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The Weight of Progress: A Sequential Mediation Analysis of Socioeconomic Drivers, the Nutrition Transition, and the Hypertension Epidemic in Nepal

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Abstract

The rising burden of hypertension in low- and middle-income countries reflects complex interactions between socioeconomic change, behavioral risk factors, and physiological pathways. This study examines the mechanisms linking socioeconomic determinants to hypertension in Nepal through the lens of the nutrition transition. Using data from the nationally representative Nepal WHO STEPS Survey 2019 (n = 5 281), we conducted a survey-weighted structural equation model to assess direct and indirect pathways involving behavioral mediators i.e tobacco use, alcohol consumption, high-salt intake, stress and physiological mediator obesity.

Results demonstrate that hypertension is primarily driven by proximal factors, specifically obesity and stress, while socioeconomic determinants operate largely through indirect pathways. Obesity emerged as a key mediator linking higher education, urban residence, marital status, and Janajati ethnicity to increased hypertension risk. Significant obesity-mediated effects were observed for education (OR = 1.01), urban residence (OR = 1.03), and marital status (OR = 1.05). In contrast, total effects of these determinants were often nonsignificant, indicating offsetting pathways. Stress also showed an independent association with hypertension and was strongly patterned by marital status, highlighting an additional psychosocial pathway.

These findings support a sequential weight of progress pathway, in which socioeconomic advancement shapes behavioral exposures that contribute to adiposity, ultimately increasing hypertension risk. The results underscore the importance of distinguishing distal from proximal determinants in cardiometabolic disease epidemiology. In the context of Nepal's rapid socioeconomic transition, effective prevention strategies should prioritize obesity reduction and address psychosocial stressors, alongside strengthening primary care hypertension management.

Summary Table

What is known about the topic

- Hypertension is a major contributor to the growing burden of non-communicable diseases in low-and middle-income countries, including Nepal.
- Socioeconomic status and behavioral risk factors such as diet, physical inactivity, and substance use are associated with hypertension risk.
- Previous studies typically model socioeconomic factors as direct predictors, with limited examination of underlying pathways.

What this study adds

- Demonstrates a sequential mediation pathway linking socioeconomic determinants to hypertension via behavioral factors and obesity.
- Identifies obesity as the primary physiological mediator connecting socioeconomic change to hypertension risk in Nepal.
- Stress emerges as an independent psychosocial pathway influencing hypertension, highlighting the importance of non-metabolic mechanisms.

Introduction

Globally, non-communicable Diseases (NCDs) account for the majority of morbidity and mortality in both high and low-income countries, driven by shifts in diet, physical activity, and other lifestyle factors.[1–3] Hypertension currently affects an estimated 1.4 billion adults worldwide aged 30–79 years, disproportionately burdening low- and middle-income countries (LMICs). [4] Globally, obesity affects ~ 16% of adults, 890 million aged 18+, with projections reaching 1.1 billion by 2030, burdening LMICs.[3,5,6] Within Asia, ongoing urbanization, expansion of processed food markets, and shifts in occupational patterns have accelerated the epidemiological transition, exposing populations to both behavioral and physiological risk factors at younger ages.[7,8]

Nepal is experiencing a rapid transformation characterized by a rising burden of NCDs, which is increasingly hiding communicable diseases, in the context of ongoing socioeconomic transformations.[9,10] Hypertension and obesity have emerged as major contributors to adult morbidity and mortality, reflecting a shift in the country's health profile.[3,11,12] This represents a public health challenge, with implications for population health and healthcare systems in Nepal. Socioeconomic status (SES) has long been associated with NCD risk in LMICs, including Nepal, where higher wealth and education often confer elevated hypertension and obesity risk[11,13–15]. However, SES is frequently modeled as a simple, proximal risk factor rather than a distal determinant operating through intermediate behavioral and physiological pathways. Consequently, the mechanisms through which socioeconomic change influences hypertension remain incompletely understood.

The Nutrition Transition framework articulated by Barry Popkin [16] provides a conceptual lens for understanding these dynamics. Economic development in LMICs is associated with dietary and lifestyle shifts, specifically, increased consumption of energy-dense, salt-fortified processed foods and reduced physical activity that contribute to rising rates of obesity, hypertension, and other cardiometabolic conditions [17]. These transitions are relevant in South Asia, where the "thin–fat" phenotype characterized by disproportionate visceral adiposity at relatively low body mass index (BMI) amplifies cardiometabolic risk even in the absence of marked overweight or obesity. [18,19] This biological susceptibility underscores the plausibility of adiposity as a mediator linking socioeconomic advancement to hypertension in the region.

Nepal's epidemiological trajectory mirrors these global and regional patterns. Overweight and obesity among Nepalese women increased from 22.2% in 2016 to 29.2% in 2022[3], and higher SES individuals consistently exhibit elevated hypertension risk.[20] Yet, current literature often treats SES as a direct risk factor, with limited exploration of the behavioral and physiological pathways that may mediate this relationship. Evidence suggests that dietary practices, physical inactivity, psychosocial stress, and adiposity[15,21–24] may act as sequential mediators, translating socioeconomic advantage into cardiovascular risk.

