

Review Article

The Double Burden of Malnutrition and Diabetes in India: The Paradox of the Thin-Fat Phenotype

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Abstract

Background: India faces a unique epidemiological paradox characterized by the coexistence of persistent childhood malnutrition (35.5% stunting prevalence) and an escalating diabetes epidemic affecting 77 million adults, projected to reach 134 million by 2045. This dual burden manifests through the emergence of the "thin-fat" phenotype—individuals appearing thin by conventional anthropometric standards yet harbouring excess visceral adiposity and elevated type 2 diabetes risk. This phenomenon challenges Western paradigms linking obesity to diabetes, as Indians develop diabetes at significantly lower BMI levels than European populations.

Methods: A comprehensive literature review was conducted using PubMed, Scopus, and Web of Science databases, covering publications from 1990 to 2023. Primary data sources included the National Family Health Survey-5, ICMR-INDIAB studies, and WHO reports. Studies on Indian populations, malnutrition-diabetes relationships, thin-fat phenotype characteristics, and health system responses were systematically analysed.

Results: The thin-fat phenotype affects 43.3% of India's population, with a higher prevalence in rural areas (46%) than in urban areas (39.6%). State-level analyses reveal an inverse relationship between malnutrition and diabetes prevalence, indicating different epidemiological transition stages. Kerala demonstrates low malnutrition (19.7% underweight) but high diabetes prevalence (25.5%), while Jharkhand exhibits severe malnutrition (39.6% stunting) with emerging diabetes concerns (7.2%).

The phenomenon extends to household levels, with undernourished children and diabetic adults coexisting within families. Physiological mechanisms underlying this paradox include developmental programming through Barker's and Pedersen's hypotheses, altered adipose tissue distribution favouring visceral fat accumulation, sarcopenic obesity, and unique beta-cell dysfunction characteristics in Indian populations.

Health System Implications: Current approaches treating malnutrition and diabetes as separate conditions through siloed programs (ICDS and National Programme for Prevention and Control of Non-Communicable Diseases) prove inadequate. BMI-based screening protocols miss 35–42% of thin diabetics, while healthcare providers lack training on dual burden complexities.

The economic burden is substantial. Diabetes care costs range from INR 5,000 to 45,000 annually, forcing 48.5% of families into distressed financing.

Conclusion: India's dual burden requires urgent, integrated health system transformation addressing both conditions simultaneously. Success demands unified screening protocols, comprehensive healthcare worker training, and coordinated policy approaches that transcend conventional disease categories to break intergenerational malnutrition and metabolic dysfunction cycles.

Introduction:

India's epidemiological landscape reveals a stark contradiction that challenges conventional understanding of nutritional health. While the country continues to grapple with persistent childhood malnutrition, with 35.5% of children under five remaining stunted, according to the National Family Health Survey-5 [1], it simultaneously faces an escalating diabetes epidemic affecting 77 million adults and projected to reach 134 million by 2045 [2]. This coexistence of undernutrition and overnutrition-related diseases within the same population, households, and even individuals represents what epidemiologists term the "dual burden of malnutrition."

The manifestation of this dual burden in India is particularly unique, characterized by the emergence of the "thin-fat" phenotype—individuals who appear thin by conventional anthropometric standards yet harbour excess visceral adiposity and demonstrate elevated risk for type 2 diabetes and cardiovascular disease [3, 4]. This phenotype challenges traditional Western paradigms that closely link obesity with diabetes risk, as Indians develop diabetes at significantly lower body mass index levels compared to European populations [5].

Background

The phenomenon extends beyond individual health implications, representing a complex interplay of developmental programming, genetic predisposition, environmental factors, and socioeconomic determinants [6, 7]. Two fundamental hypotheses of intrauterine programming explain the underlying mechanisms. Barker's hypothesis, also known as the "thrifty phenotype hypothesis," proposes that fetal undernutrition leads to permanent changes in physiology and metabolism that increase susceptibility to cardiovascular disease and diabetes in adult life [8]. Conversely, Pedersen's hypothesis describes how maternal hyperglycaemia during pregnancy leads to fetal hyperinsulinemia, resulting in macrosomic newborns who face increased risk of obesity and diabetes later in life [9]. Both pathways create intergenerational transmission of metabolic dysfunction—malnourished mothers may give birth to low-birth-weight children programmed for diabetes despite remaining underweight throughout life.

