered 82.5 g of glucose (75 g of anhydrous glucose) for an oral glucose tolerance test, and the 2-h post-load CBG was measured. For individuals with self-reported diabetes, only fasting CBG was measured. In every fifth participant and individuals with diabetes, a venous sample was taken for the measurement of lipids, glycated haemoglobin (HbA1c), and creatinine. The Variant™ II Turbo machine (Bio-Rad, Hercules, CA) was utilised for high-pressure liquid chromatography to estimate HbA1c. An autoanalyzer [model 2700/480; Beckman Coulter AU (Olympus, County Clare, Ireland)] was used to measure serum cholesterol (cholesterol esterase oxidase-peroxidase-amidopyrine method), serum triglycerides (glycerol phosphate oxidase-peroxidase-amidopyrine method), and high-density lipoprotein cholesterol (direct method; polyethylene glycol-pre-treated enzymes). Serum creatinine was measured using the Jaffe Kinetic method. For biochemical assays conducted at the central laboratory, the intra-assay and inter-assay coefficients of variation ranged between 3.1 per cent and 7.6 per cent. A resting 12-lead electrocardiogram (ECG) was recorded in every fifth individual, and those with diabetes, and were graded using Minnesota coding. For the present analysis, the sample size (n) was 19,370, as lipids, creatinine, and ECG (needed to categorise individuals for metabolic obesity and assess complications) were performed only on every fifth participant.

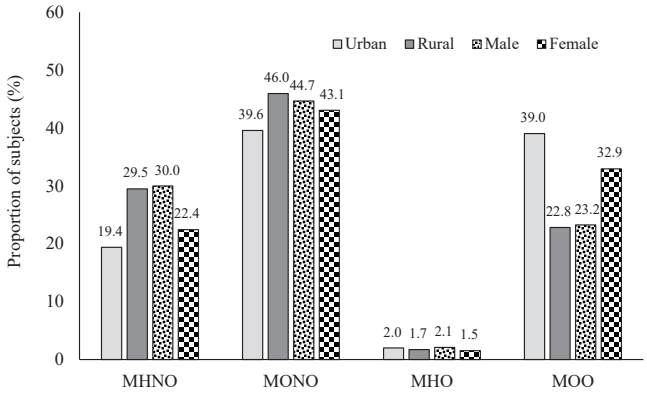

Fig. 1. Different subtypes of obesity in the Asian Indian population. MHNO, metabolically healthy non obese; MONO, metabolically obese non obese; MHO, metabolically healthy obese; MOO, metabolically obese obese.

Definitions: Metabolic obesity was defined as having ≥2 components of metabolic syndrome: (*i*) waist circumference ≥90 cm in males and ≥80 cm, in females, (*ii*) fasting blood glucose (FBG) ≥100 mg/dl, (*iii*) BP ≥130/85 mmHg or on anti-hypertensive medications, (*iv*) serum triglyceride levels ≥150 mg/dl or (*v*) HDL cholesterol <40 mg/dl for males and <50 mg/dl for females. Four obesity subtypes were defined as below: Metabolically Healthy Non-Obese (MHNO): absence of metabolic obesity and BMI <25 kg/m^2, MHO: absence of metabolic obesity and BMI ≥25 kg/m^2, Metabolically Obese Non-Obese (MONO): presence of metabolic obesity and BMI <25 kg/m^2, Metabolically Obese Obese (MOO): presence of metabolic obesity and BMI ≥25 kg/m^2.

Diabetes was defined as fasting CBG ≥126 mg/dl (7.0 mmol/l), or 2-h post-oral glucose load CBG ≥200 mg/dl (11.1 mmol/l), or a physician diagnosis of diabetes[22].

CAD was diagnosed based on a recorded history of myocardial infarction (MI) or drug treatment for CAD and/or Minnesota codes: Q wave changes (1-1-1 to 1-1-7), ST segment depression (4-1 to 4-2) T-wave abnormalities (5-1 to 5-3) on the resting ECG[23].

