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The Relationship between Obesity and Diabetes among Mexican Elderly: Results from MHAS 2001-2014

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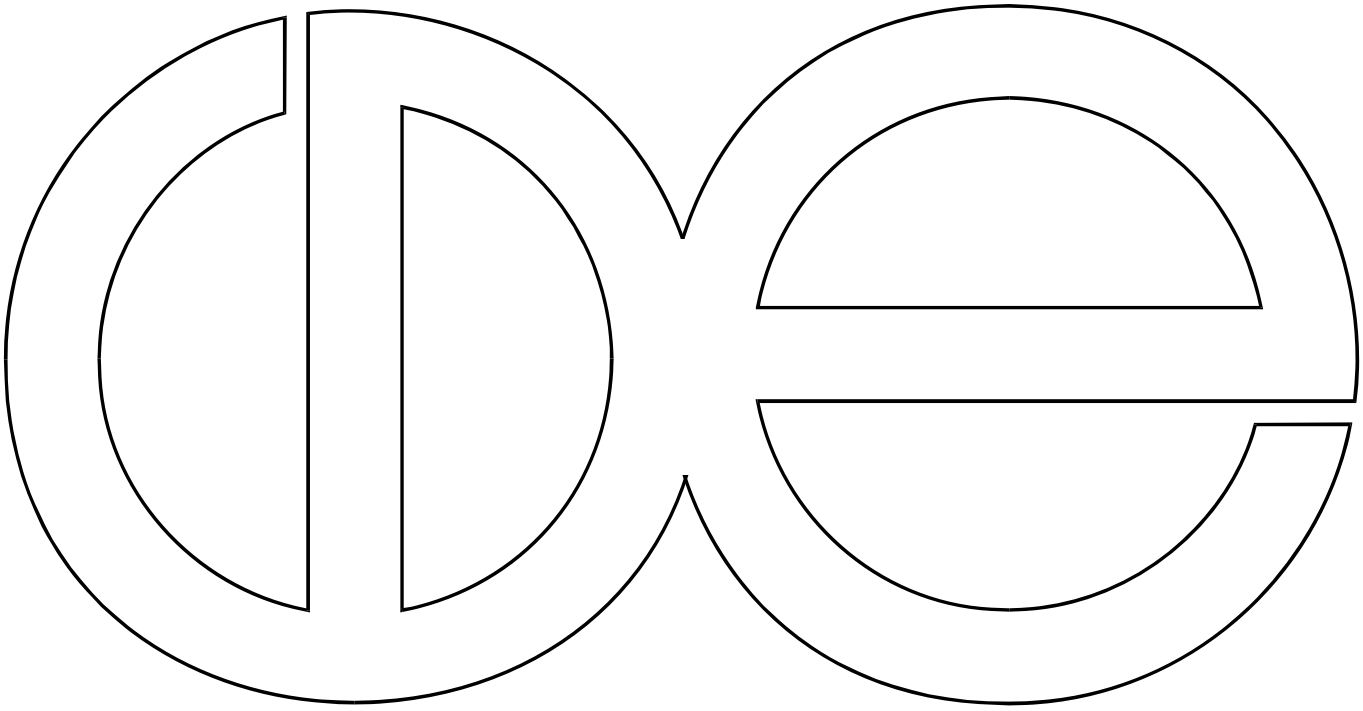

The Relationship between Obesity and Diabetes among Mexican elderly: Results from MHAS 2001-2014

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Abstract

Objective. To assess the magnitude of the relationship between obesity and type 2 Diabetes Mellitus (T2DM) among Mexican elderly population. **Material and Methods**. Data from the 2001, 2003, and 2012 Mexican Health and Aging Study (MHAS) was used and estimates were obtained using multivariate logit regression methods for panel data. **Results**. Logit models produced positive, strong, and significant estimates of the relationship between obesity and T2DM at the population-level, after adjusting for respondent's individual traits and thus, show that obese individuals, particularly those with abdominal obesity, are more likely to develop diabetes. **Conclusion**. The empirical findings gleaned in this paper provide additional evidence of the role of obesity as the primary driver of diabetes. This means that reductions in obesity rates, especially waist obesity, may lead to important declines in morbidity and mortality from diabetes, which consequently may lead to cost reductions for the health system.

