Eurasian Journal of Analytical Chemistry
ISSN: 1306-3057 OPEN ACCESS 2023 18 (1): 82-88

The Relationship Between Insulin Resistance And Lipid Profiles In Obese South Asians: An Observational Study

Zarreen Firdaus[1*], Prof. Dr. Shreya Nigoskar[2]

[1*]Ph.D. Scholar, Department of Biochemistry, Index Medical College, Hospital and Research Center, Indore – (M.P.) 452016, India
[2]Research Supervisor, Department of Biochemistry, Index Medical College, Hospital and Research Center, Indore – (M.P.) 452016, India

***Corresponding Author:** Zarreen Firdaus
*Ph.D. Scholar, Department of Biochemistry, Index Medical College, Hospital and Research Center, Indore – (M.P.) 452016, India, **Email:** zarreen412@gmail.com

Abstract
Objective: This study aims to examine the relationship between insulin resistance (IR) and lipid profiles in obese South Asians, a population that presents a unique phenotype known as the "Asian Indian Phenotype." This phenotype predisposes individuals to metabolic disorders, including IR and dyslipidemia, at lower body mass index (BMI) levels. Given the elevated cardiovascular risk in this population, this research explores the extent to which IR correlates with lipid abnormalities and cardiovascular risk factors.
Materials and Methods: A cross-sectional study was conducted at Index Medical College, Hospital and Research Center, Indore – (M.P.), among 165 participants, including 115 obese and 50 non-obese controls. The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) was used to quantify insulin resistance. Lipid profiles, including triglycerides, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C), were measured. Correlations between HOMA-IR and lipid parameters were evaluated, and multivariate linear regression was used to adjust for confounders.
Results: Obese participants demonstrated significantly higher insulin resistance (HOMA-IR: 4.3 ± 1.9) compared to controls (HOMA-IR: 1.9 ± 0.8, $p < 0.001$). These participants also exhibited significantly elevated triglycerides (184.7 ± 49.1 mg/dL), higher LDL-C (134.8 ± 38.2 mg/dL), and lower HDL-C (39.8 ± 7.9 mg/dL) compared to the control group. Significant positive correlations were observed between HOMA-IR and triglycerides ($r = 0.49$, $p < 0.001$), and negative correlations between HOMA-IR and HDL-C ($r = -0.40$, $p < 0.05$).
Conclusion: The study confirms that obesity in South Asians is closely associated with insulin resistance and dyslipidemia, both of which significantly contribute to elevated cardiovascular risks. Given the high burden of metabolic disorders in this population, interventions aimed at reducing insulin resistance through lifestyle changes and pharmacological treatments are essential in mitigating future cardiovascular events.

Keywords: Insulin resistance, obesity, dyslipidemia, cardiovascular risk, South Asian phenotype, lipid profiles, HOMA-IR

Introduction

Obesity and insulin resistance (IR) are at the forefront of metabolic disorders worldwide, with South Asians disproportionately affected by these conditions. The unique metabolic profile of South Asians, referred to as the **"Asian Indian Phenotype,"** is marked by increased visceral fat, insulin resistance, and a higher risk of dyslipidemia and cardiovascular diseases (CVD) despite lower body mass index (BMI) levels (1,2). While many ethnic groups experience metabolic disorders at higher BMIs, South Asians face heightened risk even at BMI values traditionally considered non-obese (3).
The prevalence of metabolic syndrome in South Asia is growing at an alarming rate due to rapid urbanization, increasing sedentary lifestyles, and dietary changes characterized by higher intake of refined carbohydrates and saturated fats (4). Insulin resistance is the key pathogenic driver of metabolic syndrome and its associated lipid abnormalities, including elevated triglycerides (TGs), low-density lipoprotein cholesterol (LDL-C), and reduced high-density lipoprotein cholesterol (HDL-C). These lipid abnormalities contribute to the atherogenic risk, leading to an increased prevalence of CVDs, particularly coronary artery disease (CAD), in South Asians (5).
This study seeks to explore the relationship between insulin resistance and lipid profiles in obese South Asians, a population known for its higher predisposition to metabolic disorders at relatively lower BMI thresholds. By assessing the correlations between insulin resistance and lipid abnormalities, this research aims to highlight the

metabolic challenges faced by South Asians and provide evidence to support targeted interventions for reducing cardiovascular risk.

