



---

## Exploring the Mediating Effect of Obesity in the Smoking–Diabetes Relationship: A Population-Based Study in India

**Dr. Sajni Anang Shah**

Department of Public Health, Long Island University, One University Plaza, Brooklyn, NY, USA

Email: Sajnianang.shah@my.liu.edu

**Dr. Helisse Levine\***

MPH Director, Professor, Department of Public Health, Long Island University, One University Plaza, Brooklyn  
NY, USA

Email: Helisse.levine@liu.edu

---

### ARTICLE DETAILS

**Research Paper**

**Accepted:** 24-05-2025

**Published:** 10-06-2025

**Keywords:**

*Tobacco Use, Obesity,  
Diabetes, Mediation  
Analysis*

---

### ABSTRACT

Non-communicable diseases are rapidly increasing in India, and diabetes remains a significant public health concern. While smoking has been associated with a higher risk of developing diabetes, the mechanisms by which this risk is heightened are not well understood. One known metabolic risk factor, obesity, may mediate this relationship by acting as a modifiable pathway that connects the onset of diabetes and smoking behavior. Data from 36 Indian states were utilized in this population-based cross-sectional study. Obesity was identified as the mediator, diabetes as the dependent variable, and tobacco use as the independent variable. Model 4 of the PROCESS macro in SPSS version 4.2 was employed to conduct mediation analysis. The analysis assessed both direct and indirect relationships between smoking and diabetes. Obesity partially mediates the relationship between smoking and diabetes. Public health strategies aimed at obesity prevention among smokers could potentially reduce diabetes risk within the Indian population.



## Introduction

India is experiencing a significant rise in diabetes cases, with an estimated 77 million individuals affected as of 2019—a number projected to exceed 134 million by 2045<sup>[1]</sup>. This surge is attributed to multiple modifiable risk factors, including poor diet, physical inactivity, tobacco use, and obesity<sup>[2]</sup>. Tobacco consumption remains a major public health concern in India, especially among adult males, with high rates of both smoking and smokeless tobacco use<sup>[3]</sup>. While the detrimental effects of tobacco use on cardiovascular and respiratory diseases are well documented<sup>[4]</sup>, emerging studies have also suggested an association between tobacco use and increased diabetes risk<sup>[5]</sup>.

Obesity is a well-established metabolic risk factor for diabetes, significantly increasing the likelihood of insulin resistance and glucose intolerance<sup>[6]</sup>. Interestingly, tobacco use has been linked to lower body weight, possibly due to nicotine's appetite-suppressing and metabolism-boosting effects<sup>[7]</sup>.

Given these complex interactions, this study aims to examine whether obesity mediates the relationship between tobacco use and diabetes among Indian adults. Understanding this mediation pathway could help inform public health interventions that simultaneously target tobacco cessation and obesity prevention to curb the growing diabetes burden in India.

## Literature Review

Diabetes, a rapidly growing non-communicable disease in India, has emerged as a pressing public health challenge due to its multifactorial causes. Numerous studies have demonstrated that both lifestyle behaviors and metabolic conditions significantly influence the risk of developing type 2 diabetes. Among these, tobacco use, and obesity are major and modifiable contributors.

### Tobacco use and diabetes

Tobacco consumption—through both smoking and smokeless forms—has been widely recognized for its harmful effects on health, particularly concerning cardiovascular and pulmonary diseases<sup>[8]</sup>. However, its connection to metabolic disorders such as diabetes has garnered increasing attention in recent years. According to the American Heart Association (2024), exposure to tobacco, even prenatally, can significantly heighten the risk of developing type 2 diabetes later in life<sup>[9]</sup>. Smoking contributes to insulin



resistance, chronic inflammation, and impaired glucose metabolism, all of which are mechanisms associated with diabetes development <sup>[10]</sup>.

