A structural model of wealth, obesity and health in the UK

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Abstract— **Based on a household health production framework, this paper exploits the combination of socio-economic, health and nutrition information from the UK National Diet and Nutrition Survey to analyze the endogenous relationship among wealth, nutrition, weight and the final health outcomes. Results show that higher wealth determines lower weight and better health as expected, but through a better diet rather than extra exercise or lower calorie consumption.**

Keywords—**Structural Equation Modelling, Body Mass Index, Diet, Blood Pressure, Income**

I. INTRODUCTION

The scientific debate about obesity, its determinants and consequences is still facing many open questions, mainly due to the lack of adequate, reliable and complete data. Obesity is now high in the European policy agenda, because of three main reasons. First, high obesity rates are not confined in North American and Anglo-Saxon countries any more, there are sharp increases in European countries and even developing countries are experiencing a rapid rise [1]. Second, obesity rates have been associated with serious health consequences, namely non-communicable chronic diseases like diabetes and CVD, which generate high direct health care costs and productivity losses [2]. Third, with public health care systems, these costs are borne by all taxpayers, suggesting that an externality exists and overeating might be seen as a market failure [3].

However, there is little still consensus about the policy interventions, because of a lack of clear evidence on many aspects of the lifestyle-health relationship and the conflicting interests of the various stakeholders [4]. The most convincing evidence about the causes of rising obesity rates are technical progress in food processing (and the consequent decline in energy-dense food prices) and modifications in work and life styles, although other hypotheses co-exist. For example, thanks to health care progresses, it has never been safer to be obese, asymmetric information may determine unhealthy dietary choices [5] and rational addiction, time discounting and lack of self-control may explain why individuals do not choose health-maximising lifestyles [6].

Governments' focus on obesity has also been the subject of criticism. In his best-selling book, Oliver [7] claims that diet, exercise or medical history are more likely to be direct determinants of health outcomes rather than obesity, which might be one of the outcomes of unhealthy lifestyles, but not necessarily the determinant of adverse health consequences. Oliver also argues that measurements might be flawed, especially body mass index (BMI), which is defined as 'not only a poor measure of health, it is actually a lousy measure of obesity' [7, p.21]. The issue is causality. While it is hardly debatable that unhealthy lifestyles cause poor health and unhealthy lifestyles cause obesity, it is more difficult to test whether obesity causes poor health, after accounting for unhealthy lifestyles. If the latest relationship is false, then policies against obesity may be stigmatising and undesirable, and the focus should be on lifestyles only, regardless of the body weight.

The hunt for evidence calls for a multi-disciplinary approach. Economics is relevant both because of the financial burden commonly associated with obesity-related diseases (estimated between 0.3 and 1.2% of GDP [8], and because the problem seems to be associated with social inequalities, to the extent that the UK government has recently launched a health-equality impact assessment in relation to the weight-health relationship[1].

[1] "Healthy Weight, Healthy Lives: A Cross-Government Strategy for England: Equality Impact Assessment", Cross-Government Obesity Programme, http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_082378

This paper contributes to the literature on the determinants of obesity in Europe by exploiting the most complete data set on health and nutrition available at individual level, the UK National Diet and Nutrition Survey, which collates nutrition data (collected through a dietary intake diary) and socio-economic data with medical data from blood sample and urine tests for the same individuals.

Furthermore, as emphasised by several studies [9], we explicitly allow for endogenous relationships and measurement problems by adopting a structural equation modelling (SEM) strategy, which also allows comparing different causal relationships in terms of their fit with the data.

The study attempts to provide an answer to three questions:

1. How does the wealth distribution affect lifestyles and body weight?
2. After accounting for endogeneity, what is the relationship between body weight and health outcomes?
3. Is the body-mass index a good predictor of obesity and health compared to the waist-hip ratio?

The paper is organised as follows. Section 2 illustrates the theoretical model adopted for the analysis in relation to the literature. Section 3 describes the data set, with a focus on the issues of wealth and weight measurement. Section 4 frames the economic model within the structural equation model estimation strategy. Section 5 presents the results of the quantitative analysis, while conclusions are drawn in section 6.