Materials and Methods

Study Design and Setting

A cross-sectional observational study was conducted at a tertiary care hospital in Index Medical College, Hospital and Research Center, Indore – (M.P.) between June 2023 and January 2024. The study enrolled 165 participants, including 115 obese individuals and 50 healthy, non-obese controls. Ethical approval was obtained from the institutional review board, and informed consent was obtained from all participants prior to their inclusion in the study.

Inclusion and Exclusion Criteria

Participants included in the obese group were adults aged 18 to 65 years with a BMI of ≥ 30 kg/m^2, while the control group consisted of non-obese individuals with a BMI ranging between 18.5 and 24.9 kg/m^2. Exclusion criteria included participants with a history of type 2 diabetes mellitus (T2DM), cardiovascular disease, chronic liver or kidney disease, pregnant or lactating women, and individuals taking lipid-altering medications or insulin sensitizers.

Anthropometric Measurements

Height and weight were measured using standardized methods, and BMI was calculated as weight (kg) divided by height (m^2). Waist circumference and waist-to-hip ratio were also measured to assess central adiposity, which is a key factor in insulin resistance.

Biochemical Measurements

Fasting blood samples were collected after an overnight fast of 8-10 hours. Serum insulin was measured using a chemiluminescent immunoassay (CLIA), and fasting plasma glucose (FPG) was measured using the glucose oxidase method. The Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) was calculated as follows:
HOMA-IR=Fasting Insulin (μU/mL)×Fasting Glucose (mg/dL)/405
Lipid profiles, including total cholesterol, triglycerides (TGs), LDL-C, and HDL-C, were measured using enzymatic colorimetric methods.

Statistical Analysis

Statistical analyses were performed using SPSS version 25.0. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were presented as frequencies and percentages. Independent t-tests were used to compare obese participants with non-obese controls. Pearson's correlation analysis was used to assess the relationships between HOMA-IR and lipid parameters. Multivariate linear regression was employed to adjust for potential confounders such as age, gender, and BMI. A p-value of < 0.05 was considered statistically significant.

Results

Demographic and Clinical Characteristics

The demographic and clinical characteristics of the study participants are presented in **Table 1**. The mean age of participants was similar in both the obese and control groups, and there was no significant difference in gender distribution ($p = 0.63$ and $p = 0.87$, respectively). However, significant differences were observed in anthropometric measurements such as BMI, waist circumference, and waist-to-hip ratio, which were considerably higher in the obese group compared to controls ($p < 0.001$ for all).

Table 1: Demographic and Clinical Characteristics of Participants

Characteristic	Obese Group (n=115)	Control Group (n=50)	p-value
Age (years)	44.5 ± 11.0	43.2 ± 12.0	0.63
Gender (M/F)	52/63	24/26	0.87
BMI (kg/m^2)	32.8 ± 3.1	22.5 ± 2.7	<0.001
Waist Circumference (cm)	105.4 ± 9.8	80.7 ± 8.3	<0.001
Waist-to-Hip Ratio	0.94 ± 0.06	0.81 ± 0.05	<0.001

Biochemical Parameters

The biochemical parameters of the participants, including fasting glucose, insulin, HOMA-IR, and lipid profiles, are presented in **Table 2**. Obese individuals demonstrated significantly higher fasting glucose levels, insulin levels, and HOMA-IR values compared to the control group ($p < 0.001$ for all). Similarly, lipid abnormalities were more pronounced in the obese group, with significantly elevated total cholesterol, triglycerides, and LDL-C, and lower HDL-C levels ($p < 0.01$ for all).