Key words: type 2 diabetes mellitus; obesity; abdominal obesity; elderly; Mexico

Obesity and diabetes among Mexican elderly

INTRODUCTION

The World Health Organization (WHO) on its Global Strategy on Diet, Physical Activity and Health[1] stresses that "obesity and overweight pose major risk for chronic diseases, including type 2 diabetes mellitus (T2DM), cardiovascular disease, hypertension and stroke, and certain forms of cancer." That is, obesity may be considered a major risk factor for several chronic conditions, in particular type 2 diabetes.

Worldwide figures show an upsurge in obesity and diabetes cases[2-4] which are becoming an insurmountable health problem, especially in poor and emerging countries, already strained by malnutrition and communicable diseases.[5] It is predicted that by 2030 one billion people will be obese and another 366 million will have some form of diabetes.[4]

The prevalence of obesity has consequently soared in both low-and-high income countries, causing a surge of T2DM and vascular diseases.[6] Obese persons are also more likely to suffer a deteriorated health status and tend to have shorter lifespan when compared to normal weight individuals.[7] It reduces considerable quality of life and, at the same time, causes sizeable health care costs.[8]

Prevalence of adult obesity in Mexico has grown at an unprecedented pace during the last two decades, reaching the highest level (33%) in the Latin America region in 2012.

Diabetes is a major chronic disease that not only places a high burden due to its associated comorbidities and increased medical costs but also for old-age disability and mortality. Diabetes prevalence in the adult Mexican population (aged 20 or older) has more than doubled between 1994 and 2006, from 6.7% to 14.4%, but it remained fairly constant between 2006 and 2012 at about 14%. However, the prevalence among older adults is much higher, reaching about 19% and 24% among those aged 50-59 and 60-79, respectively, in 2012. In addition, the majority of diabetic patients (85%) aged 20 or older in 2006 were poorly treated, regardless their access to health care, type of institution, or type of health insurance. As a result of poor disease control, there are sizeable economic costs associated to diabetes; some research suggests that diabetes accounts for about 7% of all health expenditures in Mexico.

Epidemiologic evidence consistently shows that being overweight or obese are major risk factors for developing diabetes. As early as 1978, a positive relationship between diabetes and obesity was found in different populations. In Mexico, evidence indicates that overweight or obese individuals have a higher likelihood of being diabetic. In addition, prevalence of overweight and obesity differs significantly by sex, socioeconomic status (SES) and place of residence, suggesting that diabetes prevalence also varies by those socioeconomic conditions. These studies, however, are based on cross-section data which limit our understanding of the relationship between overweight/obesity and diabetes as the latter condition develops slowly over the individual's life course. A handful of small epidemiologic studies have estimated the relationship between overweigh/obesity and diabetes in Mexico (e.g., in Mexico City); yet, this study is not nationally representative of the older adult Mexican population.

Epidemiological and medical literature reveals a clear connection between obesity and diabetes mellitus. As early as 1978, a positive link between diabetes and obesity in different populations was found; in fact, the association shows that the prevalence of T2DM increases as population becomes more obese.[9] Data from other studies also show a clear association between obesity and risk of diabetes.[10-13] For instance, using data from NHANES, Ford and associates found that for each kilogram increased in weight, the risk of diabetes increased by 4.5%.[12]

T2DM and obesity have also been found to be associated with insulin resistance.[14] In the last two decades, there have been important strides to disentangle the complex nature of this relationship, mostly coming from biomedical and clinical studies.[15-18] Although the precise mechanism underlying this relationship is still controversial and not fully understood; however, there is evidence of the existence of various pathways at work.[19-21]

Obesity seems to promote insulin resistance. The production of defective or insufficient insulin or the inability of body cells to use it properly and efficiently leads to diabetes, a condition known as insulin resistance. An important feature of T2DM is then insensitivity to insulin by fat and muscle cells and is also strongly implicated in β-cell dysfunction. T2DM might be responsible for the continued decline in β-cell dysfunction.[22-24]