II. THEORETICAL MODEL

The degree of endogeneity which characterizes the links among consumption choices, wealth, other lifestyle determinants and health outcomes has been the subject of various studies where it has been shown that accounting for interactions may change the intensity and direction of these relationships [9-10].

Following similar considerations, Chen et al. [11] explore the influence of prices and income on nutrient intake, exercise and blood pressure to find that accounting for endogeneity results in sodium reducing blood pressure, arguing that biomedical epidemiological studies might be flawed by treating human choice as exogenous factors. The model by Chen et al. is based on the household production function approach and can be extended to account for obesity.

Suppose that individuals derive utility from eating and drinking, smoking (S), consumption of other goods which do not contribute to health (Z), their leisure (L) and their state of health (H), which is partially a behavioural variable as it relates to smoking and weight [11-12]. For the sake of simplicity assume that the utility from food and drink consumption can be represented by calorie intake (K), a common assumption (e.g. [13-15]). Leisure may be taken as free time after working and exercise taken for health reasons.

$$U = u(K, S, L, H, Z) \qquad (1)$$

Health is related to weight (W), other aspects of diet quality (Q) (e.g. the intake of saturated fatty acids which may have an impact on health independent of weight), medical treatment (M), smoking, exercise (E), which is taken to provide health benefits independent of its impact on weight, and exogenous factors (X_H) which include genetic and socio-demographic factors, the latter including education which may affect an individual's knowledge and ability to combine health inputs to optimise the health function [11]:

$$H = h(W, S, E, Q, M, X_H) \qquad (2)$$

In this construction we abstract from dynamics which would recognise that current health also depends on past levels of weight, diet quality, smoking and exercise. It can be thought of as a long-run equilibrium relationship. Weight gain occurs when calorie intake exceeds calorie expenditure. The latter depends on activity in the workplace, in travel (by foot or bicycle) and at home for leisure and non-leisure exercise; on an individual's metabolic rate (hence the genetic component to overweight and obesity); and on one's weight. Hence, as Cutler, Glaeser and Shapiro [13] point out, there exists an equilibrium (steady state) weight associated with any level of calorie intake. The achievement of this steady state is not of course instantaneous. Within the behavioural framework established here, exercise itself is

endogenous in the sense that an individual may choose to achieve any particular weight either by consuming a large number of calories and exercising a lot or consuming a lower number of calories and exercising less.

$$W = w(K, E, X_W) \qquad (3)$$

Where X_W represents exogenous factors such as level of physical activity at work and genetic predisposition. Formally the utility function in (1) is maximised subject to the health function (2), the weight function (3) and a full income budget constraint in which time may be enjoyed as leisure or transformed into income at the prevailing wage rate or exercise for health purposes (as opposed to leisure purposes). Food, drink, cigarettes, health care and other goods up to the level of income may be purchased at prevailing prices.

$$\begin{aligned} &V + w(T - L - E) = \\ &p_K \cdot K + p_Q \cdot Q + p_S \cdot S + p_M \cdot M + p_Z \cdot Z \end{aligned} \qquad (4)$$

In (4), V is non-labour income, T is total time and L is leisure. Income, leisure and exercise are all endogenous in this framework, as of course are calorie intake, smoking, the level of medical treatment, exercise and health status. Solving the system of equations leads to a set of reduced form equations in which the optimal level of each of the endogenous variables depends on the wage rate and prices as well as the levels of the exogenous variables (X_H and X_W).

The model we adopt in this study requires some simplification, because – most unfortunately – to our knowledge there is no such thing in Europe as a data set which has individual data on nutrient intake, health outcomes, expenditure levels and prices. The UK National Diet and Nutrition Survey (described in section 3), is the data set which best approximates this ideal information set, as it includes data on individuals and merges information on wealth (income, social class, education, etc.) with nutrition data (nutrient intakes at a very detailed level) and health outcomes (namely blood pressure and cholesterol data). Thus, the structural model being estimated as a start is the following:

$$\begin{cases} K = f(Y, S, E, Q) \\ E = f(Y, K, S, Q) \\ S = f(Y, K, E, Q) \\ D = f(Y, S, K) \\ W = f(K, E, Q, Y) \\ H = f(W, E, Q, S) \end{cases} \qquad (5)$$