CKD was diagnosed if the estimated glomerular filtration rate (eGFR) was <60 ml/min/1.73m^2 at the time of study. eGFR[24] was derived using the following formula:

$$GFR=141 * \min(Scr/\kappa,1)^{\alpha} * \max(Scr/\kappa, 1)^{-1.209} * 0.993^{Age} * 1.018 \text{ (if female)}$$

[κ=0.7 if female, κ=0.9 if male; α=-0.329 if female, α=-0.411 if male; min=the minimum of Scr/κ or 1, max=the maximum of Scr/κ or 1; Scr=serum creatinine (mg/dl)]

Statistical analysis: The proc survey (frequency/mean) procedure was used to analyse the data collected from complex survey designs, ensuring that the statistical analyses and inferences drawn were accurate and representative of the target population. These procedures are used when dealing with survey data that involves stratification, clustering, and survey weights. Supplementary material (page 9) provides the sampling weights calculation to adjust for sampling at different levels within each State.

Survey-adjusted linear regression was used to compute the mean, and the Wald χ^2 test was applied to compare the proportions of variables between subtypes (MHNO *vs.* MHO, MONO, and MOO; urban *vs.* rural within each subtype of obesity). Univariate logistic regression was used to estimate risk for T2D, CAD, and CKD for individuals with different subtypes using MHNO as a reference group. The variables that were clinically relevant and/or significant in the univariate logistic regression, such as, sex, age, education, smoking, income, family history of diabetes, physical activity, and dietary nutrient densities (Carbohydrates %E, Fat %E and Protein %E) were adjusted in multiple logistic regression. A P <0·05 was considered statistically significant. To analyse data, we used Statistical Data Analysis Software (version 9.4; SAS Institute, Cary, NC, USA).

Results

Figure 1 presents the prevalence of different subtypes of obesity in the study population. The most prevalent obesity subtype was MONO, accounting for 43.3 per cent [95% confidence interval (CI): 42.6-44.%], followed by MOO (28.3%; 95% CI: 27.7-28.9%), MHNO (26.6%; 95% CI: 26.0-27.2%) and MHO (1.8%; 95% CI: 1.6-2.0%). The obese subtypes (MHO and MOO) were more common in the urban areas (urban *vs.* rural: MHO: 2%; 95% CI: 1.5-2.4% *vs.* 1.7%; 95% CI: 1.5-2%; P=0.304; MOO: 39%; 95% CI: 37.2-40.8% *vs.* 22.8%; 95% CI: 22-23.7%; P<0.001). MOO was more common among females (male *vs.* female: 23.2%; 95% CI: 22.2-24.3% *vs.* 32.9%; 95% CI: 31.7-34.2%; P<0.001), whereas MHO was more common in males (male *vs.* female: 2.1%; 95% CI: 1.8-2.4% *vs.* 1.5%; 95% CI: 1.3-1.8%; P=0.011).

Table I. Anthropometric and metabolic characteristics of the study population based on the subtypes of obesity