Obesity has been found to be consistently associated with an increased risk of developing insulin resistance, and therefore, T2DM. However, the distribution of body adiposity is a critical determinant of insulin resistance; insulin insensitivity is greater in individuals who have their fat distributed centrally, that is, in the abdominal and chest regions, which is also called visceral adiposity.[25-27]

Although the precise mechanisms involved in the etiology of obesity-related diabetes are not fully known, they are thought to work through the adipose tissue found in abdominal and chest depots, which releases free fatty acids and adipocytokines hormones (leptin, adiponectin, PAI-1, TNF -α, interlukins, etc.) and certain inflammatory cytokines (IL-6, TNF-α, TGF b1and MCP -1, etc.) promoting insulin resistance. [28-32] For instance, TNF-α, IL-6 and leptin induce insulin insensitivity in the body's cell, while adiponectin, which is secreted from adipose tissue, is inversely related to weight gain. Abdominal and chest fat is, thus, a biological active tissue that produces chemical messengers that affect the absorption of insulin by body cells. Thus, waist obesity can be considered as a marker for development of glucose intolerance and progression of T2DM.[32]

Environmental factors have been identified also as one of the culprits for the current epidemic of obesity and T2DM. The proposed mechanism seems to be clear: increased caloric intake and fatty food consumption in an environment of reduced physical activity may lead to over-nutrition, increased storage of fat and, consequently, obesity. In the long-run, dietary shifts towards consumption of fats, saturated fats and sugar, accompanied by a reduction in energy expenditure (sedentary life style), creates an imbalance that leads to obesity and thus to reduction of insulin release.[33] Conversely, increased physical activity and increased carbohydrate intake are associated with insulin sensitivity increase.[34-35] A second mechanism may be produced in utero or in early life when poor nutrition modifies individual's metabolism, which favors storage of nutritients.[36]

A genetic predisposition in the development of this form of diabetes is also present.[37] Genes have been found to be associated with dysfunction of β-cells.[38-39] First-degree relatives of individuals with diabetes mellitus (like parents or siblings) are also at

increased risk of contracting diabetes and show β-cell dysfunction.[40] Interestingly, one study suggests that lifestyle interventions causing an average weight reduction of less than 6 kilograms in a six-month period led to a 58% reduction in cumulative diabetes in treatment groups.[41]

The purpose of this paper is to assess the magnitude of the relationship between obesity and T2DM among Mexican elderly population. As shown above, the fact that obesity increases diabetes is well-established in the medical literature, which illustrates the underlying causal mechanism between obesity and diabetes mellitus and, thus, a warrant to our empirical model specification and findings.

Additionally, this paper accrues evidence to the incomplete knowledge about the magnitude of this relationship in elderly populations outside wealthy countries. Moreover, this relationship has often been studied using cross-sectional data on the prevalence of these two conditions. We intend to move forward and to estimate this relationship using robust longitudinal methods, resulting in estimates that are free of unobserved confounding effects present in cross-sectional data.

MATERIAL AND METHODS

Data

Our empirical analysis is conducted on micro-level data from a large nationally representative random sample of Mexican adults aged 50 and over. The MHAS (Mexican Health and Aging Study) dataset addresses several dimensions of individual health. The study provides data on demographic traits, self-perceived health status and chronic conditions, lifestyle choices (tobacco use, alcohol consumption, or physical activities), functional and cognitive performance, labor and economic status, income and assets, transfers, family arrangements, use of health services, health insurance and housing. This unique micro-level database was

designed following the U.S. Health and Retirement Study (HRS) protocol, to track changes on health conditions among elderly persons as well as shifts in some major bio-demographic, social, economic, and health events.

The MHAS dataset includes information from three waves. The sample baseline consists of 15,402 interviews gathered during 2001. The second wave was conducted during 2003; questionnaires were administered to 14,386 surviving respondents and a new sample of 220 new spouses. Finally, a third survey was carried out during 2012 including 12,569 respondents and a new sample of 5,896 new subjects and spouses. This survey includes sampling weights to correct for oversampling in those states with high emigration rates to the U.S. The MHAS data is available at http://mhasweb.org/. For the first and second waves, anthropometric measures (height, weight, knee height, waist and hip circumferences) were also collected by field supervisors from a 20% random subsample of respondents.

