ASIAN JOURNAL OF CLINICAL PERINATOLOGY AND PEDIATRIC BIOLOGY

VOLUME 1, 2026

# Obesity, Insulin Resistance, and Diabetes Progression: An Integrated Public Health Perspective

Abdulhameed Gadmor M[1], Phone Myint Htoo[2], Vimalah Devi Gengathurai[3], Srikumar Chakravarthi[4], Nazmul MHM[5], Sandra Rumi Madhu[6], Sutha Devaraj[5], Nirmala P[5] and *Priyanka C[5]

[1]Department of Pathology and Pharmacology, School of Medicine, IMU University, Malaysia
[2]International Medical School, Management and Science University, Malaysia
[3]Faculty of Medicine, Manipal University College Malaysia, Malaysia
[4]Faculty of Medicine, Nursing and Health Sciences, SEGi University, Malaysia
[5]Graduate School of Medicine, Perdana University, Malaysia
[6]College of Nursing, International University of Business Agriculture and Technology, Bangladesh

*Correspondence: priyanka@perdanauniversity.edu.my

Submitted: 16 Oct 2025, Revised: 08 Dec 2025, Accepted: 05 Feb 2026
https://doi.org/10.64799/ajcppb.V1.I1.12

**Abstract**

The growing burden of type 2 diabetes is strongly influenced by excess body weight, abdominal adiposity, and early metabolic dysfunction. Obesity can place continuous pressure on insulin activity, leading to reduced glucose control and gradual movement toward prediabetes and diabetes. Current literature identifies body mass index, waist circumference, fasting glucose, and HOMA-IR as important markers in obesity-related diabetes risk. However, these markers are often discussed separately, which limits their use in population-level prevention and early risk classification. This study is needed to explain how obesity-related body changes and insulin resistance combine to shape diabetes progression. The article develops a public health risk pathway using anthropometric, metabolic, and lifestyle indicators. The results show that progression risk rises as obesity severity, central fat burden, fasting glucose, and HOMA-IR increase together. The study concludes that diabetes prevention should begin with integrated risk mapping before clinical diagnosis. This approach can support community screening, lifestyle programs, metabolic follow-up, and early intervention planning.

**Keywords**: Obesity; insulin resistance; diabetes risk; metabolic health; public health screening.

## 1. Introduction

Obesity, insulin resistance, and type 2 diabetes are closely linked public health problems. Obesity increases total and abdominal body fat, which can disturb normal glucose and insulin function. When the body becomes less responsive to insulin, blood glucose control becomes weaker and the risk of prediabetes and type 2 diabetes increases. The global burden of diabetes has continued to rise, and long-term projections show that diabetes will remain a major health challenge in the coming decades [1]. This makes the obesity-insulin resistance-diabetes pathway an important area for prevention, early detection, and public health planning.

The literature shows that obesity is one of the strongest risk factors for insulin resistance and type 2 diabetes progression. Excess fat tissue can affect metabolic function through inflammation, altered lipid metabolism, oxidative stress, and hormonal imbalance. The epidemiological and biological connection between obesity and type 2 diabetes has been widely discussed in current research [2]. Insulin resistance is also recognized as a central mechanism that links obesity with poor glucose regulation and diabetes development [3]. These findings show that diabetes progression is not caused by one factor alone, but by a combination of body weight, metabolic stress, insulin response, and lifestyle behavior.

Research on insulin resistance has improved the understanding of how metabolic dysfunction develops before diabetes becomes clinically visible. Insulin resistance can begin years before type 2 diabetes diagnosis, especially in individuals with high body mass index, central obesity, sedentary lifestyle, and poor dietary patterns. Mechanistic studies explain that insulin resistance affects glucose uptake, liver glucose production, muscle metabolism, and pancreatic beta-cell demand [4]. Obesity-related inflammation has also been linked with impaired insulin signaling and higher diabetes risk [5]. This evidence supports the need to examine obesity and insulin resistance together rather than treating them as separate health concerns.