Characteristics	MHNO	MONO	MHO	MOO
n (%)	5,151 (26.6)	8,389 (43.3)	352 (1.8)	5,478 (28.3)
Male	57.1 (55.6-58.7)	50.8 (49.5-52.1)**	57.6 (51.8-63.5)	41.3 (39.6-43)**
Age (yr)	38.9 (0.24)	45.8 (0.2)**	38.5 (0.77)	45.7 (0.23)**
BMI (kg/m^2)	19.8 (0.04)	21.4 (0.03)**	27.5 (0.19)**	28.6 (0.06)**
Waist circumference - overall (cm)	72.7 (0.14)	80.0 (0.13)**	83.3 (0.72)**	94.3 (0.18)**
Waist circumference - male (cm)	75.1 (0.17)	81.8 (0.18)**	84.8 (0.92)**	96.2 (0.25)**
Waist circumference - female (cm)	69.6 (0.19)	78.1 (0.19)**	81.2 (1.08)**	93.0 (0.24)**
Systolic blood pressure (mmHg)	120 (0.28)	134 (0.29)**	121 (0.69)	135 (0.32)**
Diastolic blood pressure (mmHg)	76.0 (0.18)	83.3 (0.15)**	76.9 (0.49)	85.3 (0.2)**
Fasting plasma glucose (mg/dl)	89.8 (0.29)	109 (0.52)**	90.3 (0.64)	116 (0.74)**
HbA1c (%)	5.20 (0.01)	5.67 (0.02)**	5.30 (0.04)*	5.99 (0.03)**
Cholesterol (mg/dl)	156 (0.66)	169 (0.65)**	175 (2.72)**	185 (0.76)**
Triglycerides (mg/dl)	91.9 (0.68)	151 (1.43)**	103 (4)*	175 (2.51)**
HDL cholesterol (mg/dl)	45.6 (0.21)	38.9 (0.15)**	47.2 (0.65)*	38.7 (0.18)**
LDL cholesterol (mg/dl)	91.7 (0.56)	100 (0.56)**	107 (2.28)**	111 (0.72)**
Family history of diabetes	5.8 (5.1- 6.6)	8.8 (8.0- 9.5)**	13.6 (9.5-17.7)**	16.6 (15.5-17.8)**
Physically active	46.4 (44.6-48.1)	39.8 (38.4-41.2)**	37.5 (31.7-43.2)*	30.7 (29.2-32.2)**
Fruits & vegetables intake (≥3 servings/day)	20.2 (18.6-21.7)	19.7 (18.4-20.9)	17.9 (12.9-22.8)	17.7 (16.3-19)**
Current smokers	18.1 (16.8-19.4)	16.5 (15.4-17.6)	14.1 (10.0-18.3)	8.9 (8.0- 9.9)*

Values are presented as mean (standard error) or percentage (95% Confidence Interval, CI) as appropriate; $P^*<0.05$;$^{**}<0.001$. Compared to Metabolically Healthy Non Obese (MHNO); Metabolically Healthy Non Obese (MHNO): No metabolic obesity, BMI<25 kg/m^2; Metabolically Obese Non Obese (MONO): Metabolic obesity present, BMI<25 kg/m^2; Metabolically Healthy Obese (MHO): No metabolic obesity, BMI≥25 kg/m^2; Metabolically Obese Obese (MOO): Metabolic obesity present, BMI≥25 kg/m^2

The non-obese subtypes were more common in the rural areas (rural *vs.* urban MHNO: 29.5%; 95% CI: 28.6-30.4% *vs.* 19.4%; 95% CI: 18-20.8%; $P<0.001$; MONO: 46%; 95% CI: 45-46.9% *vs.* 39.6%; 95% CI: 37.8-41.3%; $P<0.001$) and among males (males *vs.* females MHNO: 30%; 95% CI: 28.8-31.1% *vs.* 22.4%; 95% CI: 21.4-23.4%, $P<0.001$; MONO: 44.7%; 95% CI: 43.5-45.8% *vs.* 43.1%; 95% CI: 41.8-44.4%: $P=0.079$).

The State-specific prevalence of the subtypes of obesity is depicted in supplementary figure. MONO was the most common subtype in all States, with prevalence ranging from 34.8 per cent (95% CI: 27.7-41.9%) in Delhi to 56.7 per cent (95% CI: 52.6-60.8%) in Tripura. Supplementary table I shows the contribution of various components to metabolic obesity. Among the five components of metabolic obesity, HDL-C had the highest contribution (79.2%; 95% CI: 78.5-79.9%), followed by BP (69.5%; 95% CI: 68.7-70.2%), fasting blood glucose (57.7%; 95% CI: 56.9-58.6%), WC (55.7%; 95% CI: 54.9-56.6%), and triglycerides (42.8%; 95% CI: 42-43.7%). When analysing the combination of different components of metabolic obesity, two components were present in 42.6 per cent (95% CI: 41.8-43.4%) of individuals, three in 30.7 per cent (95% CI: 29.9-31.5%), four in 19.2 per cent (95% CI: 18.6-19.9%), and all five in 7.5 per cent (95% CI: 7.1-8%).