Understanding the pathways linking SES to hypertension is critical in Nepal's context, where rapid socioeconomic change coincides with shifting dietary and physical activity patterns, growing urbanization, and emerging chronic disease burdens. The interplay of these factors may disproportionately affect populations with genetic and phenotypic susceptibility. Disentangling behavioral from physiological mediators provides insight into the mechanisms of disease emergence and identifies potential targets for intervention, policy, and prevention [25]. While higher individual SES typically elevates hypertension risk in Nepal, recent multilevel analyses reveal an inverse association at the area-level, where greater deprivation links to lower prevalence, 17.9% vs. 27.5% in affluent areas, largely mediated by BMI as a proxy for nutritional status, highlighting context-specific gradients during the nutrition transition.[26]

Despite the recognized association between SES and hypertension, quantitative analyses accounting for multiple sequential mediators remain limited. Structural Equation Modelling (SEM) allows estimation of direct and indirect effects through behavioral dietary salt intake, i.e., physical inactivity, and physiological mediators, i.e., BMI, adiposity[27] providing a comprehensive framework to assess mechanisms linking socioeconomic change to hypertension risk.

Accordingly, this study aims to clarify how SES, specifically education status, residence, marital status, and ethnicity, influence hypertension in Nepal through mediators like sequential behavioral (tobacco use, alcohol consumption, obesity, salt intake) and physiological pathways (stress). Specifically, we hypothesize that SES impacts hypertension risk via behavioral modifications, which in turn affect adiposity, ultimately influencing blood pressure. By integrating global theory, regional biological context, and national epidemiological trends, this study provides a mechanistic understanding of the hypertension epidemic in Nepal and contributes evidence applicable to other LMIC settings undergoing similar transitions.

Methods

Data source and study design

We used data from the Nepal WHO STEPS Survey 2019, a nationally representative cross-sectional survey designed using a complex sampling design. The representative national sample was selected through multistage cluster sampling. The data was collected using the WHO NCD STEPS questionnaire version 3.2, along with country-specific questions and administered in the local Nepali Language. The details about the data collection and procedure are explained and can be accessed on the WHO website.[28] Datas were downloaded from the official website after the approval as we used the secondary deidentified data no IRB approval was required.

Data cleaning and variable selection:

We selected sociodemographic, behavioral, and psychosocial variables from the raw dataset, focusing on anthropometry, blood pressure measurements, and medication use. The analysis excluded pregnant respondents to avoid pregnancy-related changes and participants with missing hypertension outcomes or missing survey weights. This resulted in a final sample size of 5 281 respondents with valid hypertension status. The primary outcome was defined as binary clinical hypertension, identified through either measured blood pressure thresholds or reported antihypertensive medication use, as per the American Heart Association guidelines.[29]

Work and home stress were classified, along with finance and life events stress items. A cumulative stress score, ranging from 0 to 4, was created by summing these four binary indicators. High-salt behavior was assessed via frequency of salt-added and processed foods, with "Don't know" responses recorded as "never." Tobacco use was coded as 1 if either smoking or smokeless tobacco was reported, and current alcohol use was noted if participants consumed alcohol in the last 30 days. Similarly, BMI was calculated as weight (kg)/ height (m^2). Obesity was categorized using Asian guidelines: 0 for BMI < 23 normal/underweight, 1 for 23 ≤ BMI < 25 overweight, and 2 for BMI ≥ 25 obese.[30]. Moreover, education was recorded for special missing codes and standardized. Age was standardized, sex was denoted as a binary indicator, and residence was classified as urban or rural according to the stratum information, with metro/sub-metro and municipality being coded as urban, and the rural municipality coded as rural. Marital status was grouped into three categories: never married, married/cohabiting, and widowed/divorced/separated. Ethnicity was divided into three groups, with Brahmin/Chhetri as the reference. Before SEM estimation, multicollinearity among key covariates was assessed using variance inflation factors (VIF) through a linear model predicting hypertension, and all continuous variables were standardized using z-score transformations.

Statistical Analysis

Statistical analysis was done using R statistical software version 4.4.2. [31]. Descriptive statistics are presented as frequencies and percentages for categorical variables and means with standard deviations for parametric numeric variables. A three-stage sequential mediation structural equation model (SEM) was conducted to examine pathways linking socioeconomic determinants to hypertension. In the first stage, socioeconomic drivers were modeled as predictors of the behavior-related mediators (high-salt behavior, stress, tobacco, alcohol), adjusting for sociodemographic covariates. In the second stage, obesity was regressed on both mediators and socioeconomic drivers. In the final stage, hypertension was regressed on obesity, selected mediators, and socioeconomic drivers. We allowed residual covariances among specific mediator disturbances and defined mediation parameters to quantify specific indirect, direct, and total effects for each socioeconomic determinant on hypertension. The SEM was estimated using a weighted Least Squares Mean and variance Adjusted (WLSMV) estimator, appropriate for binary and ordinal outcomes and predictors, using the lavaan package,[32], incorporating sampling weights and cluster-robust adjustments at the primary sampling unit level using the lavaan.survey[33] package to account for the complex survey design. Path coefficients were converted to odds ratios using the Amemiya approximation[34] (probit × 1.7) for interpretability. Statistical significance was set at $p< 0.05$. We first estimated direct-effect models for each outcome, including the hypertension and obesity models and the behavioral models (stress, salt behaviour, current tobacco use, and current alcohol use) presented in Table 2. We then evaluated mediation pathways by estimating total indirect effects, total effects