In this study, we use all respondents and their spouses aged 50 or more, interviewed in any of the three waves.

Measures

Diabetes was measured using self-reported information on chronic conditions. Self-report on diabetes was obtained using the following question: “Has a doctor or medical personnel ever told you that you have diabetes or a high blood sugar level?” Underreporting is very likely because there are probably some undiagnosed cases. If this is the case, true estimates in the regression models will be biased and underestimated. The variable “diabetes” takes the value of 1 if the respondent says “yes” to the previous question.

“Obesity” was measured using three set of indicators, one self-reported and two objective measures: 1) Body Mass Index (BMI) based on self-reported weight and

height, 2) BMI based on interviewer-performed measurements (objective measure) on height and weight, and 3) waist circumference also based on an actual measure. We also consider observed and self-reported BMI as a continuous variable as a fourth possible indicator to assess the effect of obesity on diabetes mellitus. The main advantage of the objective measures is that measurement error would not be correlated with respondent's individual characteristics.

We used the standard cut-off to define obesity according to WHO guidelines,[42] BMI ≥ 30. Similarly, cutoff points for abdominal obesity were a waist circumference of 88 centimeters for females and 102 centimeters for males.[43] The so-called central or abdominal obesity is the accumulation of adiposity around the waist.

In all analyses, the control group corresponds to non-obese individuals. Thus, coefficient estimates represent the effect of being obese (treatment group) on diabetes relative to their non-obese counterparts.

Potential confounding variables

Among the potential confounders associated with both conditions are sex, age, genetic predisposition to diabetes, physical activity, smoking and alcohol drinking habits. Since our aim is to purge for all confounding effects, we include the following variables in alternative models:

Life-styles, behavior and genetic inheritance. We consider four of these variables that might influence both obesity and diabetes. 1) Physical activity with a value of 1 if the respondent reported having exercised, or done some hard physical activity three or more times a week, and 0 otherwise. 2) Smoking corresponding to 1 if the respondent currently smokes (active smoker) and 0 otherwise. 3) Attending Mass measures the frequency of a person attending religious worship as an indicator of some unobserved behavior; it is coded as 1 if the respondent goes to church

frequently and 0 otherwise. 4) The transmission from one generation to another of some genetic factors increases an individual's propensity to acquire diabetes. This variable is coded as 1 if either the respondent's parent or sibling were told by physician or medical personnel they have diabetes and 0 otherwise.

Demographic variables. Respondent's age is represented by a continuous non-linear variable as age and age squared. Models are estimated in the full sample and a sex variable is included with 1 corresponding to males and 0 to females.

Socioeconomic variables. Six variables are used to account for socioeconomic gradients. 1) Education as binary variable with 1 for respondents who completed elementary school or higher and 0 for those with incomplete elementary or no formal schooling. 2) Income as a continuous variable consisting in the logarithm of the total monthly earnings reported by the respondent. Income included wages and salaries, pensions, transfers, and other sources. 3) Household wealth corresponding to a count of 8 household goods and conveniences, such as water and electricity connections, having a computer, a car, etc. Two categories were obtained from the breakdown of the resulting distribution of wealth scores: poor, middle, and rich, which were included in the equations as a binary variable (Poor). 4) Early-life economic status refers to respondent's economic situation during the first 10 years of life. As a binary variable takes the value of 1 if the respondent's family slept in the kitchen and/or did not wear shoes before he/she was 10 years old and 0 otherwise. 5) Early-life health problems refers to respondent's health problems; it was coded 1 if the respondent had serious health problems before he/she was 10 years old, 0 otherwise. 6) Rural origin refers to original parental residence; it was coded as 1 if respondent's parents lived in a rural area and 0 otherwise.

Empirical model specification

Since our objective is to model the propensity of diabetes as a function of obesity and various demographic, socioeconomic, and individual lifestyle characteristics, we used a random effects logistic regression model for longitudinal data. We use random effects to take into account differences across individuals that could influence our dependent variable.