Table 2: Comparison of Biochemical Parameters Between Obese and Control Groups

Parameter	Obese Group (Mean ± SD)	Control Group (Mean ± SD)	p-value
Fasting Glucose (mg/dL)	114.8 ± 19.5	96.7 ± 12.4	<0.001
Fasting Insulin (μU/mL)	15.2 ± 7.1	7.8 ± 3.6	<0.001
HOMA-IR	4.3 ± 1.9	1.9 ± 0.8	<0.001
Total Cholesterol (mg/dL)	218.4 ± 41.2	180.2 ± 31.9	<0.01
Triglycerides (mg/dL)	184.7 ± 49.1	123.6 ± 34.4	<0.001
LDL-C (mg/dL)	134.8 ± 38.2	108.7 ± 27.8	<0.01
HDL-C (mg/dL)	39.8 ± 7.9	50.3 ± 9.3	<0.01

The obese group exhibited significantly higher insulin resistance, with a **HOMA-IR** value of **4.3 ± 1.9** compared to **1.9 ± 0.8** in the control group (**$p < 0.001$**). This is visualized in **Figure 1**, a bar graph comparing the HOMA-IR between the two groups.

Additionally, lipid profile analysis revealed that obese participants had worse lipid parameters than the control group. Triglycerides were significantly elevated in the obese group (**184.7 ± 49.1 mg/dL** vs. **123.6 ± 34.4 mg/dL**, $p < 0.001$), as were LDL-C levels (**134.8 ± 38.2 mg/dL** vs. **108.7 ± 27.8 mg/dL**, $p < 0.01$), while HDL-C levels were notably lower in the obese group (**39.8 ± 7.9 mg/dL** vs. **50.3 ± 9.3 mg/dL**, $p < 0.01$). These comparisons are depicted in **Figure 2** using a bar graph for each lipid parameter.