Where Y is a measure of wealth which reflects income, education and social class (see section 3). The first three equations in (5) reflect the amount of calories, exercise and smoking that individual choose in order to maximise their utility, considering substitutions and complementarities. Within the same maximisation process, individuals also choose the allocation of their diet and the fourth equation reflect the quality of diets as a function of wealth, calorie intake and smoking. Weight is determined by decisions on calorie intake, exercise diet quality, plus wealth as an exogenous determinant, which also reflects the distinction between manual and non-manual work as discussed later. Finally, the last equation measures the health outcomes as a function of weight, exercise, diet quality and smoking. The model is consistent with the household production derivation, although it necessarily omits prices and medical treatments.

III. DATA

Data are taken from the 2000-2001 UK National Diet and Nutrition Survey[2] and variables relevant to the modelling effort are illustrated in table 1.

Diet quality. The valuable amount of information in the NDNS data set allows building a composite indicator of compliance with the WHO norms [17], as the one described in Mazzocchi et al. [18]. The norms being taken into account are listed in Table 2. The indicator measures the distance from each individual norm, and individual distances are aggregated into a composite indicator. After appropriate weighting and rescaling, the recommendation compliance index

[2] See Swan [16] for an accurate discussion of the survey and a description of the data

(RCI) is bounded between 0 and 1 and it can be shown that it is robust to modifications in the relative weight of individual norms.

Table 1. Model variables and NDNS data

Variable	Description	NDNS data
Q	Diet quality (according to WHO recommendations)	% Energy from fats
		% Energy from saturated fats
		% Energy from trans-fats
		% Energy from proteins
		% Energy from carbohydrates
		% Energy from sugar (all foods)
		Daily intakes of fruit and vegetables
		Daily intakes of sodium (all foods)
E	Exercise	Physical activity score
H	Health	Diastolic blood pressure
		Systolic blood pressure
		Total cholesterol to HDL ratio
K	Calorie intake	Total calorie intake
S	Smoking	Daily number of cigarettes
W	Weight	Body Mass Index
		Waist-hip ratio
Y	Wealth	Income
		Social class
		Education

Table 2. WHO recommendations for a healthy diet

NDNS variable	WHO recommendation	
	Lower limit (l_i)	Upper limit (u_i)
% of calorie intake from fats	15%	30%
% of calorie intake from proteins	10%	15%
% of calorie intake from carbohydrates	55%	75%
% of calorie intake from saturated fats	None	10%
% of calorie intake from trans-fats	None	1%
% of calorie intake from sugar	None	10%
Fruit and vegetables intake	400g	None
Sodium intake	None	2g

Figure 1 shows the distribution of diet quality across individuals in the NDNS sample. The distribution has two major peaks, a virtuous one (RCI between 0.90 and 1) and a less healthy one (RCI between 0.70 and 0.80).

Exercise. Measurement of exercise is based on the aggregate NDNS physical activity score, which weights daily activities according to their intensity (sleep, light, moderate, vigorous) and includes both leisure and work physical activity as they result from the NDNS diary keeping for each individual[3].

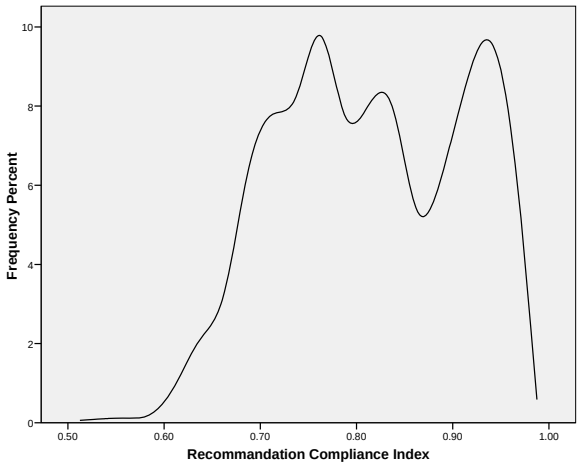

Fig. 1. Distribution of diet quality in the UK

Health. Three health indicators are taken and they are derived from the medical tests for the NDNS individuals (which involves blood and urine samples): a) diastolic blood pressure; b) systolic blood pressure; c) total cholesterol to HDL cholesterol ratio. High values of these indicators are health risk factors, especially for cardiovascular diseases.