(direct + indirect), and the specific indirect effects through obesity to assess how sociodemographic determinants influence hypertension through behavioural factors and adiposity these results are presented in Table 3.The final structural equation model is presented in the SEM path diagram in figure 1.

Results

Table 1 presents the socio-demographic characteristics of our sample participants (n = 5,281; weighted n = 16,197,495). The distribution of participation of study participants across provinces was even, with Koshi (14.3%), Madhesh (14.5%), Bagmati (13.5%), Gandaki (14.4%), Lumbini (14.3%), Karnali (14.3%), and Sudurpashchim (14.7%). Females 63.9% shared a larger representation compared to males 36.1%, though the weighted distribution was more balanced by gender. The majority of the population was residing in rural areas (87.5%), compared to urban residents (12.5%), whereas the weighted estimates indicated a predominantly urban population (62.5%). 37.9% of participants belonged to the brahmin/Chhetri group, 33.1% to Janajati, and 29.0% were identified as a marginalized ethnic group. Most of the respondents were married 85.0%, while 9.6% had never married and 5.5% were previously married. Hypertension was prevalent in 64.4% of individuals, and 36.0% in the weighted population, whereas 35.6% reported no hypertension.

Participants showed nearly equal distribution in salt consumption behavior, with 50.6% reporting low or normal salt intake and 49.4% reporting high salt consumption. Most respondents did not currently use tobacco (70.2%) or alcohol (79.3%), while 29.8% and 20.7% reported current use of tobacco and alcohol, respectively. In terms of BMI status, 54.4% were normal or underweight, 18.6% overweight, and 27.0% obese. The mean age of participants was 40.2 ± 14.1 years, with a weighted mean age of 35.2 years (SE=0.34), and their mean education level was 4.9 ± 4.9 years. The average stress score was 1.8 ± 1.3, while mean systolic and diastolic blood pressures were 125.7 ± 17.8 mmHg and 82.9 ± 11.0 mmHg, respectively. The mean BMI was 23.1 ± 4.4 kg/m², and the weighted mean BMI was 22.7 kg/m².

Table 2: Direct effects SEM table

Table 2 summarizes the structural equation model for hypertension (HTN), obesity, and behavioral factors (salt consumption behavior, tobacco use, and alcohol use). The SEM demonstrated an acceptable overall fit (CFI = 0.963; RMSEA = 0.051, 90% CI [0.036, 0.068]; SRMR = 0.023; WRMR = 0.638), although the TLI was slightly lower (0.722), which can occur in complex WLSMV models with multiple categorical outcomes, but overall, it meets the recommended thresholds for WLSMV estimation.

Hypertension model

In the hypertension model, age was a strong predictor of higher odds of hypertension (OR = 1.18, 95% CI: 1.127–1.227, p < 0.001), followed by the male gender, which also increased hypertension odds (OR = 1.12, 1.015–1.233, p = 0.02). Obesity remained an important proximal determinant of hypertension (OR = 1.16, 1.109–1.211, p < 0.001). Among behavioral covariates, the stress scale showed a statistically significant direct association with hypertension (OR = 1.05, 1.009–1.082, p = 0.01), whereas high-salt behaviour did not reach significance at our setted alpha (OR = 0.94, 0.871–1.007, p = 0.08), and tobacco use was not significantly associated (OR = 1.08, 0.977–1.198, p = 0.13). Alcohol use was borderline (OR = 1.08, 0.998–1.175, p = 0.06). Education showed no direct association with hypertension (OR = 1.00, 0.951–1.044, p = 0.89). Urban residence was not statistically significant at p < 0.05 (OR = 0.93, 0.851–1.015, p = 0.10) as well. Overall, our survey weighted analysis emphasizes that hypertension is most strongly linked to age, obesity, and stress, with smaller or borderline contributions from alcohol and high-salt consumption behavior.