Table I presents the anthropometric and metabolic characteristics of the study population characterised by obesity subtypes. The MOO subtype was more prevalent in females, while other subtypes were more prevalent in males. Compared to the healthy reference group, *i.e.*, MHNO, all other subtypes were older (except MHO), had significantly higher BMI, WC, systolic and diastolic BP (except MHO), fasting blood glucose (except MHO), HbA1c, triglycerides, and total and LDL cholesterol. As expected, the MOO subtype

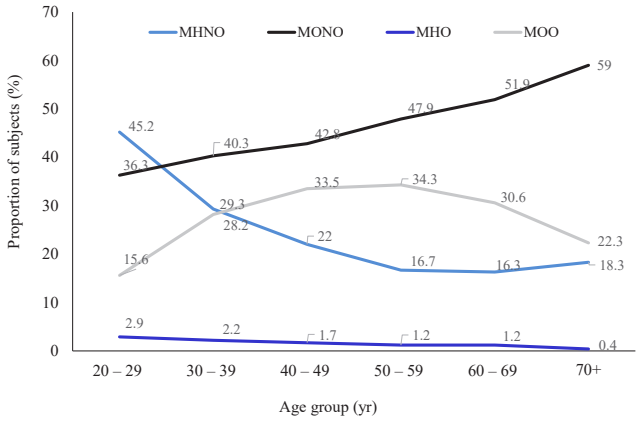

Fig. 2. Age-specific prevalence of subtypes of obesity in the study population.

had the highest values of all these parameters. While physical inactivity and family history of diabetes were higher in the obese subtypes (MHO and MOO), smoking rates were higher in the non-obese subtypes (MHNO and MONO). Compared to the reference group (MHNO), MOO reported significantly lower intake of fruits and vegetables.

Figure 2 presents the age-specific prevalence of the various obesity subtypes. With increasing age, the prevalence of MHNO and MHO decreased, and that of MONO increased. The prevalence of MOO rose until the age of 60, after which it declined.

The risk of T2D, CAD and CKD among the various obesity subtypes, using MHNO as a reference group, is presented in table II. The risk of T2D was highest in MOO [Odds ratio (OR)=12.89; 95% CI: 9.54-17.43; P<0.001], especially among females, followed by MONO (OR=6.90; 95% CI: 5.10-9.34; P<0.001) after adjusting for confounders. Similarly, the risk of CAD was highest for MOO (OR=1.92; 95% CI: 1.27-2.91; P=0.002), followed by MONO (OR=1.77; 95% CI: 1.22-2.57; P=0.003) after adjusting for age, sex, education, physical activity, smoking, income, family history of diabetes and dietary nutrient densities (Carbohydrates %E, Fat %E, and Protein %E). Among both MONO and MOO, the risk for CAD was higher in males than in females. For CKD, after adjusting for confounders, MONO had the highest risk followed by MOO. Among MONO, females were shown to have a higher risk of CKD, while among MOO, males were found to have a higher risk. Sensitivity analysis with different BMI cut points (27.5 and 30 kg/m^2) showed similar results with MONO and MOO having significantly higher risk for T2D, CAD and CKD (Supplementary Table II).

Discussion

In this nationally representative study of India, we show that (*i*) the largest proportion (43.3%) of the population in India is metabolically obese but with BMI<25 kg/m^2 (referred to as MONO), (*ii*) MONO is more prevalent in rural areas and among males, (*iii*) MOO showed highest risk for diabetes and CAD, while MONO showed the highest risk of CKD especially among females.

One of the simplest ways to identify obesity is by anthropometric measures, such as the BMI, WC, WHR, and WHtR. BMI is the most commonly used and is recommended by the International Obesity Task Force (IOTF) and the WHO[25]. However, BMI does have certain drawbacks, as it cannot be used to assess body composition and cannot distinguish between lean and fat mass[7]. Therefore, a normal BMI may conceal the presence of excess body fat. WC, WHR, and WHtR are considered better predictors of health risks associated with obesity than BMI, as they provide information on fat distribution and central adiposity.