Calorie intake. Calorie intake is a derived variable provided by the NDNS survey, based on the food items recorded in the diary kept by the surveyed individuals, then translated into calories using Atwater conversion factors.

Smoking. The average number of cigarette smoked per day is recorded in the NDNS and is obtained by averaging week-days and week-ends figures.

Weight. Two indicators of weight are provided in the NDNS data set. One is the usual body mass index, which is defined as weight in kilograms divided by height in metres squared. A person is overweight if their BMI is between 25 and 30, obese if BMI exceeds 30. Normal weight is usually defined as being in the BMI range 18.5 to 25 with BMI below 18.5 being underweight. An alternative measure of body weight is the waist-hip ratio (WHR), i.e. the ratio of the

[3] See NDNS Appendix 1, www.food.gov.uk/multimedia/pdfs/ndnsappendixi01

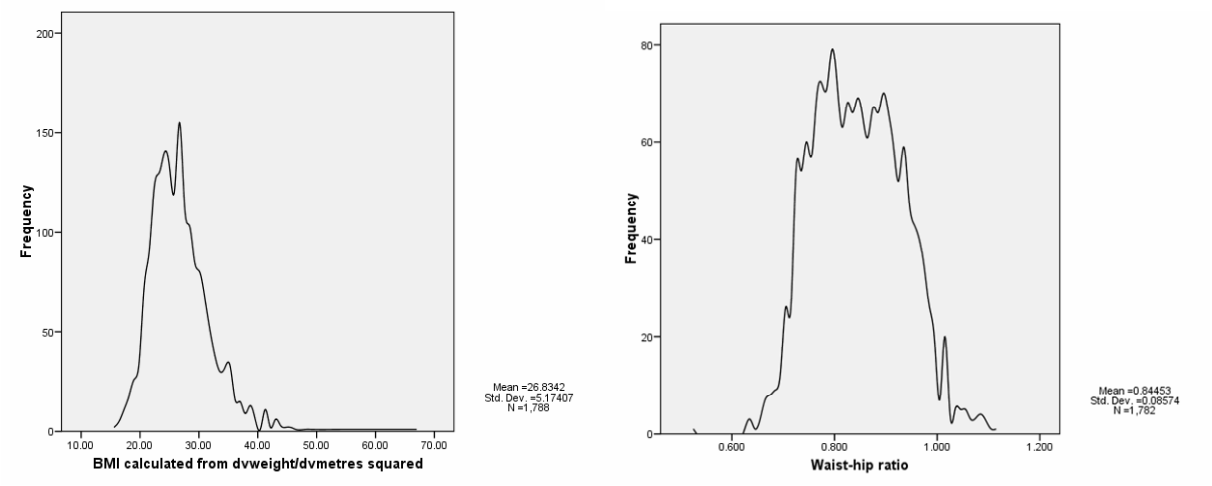

Fig. 2. BMI versus WHR distribution in the NDNS sample

circumference of the waist to that of the hips. Recent epidemiological research has shown the failure of BMI in predicting health outcomes, especially cardio-vascular diseases, suggesting that WHR is a better predictor of health [19]. The two distributions over the sample are shown in Figure 2.

Wealth. The NDNS records a set of socio-demographic variables and is subject to the usual problems of potential response biases income surveys. Thus, two additional variables besides gross annual income of the individual are considered here, that is social class according to the UK classification scheme (i.e. manual and non-manual workers, each subdivided in three levels) and education of the respondent.

IV. STRUCTURAL EQUATION MODEL

The model in (5) is clearly unidentified, as all variables but wealth are endogenous. Some simplification is necessary, but the SEM approach (see Hair et al. [20], chapter 11) has two clear advantages:

a) We can adopt a competing model strategy, where correlation and causation links can be included/deleted according to goodness-of-fit indicators
b) The variables for weight (body mass index, waist-hip ratio), wealth (income, education and social class) and health (systolic and diastolic blood pressure and the cholesterol ratio) can be seen as the manifest indicators of unmeasured latent variables, exploiting the measurement model part of the SEM approach (see figure 3).