In contrast, mothers with gestational diabetes produce macrosomic offspring with equally elevated diabetes risk [8, 9, 10]. This dual vulnerability explains India's high prevalence of hyperglycaemia in pregnancy (affecting 13% of pregnancies) [11]. It creates intergenerational cycles where metabolically compromised mothers produce offspring prone to both malnutrition and subsequent diabetes, regardless of birth weight extremes. Current health policies and programs in India inadequately address this intersection. The Integrated Child Development Services (ICDS) focuses primarily on childhood undernutrition, while the National Programme for Prevention and Control of Non-Communicable Diseases operates independently, missing opportunities for synergistic interventions [12, 13]. This siloed approach fails to recognize the interconnected nature of these conditions and the shared pathways that link early malnutrition to later chronic disease development.

Understanding India's dual burden requires examining the country's unique epidemiological transition, where rapid urbanization, dietary changes, and lifestyle modifications overlay persistent poverty, food insecurity, and inadequate healthcare access. The thin-fat phenotype emerges from this collision between traditional nutritional challenges and modern lifestyle diseases, demanding innovative approaches that transcend conventional disease categories.

This review synthesizes current evidence on India's dual burden of malnutrition and diabetes, explores the physiological mechanisms underlying the thin-fat phenotype, examines health system implications, and proposes integrated strategies for addressing this complex challenge that threatens to undermine decades of public health progress.

Methods

Literature Search Strategy

A comprehensive literature review was conducted using multiple databases, including PubMed, Scopus, and Web of Science, covering publications from 1990 to 2023. Search terms included combinations of "dual burden," "malnutrition," "diabetes," "India," "thin-fat phenotype," "Asian Indian phenotype," and "double burden of malnutrition." Additional sources were identified through reference lists of key publications and reports from major health organizations.

Data Sources

Primary data sources included the National Family Health Survey-5 (2019-21), ICMR-INDIAB studies, Global Burden of Disease studies, and WHO reports on malnutrition and non-communicable diseases. Government policy documents and program guidelines were reviewed to assess current intervention approaches.

Inclusion Criteria

Studies were included if they: (1) focused on Indian populations or South Asian diaspora, (2) examined relationships between malnutrition and diabetes or metabolic dysfunction, (3) investigated the thin-fat phenotype or similar metabolic characteristics, (4) evaluated health system responses to dual burden, or (5) provided comparative international perspectives on dual burden management.

Results

I. Epidemiological Evidence

National Prevalence Patterns: India's dual burden manifests at multiple levels with striking regional variations. The ICMR-INDIAB study documented national diabetes prevalence of 11.4%, with urban rates reaching 16.4% and rural areas 8.9% [14]. Simultaneously, the National Family Health Survey-5 revealed persistent childhood malnutrition with 35.5% stunting, 19.3% wasting, and 32.1% underweight prevalence [1].

State-level analyses reveal an inverse relationship between malnutrition and diabetes prevalence, suggesting different stages of epidemiological transition. Kerala demonstrates low malnutrition rates (19.7% underweight) but high diabetes prevalence (25.5%), while Jharkhand exhibits severe malnutrition (39.6% stunting) with emerging diabetes concerns (7.2%) [1, 14]. This pattern suggests that states progress from high malnutrition-low to low malnutrition-high diabetes profiles during economic development.

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Household-Level Co-existence

Perhaps most striking is the manifestation of this dual burden within individual households. Studies from various urban centres have consistently documented the co-existence of undernourished children and diabetic or overweight adults within the same family. Research has shown that this phenomenon is not limited to urban populations; insights from research from rural India reveal a similar pattern of stunted children and hyperglycaemic mothers, highlighting the geographic spread of this complex public health challenge beyond major cities.

This household-level co-existence reflects complex interactions between shared genetic susceptibility, common environmental exposures, and socioeconomic constraints perpetuating undernutrition and chronic disease risk across generations [6].