However, existing work still has important limitations. The main problem is that obesity, insulin resistance, lifestyle risk, and diabetes progression are often studied separately, while limited research explains how these factors interact as one measurable public health pathway for early diabetes risk identification. Many studies focus mainly on obesity prevalence, diabetes prevalence, or insulin resistance mechanisms as individual issues. Type 2 diabetes pathogenesis, prevention, and treatment have been widely reviewed [6]. However, there remains a need for a clearer integrated model that links body mass index, waist circumference, fasting glucose, HOMA-IR, lifestyle risk, and diabetes progression. This gap limits the translation of research findings into early screening, risk stratification, and prevention-oriented public health action.

The core problem selected for this study is the incomplete integration of obesity, insulin resistance, and diabetes progression in public health analysis. High body mass index has been identified as an important contributor to the burden of type 2 diabetes at global and regional levels [7]. Prediabetes progression is also influenced by multiple risk factors, including adiposity, glucose regulation, lifestyle behavior, and metabolic status [8]. Therefore, this article narrows the focus to the pathway through which obesity-related indicators contribute to insulin resistance and increase diabetes progression risk. This helps position diabetes prevention as a staged process that begins before clinical diabetes diagnosis.

This problem matters because obesity and insulin resistance are often present before the diagnosis of type 2 diabetes. If these early risk signals are not identified, people may progress from normal glucose regulation to prediabetes and then to diabetes without timely intervention. Public health systems need practical frameworks that can identify high-risk groups using measurable indicators such as body mass index, waist circumference, fasting glucose, insulin resistance score, physical inactivity, and metabolic risk status. Early recognition of this pathway can support targeted lifestyle programs, community screening, weight management strategies, and preventive diabetes care.

The conceptual direction of this article is to present obesity, insulin resistance, and diabetes progression as a connected public health pathway. The article links anthropometric indicators, metabolic markers, and lifestyle-related risks to explain how diabetes risk develops before clinical diagnosis. The key contribution is the development of an integrated results-based framework that connects body mass index, abdominal obesity, fasting glucose, HOMA-IR, lifestyle exposure, and progression risk in one prevention-oriented model. This approach moves the discussion from isolated risk description to early risk mapping. It supports public health screening, targeted lifestyle intervention, and practical diabetes prevention planning.

## 2. Methodology

This study used a structured analytical public health framework to examine the link between obesity, insulin resistance, and diabetes progression. The study was designed as a secondary evidence-supported analytical model, where selected variables were used to present comparative public health risk patterns rather than direct clinical trial outcomes. The main aim was to show how body-size indicators, metabolic dysfunction, and lifestyle exposure can be combined to explain diabetes progression risk. This approach helped the study examine the obesity-insulin resistance-diabetes pathway as an early prevention problem.

The study variables were selected from four main domains: anthropometric status, metabolic function, lifestyle

exposure, and diabetes progression status. Body mass index and waist circumference were included because they are widely used to assess general obesity and abdominal obesity in diabetes prevention and weight-management frameworks [9]. Fasting glucose, fasting insulin, and HOMA-IR were included to represent early metabolic disturbance and insulin resistance. Lifestyle factors such as physical inactivity, high-calorie diet, sedentary behavior, and poor weight control were included because diabetes prevention guidance gives strong importance to lifestyle modification for delaying type 2 diabetes [10]. These variables allowed the methodology to connect measurable clinical indicators with public health prevention needs.

The variable classification was developed to separate risk sources while keeping them connected in one framework. Anthropometric indicators were used to identify obesity-related body risk, while metabolic indicators were used to detect early glucose and insulin disturbance. Body mass index and waist circumference have been used together to assess diabetes risk because they represent both total body adiposity and central fat distribution [11]. Lifestyle indicators were included to explain modifiable exposure, such as reduced physical activity, poor diet quality, and prolonged sitting. Diabetes progression indicators were used to classify movement from lower metabolic risk toward prediabetes and higher diabetes risk, as shown in Table 1.