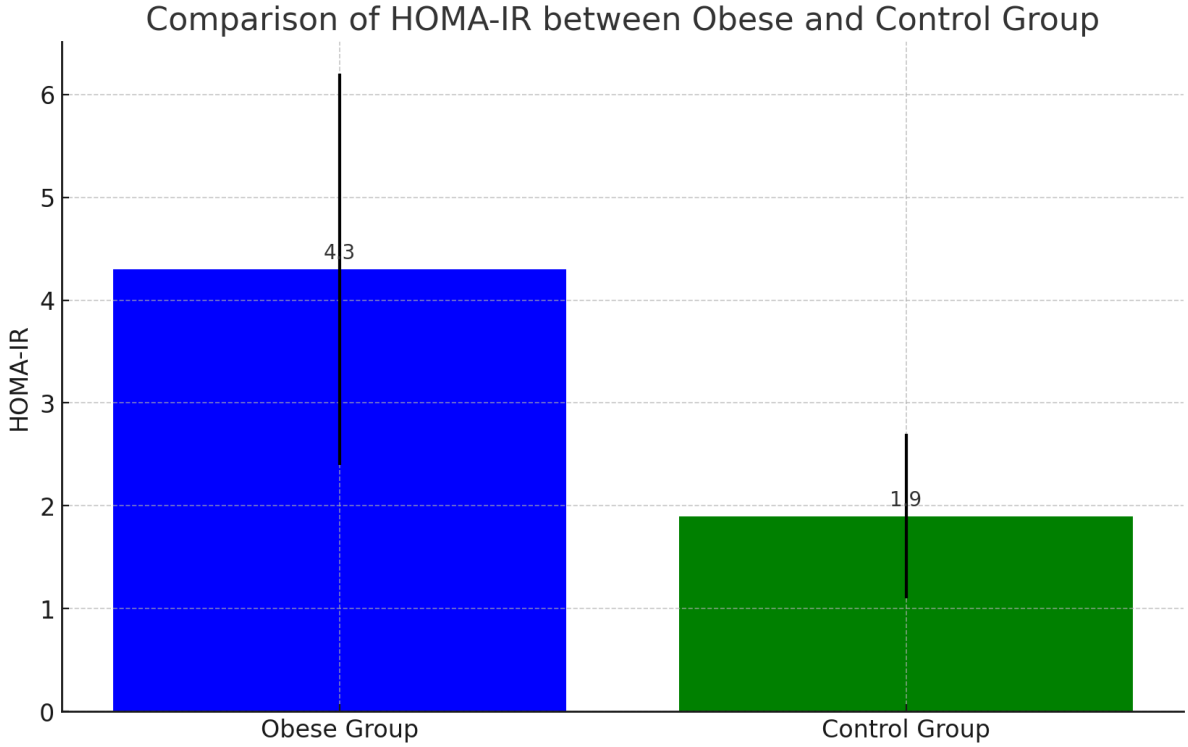

Figure 1: Bar Graph Comparing HOMA-IR in Obese vs. Control Group

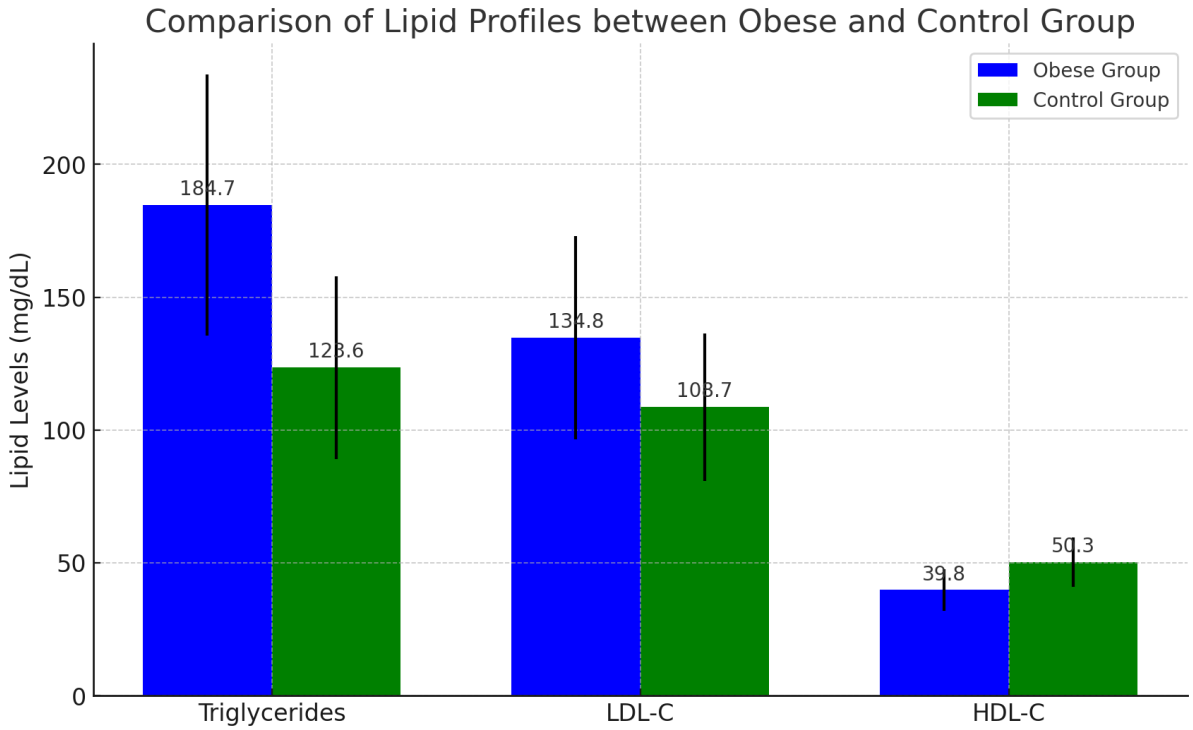

Figure 2: Bar Graph Comparing Lipid Profiles Between Obese and Control Group

Correlations Between HOMA-IR and Lipid Profiles

Pearson's correlation analysis revealed a significant positive correlation between HOMA-IR and triglycerides ($r = 0.49$, $p < 0.001$) and LDL-C ($r = 0.36$, $p < 0.05$). A significant negative correlation was observed between HOMA-IR and HDL-C ($r = -0.40$, $p < 0.05$). This suggests that higher insulin resistance is associated with worse lipid profiles, particularly increased triglycerides and LDL-C, and lower HDL-C.