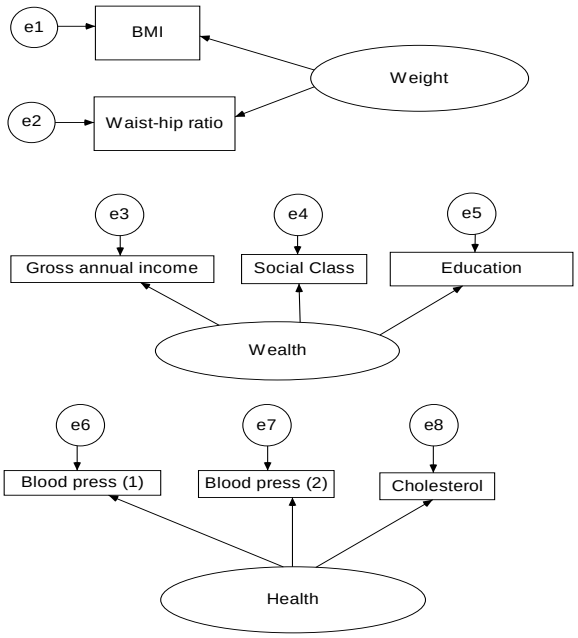

Fig. 3. Measurement models

The structural model is shown in Figure 4 and includes all of the relationships assumed in model (5), although some of the relationships are expressed as correlations rather than bidirectional causations.

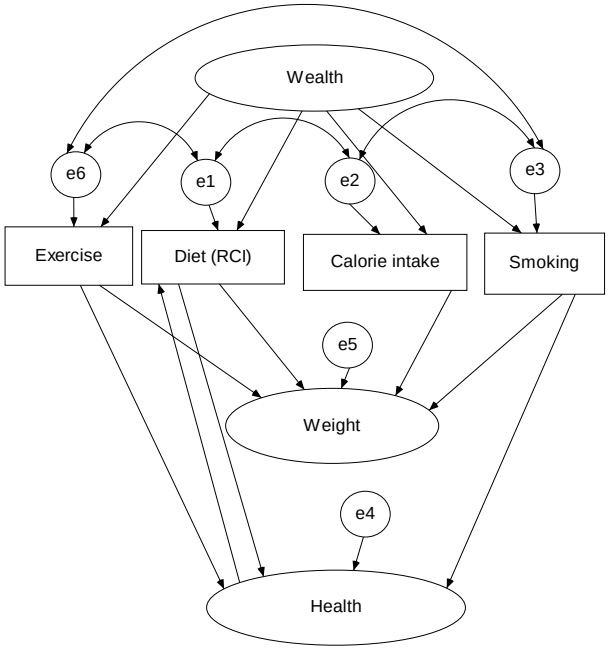

Fig. 4. Structural model

V. RESULTS

Maximum likelihood estimates of the model made by the measurement component of figure 3 and the structural component of figure 4 were obtained using AMOS 7.0. Among these relationships, only two emerged as non-significant at the 95% confidence level, the casual link between smoking and calorie intake and the correlation between exercise and smoking. These were removed from the model, which resulted in improved goodness-of-fit. Model evaluation is based on a selection of goodness-of-fit indicators, whose values are summarized in table 3.

Diagnostics are not excellent, but more than acceptable compared to the standards in published literature. The minimum sample discrepancy (CMIN) simply tests whether the model perfectly fits the data (very unlikely and not really useful as a test). When this measure is divided by the degree of freedom (CMIN/DF), one obtains a chi-square statistic, which

Table 3. Goodness-of-fit diagnostics

Parameters	47
Degrees of freedom	43
CMIN/DF	11.44
NFI	0.89
RFI	0.79
IFI	0.90
TLI	0.81
CFI	0.89
RMSEA	0.068
Hoelter .05	272
Hoelter .01	309

for optimal models is below 5. Other indices are the normed fit index (NFI), the relative fit index (RFI), the incremental fit index (IFI), the Tucker-Lewis coefficient (TLI) and the comparative fit index (CFI). In good models, these should be above 0.90, while the root mean standard error of approximation (RMSEA) is below 0.08 for acceptable models and less than 0.05 for a very good model. The hypothesis that RMSEA<0.05 is tested through the PCLOSE test.