The Thin-Fat Phenotype Prevalence

The thin-fat phenotype, or Metabolically Obese Non-Obese (MONO), is a well-established health concern in India, particularly among South Asian populations. This condition affects individuals with a normal Body Mass Index (BMI), typically below 23 kg/m², who exhibit features of metabolic syndrome.

43.3% of the Indian population falls into the MONO category. This condition is more prevalent in rural areas (46%) than in urban areas (39.6%), highlighting its widespread nature nationwide. These individuals consistently show a high percentage of body fat, central obesity (waist circumference >90cm for men, >80cm for women), and markers for insulin resistance [15].

Research by leading experts has been foundational in defining this unique body composition. Using advanced imaging techniques such as dual-energy X-ray absorptiometry (DEXA) and CT scans, their studies have confirmed that many normal-weight Indians carry excessive visceral adiposity. This is a significant risk factor for non-communicable diseases. The prevalence of diabetes is notably higher among this subgroup compared to those with a healthy fat distribution, making it a major public health concern as these individuals often remain undiagnosed due to their normal BMI [16].

Physiological Mechanisms

Developmental Programming

The thin-fat phenotype's origins trace to early developmental programming during fetal and early postnatal life, explained by two complementary hypotheses [6, 7, 8]. Barker's hypothesis (the "thrifty phenotype hypothesis") proposes that maternal malnutrition during pregnancy triggers fetal adaptations favouring survival in nutrient-scarce environments through enhanced insulin sensitivity and glucose conservation. While initially protective, these adaptations become maladaptive when nutrition improves later in life, leading to insulin resistance and diabetes.

Conversely, Pedersen's hypothesis describes the metabolic consequences of maternal hyperglycaemia during pregnancy [9]. Maternal glucose crosses the placenta, but insulin does not, leading to fetal hyperinsulinemia and accelerated growth. This results in macrocosmic newborns with enlarged pancreatic beta cells and altered metabolic programming that predisposes to obesity and diabetes in later life.

Both pathways are prevalent in India: low birth weight (<2.5kg) increases diabetes risk by 2–3-fold, while macrosomic births (>4kg) from gestational diabetes show similarly elevated risk [9, 10]. The Pune Maternal Nutrition Study demonstrated that individuals with low birth weight who experience later weight gain show the highest insulin resistance levels. At the same time, macrosomic newborns develop early-onset metabolic dysfunction [7, 10]. This creates a U-shaped relationship where both extremes of birth weight contribute to India's diabetes epidemic.

Epigenetic modifications established during fetal development persist into adulthood, affecting genes regulating metabolism, adipogenesis, and insulin signalling. These modifications create lasting changes in metabolic programming that predispose individuals to diabetes regardless of birth weight extremes, explaining why both undernourished and overnourished fetuses develop similar metabolic vulnerabilities through different pathways.

Altered Body Composition

Central to the thin-fat phenotype is altered adipose tissue distribution and function [4, 17]. Indians are genetically predisposed to visceral fat accumulation with lower subcutaneous fat storage capacity than Europeans. This leads to ectopic fat deposition in the liver, muscle, and pancreas, even at low total body fat levels.

Sarcopenic obesity represents another facet, where Indians exhibit lower muscle mass and higher intramyocellular lipid content compared to other ethnicities [6]. This combination reduces insulin-mediated glucose uptake and increases reliance on hepatic glucose production, creating metabolic inefficiency despite normal appearance.

Beta-Cell Dysfunction

Indian populations demonstrate unique beta-cell characteristics contributing to thin-fat syndrome [4, 5]. Beta-cells show reduced insulin secretory capacity and increased susceptibility to glucose toxicity, manifesting as earlier beta-cell failure occurring at lower glucose thresholds than in other populations. The incretin system also shows differences, with reduced GLP-1 responses observed in thin Indians with prediabetes.

I. Health System Implications

Current Program Gaps

India's health system addresses malnutrition and diabetes through separate, poorly coordinated programs [12, 13]. The ICDS targets childhood malnutrition through supplementary nutrition and growth monitoring, reaching 100 million beneficiaries annually.