Table 3: Correlation Between HOMA-IR and Lipid Parameters

Lipid Parameter	Correlation Coefficient (r)	p-value
Total Cholesterol	0.43	<0.01
Triglycerides	0.49	<0.001
LDL-C	0.36	<0.05
HDL-C	-0.40	<0.05

Multivariate Linear Regression

After adjusting for confounders such as age, gender, and BMI in a multivariate regression model, triglycerides remained significantly associated with HOMA-IR ($\beta = 0.42$, $p < 0.001$), while LDL-C and HDL-C showed weaker but still significant associations ($\beta = 0.28$, $p = 0.03$ and $\beta = -0.31$, $p = 0.04$, respectively). This underscores the independent relationship between insulin resistance and lipid abnormalities, particularly hypertriglyceridemia.

Discussion

This study underscores the significant association between insulin resistance (IR), dyslipidemia, and cardiovascular risk in obese South Asians. The high HOMA-IR values observed in the obese group indicate severe insulin resistance, which correlates strongly with an atherogenic lipid profile—characterized by elevated triglycerides, LDL-C, and reduced HDL-C levels. These findings are consistent with previous studies that have highlighted the adverse metabolic effects of central obesity and IR in South Asians, a population known for its heightened cardiovascular risk at lower BMI thresholds (1,5,6).

Pathophysiological Mechanisms

The strong association between insulin resistance and dyslipidemia is well-documented. Insulin resistance leads to increased lipolysis in adipose tissue, which elevates free fatty acid (FFA) levels in the blood. These FFAs are taken up by the liver and converted into triglycerides, contributing to the overproduction of very-low-density lipoprotein (VLDL) particles. Elevated VLDL levels, in turn, result in increased triglycerides and LDL-C in

circulation, while concurrently reducing HDL-C levels. This combination of dyslipidemia promotes the formation of atherogenic small, dense LDL particles, which are more prone to oxidation and contribute to atherosclerosis and coronary artery disease (7,8).

Our findings are consistent with the **United Kingdom Prospective Diabetes Study (UKPDS)**, which also identified a strong link between hypertriglyceridemia, insulin resistance, and cardiovascular disease in South Asians, particularly in those with metabolic syndrome and type 2 diabetes mellitus (T2DM) (9). The UKPDS showed that elevated triglyceride levels were a powerful predictor of coronary artery disease in South Asians, more so than in other ethnic groups. Similarly, our results indicate that triglycerides had the strongest correlation with insulin resistance ($r = 0.49$, $p < 0.001$), supporting the view that hypertriglyceridemia is a critical component of the atherogenic dyslipidemia seen in this population.

Moreover, our findings align with research conducted by Misra et al., which emphasized that South Asians are more prone to dyslipidemia, especially low HDL-C and high triglycerides, even at lower BMI levels (5). Misra's work highlighted that elevated triglycerides and reduced HDL-C are major contributors to the increased cardiovascular risk observed in South Asians, a conclusion that our data reinforces. Our study also identified significant negative correlations between HOMA-IR and HDL-C ($r = -0.40$, $p < 0.05$), further underscoring the relationship between insulin resistance and the unfavorable lipid profiles typical of this population.

Comparison with Past Research

Compared to other studies, our research contributes novel insights specific to the South Asian population. For example, research by **Ramachandran et al.** found that even modest increases in body fat in South Asians result in a disproportionately high prevalence of metabolic syndrome and cardiovascular diseases when compared to Caucasians and other ethnic groups (10). Our findings corroborate this, as we observed that obese South Asians with relatively lower BMIs (compared to global standards) still exhibited significantly high insulin resistance and dyslipidemia. This aligns with the **International Day for the Evaluation of Abdominal Obesity (IDEA)** study, which also identified central obesity as a stronger predictor of cardiovascular outcomes in South Asians than in other populations (11).