Finally, the Hoelter's critical N shows the largest sample size necessary to accept the model and it is a useful complement to the Chi-square test, which tends to reject the model when the sample size is large. Better models require larger sample sizes to be rejected and generally one would expect a critical N of at least 200 for a good model.

Parameter estimates (table 4), including correlations and variances, are all significant at the 99% levels and the standardized regression weights and correlations are reported in Table 4. Most of the signs are as expected, but some of the results are striking. First, looking at measurement models the waist-hip ratio loads more on the latent weight variable than BMI, confirming previous results that within a nutrition-health model, the former is a more important predictor of the health outcome, where the latter is mainly determined by blood pressure measures compared to the cholesterol indicator.

Second, wealth expressed as a combination of income, education and social class plays a major role in the model, but after accounting for endogeneity some of the results contrast with previous findings. For example, wealth does not lead to more exercise. This is easily explained when one considers that the NDNS activity scores accounts for all activities, aggregating leisure and work exercise. Previous

studies have claimed that the rise in obesity rates is related with the decline of manual work and our results seem to confirm the trade off between higher income (positively associated with leisure exercise) and the amount of work-related physical activity.

While higher wealth in itself determines a higher demand for calories, it is also associated with less smoking and a healthier diet as measured by the RCI. In turn, a higher attention to the diet reduces the calorie intake and the standardized effect. Thus, the overall effect in terms of calorie intake depends on whether the positive effect of wealth on diet quality is larger than the negative effect of increased calorie intake. With some simple multiplication, the overall effect of wealth on calories through diet quality is -1.51 × 0.47 = -0.71, while the direct impact of wealth on calorie intake is +0.73. It would seem that wealth improvements are associated with slightly higher calorie intakes (+0.02) and better diets (-1.51). Let us consider the final effect on weight of a wealth improvement.

Reduced activity determines a weight increase (-.11 × -0.44 = +0.05). The small increase in calorie also determines a weight increase (0.02 × 1.76 = +0.04), but these are largely offset by the improved diet quality (-1.51 × -1.01 = - 1.53). Thus while wealth improvement are associated with lower weight as observed in most developed countries, these results show that the main determinant is the improvement of diet quality.

Finally, it is possible to evaluate the overall effect on health of the various components.

As expected, diet quality and exercise have a direct positive effect on health, as the negative sign suggests a reduction in blood pressure and cholesterol. The impact of is slightly higher for exercise compared to diet quality. Weight, on the other hand, has a very strong negative influence on health. These results suggest that even after accounting for the influence of bad diet and little exercise on health, excess weight has direct unhealthy consequences, which conflicts with the thesis by Oliver (2006) mentioned in the introduction to this paper. One effect stands out as implausible, the fact that smoking seems to have a positive effect on health. However, further investigations have shown that the sign of the smoking-health relationship is quite unstable, and turns to positive and non-significant when smoking is treated as exogenous or some correlations are omitted. This suggests that the smoking component of the model should be taken with great care, while all other signs and magnitudes emerges as very robust and do not change with simplified models.

Table 4. Standardised regression weights

Relationship			Estimate
Wealth measurement model			
educ	<---	wealth	.702
income	<---	wealth	.574
classresp	<---	wealth	.734
Health measurement model			
bloodpress2	<---	health	.814
bloodpress1	<---	health	.854
cholesterol	<---	health	.252
Weight measurement model			
bmi	<---	weight	.452
whratio	<---	weight	.869
Structural model			
exercise	<---	wealth	-.113
smoking	<---	wealth	-.261
calories	<---	wealth	.727
diet	<---	wealth	.466
health	<---	exercise	-.275
weight	<---	exercise	-.444
health	<---	smoking	-.446
health	<---	weight	1.761
weight	<---	calories	1.756
diet	<---	health	2.020
weight	<---	diet	-1.007
calories	<---	diet	-1.511
health	<---	diet	-.194
Correlations			
calories	<-->	exercise	.107
smoking	<-->	diet	-.238
calories	<-->	diet	.227