Studies worldwide have attempted classifications of individuals based on obesity and metabolic abnormalities. A systematic analysis of 27 prospective and cross-sectional studies revealed around 30 definitions for metabolic health[26]. For instance, MONO was characterised by Lee *et al*[27] as having a BMI <25 kg/m^2 and at least two metabolic risk factors among high fasting glucose, hypertension, high triglycerides, low HDL, and HOMA-IR ≥90th percentile. MONO was described by Goday *et al*[28] as having a normal BMI (18.5-25 kg/m^2) and poor metabolic health as determined by the NCEP-ATP III guidelines (high fasting glucose, high triglycerides, low HDL-C, and high WC). MONO was defined as BMI<25 kg/m^2 who developed T2D during follow up by Eckel *et al*[29]. Triglyceride glucose (TyG) index was suggested as a parameter for classification by Lee *et al*[30] who defined MONO as individuals with a normal BMI (18.5-25 kg/m^2), a high metabolic syndrome score, an abnormal TyG index value, or a HOMA-IR score in the highest quartile. The use of varying criteria for defining metabolic abnormalities makes comparing results between different studies challenging. It may underlie the wide variations in reported prevalence rates of the MHO phenotype (ranging from 6 to 75%). The United States National Health and Nutrition Examination

Table II. Risk of type 2 diabetes, coronary artery disease (CAD), and chronic kidney disease (CKD) among the subtypes of obesity

	Risk of type 2 diabetes		
	Overall, n (95% CI)	Male, n (95% CI)	Female, n (95% CI)
Unadjusted model			
Metabolically Healthy Non Obese (MHNO)	1 (Reference)	1 (Reference)	1 (Reference)
Metabolically Obese Non Obese (MONO)	10.03 (7.74-13.01), *P*<0.001	9.14 (6.74-12.41), *P*<0.001	13.65 (8.1-22.99), *P*<0.001
Metabolically Healthy Obese (MHO)	0.77 (0.27-2.14), *P*=0.613	0.49 (0.12-2.07), *P*=0.335	1.56 (0.35-6.96), *P*=0.561
Metabolically Obese Obese (MOO)	19.79 (15.21-25.75), *P*<0.001	14.92 (10.92-20.39), *P*<0.001	33.38 (19.87-56.07), *P*<0.001
Adjusted model			
Metabolically Healthy Non-Obese (MHNO)	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Metabolically Obese Non-Obese (MONO)	6.90 (5.10-9.34), *P*<0.001	6.38 (4.48-9.08), *P*<0.001	8.79 (4.65-16.64), *P*<0.001
Metabolically Healthy Obese (MHO)	0.75 (0.26-2.21), *P*=0.605	0.47 (0.10-2.17), *P*=0.336	1.52 (0.32-7.24), *P*=0.601
Metabolically Obese Obese (MOO)	12.89 (9.54-17.43), *P*<0.001	9.94 (6.91-14.28), *P*<0.001	19.69 (10.53-36.82), *P*<0.001
	Risk of Coronary Artery Disease		
Unadjusted model			
Metabolically Healthy Non-Obese (MHNO)	1 (Reference)	1 (Reference)	1 (Reference)
Metabolically Obese Non-Obese (MONO)	2.18 (1.53-3.11), *P*<0.001	2.79 (1.77-4.4), *P*<0.001	1.64 (1.01-2.68), *P*=0.045
Metabolically Healthy Obese (MHO)	1.39 (0.42-4.58), *P*=0.591	0.93 (0.21-4.09), *P*=0.923	1.86 (0.36-9.47), *P*=0.456
Metabolically Obese Obese (MOO)	2.28 (1.57-3.29), *P*<0.001	2.72 (1.66-4.48), *P*<0.001	1.88 (1.16-3.04), *P*=0.010
Adjusted model			
Metabolically Healthy Non-Obese (MHNO)	1 (Reference)	1 (Reference)	1 (Reference)
Metabolically Obese Non-Obese (MONO)	1.77 (1.22-2.57), *P*=0.003	1.92 (1.17-3.16), *P*=0.010	1.52 (0.84-2.76), *P*=0.168
Metabolically Healthy Obese (MHO)	1.74 (0.44-6.81), *P*=0.429	1.24 (0.27-5.74), *P*=0.786	2.35 (0.3-18.67), *P*=0.418
Metabolically Obese Obese (MOO)	1.92 (1.27-2.91), *P*=0.002	1.99 (1.12-3.52), *P*=0.019	1.84 (0.99-3.42), *P*=0.054
	Risk of Chronic Kidney Disease		
Unadjusted model			
Metabolically Healthy Non-Obese (MHNO)	1 (Reference)	1 (Reference)	1 (Reference)
Metabolically Obese Non-Obese (MONO)	2.35 (1.73-3.2), *P*<0.001	1.9 (1.34-2.68), *P*<0.001	4.33 (2.2-8.5), *P*<0.001
Metabolically Healthy Obese (MHO)	0.42 (0.1-1.73), *P*=0.228	0 (0-0), *P*<0.001	2.14 (0.46-9.99), *P*=0.332
Metabolically Obese Obese (MOO)	1.88 (1.32-2.69), *P*<0.001	1.92 (1.22-3), *P*=0.005	2.81 (1.42-5.55), *P*=0.003
Adjusted model			
Metabolically Healthy Non-Obese (MHNO)	1 (Reference)	1 (Reference)	1 (Reference)
Metabolically Obese Non-Obese (MONO)	1.81 (1.21-2.7), *P*=0.004	1.70 (1.08-2.68), *P*=0.021	8.79 (4.65-16.64), *P*<0.001
Metabolically Healthy Obese (MHO)	0.71 (0.16-3.1), *P*=0.654	0	1.52 (0.32-7.24), *P*=0.601
Metabolically Obese Obese (MOO)	1.63 (1.04-2.56), *P*=0.033	1.85 (1.08-3.16), *P*=0.026	19.69 (10.53-36.82), *P*<0.001

