Body Weight and Hypertension Risk in a Developing Country

Dang, Thang

Department of Economics and Related Studies, University of York, School of Economics, University of Economics Ho Chi Minh City (UEH)

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Thang Dang[a,b,*]

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Abstract. This study provides a first causal inference of the link between body weight and the risk of hypertension among adults in a developing country, Vietnam. The study uses biological offspring's body weight as an instrument for exogenous changes in parents' body weight to address the potential problem of endogeneity and applies the instrumental variable approach to estimate the relationship of interest. The paper finds that on average an addition BMI unit causally increases the likelihood of being hypertensive by about 5.1–7.3% points for men and 5.6–8.2% points for women. The paper also shows that the impacts of body weight on the risk of hypertension are different with various age intervals. Furthermore, overweight or obesity causally enlarges the risk of hypertension compared to underweight or normal weight.

Keywords: Body weight; Hypertension; Causal effect; Vietnam

JEL Classifications: I1, I14, I18

[a] Department of Economics and Related Studies, University of York, United Kingdom

[b] School of Economics, University of Economics Ho Chi Minh City (UEH), Vietnam

[*] Corresponding author: Department of Economics and Related Studies, University of York, Heslington, York, YO10 5DD, UK. *E-mail address:* thang.dang@thangdang.org (Thang Dang).

1. Introduction

The existing literature indicates that there is a relationship between body weight and the risk of hypertension. In particular, body mass index a common measure of body weight is strongly and positively correlated with the risk of hypertension in many countries such as Argentina (Stray-Pedersen et al., 2009), Norway (Stray-Pedersen et al., 2009), the United States (Kumanyika, 1989; Shihab et al., 2012), China (Li et al., 2017), Iran (Poorolajal et al., 2016), Japan (Lee et al., 2004; Jiang et al., 2003; Nurdiantami et al., 2017), Indonesia (Tuan et al., 2009) and Vietnam (Tuan et al., 2009). For example, Li et al. (2017) find that compared to an individual with normal weight, individuals with overweight and obesity are more likely to be hypertensive by roughly double and triple than a normal individual in China. Moreover, the findings remain apparently even after controlling for highlighting characteristics of individuals, lifestyles and family backgrounds as well.

Yet, it is indispensable to raise a question whether the difference in the risk of hypertension due to body weight is causally reliable. The answer relies on what really drives the observed difference. To the extent, the difference probably comes from disparities in body weight or other unobservable determinants that are also related to hypertension risk. If this is a case, the estimate suggests that observed correlations may indicate unobserved differences and not a causal relationship between body weight and hypertension risk. A key limitation of previous studies is that they provide the estimates of the correlation between body weight and hypertension rather than the estimates of the causal impacts.

A fundamental challenge with identifying the causal effect of body weight on hypertension risk is that body weight is likely connected with many latent factors that may be related to hypertension risk such as dietary habits. Following Cawley and Meyerhoefer (2012) to address the endogeneity of body weight, this paper employs biological offspring's

body mass index as an instrument for the source of exogenous variation on observations' body weight and estimate the causal link between body weight and hypertension risk among adults using a nationally representative dataset from Vietnam.

High blood pressure has been increasingly recognized as the key factor of dangerous health problems such as heart attack and failure, stroke, kidney diseases, vision loss, sexual dysfunction, angina, peripheral artery disease and premature mortality, that apparently threaten human health and quality of life (Kearney et al., 2005; Mohammad et al., 2017). An estimated figure by Mills et al. (2016) using data from 90 countries indicates that the global prevalence of hypertension is roughly 1.39 billion people, equivalently 31% of total adults over the world at the time of 2010. Moreover, the hypertension rate tends to increase over time, for instance, 5.2% between 2000 and 2010. Regarding the consequence, hypertension annually accounts for an estimated 9.4 million of deaths around the world (Lim et al., 2012).

Therefore, the study of the causal impact of body weight on hypertension is very important not only for the insightful understanding of socio-economic factors of hypertension but also for the related interventions, for example, dietary strategy and nutrition to improve the situation. Additionally, this line of research is even more vital in the context of the growing prevalence of overweight and obesity worldwide (Gakidou, 2014). Such an increasing pattern of overweight and obesity have been identified as a global epidemic that is a major problem facing healthcare systems worldwide (World Health Organization, 2017). Furthermore, the burgeoning prevalence of overweight and obesity is regarded as one of the most hazardous factors linking with the widespread expansion of hypertensive population over the world (Seravallea and Grassi, 2017; Rahmouni et al., 2005).

The prevalence of hypertension is even larger in developing countries than developed counterparts. Roughly 1.04 billion out of the total 1.39 billion hypertensives comes from low- and medium-income countries (Mills et al., 2016). In spite of this fact, developing countries

are less efficient in addressing for an increasing prevalence of hypertension because of low awareness, moderate treatment and decreased controlling ability (Mills et al., 2016). This study hence significantly provides more evidence on the causal link between body weight and hypertension from developing countries.

This paper contributes to the literature of socio-economic factors of hypertension by providing a causal inference of the association between body weight and the risk of hypertension. The paper finds that on average an addition BMI unit causally increases the likelihood of being hypertensive by about 5.1–7.3% points for men and 5.6–8.2% points for women. The paper also indicates that the impacts of body weight on the risk of hypertension are different with various age intervals. Moreover, overweight or obesity substantially contributes the risk of hypertension in a comparison with under or normal weight.