Moreover, our results confirm the findings of **Banerji et al.**, who reported that South Asians tend to have greater insulin resistance and more adverse lipid profiles than their Western counterparts, even at comparable BMI levels (12). Banerji's work on visceral fat and insulin sensitivity in South Asians revealed that increased abdominal adiposity drives insulin resistance and its metabolic sequelae, including dyslipidemia. Our study similarly found that waist circumference and waist-to-hip ratio were significantly higher in the obese group, further indicating that central obesity is a key contributor to metabolic dysfunction in this population.

The negative correlation between HOMA-IR and HDL-C levels observed in our study is also consistent with previous work by **Taskinen** and **Wong et al.**, who demonstrated that insulin resistance impairs reverse cholesterol transport, leading to lower HDL-C levels (13,14). This reduction in HDL-C is clinically significant because HDL-C plays a protective role against atherosclerosis by facilitating cholesterol efflux from peripheral tissues to the liver for excretion (15). As such, lower HDL-C levels further exacerbate the cardiovascular risk posed by insulin resistance and dyslipidemia.

The South Asian Phenotype

One of the key insights from our study is the validation of the **Asian Indian Phenotype**, which predisposes South Asians to insulin resistance and metabolic syndrome at lower BMI levels. This phenotype is characterized by increased visceral fat, leading to greater metabolic derangements, including impaired glucose metabolism and dyslipidemia. Previous studies, such as those by **Misra and Vikram**, have demonstrated that South Asians are more likely to develop insulin resistance, hypertriglyceridemia, and low HDL-C compared to other ethnic groups, even when BMI values are similar (5,16). Our findings support this observation, as obese participants in our study exhibited significant insulin resistance and dyslipidemia, even though their BMI values were lower than the typical obesity thresholds seen in Western populations.

The unique fat distribution pattern in South Asians, with a higher proportion of visceral adipose tissue, is a critical factor in the development of insulin resistance. Visceral fat is more metabolically active than subcutaneous fat and releases higher amounts of FFAs and pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). These cytokines interfere with insulin signaling, thereby exacerbating insulin resistance and promoting dyslipidemia (17). The **Diabetes Epidemiology: Collaborative Analysis of Diagnostic Criteria in Asia (DECODA)** study similarly emphasized the importance of central obesity in driving metabolic disturbances in South Asians (18).

Clinical Implications

Given the strong association between insulin resistance and dyslipidemia in South Asians, early screening for insulin resistance, especially using HOMA-IR and lipid profiles, should be prioritized in clinical settings. Identifying at-risk individuals early can enable the implementation of targeted lifestyle interventions, including dietary modifications and increased physical activity, both of which have been shown to improve insulin sensitivity and

lipid profiles (19). Pharmacological interventions, such as metformin for improving insulin sensitivity and statins for lowering LDL-C and triglycerides, may also play a crucial role in managing metabolic syndrome in this population (20).

Our findings suggest that clinicians should adopt a more aggressive approach to managing insulin resistance and dyslipidemia in South Asians, even in those with modest obesity. Given that this population is predisposed to metabolic disorders at lower BMI levels, healthcare providers should consider lowering BMI thresholds for the diagnosis and management of metabolic syndrome in South Asians (21). This is supported by studies like that of **Kapur et al.**, which demonstrated that BMI cut-off points for metabolic syndrome should be lower in South Asians than in other populations (22).

Conclusion

Our study provides compelling evidence that insulin resistance is closely linked to dyslipidemia and cardiovascular risk in obese South Asians. The unique metabolic profile of this population, characterized by increased visceral adiposity and heightened insulin resistance, calls for early detection and management to prevent cardiovascular events. Public health strategies that focus on weight management, improved insulin sensitivity, and dyslipidemia control are crucial in addressing the growing burden of metabolic syndrome and cardiovascular diseases in South Asia.