The model assumes that diabetes is not directly observable but instead is reported by the respondents themselves and represented by a binary variable. The propensity-to-diabetes equation is specified over individuals (i) and time (t) as follows:

$$\log [Diabetes_{it}/(1-Diabetes_{it})] = \beta_0 + \beta_1 X_{1it} + \beta_2 X_{2it} + \ldots + \beta_k X_{kit} + \mu_i + \varepsilon_{it}$$

"*Diabetes*" is the chronic condition reported by the respondent. This empirical specification monitors respondent's individual traits such as age, gender, education, socioeconomic status, income, smoking, physical activities, heredity, and religiosity (behavioral component).

RESULTS

Prevalence of diabetes and obesity

Prevalence rates of diabetes, overall obesity and abdominal obesity during the three waves of the MHAS study are displayed in Table 1. Since these values are calculated using self-reported information, it is very likely the true values are underestimated, because there are, probably, some undiagnosed cases.

Table 1 about here

These figures show clear patterns in prevalence rates. First, diabetes prevalence rates are rather constant between 2001 and 2003, but increase by 2012. For instance, diabetes rates increase from 16.2 in 2001 to 25.2 in 2012. A different

pattern is observed for self-reported obesity, there is a slight increase in obesity from 23.3 to 25.2 between 2001 and 2003 but a decrease during the next period. Both objective indicators of obesity show a noticeable increase from 2001 to 2003 by about 6%.

Diabetes prevalence by obesity—crude associations

Table 2 displays diabetes prevalence rates among obese and non-obese individuals. These figures show that obese respondents are more vulnerable to this chronic condition than non-obese persons. According to these figures, obese individuals show larger prevalence rates of overall and abdominal obesity than non-obese persons. For instance, in wave 2, the prevalence rate of diabetes among abdominal obese persons is 1.6 times larger than in non-obese individuals. In sum, prevalence rates show that obese individuals are more likely to be diabetics than their non-obese counterparts. Thus, it can be concluded that overall obesity and abdominal obesity increases, on average, the risk of diabetes in this population.

Table 2 about here

The net effect of obesity on diabetes

Results from multivariate logit random effect models assessing the relationship between self-reported diabetes and different measures of obesity are shown in Tables 3 and 4 for self-reported and objective measures, respectively. These results show net effects representing the association between measures of obesity and diabetes that are not “contaminated” or “confounded” by all other factors included in the model.

Table 3 about here

Table 3 reports two alternative models for the effects of self-reported obesity on the prevalence of diabetes between 2001 and 2012. Model 1 uses a dummy obesity indicator (BMI≥30) and Model 2 includes BMI as a continuous variable. Similarly, Table 4 shows three alternative models for objective measures of obesity on the prevalence of diabetes between 2001 and 2003. Model 1 includes a dummy indicator for abdominal obesity, Model 2 uses a dummy indicator of overall obesity (BMI≥30) and Model 3 includes BMI as a continuous variable.

Results indicate that the net effects of obesity on diabetes are all statistically significant and in the expected direction. That is, obese persons are more likely to be diabetics, after controlling for confounding variables.

For instance, in Table 3 Model 1 shows that **self-reported obesity** is positively associated with T2DM, even after controlling for demographic, socioeconomic, behavioral and hereditary factors. That is, being obese increases the likelihood of having diabetes, when compared to non-obese persons, everything else being equal. In fact, in Model 1, obese persons are 1.6 times more likely to have diabetes than non-obese persons, after controlling for other variables in the model. The remaining variables have significant effects on diabetes and in the expected direction: the presence of some “Physical Activity” reduces the odds of being diabetic, while having parents or siblings diagnosed with diabetes has the strongest effect: the odds increase 14 times. Model 2 also indicates that self-reported BMI is positively and statistically significantly associated with diabetes. In relative terms, its effect can be interpreted as follows: a one-unit increase in the BMI score is expected to increase the odds of having diabetes by about 9 percent, everything else being equal. That is, higher values of BMI score (overweight and obesity) make type 2 diabetes more likely to occur after controlling for different socioeconomic,

demographic and behavior variables in the models. Thus, even moderate gains in weight are associated with increased risk of developing diabetes suggesting that even a slight degree of overweight may affect this chronic condition.