Furthermore, the relationship between tobacco use and body weight adds complexity to this association. Several studies suggest that nicotine has appetite-suppressing and metabolism-enhancing effects, often resulting in lower body weight among smokers <sup>[11]</sup>. For instance, Winslow (2015) confirmed a significant negative association between high tobacco consumption and body weight through a Mendelian randomization study <sup>[11]</sup>. However, despite potential reductions in body weight, smoking is also linked to negative metabolic changes that increase diabetes risk, such as abdominal fat accumulation and insulin resistance <sup>[10]</sup>.

### **Obesity and diabetes**

Obesity is a well-documented risk factor for type 2 diabetes. Excess adiposity, especially central obesity, leads to metabolic dysfunction including insulin resistance, impaired glucose tolerance, and systemic inflammation <sup>[12]</sup>. A 2023 StatPearls publication outlines that the presence of obesity significantly increases the likelihood of developing diabetes, reinforcing the critical need for obesity management in diabetes prevention efforts <sup>[12]</sup>.

### **Mediation framework linking tobacco, obesity, and diabetes**

While both tobacco use and obesity have independently been associated with diabetes, the **mediation role of obesity** in the tobacco-diabetes pathway has not been extensively studied, particularly in the Indian context. Given nicotine's paradoxical role in reducing body weight but promoting insulin resistance, it is plausible that obesity may serve as a **mediating factor** that influences whether or how tobacco use ultimately affects diabetes risk.

Previous research has also emphasized the need to explore indirect pathways to uncover the underlying mechanisms in health behavior relationships. Chiolero et al. (2023) suggest that looking at only the direct relationship between smoking and diabetes may obscure the pathways through which smoking exerts its health impact, such as body weight and fat distribution <sup>[10]</sup>.

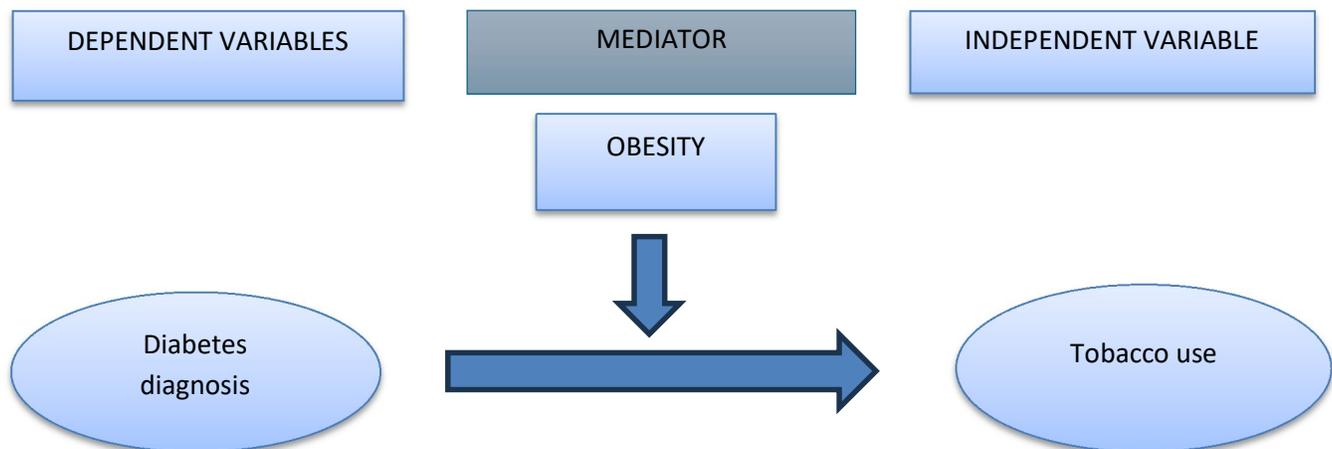
While the individual relationships between smoking, obesity, and diabetes are established, a mediation model that positions obesity as a pathway between tobacco use and diabetes provides a nuanced

understanding. This study aims to contribute to the growing body of research by empirically testing this relationship in the Indian population using secondary cross-sectional data.