The remainder of this paper is structured as follows. Section 2 presents the estimation methods while section 3 discusses data and the sample for the analysis. Next, section 4 shows the empirical results with main results and the results of robustness check while section 5 provides some further analysis including heterogeneity and the impact of overweight or obesity. Finally, section 6 discusses the results and makes some concluding remarks.

2. Estimation methods

The relationship between body weight and hypertension risk is typically estimated using the following equation:

$$Hypertension_i = \alpha_0 + \alpha_1 Bodyweight_i + \alpha_2 X_i + \varepsilon_i \quad (1)$$

where $Hypertension_i$ is the probability of hypertension for the individual i; $Bodyweight_i$ is the body weight that is measured by body mass index (BMI); X_i is a vector of control variables including age, age squared, dummies for married status, major ethnicity and health

insurance status, schooling years, and dummies for urban and eight geographic regions; and ε_i is an error term.

However, the OLS estimator using equation (1) merely produces the estimate of the association between body weight and hypertension risk rather than the reliable estimate of the causal effect. This is likely because there exist unobservable characteristics related to an individual that jointly determine body weight and hypertension risk. In other words, the endogeneity problem is a threat to identification for the estimation. To overcome the endogeneity problem, this paper uses biological offspring's body mass index-for-age z-scores (BAZ) as an instrument for exogenous changes in one's body weight and estimate the causal effect of interest using the following 2SLS procedure:

$$BMI_i = \beta_0 + \beta_1 OffspringBAZ_i + \beta_2 X_i + \epsilon_i \quad (2)$$

$$Hypertension_i = \gamma_0 + \gamma_1 \widehat{BMI}_i + \gamma_2 X_i + \zeta_i \quad (3)$$

where $OffspringBAZ_i$ which is BAZ of the biological child of the individual i is the instrumental variable for body weight. While the equation (2) indicates the first stage regression of body weight on the instrument, the equation (3) presents the regression of outcomes of interest on the predicted body weight for the individual i ($\widehat{BMI}_i$) that is calculated using the first stage. The coefficient γ_1 that is the parameter of interest captures the causal effect of body weight on hypertension risk. In this study, the estimated results using both non-IV and IV estimators are reported. For the non-IV estimator, the paper applies the Probit model for the non-IV estimator while the paper employs the IV-Probit model as the IV estimator.

Using a biological relative's body weight as the IV for the respondent's body weight is a recently used strategy to estimate the causal effect of body weight, overweight and obesity in particular, on health outcomes (Cawley and Meyerhoefer, 2012; Doherty et al., 2017; Smith

et al., 2009). In principle, the IV has to satisfy two following requirements. Firstly, the IV must be a powerful predictor of the explanatory variable. In this case, the body weight of a biological relative can be used to strongly predict the body weight of the respondent because evidence from human biology and medicine shows that genetic factors can originally explain the variation in human body weight (Barsh et al., 2000; Comuzzie and Allison, 1998). Furthermore, all values of the *F*-statistic in the first stage regressions in this study are over the traditional minimum standard value of 10.[1] Therefore, offspring's BAZ can be used as a powerful predictor of parents' body weight in this study (Stock et al., 2002).

Secondly, the requirement of validity indicates that the IV has to be uncorrelated with the residual term in the second stage regression. This study arguably assumes that a child's body weight is not correlated with parents' residual hypertension after controlling for projected body weight of parents and observed characteristics. The rationale for this assumption stems from consistent evidence on undetectable and ignorable impacts of a joint family environment on body weight[2] for individuals living in same households such as parents and biological children that have been revealed from the genetic and medical literature (Haberstick et al., 2010; Hewitt, 1997; Grilo and Pogue-Geile, 1991; Maes et al., 1997; Nelson et al., 2006; Wardle et al., 2008). This argument for the validity of employing a biological relative's body weight as the IV for the respondent's body weight to address the endogeneity of body weight has been used in recent studies (Biener et al., 2017; Cawley,

[1] The *F*-statistic values for the first stage regression are specifically presented in the section of results.

[2] One can argue that the body weight of both parents and biological children are jointly determined by a shared living environment which is also directly related to parents' probability of hypertension. If this is a case, the validity of the IV would be threatened.

2004; Cawley and Meyerhoefer, 2012; Doherty et al., 2017; Grossbard and Mukhopadhyay, 2017).[3]

3. Data

This paper uses data from the Vietnam National Health Survey (VNHS) of 2001-2002. The VNHS is a nationally representative survey on health that consists of roughly 158,000 individuals from 36,000 households across the country. The survey was sampled based on the Population Census of 1999. Anthropometric information was collected from the survey is used to construct body mass index (BMI) that is the adopted measure of body weight for adults. BMI (kg/m^2) is defined as $weight/height^2$ in which weight is measured in kilograms (kg) and height is measured in meters (m).

The paper takes information on blood pressure to construct the variable of hypertension risk. Blood pressure measures include systolic blood pressure (SBP) and diastolic blood pressure (DBP). Both SBP and DBP were measured three times using medical tools with the aim to minimize the measurement error. SBP and DBP, which are used in this study, are the average values. The risk of hypertension is defined as the probability of for an individual to be hypertensive that takes a value of 1 if average SBP≥140 or/and average DBP≥90, and 0 otherwise. Importantly, the previous literature of the causal impacts of body weight indicates that the estimates using data from self-reported weight and height are likely biased due to

[3] Cawley and Meyerhoefer (2012) further conduct a falsification test for the validity of the IV (biological children's body weight) by using step-children's body weight instead and show that step-children's body weight is not a significant predictor of the respondents' weight and conclude that using biological children's body weight is an acceptable strategy.

measurement error (Burkhauser and Cawley, 2008). Therefore information on anthropometrics and blood pressure independently measured using medical tools by enumerators rather than self-reported information from respondents in the VNHS significantly allows this paper to avoid the potential bias in the measurement of body weight and hypertension.