Table 4 about here

Results displayed in Table 4, using objective obesity measures, provide further support for the obesity-diabetes link. Model 1 shows that the presence of abdominal obesity makes diabetes almost 5 times more likely to occur, even when controlling for BMI. That is, abdominal obesity seems to be a major risk factor for developing diabetes above and beyond that of BMI. In fact, BMI is no longer significant when abdominal obesity is accounted for, suggesting that the latter is a stronger predictor of diabetes incidence in this population. It is also noticeable that the coefficients of "Physical Activity" and "Hereditary," are in the expected direction and statistically significant, indicating that they have a negative and positive significant effect on diabetes incidence, respectively. Moreover, Model 2 shows that objective measures of weight and height (overall obesity) doubles in size the effects of its self-reported counterpart shown in Table3, albeit not significantly.

Model 3 also indicates that a one-unit increase in the objective BMI score is expected to increase the odds of having diabetes by about 112 percent, holding constant everything else in the model. As we move to higher values of the BMI score, type 2 diabetes is more likely to occur after controlling for different socioeconomic, demographic and behavior variables. Thus, we can say that any gain in weight is significantly associated with an increased risk of developing diabetes.

All models also predict a significant nonlinear effect of age on the odds of diabetes. In fact, age and squared age have a significant effect on the propensity of having diabetes. Age has a positive coefficient and squared age a negative one. That is, the presence of diabetes increases with age up to a certain value and decreases afterwards. For instance, in Model 1 displayed in Table 4, the odds ratio associated with age can be expressed as $e^{1.0633+2*(-0.0084)*Age} = 2.8959* e^{-0.0084*Age}$, which depends solely on age. At age 70, the odds ratio of having diabetes is about 1.61, while at age 80 it decreases to around 1.48. Similar results can be obtained for all other regression equations. This quadratic effect could be explained by the fact that after certain age healthier individuals survive with lower levels of chronic conditions. Therefore, results from all models show that obese individuals, particularly those with abdominal obesity, are more likely to develop diabetes, and thus obesity is rather a strong and useful predictor of the incidence of T2DM among elderly persons in Mexico.

Conclusion

The data used in this paper confirms previous findings indicating a higher probability of developing diabetes among obese individuals. Logit regression models using data from MHAS produced positive, strong, and significant estimates of the relationship between obesity and T2DM at the population-level, after adjusting for respondent's individual traits. That is, as expected, overall obesity and abdominal obesity are strongly and significantly associated with T2DM in the elderly Mexican population. Moreover, our results indicate that people with waist obesity are more likely to have and develop diabetes mellitus, after controlling for inter-individual variability.

The clear-cut relationship between obesity and diabetes found in this paper is warranted by the underlying mechanism described in the introduction section. That

is, these results are consistent with findings showing that in individuals with visceral or abdominally obesity, adipose tissue releases more frequently fatty acids and glyrecol hormones as well as pro-inflammatory cytokines and other factors that may encourage insulin resistance,[21,31,44] and when insulin insensitivity is accompanied by dysfunction of β-cells, failure to control glucose levels in the blood may result.[23-24]

Additionally, our results are consistent with the hereditary link found in neuroendocrine studies. [37-40] We found that individuals who had a parent or sibling with diabetes are about 14 times more likely to develop diabetes, net of obesity and other inter-individual traits, which is consistent with the underlying idea that malfunction of β-cells is critical for developing type 2 diabetes, has a genetic component. The results are also germane to the fact that decreased or lack of physical activity is both a cause and consequence of weight gain and also contributes to insulin resistance. In all models, physical activity decreases the odds of being diabetic, which is also compatible with the idea that impaired insulin signals the hypothalamus to change body's weight.[47]

Thus, the empirical findings gleaned in this paper provide additional evidence of the role of obesity as one of the primary drivers of diabetes. This means that reductions in obesity rates, especially waist obesity, may lead to important declines in morbidity and mortality from diabetes, which consequently may lead to cost reductions for the health system. For instance, diabetes is the leading cause of death among women and the third among men in Mexico.[45]

We can conclude that even modest decreases in body weight may produce a significant reduction in adipose tissue, which make the largest contributions to diabetes upsurge.[46]

However, there are some limitations in this study. We rely on self-reported data on diabetes, which may be producing some bias in our estimates due to undiagnosed cases. Nonetheless, underreports of diabetes will likely lead to lower than expected coefficient estimates which suggest that our results are conservative. Despite this problem, the results obtained are robust and consistent with previous observational studies and are very likely to reflect the true association in the elderly Mexican population.