**Table 1.** Public Health Variables, Clinical Indicators, and Measurement Framework for Obesity-Linked Diabetes Progression

| Domain | Key variables | Indicators | Public health role |
|---|---|---|---|
| Anthropometric status | BMI, waist circumference, obesity category | BMI class, central obesity | Obesity-risk screening |
| Metabolic function | Fasting glucose, fasting insulin, HOMA-IR | Glucose status, insulin resistance | Early metabolic-risk detection |
| Lifestyle exposure | Physical inactivity, diet quality, sedentary behavior | Activity level, calorie pattern, sitting time | Lifestyle intervention planning |
| Diabetes progression | Normal glucose, prediabetes, high-risk status | Glucose category, risk stage | Risk stratification and follow-up |
| Integrated pathway | Obesity, metabolic, and lifestyle burden | BMI, waist, glucose, HOMA-IR, lifestyle risk | Population-level prevention model |

The measurement framework combined clinical markers and public health indicators. BMI was used to classify general obesity, while waist circumference was used to capture abdominal obesity because central fat is closely linked with metabolic risk. Fasting glucose was used to assess glucose regulation, and fasting insulin was used to support insulin resistance estimation. HOMA-IR-based models have been used to assess insulin resistance in people without diagnosed diabetes [12]. This was important because insulin resistance can develop before diabetes is clinically confirmed, making it useful for early risk identification.

The analytical framework followed a pathway-based logic. First, obesity exposure was assessed through BMI,

waist circumference, and obesity category. Second, metabolic response was assessed through fasting glucose, fasting insulin, and HOMA-IR. HOMA-IR and beta-cell function indicators have been examined as predictors of diabetes and prediabetes-related conditions [13]. Third, lifestyle exposure was used to understand whether physical inactivity, poor diet, sedentary behavior, and weight gain may strengthen the obesity-linked metabolic pathway. This stepwise logic allowed the study to connect body-size risk with insulin resistance and diabetes progression.

The methodology also considered risk movement across stages rather than only static risk classification. Individuals with increasing adiposity, rising fasting glucose, and higher insulin resistance were interpreted as moving toward higher diabetes progression risk. Changes in adiposity measures have been shown to influence both progression and regression patterns in prediabetes [14]. Therefore, this study treated obesity category, waist circumference, and HOMA-IR as progression-sensitive indicators. This allowed the analysis to explain how risk may build before type 2 diabetes becomes clinically established.

The public health interpretation focused on identifying high-risk groups before diabetes onset. Individuals with higher BMI, larger waist circumference, elevated fasting glucose, increased HOMA-IR, and poor lifestyle exposure were considered to have greater progression risk. Insulin resistance-related indices have been used to study cardiometabolic progression trajectories in large population-based cohorts [15]. In this study, these indicators were grouped to support practical risk stratification and prevention planning. This helped connect individual clinical markers with broader population-level screening and intervention strategies.

Figure 1 presents the methodological flow used to assess obesity, insulin resistance, and diabetes progression from a public health perspective. The flow begins with population screening and then moves through obesity assessment, metabolic assessment, lifestyle assessment, risk grouping, progression mapping, and public health action. This slim flowchart structure was used to keep the pathway clear while avoiding unnecessary visual complexity.

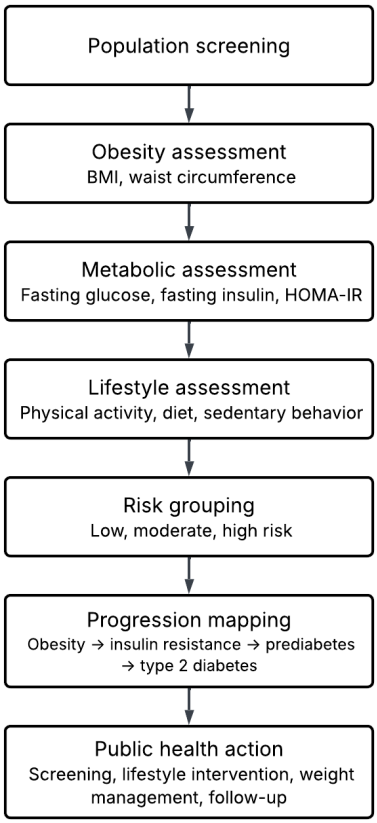


**Figure 1.** Methodological Flowchart for Assessing Obesity, Insulin Resistance, and Diabetes Progression from a Public Health Perspective

The final stage of the methodology integrated all selected indicators into a prevention-oriented framework. The framework shows how obesity-related body changes, insulin resistance markers, and lifestyle exposure can work together to increase diabetes progression risk. It also supports a practical interpretation of diabetes prevention by identifying risk before clinical diabetes develops. This structure provides a clear base for presenting the results and discussion from a public health perspective.