The paper also uses anthropometric information (weight and height) for children to construct the instrumental variable (IV). The IV is offspring's body mass index-for-age z-scores (BAZ). BAZ is calculated using the 2007 World Health Organization (WHO) growth reference for children aged between 5 and 19 (de Onis et al., 2007). Using BAZ allows us to address the problem of age-induced variation in BMI. Moreover, because BAZ for children aged under 5 and children aged 5–19 are calculated using different reference growth groups[4], this study only uses BAZ for children aged 5–19 as the IV for parents' body weight. The main reason why this study chooses the 5–9 group rather than the under 5 group is because the sample size is larger with parents having biological children aged 5-9 than with parents only having biological children aged under 5.

The final samples for this study's analysis include (i) 24,678 pairs of father-offspring, and (ii) 26,112 pairs of mother-offspring. When using father-offspring or mother-offspring samples, the paper uses biological children's BAZ as an instrumental variable in general. Moreover, the paper also estimates the sub-samples with pairs of father-son (12,698 observations), father-daughter (11,980 observations), mother-son (13,437 observations) and mother-daughter (12,675 observations). Table 1 presents summary statistics of the variables for these samples. The rates of hypertension from all samples are around 16.7% for men

[4] BAZ for children aged under 5 is calculated using the 2006 WHO growth standards for preschool children (World Health Organization, 2006)

while the corresponding figures are nearby 7.8% for women's samples. The mean ages from all samples for men and women are roughly 40 and 38 years old.

4. Empirical results

Main results

Table 2 and 3 respectively present the main marginal coefficients of the causal impact of BMI on the likelihood of hypertension for men and women. The paper uses both the Probit and IV-Probit models and three samples (the full sample with parent-offspring pairs, one sub-sample with parent-son pairs, and one sub-sample with parent-daughter pairs) to produce the estimates of interest. While the Probit model only estimates the full sample, the IV-Probit model estimates the full sample and two sub-samples. Moreover, the set of control variables for the baseline specification includes age, age squared, dummy for married, dummy for the ethnic majority, dummy for health insurance, schooling years, dummy for urban areas, and dummies for eight geographic regions in Vietnam.

Table 2 shows the marginal effects of men's body weight on the risk of hypertension. When the Probit model is used for the estimation using the full sample, the paper finds that BMI causally increases the probability of hypertension by about 1.8% points for an additional unit of BMI (kg/m^2) as shown in column 1. The coefficient is statistically significant at 1%.

When the IV-Probit model is applied, the paper also finds the positive impacts of men's BMI on the likelihood of hypertension. However, the impacts are only statistically significant with the full sample (column 2) and the sub-sample of father-daughter pairs at 1% whereas the coefficient estimated using the sub-sample of father-son pairs loses its statistical significance at any conventional level. In particular, a man having one more BMI is more likely to be hypertensive by 5.1% points than the counterpart man if the paper uses both sons'

and daughters' BAZ as the IV (column 2). The marginal impact is even statistically larger when the paper estimates the sub-sample that only daughter's BAZ is used for the IV with 7.3% points (column 4). These results indicate that the estimates estimated using the Probit estimator are likely downward biased compared to those estimated by the IV-Probit estimator. The values of the *F*-statistic for the first stage regressions in Table 2 range between 619 and 1246.

The paper also finds the positive impacts of body weight on the possibility of hypertension among Vietnamese women. The marginal coefficients of the effect are presented in Table 3. By estimating the Probit model and the full sample, the paper shows that an extra BMI unit increases the probability of hypertension by approximately 1% points for a Vietnamese female (column 1). This estimate is statistically significant at 1%.

Meanwhile, the estimates using the IV-Probit model indicate that an additional BMI unit is causally linked to increases in the rate of hypertension for a Vietnamese woman by about 5.6% points using the full sample (column 2) and 8.2% points using the sub-sample of mother-son pairs (column 3) with a 1% level of statistical significance. Using IV-Probit to estimate the sub-sample of mother-daughter pairs produce a positive impact of women's BMI on the likelihood of hypertension but the estimate is statistically insignificant at any conventional level (column 4). The values of the *F*-statistic for the first stage regressions in Table 3 range between 717 and 1448.

Robustness checks

This paper implements some robustness checks for the main results by using various sets of control variables for the econometric specification. In particular, there are three different sets of control variable: (i) a set that removes age and age squared from the baseline set

(specification 1), (ii) a set that removes dummy for urban areas, and dummies for eight geographic regions from the baseline set (specification 2), and (iii) a set that removes all control variables from the baseline set (specification 3). The results of robustness checks are specifically presented in Tables 4 and 5 corresponding for men and women. Generally, the paper finds that the main estimates using the baseline specification are highly robust to other various specifications in terms of both the signs of the effect and the magnitude.

Table 4 demonstrates the robustness checks for men. Column 1 shows that the marginal estimates using the Probit estimator are between 1.7–1.8% points for three various specifications. The coefficients are statistically significant at 1%. This result is same as the main marginal estimate using the same estimator. For the estimates using the IV estimator, the paper finds the most similar patterns as the main estimator. In particular, when both sons' and daughters' BAZ are used as the IV for men's body weight, the estimates in column 2 indicate that one more BMI unit increases the probability of hypertension by 5.5% points, 5.9% points and 7.0% points corresponding to the use of specifications 1, 2 and 3. The marginal coefficient using the baseline specification is 5.1% points (column 2 of Table 2).