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Table 1. Prevalence Rates of Diabetes and Obesity, MHAS 2001, 2003, 2012

Wave	Diabetes	Overall Obesity*	Overall Obesity**	Abdominal Obesity
2001	16.18	23.34	27.15	58.46
2003	16.69	25.23	28.72	61.70
2003	25.24	24.00	N/A	N/A

* Self-Reported

** Anthropometric measures

Table 2. Prevalence Rates of Diabetes and Obesity, MHAS 2001, 2003, 2012

Obesity/Wave	Diabetes		
	2001	2003	2012
Obese*	18.04	16.33	30.83
Non-Obese	15.71	19.19	24.31
Obese**	19.90	19.80	N/A
Non-Obese	15.62	16.58	N/A
Abdominal Obese**	19.04	20.40	N/A
Non-Obese	13.66	12.70	N/A

* Self-Reported

** Anthropometric measures

Table 3. Alternative Logit Models for Incidence of Diabetes, MHAS 2001-2012

Predictors	**Model 1**	**p-value**	**Model 2**	**p-value**
Overall Obesity*	0.4778	0.007		
BMI*			0.0866	0.000
Physical Activity	-0.4489	0.004	-0.4236	0.007
Hereditary	2.6630	0.000	2.6515	0.000
Sex	0.0760	0.647	0.0780	0.640
Age	1.1849	0.000	1.1702	0.000
Age2	-0.0083	0.000	-0.0082	0.000
Early Health Problem	0.4705	0.068	0.4700	0.070
Poor Economic Status	-1.0801	0.000	-0.9998	0.000
Education	0.9402	0.000	0.9013	0.000
Log(Income)	0.0007	0.977	-0.0003	0.989
Smoking	-1.3391	0.000	-1.2700	0.000
Attends Mass	-0.1120	0.561	-0.1177	0.543
Rural origin	-0.2992	0.107	-0.2680	0.151
Poor Childhood	0.2002	0.501	0.2144	0.473
Constant	-47.1168	0.000	-49.0676	0.000
Wald Chi	377.58	0.000	385.53	0.000
Rho	0.8946	0.000	0.8941	0.000
N	8255		8255	

* Self-Reported

Table 4. Alternative Logit Models for Incidence of Diabetes, MHAS 2001-2003

Predictors	**Model 1**	**p-value**	**Model 2**	**p-value**	**Model 3**	**p-value**
Abdominal Obesity	1.5505	1.019			0.1163	0.022
Overall Obesity*			0.8506	0.109		
BMI	0.0544	0.359				
Physical Activity	-1.5393	0.004	-1.5121	0.003	-1.5129	0.004
Hereditary	2.6785	0.000	2.4966	0.000	2.5315	0.000
Sex	-0.3306	0.575	0.2751	0.584	0.2838	0.581
Age	1.0663	0.027	0.9911	0.028	0.9998	0.032
Age2	-0.0084	0.025	-0.0079	0.026	-0.0079	0.031
Early Health Problem	0.5152	0.497	0.6650	0.362	0.5712	0.446
Poor Economic Status	-1.1593	0.165	-1.4197	0.088	-1.3103	0.126
Education	0.5822	0.389	0.6490	0.316	0.5351	0.418
Log(Income)	-0.1001	0.204	-0.0980	0.188	-0.0960	0.209
Smoking	-0.3903	0.426	-0.4834	0.298	-0.4202	0.378
Attends Mass	-0.1946	0.778	-0.1854	0.777	-0.1840	0.784
Rural origin	-0.9938	0.089	-0.9411	0.096	-0.8747	0.130
Poor Childhood	1.3027	0.170	1.2215	0.185	1.3082	0.164
Constant	-40.9398	0.008	-36.1798	0.012	-39.8039	0.008
Wald Chi	47.15	0.000	33.82	0.002	-39.8039	0.008
Rho	0.8999	0.000	0.8636	0.000	0.8674	0.000
N	2049		2053		2053	

*Anthropometric measures

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