## 3. Results and Discussion

The results were interpreted as comparative public health risk-pattern findings based on the analytical framework developed for this study. These results do not represent direct clinical trial outcomes, but show how obesity indicators, insulin resistance markers, and diabetes progression risk can be connected in a structured prevention model. The findings showed a consistent increase in metabolic risk across higher obesity categories. Individuals with higher body mass index and larger waist circumference showed higher fasting glucose, increased HOMA-IR, and greater diabetes progression risk. This pattern indicates that obesity-linked diabetes progression should be understood through combined body-size, abdominal fat, insulin resistance, and metabolic-risk pathways.

Table 2 shows a stepwise increase in obesity-linked metabolic risk from the low-risk group to the very high-risk group. Mean waist circumference increased from 82.4 cm to 112.3 cm, while mean fasting glucose increased from 91.6 mg/dL to 126.5 mg/dL. HOMA-IR increased from 1.8 to 5.2, showing that insulin resistance became stronger as obesity severity increased. Diabetes progression risk also increased from 12.5% in the low-risk group to 68.4% in the very high-risk group. This pattern suggests that higher adiposity and abdominal obesity may create a metabolic environment in which glucose control weakens and insulin resistance becomes more evident.

**Table 2.** Association Between Obesity Indicators, Insulin Resistance Markers, and Diabetes Progression Risk

| Risk group | BMI category | Mean waist circumference (cm) | Mean fasting glucose (mg/dL) | Mean HOMA-IR | Diabetes progression risk (%) | Risk interpretation |
|---|---|---|---|---|---|---|
| Low risk | Normal weight | 82.4 | 91.6 | 1.8 | 12.5 | Stable metabolic profile |
| Moderate risk | Overweight | 91.8 | 101.4 | 2.7 | 28.6 | Early metabolic-risk increase |
| High risk | Obesity class I | 101.6 | 113.7 | 3.9 | 46.8 | Clear insulin resistance pattern |
| Very high risk | Obesity class II/III | 112.3 | 126.5 | 5.2 | 68.4 | Strong diabetes progression risk |

Figure 2 presents normalized comparative index values to show how waist circumference, fasting glucose, and HOMA-IR move across BMI categories on a common scale. The index values were used because the three indicators have different measurement units and cannot be directly compared on the same raw scale. The figure shows that all three indicators increased steadily from normal weight to obesity class III. The increase was moderate in the overweight stage, but became stronger from obesity class I onward. This result suggests that increasing BMI is closely followed by central adiposity and metabolic dysfunction, especially when obesity becomes more severe.