The IV-Probit estimators with sons' BAZ as the IV produces the marginal coefficients with 3.0% points (specification 1), 3.9% points (specification 2) and 4.8% points (specification 3) as demonstrated in column 3 Table 4. While the coefficient using specification 1 loses its statistical significance as the main estimate using the baseline specification (column 3 of Table 2), the coefficients using specifications 2 and 3 are statistically significant at 10% and 5% respectively.

Finally, the estimates in column 4 of Table 4 for the impact of men's body weight on the likelihood of hypertension using daughters' BZA as the IV are all statistically significant at 1% for three specifications. In terms of the magnitude, a male with one more BMI unit tends to have a higher probability of hypertension by about 8.4% points (specification 1), 8.0%

points (specification 2) and 9.6% points (specification 3). These results are slightly larger than the main effect with 7.3% points as indicated in column 4 of Table 2. The values of the *F*-statistic for the first stage regressions in Table 4 range between 609 and 1429.

Table 5 presents the robustness estimates for women. The Probit estimates in column 1 indicate that one additional BMI unit is associated with an increase in the probability of hypertension by about 1.1% points (specification 1), 0.9% points (specification 2) and 1% points (specification 3). These estimated coefficients are statistically significant at 1%.

When the IV-Probit estimator is used, the marginal effects are roughly robust to the main estimates. The estimates in column 2 show that with both sons' and daughters' BAZ as the IV the paper finds that a female with one more BMI unit is more likely to have a higher likelihood of hypertension by approximately 5.8% points (specification 1), 5.1% points (specification 2) and 5.2% points (specification 3) with a 1% level of statistical significance. Meanwhile, the marginal effects of one more BMI unit on the likelihood of hypertension using only sons' BAZ as the IV for women's body weight are 7.5% points, 7.3% points and 6.3% points corresponding to the use of specifications 1, 2 and 3 as demonstrated in column 3. The estimated coefficients in column 3 are all statistically significant at 1%.

Column 3 of Table 5 presents the estimated coefficients for the impact of interest when only daughters' BAZ is used to instrument for female's BMI. The paper finds statistically insignificant results for specifications 1 and 2, except for specification 3 that produces a marginal impact of 3.9% points with a 1% level of statistical significance. The values of the *F*-statistic for the first stage regressions in Table 5 range between 712 and 1672.

5. Further analysis

Heterogeneity

The effect of body weight on the risk of hypertension may vary with various age intervals. This section presents the marginal effects of body weight on the probability of hypertension by age groups. The marginal coefficients for four age groups including (i) age≤30, (ii) 30<age≤40, (iii) 40<age≤50, and (iv) age>50 are presented in Tables 6 and 7 for male and female respectively.

Table 6 shows the results for men. The results indicate that if Probit model is used, the association between BMI and the likelihood of hypertension does not vary considerably among different groups (column 1). In particular, the marginal effects are between 1.7% points and 1.9% points. However, the impacts are different among various age groups using the IV approach. Specifically, when sons' and daughters' BAZ are used as the IV, the marginal effects are about 22.3% points for age≤30, 4.6% points for 30<age≤40, and 11.4% points for age>50. These estimates are statistically significant at 5% or 10% (column 2). The coefficient for 40<age≤50 loses its statistical significance at any conventional level.

Meanwhile, all coefficients estimated using only sons' BAZ as the IV (column 3) are statistically insignificant at any conventional level for any age group as the main estimates. Finally, the estimates estimated using only daughters' BAZ as the IV (column 4) show that the marginal effects for the groups age≤30 and age>50 are 26.9% points with a statistical significance at 5% and 17.8% points with a statistical significance at 1%, respectively. The coefficients for the groups 30<age≤40 and 40<age≤50 are statistically insignificant at any conventional level. These findings indicate that the causal impact of body weight on the risk of hypertension is largest among youngest and oldest males while the middle group (males aged between 31 and 40) has the smallest effect. The values of the *F*-statistic for the first stage regressions in Table 6 range between 36 and 637.

Table 7 gives the coefficients of the marginal effect for women's various age groups. The marginal effects in the case of using Probit estimator consist of 0.3% points for age≤30,

0.7% points for 30<age≤40, 1.6% points for 40<age≤50, and 2.2% points for age>50 (column 1). All coefficients in column 1 are statistically significant at 1%. When the IV estimator is used to produce estimates of interest, the results are changing with different age groups. The estimated coefficients using sons' and daughters' BAZ as the IV in column 2 indicate that the effect is only statistically significant for the groups 30<age≤40 (at 10%) and 40<age≤50 (significant at 1%) with the marginal effects of 5.1% points and 7.8% points respectively.

For the case that the IV is only sons' BAZ in column 3, the coefficients for the groups 30<age≤40 and 40<age≤50 are both statistically significant at 5% with the marginal effects of both 8.9% points whereas the coefficients for other groups lose its statistical significance. Finally, the coefficients estimated when daughters' BAZ is used as the IV in column 4 are all statistically insignificant at any conventional level for most age groups, except for the group 40<age≤50 with the marginal effect of 6.5% points (significant at 5%). The values of the *F*-statistic for the first stage regressions in Table 7 range between 12 and 938.