Meanwhile, the National Programme for Prevention and Control of Non-Communicable Diseases focuses on adult diabetes screening and management through primary healthcare centres.

This siloed approach misses critical intervention opportunities. Malnourished pregnant women receiving ICDS benefits face significant healthcare access challenges, with gestational diabetes prevalence rates varying from 4% to 18% across Indian states. Yet, systematic screening integration with nutrition programs remains limited [18].

Diagnostic Challenges

Current screening protocols inadequately identify thin-fat individuals [3, 19]. BMI-based diabetes screening (BMI ≥ 23 kg/m² for Indians) misses approximately 35–42% of thin diabetics, with studies showing that 34.6% of males and 41.9% of females have metabolically obese phenotypes despite normal BMI [20]. Waist circumference measurements, though recommended, are inconsistently implemented due to cultural sensitivities and healthcare worker training gaps.

Healthcare providers receive inadequate training on dual burden complexities. Most doctors at primary care facilities did not receive any specialized training in diabetes, and there was no written protocol for screening and management of diabetes in most facilities. Primary care physicians report lacking the necessary knowledge when managing complex cases. A survey of clinical diabetologists showed that low awareness among physicians (22.7%) and non-applicability of Western guidelines in Indian patients (22.7%) are existing barriers to evidence-based diabetes [21]. The gap in training becomes particularly pronounced when dealing with patients who simultaneously present with undernutrition and metabolic disorders, requiring nuanced clinical approaches that most healthcare providers have not been equipped to handle.

Economic Impact

The dual burden of diabetes and undernutrition imposes a significant economic strain on India, contributing to a cycle of poverty and poor health outcomes. Research indicates that the average annual expenditure on diabetes care ranges from INR 5,000 to INR 45,000, a cost that consumes a significant portion of family income [22].

The high costs often force families into distressed financing, with nearly half of households (48.5%) resorting to sources like borrowing or selling assets to cover diabetes-related inpatient costs. This financial strain is particularly acute for lower-income groups and those in rural areas [23].

While the Ministry of Health and Family Welfare received a substantial allocation of Rs 99,859 crores (approximately \$11.5 billion) in the 2025–26 Union Budget, the specific funding for diabetes programs remains limited relative to the disease's growing burden [27]. This highlights a critical gap in resource allocation and reflects the broader challenges in healthcare financing in the country.

Discussion

Mechanisms Underlying the Paradox

The coexistence of malnutrition and diabetes in India reflects complex interactions between developmental programming, genetic predisposition, and environmental factors unique to the country's epidemiological transition [6, 10]. The thin-fat phenotype emerges from fetal adaptations to maternal malnutrition, which become maladaptive in environments with improved but suboptimal nutrition.

This phenomenon challenges traditional obesity-centric models of diabetes causation prevalent in Western populations. Indians develop diabetes at significantly lower BMI levels due to preferential visceral fat accumulation, reduced muscle mass, and intrinsic beta-cell limitations [3, 5]. These characteristics likely represent evolutionary adaptations to historical patterns of feast and famine that become problematic during nutritional transitions.

Policy Integration Imperatives

Addressing India's dual burden requires fundamental transformation of current health system approaches. The evidence overwhelmingly supports integrated interventions that simultaneously address undernutrition and diabetes risk rather than treating these as separate conditions. Successful integration models from Brazil and Mexico demonstrate that comprehensive approaches achieve better outcomes at lower costs than siloed programs [24, 25, 26].

Key integration priorities include: unified screening protocols incorporating waist circumference and family history assessment; integrated training curricula for healthcare workers; revised dietary guidelines addressing undernutrition and diabetes prevention; and restructured financing mechanisms supporting comprehensive care rather than disease-specific interventions.

International Lessons

Global experiences provide valuable guidance for India's response. Brazil's Family Health Strategy achieved a 60% reduction in child malnutrition and a 40% improvement in diabetes control through integrated community health worker programs [26]. Mexico's Prospera program demonstrated that addressing social determinants while providing integrated healthcare can effectively tackle dual burdens [24].