Obesity model

In our obesity model, A 1 SD increase in educational attainment was associated with 13 percentage higher odds of being in a higher obesity category (OR = 1.13, 95% CI: 1.034–1.226, p 0.01), and a 1 SD increase in age was associated with 17% higher odds of higher obesity category (OR = 1.17, 95% CI: 1.093–1.255, p < 0.001). Urban residence predicted substantially higher odds of obesity (OR = 1.44, 1.188–1.746, p < 0.001), consistent with nutrition transition processes in urban settings. Marital status was strongly related to obesity: married/cohabiting individuals had higher obesity odds than never married (OR = 1.78, 1.476–2.140, p < 0.001), while wid/div/sep did not differ significantly (OR = 1.03, 0.752–1.401, p = 0.87). Janajati ethnicity was associated with higher odds of obesity (OR = 1.24, 1.023–1.511, p = 0.03), whereas marginalized ethnicity was not (OR = 0.87, 0.692–1.083, p = 0.21). Behaviorally, tobacco use was associated with lower odds of obesity (OR = 0.82, 0.703–0.946, p = 0.01), while alcohol use was borderline (OR = 1.15, 0.980–1.347, p = 0.09). Neither high-salt behaviour nor stress scale was directly associated with obesity in this weighted model (p = 0.50 and 0.27, respectively).

Behavioral Model

As the stress is modeled as an ordinal outcome in this weighted SEM, the stress model ORs reflect shifts toward higher stress categories. Urban residence was associated with lower odds of higher stress (OR = 0.58, 0.386–0.864, p < 0.001), while marital status was the dominant predictor compared with never married, married/cohabiting individuals had markedly higher odds of higher stress (OR = 2.46, 1.836–3.284, p < 0.001), and wid/div/sep had even higher odds (OR = 4.39, 2.362–8.141, p < 0.001). Education, age, sex, and ethnicity were not statistically significant (p > 0.05). Suggesting that marital disruption/transition and partnership status are more strongly linked to stress than education and ethnicity.

In the salt behaviour model, age was strongly protective (OR = 0.91, 0.864–0.952, p < 0.001), indicating lower odds of high-salt behaviour with increasing age (per 1 SD). Education, sex, residence, marital status, and ethnicity were not significant. In the current tobacco use model, education reduced tobacco use (OR = 0.92, 0.889–0.962, p < 0.001), while age increased tobacco use (OR = 1.07, 1.024–1.113, p < 0.001), and male gender strongly increased tobacco use (OR = 1.96, 1.807–2.129, p < 0.001). Marital status predicted higher tobacco use (married/cohabiting: OR = 1.29, 1.153–1.444, p < 0.001; wid/div/sep: OR = 1.61, 1.298–2.004, p < 0.001), while urban residence was borderline protective (OR = 0.92, 0.830–1.012, p = 0.08). In the current alcohol use model, education was protective (OR = 0.96, 0.917–0.996, p = 0.03), male gender increased odds (OR = 1.61, 1.503–1.721, p < 0.001), and marital status again increased alcohol use (married/cohabit: OR = 1.27, 1.152–1.393, p < 0.001; wid/div/sep: OR = 1.45, 1.183–1.780, p < 0.001). Ethnic differences were most apparent for alcohol

use, with Janajati ethnicity showing higher odds (OR = 1.38, 1.239–1.537, p < 0.001), while marginalized ethnicity was not significantly associated with alcohol use.Table 3 summarizes survey-weighted indirect, total, and obesity-mediated effects of key sociodemographic determinants on hypertension. Obesity-mediated pathways were statistically significant for education (OR = 1.01, p = 0.02), urban residence (OR = 1.03, p < 0.001), married/cohabiting status (OR = 1.05, p < 0.001), and Janajati ethnicity (OR = 1.02, p = 0.03), indicating that obesity acts as an important sequential mediator for several upstream determinants. Total indirect effects were significant for married/cohabiting (OR = 1.10, p < 0.001), wid/div/sep (OR = 1.09, p < 0.001), and Janajati (OR = 1.04, p = 0.01), but total effects were non-significant across determinants, suggesting offsetting direct and indirect pathways in the weighted SEM. Notably, wid/div/sep showed a significant total indirect effect despite a non-significant obesity-mediated component, implying mediation through other behavioural/psychosocial pathways rather than obesity alone. Marginalized ethnicity showed no significant indirect or obesity-mediated effects and only a borderline protective total effect (OR = 0.91, p = 0.10).