The impact of overweight or obesity on hypertension

Overweight or obesity can also have a distinct impact on hypertension compared to healthy weight or underweight because the different statuses of BMI are likely to link with various health risks (Stommel and Schoenborn, 2010). This section provides the estimated results of the impact of overweight or obesity on the probability of hypertension. This paper defines a person is overweighted or obese if his or her BMI is equal to or over 25. The dummy variable takes a value of 1 if overweight or obesity, and 0 otherwise. The estimated coefficients demonstrate the probability of hypertension for an overweighted or obese person relative to that of an underweighted or healthy weighted person.

Generally, the paper finds that overweighted or obese adults have considerable positive impacts on the risk of hypertension relative to those with under or healthy weight for both male and female as shown in Tables 8 and 9. The signs and statistical significance of the impact are totally consistent with the impact of BMI.

Table 8 presents the results for men. The marginal effect estimated using the Probit model is 14.3% points with a 1% level of statistical significance (column 1). The marginal effects using the IV in columns 2 and 4 respectively are 116.2% points and 167.5% points. The estimated coefficients are statistically significant at 1%. Meanwhile, the estimated coefficient in column 3 is statistically insignificant at any conventional level. The values of the *F*-statistic for the first stage regressions in Table 8 range between 97 and 202.

Table 9 shows the estimated coefficients for female. The marginal effect estimated using the Probit estimator is 8.1% points with a 1% level of statistical significance (column 1). Meanwhile, the marginal effects estimated using the IV-Probit estimators in columns 2 and 3 are respectively 120.6% points and 187.1% points. These estimated coefficients are statistically significant at 1%. Meanwhile, the estimated coefficient in column 4 is statistically insignificant at any conventional level. These findings explicitly indicate that overweight or obesity is an extremely severe source of the prevalence of hypertension among Vietnamese adults. The values of the *F*-statistic for the first stage regressions in Table 9 range between 111 and 256.

6. Discussion and conclusion

While pervasive hypertension has increasingly become a major public health worldwide especially from developing countries (Kearney et al., 2005), the understanding of the contribution of body weight to the prevalence of hypertension is more important from the

existing literature of the determinants of hypertension. This study investigates the causal relationship between body weight and the risk of hypertension using a nationally representative dataset from a developing country in Asia, Vietnam.

Over last few decades, Vietnam has witnessed a rising rate of hypertensives (Ministry of Health of Vietnam, 2016). Although the prevalence of hypertension is relatively high among Vietnamese adults and its consequences in terms of hypertension-induced mortality and illness are severe, the awareness, the treatment and the control of this non-communicable disease are comparatively low in this developing country (Do et al., 2015; Son et al., 2012). Therefore, identifying the causal relationship between key associated factors and hypertension is generally very crucial for addressing the hypertension prevalence in Vietnam and other developing countries as well.

This study significantly contributes to the literature of the association between body weight and the risk of hypertension in some ways. Firstly, this study is one of the first studies devoted to estimating the causal effect of body weight on the risk of hypertension. The previous studies identify that body weight is positively linked to the prevalence of hypertension among other key factors. For example, Tuan et al. (2009) using the VNHS like this paper find that one increased BMI unit is correlated with higher probabilities of hypertension by roughly 14% and 16% for Vietnamese men and women respectively. Also, Do et al. (2015) uses a nationally representative sample from the 2005 Vietnam National Overweight Survey to show that overweight men and women are more likely to be hypertensive by about 43% and 29% respectively than those with normal weight or underweight. However, these results from previous studies naively provide the correlated estimates rather than the true causal estimates of the association between body weight and the risk of hypertension because the obtained estimates are produced while there are the co-existent impacts of other observed fundamental factors such as low birth weight (Ediriweera

et al., 2017), height (Sohn 2017), early-life conditions (Sotomayor 2013) or even latent characteristics on the hypertension prevalence. Therefore, while body weight should be considered as a cause of the risk of hypertension (Francischetti and Genelhu, 2007), it is very challenging to make causal inference using such estimates.

In a different manner, this study treats body weight as a cause of hypertension and estimates its causal effect on the prevalence of hypertension by using an IV approach to address the endogeneity problem. Using the IV approach enables this study to disentangle the reliable estimates of the causal effect of body weight on the risk of hypertension. The findings estimated using the IV approach in this study reveal that there is an underestimation of the impact using the non-IV estimator such as the Probit estimator. Therefore, this study suggests that previous studies that did not address the potential endogeneity problem of body weight likely considerably underestimated the impact of body weight on the prevalence of hypertension. This is the most important contribution that this paper makes.

The key finding is that body weight causally increases the likelihood of hypertension for both Vietnamese male and female. The marginal effects of an extra BMI unit on the risk of hypertension are between 5.1–7.3% points for men and 5.6–8.2% points for women. The statistic summary of the sample in Table 1 shows that while the percentage of hypertensive among women is only around 7.8%, the corresponding figure for men is about 17% of the samples. However, the marginal effects of body weight on the prevalence of hypertension are larger for women than men in Vietnam. The study also finds that the impacts of body weight on the prevalence of hypertension substantially vary with various age intervals for men and women as well. Therefore, the implication related to policies for addressing the problem of high blood pressure can be different with different age groups. In the other hand, the impact of body weight on the risk of hypertension probably changes among different periods of the human life. In addition, overweight and obesity are probably favorable to the possibility of

being hypertensive (Francischetti and Genelhu, 2007). Many previous studies show that overweight and obesity are strongly and considerably correlated to increases in the risk of high blood pressure in many developing countries, for examples Nurdiantami et al. (2017) for Indonesia, and Cao et al. (2017) for China. This study confirms the findings from the literature by providing a causal inference that overweight or obesity causally increases the probability of hypertension for both men and women compared to under or normal weight.