### Conceptual framework

This study investigates the potential mediating role of obesity in the relationship between tobacco use and diabetes diagnosis within a population-based context in India. The conceptual framework, as shown in Figure 1 below, is structured as follows:

Figure 1: A Conceptual Framework of Tobacco Use and Diabetes Diagnosis



#### Independent Variable (IV): Tobacco Use

Tobacco use serves as the primary exposure variable. It is hypothesized to exert both direct and indirect effects on the risk of developing diabetes. The direct effect refers to the potential physiological impact of tobacco on glucose metabolism and insulin resistance. In contrast, the indirect effect operates through its influence on body weight and fat distribution.

#### Mediator (M): Obesity

Obesity is posited as the mediating variable in the pathway between tobacco use and diabetes. It is theorized that tobacco use may influence obesity through behavioral and metabolic mechanisms (e.g., appetite regulation, physical activity patterns), and that obesity, in turn, increases the risk of diabetes. This establishes a casual pathway

**Tobacco Use → Obesity → Diabetes Diagnosis**

#### Dependent Variable (DV): Diabetes Diagnosis



Diagnosis of diabetes represents the primary outcome of interest. The framework allows for the examination of both direct effects of tobacco use on diabetes and indirect effects that are mediated by obesity.

This framework facilitates a mediation analysis approach, enabling a nuanced understanding of the extent to which obesity explains the association between tobacco use and diabetes, and contributes to public health strategies aimed at addressing interconnected lifestyle risk factors.

## Hypotheses

### Primary Hypothesis (Total Effect)

Berlin et al. (2024) report that among individuals without diabetes, smoking not only increases the likelihood of Type 2 diabetes but also increases the likelihood of all-cause mortality among those with diabetes [13]. Similarly, Lee and Coombs' systematic review concludes that there is a modest increased risk of type 2 diabetes associated with current smoking, which is greater in heavier smokers and reduced following quitting [14]. We hypothesize that:

### Direct Effect Hypotheses

**H<sub>1</sub>:** Tobacco use is significantly associated with an increased risk of diabetes diagnosis.

Further, in 2024, the CDC suggested that those who smoke have a higher risk of belly fat, which also increases the risk of type 2 diabetes, even if they are not overweight [15].

**H<sub>2</sub>:** Tobacco use has a direct effect on the risk of diabetes diagnosis, independent of obesity.

### Indirect Effect (Mediation) Hypotheses

Based on Durlach et al.'s (2002) conclusion that smoking is a risk factor for the development of both pre-diabetes and diabetes, it is hypothesized [16]

**H<sub>3</sub>:** Tobacco use is significantly associated with increased obesity.

Popkin, Adair, and Ng (2012) have demonstrated that one of the key elements for the emergence of diabetes is the obesity pandemic brought on by changes in people's food and lifestyle. We therefore hypothesize [17]

**H<sub>4</sub>:** Obesity is significantly associated with an increased risk of diabetes diagnosis



According to Duan et al. (2025), while smoking may reduce weight through various mechanisms, alterations in gut microbiota related to smoking are associated with weight gain, as hypothesized below..<sup>[18]</sup>

**H5:** The relationship between tobacco use and diabetes diagnosis is partially mediated by obesity

### Materials and Method

The cross-sectional study was based on secondary data obtained from the Open Government Data (OGD) Platform India <sup>[19]</sup>, a website that offers a repository of datasets by Indian government departments. The subset of data used for the analysis included health-related information such as status of diabetes, tobacco use behavior, and obesity, which served as a dependent, independent, and mediating variable, respectively. For preliminary analysis, a total sample of 36 Indian states was obtained. The criteria for inclusion were participants with complete data for the three relevant variables: Tobacco use as an independent variable, obesity as a mediator, and diabetes diagnosis as the dependent variable.

Statistical analyses were performed using the PROCESS macro (Model 4) for SPSS Version 4.2, authored by Andrew F. Hayes. This model enables estimation of both direct and indirect effects of an independent variable on an outcome through a proposed mediator. Bias-corrected confidence intervals for the indirect effect were calculated using bootstrapping with 5,000 resamples. A threshold of  $p < .05$  was used to assess statistical significance.