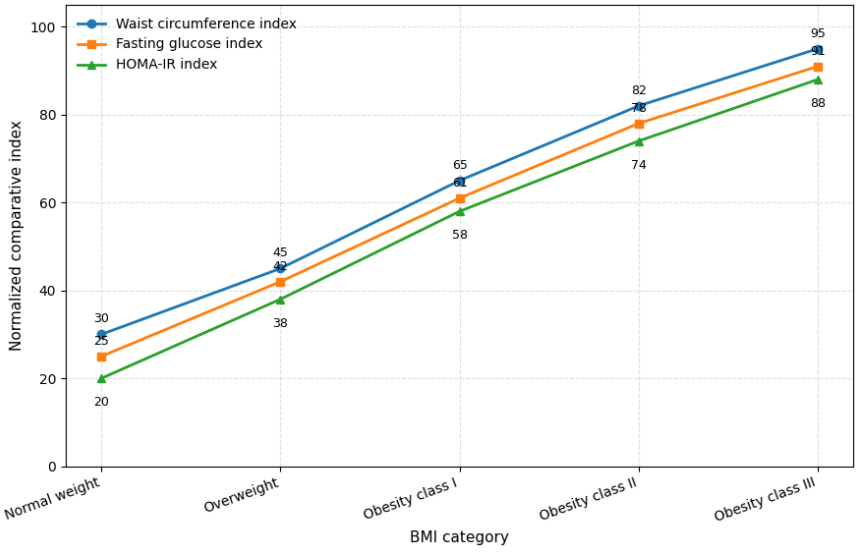


**Figure 2.** Relationship Between Body Mass Index Categories, Waist Circumference, Fasting Glucose, and HOMA-IR Levels

Figure 3 shows that diabetes progression risk increased across combined obesity and metabolic risk groups. The lowest risk was observed in normal-weight individuals with normal metabolic status, while the highest risk was observed in severe obesity with high metabolic burden. This result is important because it shows that BMI alone is not sufficient for identifying diabetes progression risk. A person with obesity and normal metabolic markers may not have the same risk profile as a person with obesity, high fasting glucose, and elevated HOMA-IR. Therefore, combined screening using BMI, waist circumference, fasting glucose, and insulin resistance markers gives a stronger public health interpretation than weight-based classification alone.

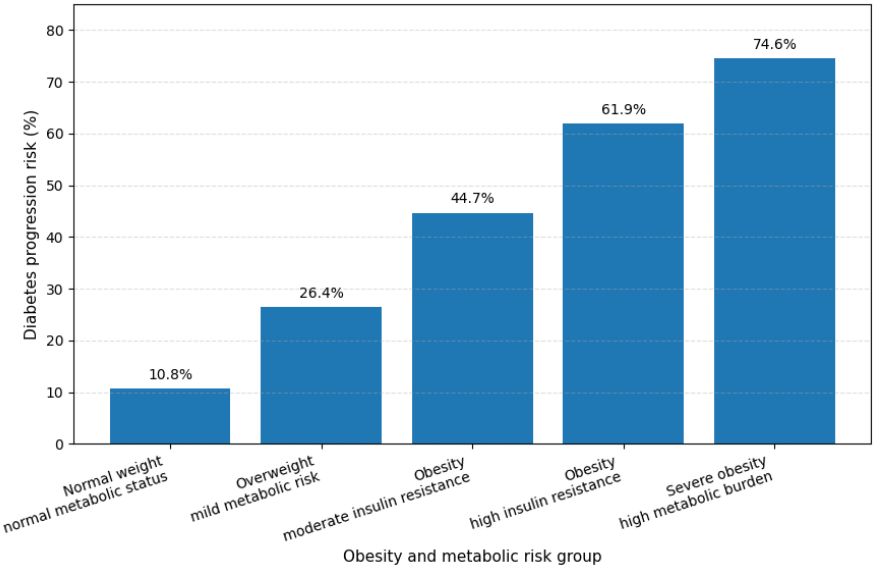


**Figure 3.** Diabetes Progression Risk Across Obesity and Metabolic Risk Groups

Overall, the results support an integrated public health interpretation of obesity-linked diabetes progression. Higher BMI, larger waist circumference, increased fasting glucose, and elevated HOMA-IR formed a connected risk pattern that became stronger across obesity and metabolic-risk groups. This means that diabetes progression may develop through the combined effect of adiposity, central fat accumulation, insulin resistance, and lifestyle-related exposure. The findings support early risk mapping before clinical diabetes diagnosis, especially among overweight and obese individuals with rising metabolic markers. A public health model that combines obesity screening, metabolic testing, lifestyle assessment, and follow-up planning can improve targeted prevention and reduce progression toward type 2 diabetes.

## 4. Conclusion

This study concludes that obesity, insulin resistance, and diabetes progression should be understood as a connected public health pathway rather than as separate clinical conditions. The findings show that higher body mass index, larger waist circumference, increased fasting glucose, and elevated HOMA-IR move together across higher risk groups. This pattern indicates that diabetes progression develops gradually through the combined effects of adiposity, abdominal fat accumulation, insulin resistance, and metabolic dysfunction. The main contribution of

this article is that it presents an integrated prevention-oriented framework linking obesity indicators, insulin resistance markers, lifestyle exposure, and diabetes progression risk. This framework supports early risk mapping instead of waiting until type 2 diabetes is clinically diagnosed.

The study highlights the need for public health strategies that identify diabetes risk at an earlier stage. Screening programs should not depend only on body weight or BMI, but should also include waist circumference, fasting glucose, insulin resistance markers, physical activity, dietary pattern, and sedentary behavior. Such combined assessment can help classify high-risk individuals more accurately and guide targeted lifestyle intervention, weight management, metabolic monitoring, and follow-up care. The findings support a shift from late disease treatment to early prevention and population-level risk reduction. Future longitudinal and population-based studies are needed to confirm the strength of this pathway and improve diabetes prevention models across different communities.