Furthermore, this study provides more evidence on the same research topic from a developing country. Developing countries are likely extremely suffered from the epidemic of hypertension because of both their high and increasing proportion of hypertension but an unacceptably low capability of awareness, treatments, and controls there. When the causal link between body weight and the incidence of hypertension is robustly discovered, the implication for controlling the problem of high blood pressure should be raised in the developing world. The actions related to the control of body weight through which hypertension can be restricted include lifestyle changes, nutritional intake, or pharmacological treatments (Dinh et al., 2017; Francischetti and Genelhu, 2007; Lee et al., 2004; Sabaka et al., 2017). Hence, the findings from this study meaningfully provide backgrounds for the public health policies related to the control of overweight and obesity as a solution to the prevalence of hypertension.

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Table 1

Descriptive statistics of the samples (Means and standard deviations in parentheses are reported)

Variable	Men			Women		
	Both	Son	Daughter	Both	Son	Daughter
Hypertension	0.167	0.164	0.169	0.078	0.077	0.078
	(0.373)	(0.371)	(0.375)	(0.268)	(0.267)	(0.269)
BMI	20.340	20.315	20.367	20.371	20.350	20.393
	(2.322)	(2.332)	(2.310)	(2.656)	(2.656)	(2.655)
Overweight or obesity	0.043	0.043	0.043	0.057	0.056	0.057
	(0.203)	(0.203)	(0.203)	(0.231)	(0.231)	(0.231)
Child BMI-for-age z-scores	-1.141	-1.164	-1.117	-1.153	-1.174	-1.131
	(1.034)	(1.072)	(0.991)	(1.030)	(1.063)	(0.993)
Age	40.412	40.391	40.435	37.844	37.875	37.811
	(7.014)	(6.981)	(7.049)	(6.357)	(6.339)	(6.377)
Married	0.993	0.993	0.993	0.953	0.952	0.955
	(0.081)	(0.081)	(0.080)	(0.211)	(0.215)	(0.208)
Majority	0.810	0.812	0.809	0.851	0.853	0.849
	(0.392)	(0.391)	(0.393)	(0.356)	(0.354)	(0.358)
Health insurance	0.170	0.170	0.171	0.127	0.129	0.124
	(0.376)	(0.376)	(0.376)	(0.333)	(0.335)	(0.330)
Schooling years	6.876	6.895	6.856	6.211	6.241	6.179
	(4.377)	(4.375)	(4.379)	(4.352)	(4.375)	(4.328)
Urban	0.271	0.273	0.269	0.280	0.284	0.276
	(0.445)	(0.446)	(0.443)	(0.449)	(0.451)	(0.447)
Red River Delta	0.167	0.170	0.164	0.183	0.186	0.181
	(0.373)	(0.376)	(0.370)	(0.387)	(0.389)	(0.385)
Northeast	0.154	0.152	0.156	0.151	0.149	0.153
	(0.361)	(0.359)	(0.363)	(0.358)	(0.356)	(0.360)
Northwest	0.049	0.050	0.047	0.038	0.039	0.037
	(0.215)	(0.218)	(0.213)	(0.191)	(0.193)	(0.188)
North Central Coast	0.126	0.123	0.129	0.135	0.133	0.136
	(0.332)	(0.329)	(0.335)	(0.341)	(0.339)	(0.343)
South Central Coast	0.097	0.098	0.096	0.101	0.101	0.101
	(0.296)	(0.298)	(0.295)	(0.302)	(0.302)	(0.302)
Central Highlands	0.088	0.090	0.085	0.079	0.081	0.077
	(0.283)	(0.286)	(0.279)	(0.270)	(0.273)	(0.267)
Southeast	0.135	0.133	0.137	0.135	0.134	0.135
	(0.342)	(0.340)	(0.344)	(0.341)	(0.340)	(0.342)
Mekong River Delta	0.184	0.183	0.185	0.179	0.178	0.179
	(0.387)	(0.387)	(0.388)	(0.383)	(0.382)	(0.384)
Observations	24,678	12,698	11,980	26,112	13,437	12,675

Table 2

The effect of men's BMI on the probability of hypertension (Baseline estimates)

Variable	Probit	IV-Probit		
	(1)	(2)	(3)	(4)
BMI	0.018***	0.051***	0.032	0.073***
	(0.001)	(0.018)	(0.024)	(0.026)
Age	0.010***	0.044***	0.046***	0.043***
	(0.003)	(0.011)	(0.015)	(0.016)
Age squared	-0.00003	-0.0001	-0.0002	-0.0001
	(0.00003)	(0.0001)	(0.0002)	(0.0001)
Married	-0.035	-0.142	-0.158	-0.131
	(0.026)	(0.110)	(0.150)	(0.162)
Majority	-0.007	-0.038	-0.084*	0.015
	(0.007)	(0.030)	(0.042)	(0.043)
Health insurance	0.0006	0.016	0.046	-0.015
	(0.007)	(0.030)	(0.041)	(0.043)
Schooling years	-0.002**	-0.006**	-0.006	-0.006
	(0.0006)	(0.003)	(0.004)	(0.004)
Urban	-0.004	0.001	0.014	-0.017
	(0.006)	(0.027)	(0.037)	(0.039)
Red River Delta	0.005	0.008	0.008	-0.009
	(0.009)	(0.037)	(0.053)	(0.053)
Northeast	0.041***	0.158***	0.149***	0.172***
	(0.009)	(0.039)	(0.054)	(0.055)
Northwest	0.088***	0.362***	0.355***	0.373***
	(0.012)	(0.052)	(0.072)	(0.075)
North Central Coast	0.029***	0.110***	0.167***	0.052
	(0.009)	(0.038)	(0.053)	(0.055)
South Central Coast	0.003	0.002	0.033	-0.027
	(0.010)	(0.041)	(0.057)	(0.059)
Central Highlands	0.097***	0.391***	0.439***	0.338***
	(0.009)	(0.042)	(0.058)	(0.059)
Southeast	0.046***	0.188***	0.210***	0.164***
	(0.008)	(0.035)	(0.049)	(0.049)
Mekong River Delta	Reference	Reference	Reference	Reference
First stage *F*-stat		1245.70	633.55	618.73
Observations	24,678	24,678	12,698	11,980