Alignment with all statistical assumptions was achieved, and the results were interpreted using the mediation analysis guidelines, which placed particular emphasis on standard procedures.

### Results

#### Model Summary for Obesity (Mediator) (Table 1)

Variable	Coefficient	SE	t	p	LLCI	ULCI
Constant	34.6863	2.962	11.7098	0.0	28.6664	40.7063
Tobacco use	-0.2775	0.102	-2.7101	0.0105	-0.4855	-0.0694



Interpretation: The regression model predicting Obesity from Tobacco Use was statistically significant ( $F(1, 34) = 7.34, p = .0105, R^2 = .178$ ). Tobacco Use had a significant negative effect on Obesity ( $B = -0.2775, p = .0105$ ), suggesting that as tobacco use increases, obesity levels tend to decrease.

**Model Summary for Diabetes (Outcome) (Table 2)**

Variable	Coefficient	SE	t	p	LLCI	ULCI
Constant	8.3402	3.000	2.7792	0.0089	2.2347	14.4457
Tobacco use	-0.0034	0.051	-0.0672	0.9468	-0.1072	0.1003
Obesity	0.2402	0.077	3.1013	0.0039	0.0826	0.3977

Interpretation: The model predicting Diabetes from Tobacco Use and Obesity was significant ( $F(2, 33) = 5.96, p = .0062, R^2 = .265$ ). Obesity significantly predicted Diabetes ( $B = 0.2402, p = .0039$ ), while the direct effect of Tobacco Use was not significant ( $B = -0.0034, p = .9468$ ).

**Direct Effect of Tobacco Use on Diabetes (Table 3)**

Effect	SE	t	p	LLCI	ULCI
-0.0034	0.051	-0.0672	0.9468	-0.1072	0.1003

Interpretation: The direct effect of Tobacco Use on Diabetes was not statistically significant ( $B = -0.0034, p = .9468$ ), indicating no direct relationship when Obesity is accounted for.

**Indirect Effect of Tobacco Use on Diabetes via Obesity (Table 4)**

Mediator	Effect	BootSE	BootLLCI	BootULCI
Obesity	-0.0666	0.0395	-0.1668	-0.017

Interpretation: The indirect effect of Tobacco Use on Diabetes through Obesity was statistically significant ( $B = -0.0666, BootCI = [-0.1668, -0.0170]$ ). This indicates that Obesity mediates the relationship between Tobacco Use and Diabetes, such that increased tobacco use reduces obesity, which in turn reduces the likelihood of diabetes.



## Discussion

This study aimed to investigate the mediating function of obesity for the relation between tobacco use and diabetes among a sample of Indian adults. The analysis of data revealed a statistically significant indirect effect: Tobacco use was predictive of lower obesity levels, whereas obesity was predictive of diabetes. This means that tobacco users would have lower levels of obesity, which in turn are associated with reduced risk of diabetes. The 95% bootstrap confidence interval of the indirect effect was not zero, confirming the significance of the mediation pathway. It shows that obesity is an important intermediate variable in elucidating how tobacco smoking could influence metabolic outcomes such as diabetes.

Interestingly, the direct effect of tobacco on diabetes was not significant. This indicates that controlling obesity, tobacco smoking in itself does not have a clinically significant rise or fall on diabetes risk in the population in question. One possible explanation is that tobacco may influence diabetes through physiological alterations in weight or fat distribution, rather than an immediate metabolic effect <sup>[20]</sup>.

Previous research has shown that smoking potentially causes weight loss due to the fact that it can repress hunger and accelerate the rate at which the body uses fuel. But at the same time, smoking is not just harmful in this respect — it can be harmful in other regards as well — and includes problems like insulin not working properly, ongoing inflammation, and trouble with blood sugar regulation. These impacts will balance each other out: whereas smoking lowers weight (which tends to reduce the risk of diabetes), it also creates diseases that will raise the risk of diabetes <sup>[10]</sup>. Therefore, we may not see an apparent or significant connection between diabetes and smoking when we look at the direct impact only. Hence, it is essential to look at the whole picture, e.g., how smoking affects body weight, to fully appreciate the extent to which it is related to diabetes. Overall, this study highlights the importance of examining mediating variables in health behavior research. Obesity should be a focus target among tobacco quit programs, and truly among diabetes prevention programs. Future research with larger and more diverse samples is needed to examine these complex relationships further and determine the generalizability of these findings.