Values are presented as Odds ratio (95% Confidence Interval, CI); Adjusted for age, sex, education, physical activity, smoking, income, family history of diabetes and dietary nutrient densities (Carbohydrates %E, Fat %E and Protein %E)

Survey (NHANES, 1999-2004) data revealed that among men and women of normal weight, 30.1 and 21.1 per cent, respectively, had abnormal metabolic status[31].

Results from a study on 2,350 urban Asian Indian adults suggested that metabolic obesity had different clinical implications than generalised obesity[32]. The MONO phenotype is characterised by increased

subcutaneous and visceral fat and insulin-resistant glucose metabolism. This makes the pancreas produce more insulin, exhausting it over time and reducing its ability to make insulin[33,34]. It is therefore not surprising that individuals with MONO in our study were found to have an elevated risk of T2D. Studies have also suggested that the high prevalence rates of MONO may explain the increased risk for T2D in Asian Indians[35]. Our study findings showed that MONO was also associated with elevated risk of CAD and CKD. Metabolic risk factors such as insulin resistance, hypertension, dyslipidaemia, and inflammation, common in MONO, might play a synergistic role in the development and progression of atherosclerosis, the underlying cause of CAD. Insulin resistance promotes a pro-inflammatory and pro-thrombotic State, fostering the formation of atherosclerotic plaques in the coronary arteries. Elevated triglycerides and decreased HDL-cholesterol further exacerbate atherosclerosis, and hypertension adds to the burden by accelerating endothelial dysfunction and vascular damage. Insulin resistance and associated hyperinsulinemia contribute to renal damage through glomerular hyperfiltration and inflammation. Dyslipidaemia exacerbates renal injury by promoting glomerulosclerosis and tubulointerstitial fibrosis, and hypertension contributes to renal damage via increased glomerular pressure and renal vascular injury. According to the Tehran Lipid Glucose Study (TLGS)[36], compared to individuals with normal weight and normal metabolic status, those with MHO and MONW (MONO) had a 23 and 43 per cent increased risk, respectively, of developing CKD.