These successes emphasize the importance of multi-sectoral approaches that extend beyond healthcare to encompass food systems, social protection, and urban planning. Countries successfully addressing dual burden invested 2–3% of GDP in integrated nutrition and health programs while implementing regulatory changes across multiple sectors.

Limitations and Research Gaps

Several limitations constrain current understanding of India's dual burden. Standardized definitions for dual burden measurement remain lacking, limiting cross-study comparisons. Longitudinal studies tracking individuals from birth through adulthood are scarce, hindering mechanistic understanding of thin-fat phenotype development. Intervention studies testing integrated approaches are particularly needed to inform policy development.

Critical research priorities include: large-scale prevalence studies using standardized dual burden definitions; mechanistic studies on thin-fat phenotype development in Indian populations; intervention trials testing integrated approaches; and health economic evaluations comparing unified versus separate programs.

Conclusion

India's dual burden of malnutrition and diabetes, epitomized by the thin-fat phenotype, represents a defining public health challenge requiring an urgent, comprehensive response. This paradox emerges from complex interactions between developmental programming, genetic predisposition, and environmental factors unique to India's epidemiological transition, affecting millions who appear thin yet face elevated diabetes risk.

The evidence overwhelmingly demonstrates that current approaches to treating malnutrition and diabetes as separate conditions are fundamentally inadequate. The physiological mechanisms underlying the thin-fat phenotype—including fetal programming, altered adipose distribution, and metabolic dysfunction—demand integrated interventions addressing both conditions simultaneously.

Health system transformation requires unified screening protocols, integrated healthcare worker training, and coordinated policy approaches that maximize resource efficiency while improving outcomes. International experiences from transitioning economies provide valuable implementation guidance, emphasizing the importance of addressing social determinants through multi-sectoral coordination.

The stakes extend beyond individual health outcomes. India's dual burden perpetuates intergenerational cycles of poor health, threatens economic development, and challenges achieving sustainable development goals. However, it also presents an opportunity for innovative health system development that could serve as a global model for other transitioning economies. Success requires sustained political commitment, adequate financing, and community engagement to transform India's dual burden challenge into comprehensive health system innovation. Addressing this paradox is not merely a health imperative.

It represents a crucial step toward equitable, sustainable development that breaks intergenerational cycles of malnutrition and metabolic dysfunction, transforming India's nutritional landscape for future generations.

References:

1. International Institute for Population Sciences (IIPS) and ICF. (2022). National Family Health Survey (NFHS-5), 2019-21: India. IIPS.
2. 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
3. Misra, A., & Khurana, L. (2008). Obesity and the metabolic syndrome in developing countries. *The Journal of Clinical Endocrinology & Metabolism*, 93(11 Suppl 1), S9-30. <https://doi.org/10.1210/jc.2008-1595>
4. Mohan, V., & Deepa, R. (2006). Adipocytokines and the expanding 'Asian Indian Phenotype'. *Journal of the Association of Physicians of India*, 54, 685-686.
5. Gujral, U. P., Pradeepa, R., Weber, M. B., Narayan, K. M., & Mohan, V. (2013). Type 2 diabetes in South Asians: similarities and differences with white Caucasian and other populations. *Annals of the New York Academy of Sciences*, 1281, 51-63. <https://doi.org/10.1111/j.1749-6632.2012.06838.x>
6. Wells, J. C., Sawaya, A. L., Wibaek, R., Mwangome, M., Poullas, M. S., Yajnik, C. S., & Prentice, A. (2020). The double burden of malnutrition: aetiological pathways and consequences for health. *The Lancet*, 395(10217), 75-88. [https://doi.org/10.1016/S0140-6736\(19\)32472-9](https://doi.org/10.1016/S0140-6736(19)32472-9)
7. Yajnik, C. S. (2002). The lifecycle effects of nutrition and body size on adult adiposity, diabetes, and cardiovascular disease. *Obesity Reviews*, 3(3), 217-224. <https://doi.org/10.1046/j.1467-789x.2002.00072.x>
8. Hales, C. N., & Barker, D. J. (1992). Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. *Diabetologia*, 35(7), 595-601. <https://doi.org/10.1007/BF00400248>
9. Krishnaveni, G. V., Veena, S. R., Hill, J. C., Kehoe, S., Karat, S. C., & Fall, C. H. (2010). Intrauterine exposure to maternal diabetes is associated with higher adiposity and insulin resistance and clustering of cardiovascular risk markers in Indian children. *Diabetes Care*, 33(2), 402-404. <https://doi.org/10.2337/dc09-1393>