## Conclusion

The relationship between tobacco use and diabetes in Indian adults is significantly mediated by obesity, according to empirical evidence from this study. Although there was no direct correlation between tobacco use and diabetes, there was an indirect effect due to its impact on obesity. These results emphasize the significance of taking intermediary variables into account in public health research and the intricate



interaction between behavioral and metabolic factors in determining diabetes risk. The study's identification of obesity as a significant modifiable mediator raises the possibility that combined interventions aimed at reducing obesity and quitting smoking could be more successful in preventing diabetes than addressing either factor separately. The increasing prevalence of non-communicable diseases in India necessitates population-level approaches that take these pathways of mediation into consideration.

## Declaration

### Funding:

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

### Conflicts of Interest:

The authors declare that there are no conflicts of interest related to this study.

### Ethical Approval:

As the study used publicly available secondary data from the Open Government Data (OGD) Platform India, no formal ethical approval was required. All data were anonymized and used in accordance with relevant data use guidelines.

## Acknowledgement

I would like to express my sincere gratitude to **Dr. Helisse Levine** for her invaluable support, guidance, and encouragement throughout this research. Her thoughtful feedback, mentorship, and continuous motivation have been instrumental in shaping this study and enhancing the quality of my academic work. I am truly thankful for the opportunity to learn under her supervision.

## References

- 1) Pradeepa, R., & Mohan, V. (2021). Epidemiology of type 2 diabetes in India. *Indian Journal of Ophthalmology*, 69(11), 2932–2938. [https://doi.org/10.4103/ijo.IJO\\_1627\\_21](https://doi.org/10.4103/ijo.IJO_1627_21)
- 2) Cheng, Y., Wang, L., Li, Y., & Zhang, H. (2024). The impact of urban green spaces on mental health: A systematic review. *Urban Forestry & Urban Greening*, 85, 127-138. <https://doi.org/10.1016/j.ufug.2024.127138>