Our findings that more than a third of individuals, even in the 20-29 yr age group, had MONO is a concerning trend given the long-term health risks associated with metabolic dysfunction. The differences in MONO and MOO prevalence between sexes are likely driven by the complex interplay of hormones and metabolism. They may have different health consequences, as females have proportionally more fat mass and males, more muscle mass. Differences in body shape and fat distribution between males and females may result in different patterns of abdominal fat accumulation and its relationship with cardiometabolic risk factors.

Based on studies[37-42] that examined the changes in metabolic phenotype associated with obesity over time, MHO is suggested to be a transient phase, which progresses to MOO subtype with time. The Coronary Artery Risk Development in Young Adults (CARDIA) cohort[43], with a mean follow up of 9.1 yr, reported that individuals with MHO had a significantly higher risk of coronary artery calcium development compared to MHNW individuals. Between 15 and 25 yr, up to 60 per cent of individuals with MHO transitioned to metabolically unhealthy obesity, suggesting that MHO serves as an intermediate phenotype between metabolically low- and high-risk obese individuals.

As individuals with MONO are at higher risk of adverse metabolic consequences, early identification is crucial for targeted interventions. However, when BMI is used as the sole measure for identifying obesity, individuals with MONO may not be detected, and thereby lose out on opportunities for early intervention and prevention of NCDs. While diagnostic tests like insulin resistance assessments and detailed lipid panels are available to identify individuals at risk, simpler and more accessible screening tools, such as WC, WHR, WHtR, and BP measurements, can help detect this unfavourable phenotype early. Raising awareness among healthcare professionals that a ‘normal’ body weight or BMI does not necessarily mean healthy will sensitise them to screen individuals for metabolic ill-health using other simple, readily available tools, particularly in rural and other resource-constrained settings. This will also enable early interventions in the form of lifestyle modifications with or without medication to reduce cardiometabolic risk. Regular monitoring of metabolic markers is also essential to monitor changes and appropriately modify interventions in these at-risk individuals. A multifaceted approach, involving national, regional, State, and district-level action and community participation, is needed to combat the obesity and cardiometabolic disease challenge. In India, national policies are being established to tackle obesity and cardiometabolic diseases. The Food Safety and Standards Authority of India (FSSAI) has introduced initiatives to regulate the advertising and labelling of unhealthy foods, particularly those high in fat, sugar, and salt. The National Programme for Prevention and Control of Non-Communicable Diseases (NP-NCD) focuses on early detection and prevention of NCDs through lifestyle interventions, including promoting physical activity, dietary modifications, and smoking cessation. Some Indian States have implemented targeted measures such as the ‘fat tax’ on high-calorie foods, including burgers, pizzas, and sugary drinks, to discourage unhealthy eating and encourage healthier food choices.

The strengths of our study include the survey of a large, community-based, nationally representative

sample of men and women spanning a wide age range, as well as the standardised assessment of cardiometabolic risk factors. Some of the limitations of our study include the fact that it is cross-sectional, which prevents us from assessing causal implications or looking at long-term outcomes. For example, it is unclear whether MONO precedes disease onset or is a consequence; longitudinal studies are needed to confirm causality. While we have based the diagnosis of diabetes on CBG, which has a higher coefficient of variation than venous plasma, we have previously demonstrated a strong correlation between CBG and venous plasma estimations[44]. Finally, different study phases were carried out at different time points, which was unavoidable due to sampling across a vast geographical terrain.

In conclusion, clear definitions are necessary to classify obese individuals across clinical and research studies. Individuals with MONO have a unique phenotype with adverse metabolic consequences. They represent a significant public health challenge in India as they constitute more than 43 per cent of the adult population, have an elevated risk of T2D, CAD, and CKD, and can only be identified through targeted and specific screening tests, as they do not have any of the easily identifiable phenotypic indicators of obesity. Weight-centric paradigms must shift to a more holistic understanding of metabolic health to empower individuals to make informed choices and take proactive steps toward a healthier future. In the clinical scenario, identifying individuals with MONO during a routine health check-up will help initiate suitable interventions before developing undesired outcomes, such as CAD and CKD.

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