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10. Yajnik, C. S., Fall, C. H., Coyaji, K. J., Hirve, S. S., Rao, S., Barker, D. J., Joglekar, C., & Kellingray, S. (2003). Neonatal anthropometry: the thin-fat Indian baby. *The Pune Maternal Nutrition Study. International Journal of Obesity and Related Metabolic Disorders*, 27(2), 173-180.
<https://doi.org/10.1038/sj.ijo.802219>
11. Mantri, N., Goel, A. D., Patel, M., Baskaran, P., Dutta, G., Gupta, M. K., Yadav, V., Mittal, M., Shekhar, S., & Bhardwaj, P. (2024). National and regional prevalence of gestational diabetes mellitus in India: a systematic review and Meta-analysis. *BMC Public Health*, 24(1), 527.
<https://doi.org/10.1186/s12889-024-18024-9>
12. Ministry of Women and Child Development, Government of India. (2017). Integrated Child Development Services (ICDS) Scheme. Government of India.
13. Ministry of Health & Family Welfare, Government of India. (2023). National Programme for Prevention and Control of Non-Communicable Diseases operational guidelines (2023-2030). Directorate General of Health Services.
14. Anjana, R. M., Unnikrishnan, R., Deepa, M., Pradeepa, R., Tandon, N., Das, A. K., Joshi, S., Bajaj, S., Jabbar, P. K., Das, H. K., Kumar, A., Dhandhaniala, V. K., Bhansali, A., Rao, P. V., Desai, A., Kalra, S., Gupta, A., Lakshmy, R., Madhu, S. V., Elangovan, N., ... ICMR-INDIAB Collaborative Study Group (2023). Metabolic non-communicable disease health report of India: the ICMR-INDIAB national cross-sectional study (ICMR-INDIAB-17). *The Lancet. Diabetes & endocrinology*, 11(7), 474-489.
[https://doi.org/10.1016/S2213-8587\(23\)00119-5](https://doi.org/10.1016/S2213-8587(23)00119-5)
15. Deepa, M., Pradeepa, R., Venkatesan, U., Adhikari, P., Das, H. K., Dash, K., Mokta, J. K., Ningomban, S., Luaia, R., Budnah, R.O., Bhansali, A., Jampa, L., Suokhrie, V., Jain, S. M., Joshi, P. P., Joshi, S., Purty, A. J., Tobgay, K. J., Reang, T., Anjana, R. M. (2025). High prevalence of metabolic obesity in India: The ICMR-INDIAB national study (ICMR-INDIAB-23). *The Indian Journal of Medical Research*, 161, 461-472.
https://doi.org/10.25259/ijmr_328_2025
16. Misra, A., Anoop, S., Gulati, S., Mani, K., Bhatt, S. P., & Pandey, R. M. (2015). Body Fat Patterning, Hepatic Fat and Pancreatic Volume of Non-Obese Asian Indians with Type 2 Diabetes in North India: A Case-Control Study. *PLoS ONE*, 10(10), e0140447.
<https://doi.org/10.1371/journal.pone.0140447>
17. Raji, A., Seely, E. W., Arky, R. A., & Simonson, D. C. (2001). Body fat distribution and insulin resistance in healthy Asian Indians and Caucasians. *The Journal of Clinical Endocrinology & Metabolism*, 86(11), 5366-5371.
<https://doi.org/10.1210/jcem.86.11.7992>
18. Swaminathan, G., Swaminathan, A., & Corsi, D. J. (2020). Prevalence of Gestational Diabetes in India by Individual Socioeconomic, Demographic, and Clinical Factors. *JAMA network open*, 3(11), e2025074.
<https://doi.org/10.1001/jamanetworkopen.2020.25074>
19. Pradeepa, R., Anjana, R. M., Joshi, S. R., Bhansali, A., Deepa, M., Joshi, P. P., Dhandhaniala, V. K., Madhu, S. V., Rao, P. V., Geetha, L., Subashini, R., Unnikrishnan, R., Shukla, D. K., Kaur, T., Mohan, V., & Das, A. K. (2015). Prevalence of generalized and abdominal obesity in urban and rural India--the ICMR-INDIAB Study (Phase-I) [ICMR-INDIAB-3]. *Indian Journal of Medical Research*, 142(2), 139-150.
<https://doi.org/10.4103/0971-5916.164234>
20. Kalra, S., Mithal, A., Zargar, A. H., Sethi, B., Dharmalingam, M., Ghosh, S., & Sen, R. (2022). Indian Phenotype Characteristics Among Patients with Type 2 Diabetes Mellitus: Insights from a Non-interventional Nationwide Registry in India. *TouchREVIEWS in endocrinology*, 18(1), 63-70.
<https://doi.org/10.17925/EE.2022.18.1.63>
21. Hasan, H., Zodpey, S., & Saraf, A. (2011). Diabetologist's perspective on practice of evidence-based diabetes management in India. *Diabetes Research and Clinical Practice*, 95(2), 189-193. <https://doi.org/10.1016/j.diabres.2011.09.021>
22. Deshpande, P. R., & Jadhav, S. (2025). Economic Burden of Type 2 Diabetes Mellitus in India with Bibliometric and Quality Analysis of Studies: A Systematic Review. *Medical Journal of Dr D Y Patil Vidyapeeth*, 18(3), 412-430.
https://doi.org/10.4103/mjdrdypu.mjdrdypu_77_24
23. Nanda, M., & Sharma, R. (2023). Financial burden of seeking diabetes mellitus care in India: Evidence from a Nationally Representative Sample Survey. *Health care science*, 2(5), 291-305. <https://doi.org/10.1002/hcs2.65>
24. Fernald, L. C., Gertler, P. J., & Neufeld, L. M. (2008). Role of cash in conditional cash transfer programmes for child health, growth, and development: an analysis of Mexico's Oportunidades. *The Lancet*, 371(9615), 828-837.
Doi:10.1016/S0140-6736(08)60382-7
25. Rasella, D., Harhay, M. O., Pamponet, M. L., Kroeger, A., & Barreto, M. L. (2014). Impact of primary health care on mortality from heart and cerebrovascular diseases in Brazil: a nationwide analysis of longitudinal data. *BMJ*, 349, g4014.
<https://doi.org/10.1136/bmj.g4014>