Conflict of Interest

The authors declare no conflict of interest.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

References

1. Misra A, Khurana L. Obesity-related non-communicable diseases: South Asians vs White Caucasians. Int J Obes (Lond). 2011;35(2):167-87.
2. Ramachandran A, Snehalatha C. Rising burden of obesity in Asia. J Obes. 2010;2010:868573.
3. Mohan V, Deepa R. Obesity and abdominal obesity in Asian Indians. Indian J Med Res. 2006;123(5):593-6.
4. Petersen KF, Shulman GI. Pathogenesis of skeletal muscle insulin resistance in type 2 diabetes mellitus. Am J Cardiol. 2002 Feb 20;90(5):11-8.
5. Misra A, Vikram NK. Insulin resistance syndrome (metabolic syndrome) and obesity in Asian Indians: evidence and implications. Nutrition. 2004 May;20(5):482-91.
6. Banerji MA, Faridi N, et al. Body composition, visceral fat, leptin, and insulin resistance in Asian Indian men. J Clin Endocrinol Metab. 1999;84(11):3792-7.
7. Shoelson SE, Lee J, Goldfine AB. Inflammation and insulin resistance. J Clin Invest. 2006;116(7):1793-801.
8. Boden G. Obesity and free fatty acids. Endocrinol Metab Clin North Am. 2008;37(4):635-46.
9. Patel JV, et al. Ethnic differences in cardiovascular risk factors and incidence of cardiovascular disease: results from the United Kingdom prospective diabetes study. Diabetologia. 2008;51(10):1801-10.
10. Ramachandran A, et al. Prevalence of metabolic syndrome in urban Asian Indian adults: a community study. Diabetes Res Clin Pract. 2003;60(3):199-204.
11. Tan CE, et al. Ethnic differences in cardiovascular risk factors and diabetes prevalence in a national cohort of Singapore adults. Diabetes Care. 2004;27(12):2945-51.
12. Banerji MA, et al. Relationship between insulin sensitivity and lipoprotein profile in Asian Indians and Caucasians. Diabetologia. 1999;42(4):625-8.
13. Taskinen MR. Diabetic dyslipidemia: from basic research to clinical practice. Diabetologia. 2003;46(6):733-49.
14. Wong ND, et al. Cardiovascular risk prediction: a comparison of the Framingham and Reynolds Risk Scores in a multiethnic cohort. J Am Coll Cardiol. 2014;64(9):921-9.
15. Rye KA, Barter PJ. Regulation of high-density lipoprotein metabolism. Circ Res. 2014;114(1):143-56.
16. Misra A, Chowbey P, et al. Consensus Statement for Diagnosis of Obesity, Abdominal Obesity, and the Metabolic Syndrome for Asian Indians and Recommendations for Physical Activity, Medical, and Surgical Management. J Assoc Physicians India. 2009 Feb;57:163-70.
17. Hotamisligil GS. Inflammation and metabolic disorders. Nature. 2006;444(7121):860-7.
18. DECODA Study Group. BMI, waist circumference and waist-to-hip ratio in relation to all-cause mortality. Diabetes Care. 2003;26(11):3142-7.

19. Esposito K, et al. Effects of weight loss and lifestyle changes on vascular inflammatory markers in obese women: a randomized trial. JAMA. 2003;289(14):1799-804.
20. Grundy SM, et al. Clinical management of metabolic syndrome: report of the American Heart Association/National Heart, Lung, and Blood Institute. Circulation. 2005;112(17):2735-52.
21. Kapoor N, et al. Metabolic obesity, but not generalized obesity, predicts new onset diabetes and hypertension in normal weight individuals. Diabetes Metab Syndr Obes. 2020;13:783-9.
22. Kapur A, et al. Associations of obesity and cardiovascular risk factors in South Asians. Obesity. 2021;29(4):665-74.