Figure 1 summarizes the survey-adjusted sequential mediation SEM for hypertension, through behavioral mediators and obesity. The diagram is structured into four layers (sociodemographic → behavioural mediators → obesity → hypertension), with solid-colored paths indicating statistically significant direct effects (p < 0.05) and dashed/light grey paths indicating non-significant associations. The diagram supports the hypothesized pathway in which sociodemographic determinants shape behavioral risk factors (high-salt behavior, tobacco use, alcohol use, and stress), which in turn influence obesity and ultimately hypertension. The final hypertension equation, the most prominent proximal pathway is obesity → hypertension (OR = 1.16, 95% CI: 1.109–1.211, p < 0.001), reinforcing obesity as the key physiological bridge in the model. Upstream determinants also show direct contributions to hypertension. Age is strongly associated with higher odds of hypertension (OR = 1.18, 1.127–1.227, p < 0.001), and male gender increases hypertension odds (OR = 1.12, 1.015–1.233, p = 0.02). Beyond this direct route, the figure highlights how upstream factors shape obesity and behavioural risks. Obesity is socially patterned, with higher odds among urban residents (OR = 1.44, 1.188–1.746, p < 0.001), those married/cohabiting (OR = 1.78, 1.476–2.140, p < 0.001), and among Janajati (OR = 1.24, 1.023–1.511, p = 0.03), and it also rises with education (OR = 1.13, 1.034–1.226, p = 0.01) and age (OR = 1.17, 1.093–1.255, p < 0.001). Behavioural mediators show strong demographic gradients that feed into the downstream pathway: age is associated with lower odds of high-salt behaviour (OR = 0.91, 0.864–0.952, p < 0.001), while education is protective for substance use (tobacco OR = 0.92, 0.889–0.962, p < 0.001; alcohol OR = 0.96, 0.917–0.996, p = 0.03). In contrast, male gender strongly elevates tobacco and alcohol use (tobacco OR = 1.96, 1.807–2.129, p < 0.001; alcohol OR = 1.61, 1.503–1.721, p < 0.001), and marital status increases both tobacco and alcohol use (e.g., married/cohabiting: tobacco OR = 1.29, alcohol OR = 1.27, both p < 0.001; wid/div/sep: tobacco OR = 1.61, alcohol OR = 1.45, both p < 0.001). Stress stands out as both an upstream-patterned mediator and a direct contributor to hypertension: stress is substantially higher in married/cohabiting (OR = 2.46, 1.836–3.284, p < 0.001) and wid/div/sep (OR = 4.39, 2.362–8.141, p < 0.001) compared with never married, while urban residence is associated with lower stress (OR = 0.58, 0.386–0.864, p < 0.001). Consistent with this, the stress scale → hypertension path is statistically significant (OR = 1.05, 1.009–1.082, p = 0.01). Finally, several hypothesized behavioural→hypertension links are shown as weak or non-significant after survey adjustment—high-salt behaviour → hypertension is borderline (OR = 0.94, p = 0.08) and alcohol use → hypertension is borderline (OR = 1.08, p = 0.06) suggesting that in the weighted SEM, the clearest hypertension mechanism operates through obesity (and to a lesser extent stress) rather than through strong direct effects of individual behaviors alone.

Discussion

The SEM results support a weight of progress pathway and provide evidence that Nepal's hypertension burden is shaped by a multi-step pathway consistent with the nutrition transition framework sociodemographic determinants structure behavioural risks (salt behaviour, tobacco, alcohol, stress), these contribute to obesity, and obesity as a proximal driver of hypertension supporting our central theory of the weight of progress by identifying adiposity as a mechanism linking upstream determinants to the hypertension.

The obesity category was a strong predictor of hypertension, and stress score was independently associated with hypertension, net of sociodemographic controls. This pattern is consistent with global evidence that excess adiposity is a key modifiable driver of elevated blood pressure and cardiometabolic risk[6,35] as well as consistent with the findings from Nepal.[15] Upstream predictors in the model appear to shape hypertension risk primarily by structuring behavioral exposures that feed into obesity, rather than acting only as direct causes but at the same time several sociodemographic factors i.e age, urban residence, gender came as a significant contributor as well which was consistent with the findings highlighted by Ofori et al in ghana[36] and Gupta et al in Nepal.[11]Furthermore, the weighted direct effects show that several upstream factors contribute to hypertension risk directly and indirectly. Age and male gender retain significant direct associations with hypertension, consistent with established epidemiology that hypertension risk increases with age and is influenced by biological and behavioral factors.[2,12,15] More importantly for our conceptual model, obesity is itself socially patterned in the weighted results; obesity is higher among urban residents and among those who are married/cohabiting, and it increases with education and age. These patterns closely match Nepal's documented shift toward urban lifestyles, changing diets, and rising overweight/obesity concentrated in socioeconomically advantaged and urban groups, hallmarks of the Nutrition Transition. [7,15,37]. Together, the results strengthen the argument that SES and social change should be treated as distal determinants that operate through intermediate behavioral and physiological pathways rather than only as proximal predictors.[17,38] The behavioral mediators gave a bit of mixed stratification, education is protective for tobacco and alcohol use, being male is associated with increased use of both tobacco and alcohol, and marital status is associated with higher substance use. These strong behavioral gradients mirror the behavioral risk factors emphasized by WHO for hypertension prevention[39] similarly for the obesity mediated, even though the effect size was lesser extreme for few of the predictors i.e alcohol and high salt consumers at a 95% CI, it might be suggesting that they might be operating through the obesity mediated pathway which has been so called determinants in the studies conducted previously for the hypertension. [40,41]. Interestingly, a notable finding is the prominent role of stress. In our result we found stress is strongly patterned by marital status (married/cohabiting and wid/div/sep vs never married show markedly higher odds of higher stress categories), and stress also shows a statistically significant direct association with hypertension. This supports a dual role for stress as a mediator linking social circumstances to physiological outcomes,[42] and an independent pathway to hypertension consistent with WHO's broader framing of behavioural and