- 3) Indian Council of Medical Research – National Centre for Disease Informatics and Research. (2021). *Report on sites of cancer associated with tobacco use in India: Findings from the National Cancer Registry Programme* (Updated version dated 31st May 2021). Bengaluru, India: ICMR-NCDIR. [https://ncdirindia.org/all\\_reports/trc\\_report/resources/TRC\\_Chapter\\_1.pdf](https://ncdirindia.org/all_reports/trc_report/resources/TRC_Chapter_1.pdf)
- 4) Münzel, T., Hadad, O., Kuntic, M., Keaney, J. F., Deanfield, J. E., & Daiber, A. (2020). Effects of tobacco cigarettes, e-cigarettes, and waterpipe smoking on endothelial function and clinical outcomes. *European Heart Journal*. Advance online publication. <https://doi.org/10.1093/eurheartj/ehaa460>
- 5) American Heart Association. (2024, March 20). *Exposure to tobacco before birth significantly increased risk of Type 2 diabetes in adults*. <https://newsroom.heart.org/news/exposure-to-tobacco-before-birth-significantly-increased-risk-of-type-2-diabetes-in-adults>
- 6) Yashi, K., & Daley, S. F. (2023, June 19). *Obesity and type 2 diabetes*. In StatPearls. StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK592412/>
- 7) Thorgeirsson, T. E., Geller, F., Sulem, P., Rafnar, T., Wiste, A., Magnusson, K. P., ... & Stefansson, K. (2015). High tobacco consumption lowers body weight: a Mendelian randomization study. *International Journal of Epidemiology*, 44(2), 540–550. <https://doi.org/10.1093/ije/dyu276>
- 8) Münzel, T., Hahad, O., Kuntic, M., Keaney, J. F., Deanfield, J. E., & Daiber, A. (2020). Effects of tobacco cigarettes, e-cigarettes and waterpipe smoking on endothelial function and clinical outcomes. *European Heart Journal*, 41(41), 4057–4070. <https://doi.org/10.1093/eurheartj/ehaa460>
- 9) American Heart Association. (2024, March 20). *Exposure to tobacco before birth significantly increased risk of Type 2 diabetes in adults*. <https://newsroom.heart.org/news/exposure-to-tobacco-before-birth-significantly-increased-risk-of-type-2-diabetes-in-adults>
- 10) Chiolero, A., Faeh, D., Paccaud, F., & Cornuz, J. (2008). Consequences of smoking for body weight, body fat distribution, and insulin resistance. *The American Journal of Clinical Nutrition*, 87(4), 801–809. <https://doi.org/10.1093/ajcn/87.4.801>
- 11) Winsløw, U. C., Rode, L., & Nordestgaard, B. G. (2015). High tobacco consumption lowers body weight: A Mendelian randomization study of the Copenhagen General Population Study. *International Journal of Epidemiology*, 44(2), 540–550. <https://doi.org/10.1093/ije/dyu276>
- 12) Yashi, K., & Daley, S. F. (2023). *Obesity and type 2 diabetes*. In StatPearls. StatPearls Publishing. <https://www.ncbi.nlm.nih.gov/books/NBK592412/>
- 13) Roussel, R., Travert, F., Detournay, B., Simon, D., Charbonnel, B., Fournier, C., Emery, C., Lièvre, M., Halimi, S., & Marre, M. (2024). *Association between tobacco smoking and microvascular and macrovascular complications in patients with type 2 diabetes mellitus: A real-life study in France*. *Primary Care Diabetes*, 18, 101347. <https://doi.org/10.1016/j.pcd.2024.101347>
- 14) Lee, P. N., & Coombs, K. J. (2020). *A review of the evidence on the effects of nicotine on human performance*. *World Journal of Meta-Analysis*, 8(2), 119–143. <https://www.wjgnet.com/2308-3840/full/v8/i2/119.htm>
- 15) Centers for Disease Control and Prevention. (2024, May 15). *Smoking and diabetes*. U.S. Department of Health & Human Services. <https://www.cdc.gov/diabetes/risk-factors/diabetes-and-smoking.html>



- 16) Durlach, V., Vergès, B., Al-Salameh, A., Bahougne, T., Benzerouk, F., Berlin, I., Clair, C., Mansourati, J., Rouland, A., Thomas, D., Thuillier, P., Tramunt, B., & Le Faou, A.-L. (2022). Smoking and diabetes interplay: A comprehensive review and joint statement. *Diabetes & Metabolism*, 48(6), 101370. <https://doi.org/10.1016/j.diabet.2022.101370>
- 17) Popkin, B. M., Adair, L. S., & Ng, S. W. (2012). Global nutrition transition and the pandemic of obesity in developing countries. *Nutrition Reviews*, 70(1), 3–21. <https://doi.org/10.1111/j.1753-4887.2011.00456>.
- 18) Duan, Y., Xu, C., Wang, W., Wang, X., Xu, N., Zhong, J., Gong, W., Zheng, W., Wu, Y.-H., Myers, A., Chu, L., Lu, Y., Delzell, E., Hsing, A. W., Yu, M., He, W., & Zhu, S. (2025). Smoking-related gut microbiota alteration is associated with obesity and obesity-related diseases: Results from two cohorts with sibling comparison analyses. *BMC Medicine*, 23(1), 146. <https://doi.org/10.1186/s12916-025-03969-4>
- 19) National Informatics Centre. (n.d.). *Open Government Data (OGD) Platform India*. Ministry of Electronics & Information Technology, Government of India. Retrieved May 23, 2025, from <https://data.gov.in/>
- 20) U.S. Food and Drug Administration. (n.d.). *How smoking can increase the risk for and affect diabetes*. <https://www.fda.gov/tobacco-products/health-effects-tobacco-use/how-smoking-can-increase-risk-and-affect-diabetes>