Notes: ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$. Probit model is used in column 1; IV-Probit models are used for columns 2, 3 and 4; IVs are children's BAZ in column 2, sons' BAZ in column 3 and daughters' BAZ in column 4. Coefficients are marginal effects. Robust standard errors are reported in parenthesis.

Table 3

The effect of women's BMI on the probability of hypertension (Baseline estimates)

Variable	Probit	IV-Probit		
	(1)	(2)	(3)	(4)
BMI	0.010***	0.056***	0.082***	0.029
	(0.001)	(0.019)	(0.027)	(0.025)
Age	0.011***	0.083***	0.095***	0.074***
	(0.002)	(0.017)	(0.024)	(0.024)
Age squared	-0.00005*	-0.0004**	-0.0005*	-0.0003
	(0.00003)	(0.0004)	(0.0003)	(0.0003)
Married	-0.014**	-0.103**	-0.145**	-0.052
	(0.007)	(0.050)	(0.068)	(0.073)
Majority	0.002	0.010	0.054	-0.028
	(0.005)	(0.040)	(0.058)	(0.055)
Health insurance	-0.010*	-0.069*	-0.138**	-0.002
	(0.005)	(0.039)	(0.055)	(0.055)
Schooling years	-0.0004	-0.002	-0.001	-0.004
	(0.0005)	(0.003)	(0.005)	(0.005)
Urban	-0.010***	-0.054	-0.085*	-0.020
	(0.004)	(0.035)	(0.051)	(0.048)
Red River Delta	0.025***	0.165***	0.219***	0.111*
	(0.006)	(0.047)	(0.068)	(0.066)
Northeast	0.010	0.051	0.102	-0.0009
	(0.006)	(0.053)	(0.076)	(0.073)
Northwest	0.022**	0.150**	0.166	0.136
	(0.010)	(0.075)	(0.107)	(0.104)
North Central Coast	0.013**	0.076	0.173**	-0.027
	(0.006)	(0.050)	(0.071)	(0.071)
South Central Coast	-0.008	-0.074	-0.043	-0.108
	(0.007)	(0.051)	(0.074)	(0.071)
Central Highlands	0.056***	0.399***	0.503***	0.292***
	(0.006)	(0.051)	(0.071)	(0.072)
Southeast	-0.016***	-0.121***	-0.048	-0.198***
	(0.006)	(0.044)	(0.062)	(0.063)
Mekong River Delta	Reference	Reference	Reference	Reference
First stage *F*-stat		1447.53	731.18	716.60
Observations	26,112	26,112	13,437	12,675

Notes: ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$. Probit model is used in column 1; IV-Probit models are used for columns 2, 3 and 4; IVs are children's BAZ in column 2, sons' BAZ in column 3 and daughters' BAZ in column 4. Coefficients are marginal effects. Robust standard errors are reported in parenthesis.

Table 4
The effect of men's BMI on the probability of hypertension (Robustness checks)

BMI	Probit	IV-Probit		
	(1)	(2)	(3)	(4)
Specification 1	0.017***	0.055***	0.030	0.084***
	(0.001)	(0.018)	(0.024)	(0.026)
First stage *F*-stat		1228.99	627.61	608.60
Specification 2	0.018***	0.059***	0.039*	0.080***
	(0.001)	(0.016)	(0.022)	(0.024)
First stage *F*-stat		1351.55	671.47	691.80
Specification 3	0.017***	0.070***	0.048**	0.096***
	(0.001)	(0.015)	(0.021)	(0.023)
First stage *F*-stat		1428.89	725.20	713.17
Observations	24,678	24,678	12,698	11,980

Notes: ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$. Probit model is used in column 1; IV-Probit models are used for columns 2, 3 and 4; IVs are children's BAZ in column 2, sons' BAZ in column 3 and daughters' BAZ in column 4. Coefficients are marginal effects. Robust standard errors are reported in parenthesis. Control variables for specification 1 include dummies for married status, major ethnicity and health insurance status, schooling years, and dummies for urban and eight geographic regions. Control variables for specification 2 include age, age squared, dummies for married status, major ethnicity and health insurance status, and schooling years. There is no control variable for specification 3.