environmental determinants.[39] The pattern of results in our study also reinforces this for wid/div/sep, the total indirect effect is significant while the obesity-mediated component is not, implying that non-obesity mediators (plausibly stress-related routes) account for much of the mediated association with hypertension for this group. In practical terms, hypertension prevention in Nepal may require not only diet/weight interventions as suggested by previous studies [15,4 3]but also attention to psychosocial stressors, particularly those linked to marital disruption and social vulnerability. In our indirect vs total effects, several determinants (married, wid/div/sep, Janajati) have significant total indirect effects but non-significant total effects. This is a classic SEM finding indicating offsetting pathways, for example, a determinant may increase hypertension indirectly through obesity or stress, while its direct path (or other indirect routes) counterbalances the net effect. SEM is particularly useful here because it separates the "how" (indirect pathways) from the "overall" (total effect), allowing a more mechanistic interpretation than single-equation regression. From a policy perspective, this matters: even if a determinant's total effect is not significant, identifying significant mediated pathways still highlights intervention targets (e.g., obesity prevention among urban residents; stress mitigation among wid/div/sep). Our findings from this study suggest that hypertension prevention in Nepal should emphasize interventions that disrupt the obesity pathway while also addressing behavioural and psychosocial determinants. At the health-system level, scaling diagnosis and treatment through primary care using WHO's HEARTS approach (standardized protocols, team-based care, monitoring systems) complements upstream prevention by improving control among those already hypertensive.[44]

Conclusion

This study provides survey-weighted SEM evidence that Nepal's hypertension epidemic reflects a sequential nutrition-transition pathway in which upstream sociodemographic determinants shape behavioural risks and obesity, and obesity acts as a key proximal driver of hypertension. The strongest and most consistent relationship in the model is the obesity → hypertension pathway, indicating that adiposity is a central mechanism through which social and behavioural change translates into hypertension risk. Behavioural mediators are strongly socially patterned, especially tobacco and alcohol by gender and education, and stress by marital status. Stress also shows an independent association with hypertension, highlighting the importance of psychosocial pathways alongside metabolic ones. Indirect effects are frequently significant even when total effects are not, underscoring the value of SEM for identifying mechanisms and intervention targets rather than relying solely on overall associations. Given Nepal's rapid rise in overweight/obesity and the disproportionate burden of hypertension in LMIC settings, prevention strategies should prioritize obesity-centered interventions and upstream behavioural policies, while simultaneously strengthening primary-care hypertension control using established WHO frameworks.

Study Limitations

This study has several key limitations. First, the analysis is based on cross-sectional STEPS survey data, so the sequential mediation pathways represent associational decomposition rather than confirmed causal mechanisms. Second, mediation results rely on strong assumptions, no unmeasured confounding of determinant–mediator and mediator–outcome relationships, and some relevant pathways such as detailed diet quality, physical activity, healthcare access, medication use, and early-life nutrition may not be fully captured, potentially biasing direct and indirect estimates. Third, behavioural mediators (salt behaviour, tobacco, alcohol, and stress categories) may be affected by measurement error and reporting bias, which can attenuate effects. Finally, because the STEPS data reflect the survey period Nepal STEPS 2019 among adults aged 15–69 years using a multistage design, the estimated relationships may not fully generalize to Nepal's current, rapidly evolving nutrition transition environment.

Declarations

Competing Interests:

The authors declare no competing interests.

Author Contributions

Biraj Neupane: Conceptualization, Methodology, Formal Analysis, Data access and Curation, Visualization, Writing Original Draft and Review & Editing. **Niharika Jha**: Conceptualization, Methodology, Interpretation, Writing – Review & Editing. **Bikram Adhikari**: Methodology, Statistical Guidance, Writing – Review & Editing. **Silon Pant**: Data management, data access, software, coding, Writing – Review & Editing. **Jyoti Badu**: Literature Review, project oversight, Writing – Review & Editing. **Ian Brooks**: Supervision, Methodological Oversight, Interpretation Guidance, Conceptualization, Writing – Review & Editing. **Csaba Varga**: Supervision, Statistical Guidance, Critical Revision of Manuscript, Project Administration.

Data Availability Statement :

The data used in this study are publicly available from the WHO STEPS Survey 2019 repository which can be accessed here (https://extranet.who.int/ncdsmicrodata/index.php/catalog/771).