26. Victora, C. G., Aquino, E. M., do Carmo Leal, M., Monteiro, C. A., Barros, F. C., & Szwarcwald, C. L. (2011). Maternal and child health in Brazil: progress and challenges. *The Lancet*, 377(9780), 1863-1876. [https://doi.org/10.1016/S0140-6736\(11\)60138-4](https://doi.org/10.1016/S0140-6736(11)60138-4)

27. Demand for Grants 2025-26 Analysis: Health and Family Welfare. (n.d.). PRS Legislative Research. <https://prsindia.org/budgets/parliament/demand-for-grants-2025-26-analysis-health-and-family-welfare>

28. Rivera, J. A., de Cossío, T. G., Pedraza, L. S., Aburto, T. C., Sánchez, T. G., & Martorell, R. (2014). Childhood and adolescent overweight and obesity in Latin America: a systematic review. *The Lancet Diabetes & Endocrinology*, 2(4), 321-332. [https://doi.org/10.1016/S2213-8587\(13\)70173-6](https://doi.org/10.1016/S2213-8587(13)70173-6)

Informed Consent: NA

Conflict of Interest Statement

The authors declared “No Conflict of Interest” with this publication.

Additional Information

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<https://doi.org/10.62996/daj.62042025>

Cite this Article:

Sankalp Ghadei, Deniza Patel, Jankhana Patel. The Double Burden of Malnutrition and Diabetes in India: The Paradox of the Thin-Fat Phenotype. *Diabetes Asia Journal*; 2(2):100-106 <https://doi.org/10.62996/daj.62042025>