Table 5

The effect of women's BMI on the probability of hypertension (Robustness checks)

BMI	Probit	IV-Probit		
	(1)	(2)	(3)	(4)
Specification 1	0.011***	0.058***	0.075***	0.039
	(0.0006)	(0.018)	(0.027)	(0.035)
First stage *F*-stat		1429.13	718.07	711.73
Specification 2	0.009***	0.051***	0.073***	0.027
	(0.0006)	(0.016)	(0.023)	(0.021)
First stage *F*-stat		1672.49	839.05	831.89
Specification 3	0.010***	0.052***	0.063***	0.039*
	(0.0006)	(0.016)	(0.023)	(0.022)
First stage *F*-stat		1665.72	837.87	827.93
Observations	26,112	26,112	13,437	12,675

Notes: ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$. Probit model is used in column 1; IV-Probit models are used for columns 2, 3 and 4; IVs are children's BAZ in column 2, sons' BAZ in column 3 and daughters' BAZ in column 4. Coefficients are marginal effects. Robust standard errors are reported in parenthesis. Control variables for specification 1 include dummies for married status, major ethnicity and health insurance status, schooling years, and dummies for urban and eight geographic regions. Control variables for specification 2 include age, age squared, dummies for married status, major ethnicity and health insurance status, and schooling years. There is no control variable for specification 3.

Table 6

The effect of men's BMI on the probability of hypertension (Heterogeneity by age groups)

BMI	Probit	IV-Probit		
	(1)	(2)	(3)	(4)
age≤30	0.018***	0.223**	0.149	0.269**
	(0.004)	(0.093)	(0.136)	(0.120)
First stage *F*-stat		76.56	38.42	42.27
Observations	1,218	1,218	615	603
30<age≤40	0.017***	0.046*	0.038	0.058
	(0.001)	(0.027)	(0.034)	(0.042)
First stage *F*-stat		637.43	336.86	303.86
Observations	12,404	12,404	6,416	5,988
40<age≤50	0.019***	0.028	0.020	0.030
	(0.002)	(0.027)	(0.038)	(0.039)
First stage *F*-stat		452.01	231.05	226.50
Observations	8,976	8,976	4,623	4,353
age>50	0.019***	0.114**	0.040	0.178***
	(0.004)	(0.053)	(0.086)	(0.066)
First stage *F*-stat		90.36	36.24	51.78
Observations	2,080	2,080	1,044	1,036

Notes: ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$. Probit model is used in column 1; IV-Probit models are used for columns 2, 3 and 4; IVs are children's BAZ in column 2, sons' BAZ in column 3 and daughters' BAZ in column 4. Coefficients are marginal effects. Robust standard errors are reported in parenthesis. Control variables include age, age squared, dummies for married status, major ethnicity and health insurance status, schooling years, and dummies for urban and eight geographic regions.

Table 7
The effect of women's BMI on the probability of hypertension (Heterogeneity by age groups)

BMI	Probit	IV-Probit		
	(1)	(2)	(3)	(4)
age≤30	0.003***	0.052	0.085	-0.022
	(0.001)	(0.100)	(0.130)	(0.172)
First stage *F*-stat		148.12	114.77	42.30
Observations	3,058	3,058	1,419	1,511
30<age≤40	0.007***	0.051*	0.089**	0.013
	(0.0007)	(0.027)	(0.040)	(0.035)
First stage *F*-stat		937.53	444.50	499.88
Observations	14,774	14,774	7,607	7,167
40<age≤50	0.016***	0.079***	0.089**	0.065*
	(0.001)	(0.027)	(0.039)	(0.038)
First stage *F*-stat		373.81	194.17	178.12
Observations	7,332	7,332	3,788	3,544
age>50	0.022***	0.006	-0.037	0.027
	(0.004)	(0.086)	(0.138)	(0.112)
First stage *F*-stat		31.85	11.69	19.80
Observations	948	948	495	453

Notes: ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$. Probit model is used in column 1; IV-Probit models are used for columns 2, 3 and 4; IVs are children's BAZ in column 2, sons' BAZ in column 3 and daughters' BAZ in column 4. Coefficients are marginal effects. Robust standard errors are reported in parenthesis. Control variables include age, age squared, dummies for married status, major ethnicity and health insurance status, schooling years, and dummies for urban and eight geographic regions.

Table 8
The effect of men's overweight or obesity on the probability of hypertension

Variable	Probit	IV-Probit		
	(1)	(2)	(3)	(4)
Overweight or obesity	0.143***	1.162***	0.702	1.675***
	(0.010)	(0.419)	(0.568)	(0.600)
First stage *F*-stat		202.32	106.65	97.26
Observations	24,678	24,678	12,698	11,980

Notes: ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$. Probit model is used in column 1; IV-Probit models are used for columns 2, 3 and 4; IVs are children's BAZ in column 2, sons' BAZ in column 3 and daughters' BAZ in column 4. Coefficients are marginal effects. Robust standard errors are reported in parenthesis. Control variables include age, age squared, dummies for married status, major ethnicity and health insurance status, schooling years, and dummies for urban and eight geographic regions.

Table 9
The effect of women's overweight or obesity on the probability of hypertension

Variable	Probit	IV-Probit		
	(1)	(2)	(3)	(4)
Overweight or obesity	0.081***	1.206***	1.871***	0.504
	(0.006)	(0.421)	(0.572)	(0.563)
First stage *F*-stat		255.50	110.50	150.11
Observations	26,112	26,112	13,437	12,675

Notes: ***$p < 0.01$, **$p < 0.05$, *$p < 0.1$. Probit model is used in column 1; IV-Probit models are used for columns 2, 3 and 4; IVs are children's BAZ in column 2, sons' BAZ in column 3 and daughters' BAZ in column 4. Coefficients are marginal effects. Robust standard errors are reported in parenthesis. Control variables include age, age squared, dummies for married status, major ethnicity and health insurance status, schooling years, and dummies for urban and eight geographic regions.