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Tables

Table:1 socio-demographic characteristics				
Category	**n=5281**	**Percentage**	**Weighted (n = 16197495)**	**Percentage**
Province				
Koshi	755	14.30	2,975,947	18.40
Madhesh	768	14.54	3,171,329	19.60
Bagmati	714	13.52	2,648,783	16.40
Gandaki	760	14.39	1,312,175	8.10
Lumbini	755	14.30	3,334,460	20.60
Karnali	754	14.28	894,873	5.50
Sudurpashchim	775	14.68	1,859,930	11.50
Gender				
Female	3375	63.91	8,476,079	52.30
Male	1906	36.09	7,721,416	47.70
Residence Type				
Rural	4620	87.48	6,076,389	37.50
Urban	661	12.52	10,121,107	62.50
Ethnic Group				
Advantaged Group	2000	37.87	5,407,245	33.40
Janajati	1749	33.12	5,372,883	33.20
Marginalized	1532	29.01	5,417,368	33.40
Marital Status				
Never Married	505	9.56	3,174,701	19.60
Married	4488	84.98	12,588,465	77.70
Previously Married	288	5.45	434,329	2.70
Hypertension Status				
No	1882	35.64	10,317,047	64.00
Yes	3399	64.36	5,880,449	36.00
Salt Consumption Behavior				
Low/Normal	2671	50.58	7,933,155	49.00
High Salt Behavior	2610	49.42	8,264,340	51.00
Current Tobacco Use				
No	3706	70.18	11,487,425	71.00
Yes	1575	29.82	4,710,071	29.00
Current Alcohol Use				
No	4188	79.30	12,847,776	79.30
Yes	1093	20.70	3,349,720	20.70
BMI Categorization				
Normal/Underweight	2874	54.42	9,397,084	58.00
Overweight	980	18.56	2,895,850	17.90
Obese	1427	27.02	3,904,562	24.10
	mean	SD	mean	SE
Age	40.20	14.10	35.20	0.34
Education	4.90	4.90	6.00	0.18
Stress Score	1.80	1.30	1.70	0.06

Systolic Blood Pressure	125.70	17.80	124.50	0.43
Diastolic Blood Pressure	82.90	11.00	81.60	0.33
BMI (kg/m2)	23.10	4.40	22.70	0.13

SD: standard deviation, SE: standard error, BMI: body mass index, n: frequency

Table 2. Direct effects SEM Table

Predictors	**Hypertension Model**		**Obesity Model**			**Stress Model**		**Salt Behaviour Model**			C
	OR	**95% CI**	**p-value**	**OR**	**95% CI**	**p-value**	**OR**	**95% CI**	**p-value**	**OR**	**95% CI**
Education	1.00	(0.951, 1.044)	0.89	1.13	(1.034, 1.226)	**0.01**	0.95	(0.814, 1.103)	0.49	1.01	(0.952, 1.0
Age	1.18	(1.127, 1.227)	**<0.001**	1.17	(1.093, 1.255)	**<0.001**	1.07	(0.943, 1.207)	0.30	0.91	(0.864, 0.9
Gender											
Female	**Ref**		**Ref**			**Ref**		**Ref**			R
Male	1.12	(1.015, 1.233)	**0.02**	1.03	(0.881, 1.198)	0.73	1.11	(0.921, 1.335)	0.27	0.97	(0.894, 1.0
Residency											
Rural Residency	**Ref**		**Ref**			**Ref**		**Ref**			R
Urban Residency	0.93	(0.851, 1.015)	0.10	1.44	(1.188, 1.746)	**<0.001**	0.58	(0.386, 0.864)	**<0.001**	1.01	(0.87, 1.16
Marital Status											
Never Married	**Ref**		**Ref**			**Ref**		**Ref**			R
Married/cohabit	0.95	(0.841, 1.076)	0.43	1.78	(1.476, 2.14)	**<0.001**	2.46	(1.836, 3.284)	**<0.001**	0.90	(0.786, 1.0
Wid/Div/Sep	0.91	(0.775, 1.063)	0.23	1.03	(0.752, 1.401)	0.87	4.39	(2.362, 8.141)	**<0.001**	0.83	(0.688, 1.0
Ethnicity											
Ethnicity Brahmin/chhetri	**Ref**		**Ref**			**Ref**		**Ref**			R
Ethnicity Janajati	0.96	(0.882, 1.047)	0.36	1.24	(1.023, 1.511)	**0.03**	1.25	(0.873, 1.784)	0.23	1.03	(0.905, 1.1
Ethnicity Marginalize	0.93	(0.835, 1.035)	0.18	0.87	(0.692, 1.083)	0.21	0.87	(0.551, 1.358)	0.53	1.11	(0.947, 1.2
Obesity	1.16	(1.109, 1.211)	**<0.001**								
High Salt Behaviour	0.94	(0.871, 1.007)	0.08	0.96	(0.844, 1.086)	0.50					
Stress Scale	1.05	(1.009, 1.082)	**0.01**	0.97	(0.922, 1.023)	0.27					
Tobacco Use											
No	**Ref**		**Ref**								
Yes	1.08	(0.977, 1.198)	0.13	0.82	(0.703, 0.946)	**0.01**					
Alcohol Use											
No	**Ref**		**Ref**								
Yes	1.08	(0.998, 1.175)	0.06	1.15	(0.98, 1.347)	0.09					