VI. CONCLUSIONS

Based on a household health production framework, this paper exploits the combination of socio-economic, health and nutrition information from the UK National Diet and Nutrition Survey to analyze the endogenous relationship among wealth, nutrition, weight and the final health outcomes. Using structural equation modelling, we reach three main conclusions: (1) higher wealth is associated with lower weight and

better health as expected, but because of a better diet rather than extra exercise or lower calorie consumption; (2) while reduced exercise and unhealthy diets have a direct negative effect on health, this does not rule out an additional adverse health outcome associated with larger weight; (3) the waist-hip ratio is a better predictor of health outcomes than body-mass index. The study has also limitations and model specification can be further improved by including prices and other behavioural determinants. However, there is a lack of such a comprehensive data-set.

REFERENCES

1. Lang T, Rayner G (2005) Obesity: a growing issue for European policy? Journal of European Social Policy 15: 301-327
2. WHO (2004) Food and health in Europe: a new basis for action basis. WHO Regional Publications European Series 96. WHO Europe, Copenhagen, DK.
3. Yach D, Stuckler D, Brownell KD (2006) Epidemiologic and economic consequences of the global epidemics of obesity and diabetes. Nature Medicine 12: 62-66
4. Mazzocchi M, Traill WB (2005) Nutrition, health and economic policies in Europe. Food Economics-Acta Agriculturae Scandinavica, Section C 2: 138-149
5. Seiders K, Petty RD (2004) Obesity and the role of food marketing: A policy analysis of issues and remedies. Journal of Public Policy & Marketing 23: 153-169
6. Frederick S, Loewenstein G, O'Donoghue T (2002) Time discounting and time preference: A critical review. Journal of Economic Literature 40: 351-401
7. Oliver JE (2006) Fat Politics: The Real Story Behind America's Obesity Epidemic. Oxford University Press, New York (NY), USA
8. Knai C, Suhrcke M, Lobstein T (2007) Obesity in Eastern Europe: An overview of its health and economic implications. Economics & Human Biology 5: 392-408
9. Shogren JF (2005) Economics of diet and health: Research challenges. Food Economics - Acta Agriculturae Scandinavica Section C 2: 117-127
10. Smith TG (2004) The McDonald's equilibrium - Advertising, empty calories, and the endogenous determination of dietary preferences. Social Choice and Welfare 23: 383-413
11. Chen SN, Shogren JF, Orazem PF, Crocker TD (2002) Prices and health: Identifying the effects of nutrition, exercise, and medication choices on blood pressure. American Journal of Agricultural Economics 84: 990-1002
12. Chou S, Grossman M, Saffer H (2002) An Economic Analysis of Adult Obesity: Results from the Behavioral Risk Factor Surveillance System. NBER Working Paper 9247. National Bureau of Economic Research, Cambridge (MA), USA.
13. Cutler DM, Glaeser EL, Shapiro JM (2003) Why have Americans become more obese? Journal of Economic Perspectives 17: 93-118
14. Lakdawalla D., Philipson, TJ (2002) The Growth of Obesity and Technological Change: A Theoretical and Empirical Examination. NBER Working Paper 8946. National Bureau of Economic Research, Cambridge (MA), USA.
15. Philipson TJ, Posner RA (1999) The long-run growth in obesity as a function of technological change. Perspectives in Biology and Medicine 46: S87-S107
16. Swan G (2004) Findings from the latest National Diet and Nutrition Survey. Proceedings of the Nutrition Society 63: 505-512
17. WHO (2003) Diet, Nutrition and the Prevention of Chronic Diseases. WHO Technical Report Series 916. World Health Organization, Geneva, SWI
18. Mazzocchi M, Brasili C, Sandri E (2008) Trends in dietary patterns and compliance with World Health Organization recommendations: a cross-country analysis. Public Health Nutrition 11: 535-540.
19. Kragelund C, Omland T (2005) A farewell to body-mass index? Lancet 366: 1589-1591
20. Hair JF Jr, Anderson RE, Tatham RL, Black WC (1998) Multivariate Data Analysis. Prentice-Hall, Upper Saddle River (NJ), USA