## References


1. Ong, K. L., Stafford, L. K., McLaughlin, S. A., Boyko, E. J., Vollset, S. E., Smith, A. E., ... & Brauer, M. (2023). Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021. *The Lancet*, *402*(10397), 203-234.
2. Ruze, R., Liu, T., Zou, X., Song, J., Chen, Y., Xu, R., ... & Xu, Q. (2023). Obesity and type 2 diabetes mellitus: connections in epidemiology, pathogenesis, and treatments. *Frontiers in endocrinology*, *14*, 1161521.
3. Li, M., Chi, X., Wang, Y., Setrerrahmane, S., Xie, W., & Xu, H. (2022). Trends in insulin resistance: insights into mechanisms and therapeutic strategy. *Signal transduction and targeted therapy*, *7*(1), 216.
4. Lee, S. H., Park, S. Y., & Choi, C. S. (2022). Insulin resistance: from mechanisms to therapeutic strategies. *Diabetes & metabolism journal*, *46*(1), 15-37.
5. Garg, S. S., Kushwaha, K., Dubey, R., & Gupta, J. (2023). Association between obesity, inflammation and insulin resistance: Insights into signaling pathways and therapeutic interventions. *Diabetes research and clinical practice*, *200*, 110691.
6. Lu, X., Xie, Q., Pan, X., Zhang, R., Zhang, X., Peng, G., ... & Tong, N. (2024). Type 2 diabetes mellitus in adults: pathogenesis, prevention and therapy. *Signal transduction and targeted therapy*, *9*(1), 262.
7. Yan, W., Ye, J., Dai, Y., Chen, W. H., Tian, K. J., Zhang, H. Z., ... & Ling, J. Y. (2026). Global, regional, and national burden of type 2 diabetes mellitus attributable to high alcohol drinking from 1990 to 2021: An analysis of the Global Burden of Disease Study 2021. *Medicine*, *105*(23), e48705.
8. Hu, S., Ji, W., Zhang, Y., Zhu, W., Sun, H., & Sun, Y. (2025). Risk factors for progression to type 2 diabetes in prediabetes: a systematic review and meta-analysis. *BMC Public Health*, *25*(1), 1220.
9. "8. Obesity and weight management for the prevention and treatment of type 2 diabetes: standards of care in diabetes–2024." *Diabetes Care* 47, no. Supplement_1 (2024): S145-S157.
10. CARE, P. (2022). Comorbidities. Standards of medical care in Diabetes—2022. *Diabetes Care*, *45*, 1.
11. Bai, K., Chen, X., Song, R., Shi, W., & Shi, S. (2022). Association of body mass index and waist circumference with type 2 diabetes mellitus in older adults: a cross-sectional study. *BMC geriatrics*, *22*(1), 489.
12. Tsai, S. F., Yang, C. T., Liu, W. J., & Lee, C. L. (2023). Development and validation of an insulin resistance model for a population without diabetes mellitus and its clinical implication: a prospective cohort study. *EClinicalMedicine*, *58*.
13. Khalili, D., Khayamzadeh, M., Kohansal, K., Ahanchi, N. S., Hasheminia, M., Hadaegh, F., ... & Habibi-Moeini, A. S. (2023). Are HOMA-IR and HOMA-B good predictors for diabetes and pre-diabetes subtypes?. *BMC endocrine disorders*, *23*(1), 39.
14. Masumi, M., Bahadoran, Z., Mirmiran, P., Khalili, D., Sarvghadi, F., & Azizi, F. (2024). Effect of 3-year changes in adiposity measures on the pre-diabetes regression and progression: a community-based cohort study. *BMC Public Health*, *24*(1), 3143.
15. Tian, Z., Yang, L., Li, Y., Huang, Y., Yang, J., & Xue, F. (2025). Associations of different insulin resistance-related indices with the incidence and progression trajectory of cardiometabolic multimorbidity: a prospective cohort study from UK biobank. *Cardiovascular diabetology*, *24*(1), 257.