OR: odds ratio, CI: confidence interval, ref: reference group,

Table 3. Indirect, total, and obesity-mediated effects on hypertension.				
Determinant	**Effect Type**	**OR**	**95% CI**	**p-value**
Education				
	Total Indirect	1.00	(0.991, 1.015)	0.59
	Total Effect	1.00	(0.956, 1.046)	1.00
	Obesity Mediated	1.01	[1.002, 1.019]	0.02
Urban Residence				
	Total Indirect	1.01	(0.987, 1.043)	0.30
	Total Effect	0.94	(0.86, 1.034)	0.21
	Obesity Mediated	1.03	(1.014, 1.051)	**<0.001**
Married Vs Never Married				
	Total Indirect	1.10	(1.067, 1.140)	**<0.001**
	Total Effect	1.05	(0.922, 1.194)	0.47
	Obesity Mediated	1.05	(1.027, 1.076)	**<0.001**
Widow/Div/Sep Vs Never Married				
	Total Indirect	1.09	(1.031, 1.145)	**<0.001**
	Total Effect	0.99	(0.843, 1.153)	0.86
	Obesity Mediated	1.00	(0.976, 1.030)	0.87
Janajati Vs Upper Castes				
	Total Indirect	1.04	(1.012, 1.069)	**0.01**
	Total Effect	1.00	(0.917, 1.089)	0.99
	Obesity Mediated	1.02	(1.001, 1.037)	**0.03**
Marginalized Vs Upper Caste				
	Total Indirect	0.98	(0.953, 1.005)	0.11
	Total Effect	0.91	(0.813, 1.018)	0.10
	Obesity Mediated	0.99	(0.968, 1.008)	0.23

OR: odds ratio, CI: confidence interval, ref: reference group,

Figures

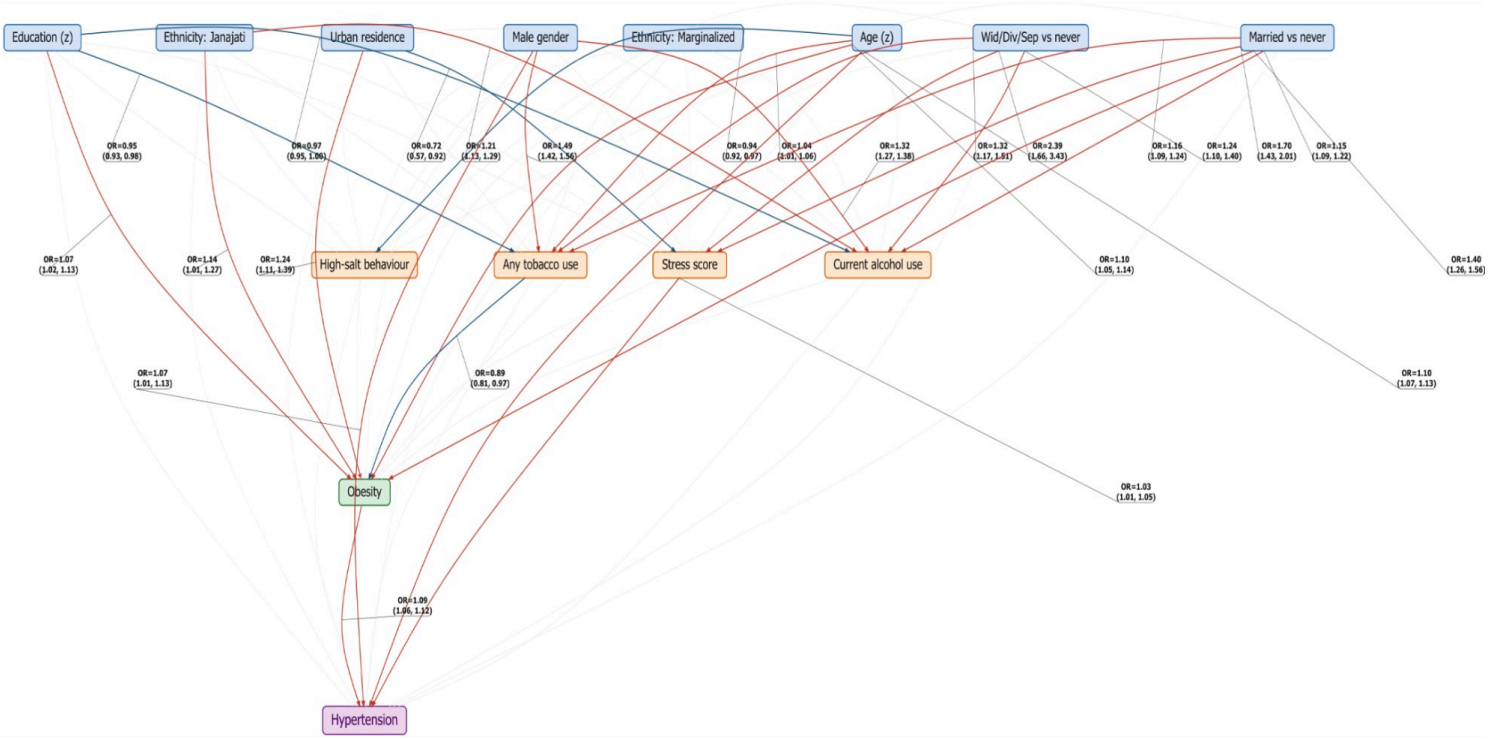


Figure 1

Legend not included with this version.

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