Dynamics of obesity in Finland

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Abstract

The purpose of this research is to study empirically the dynamics of obesity in Finland and provide empirical evidence of temporal causality between obesity, health expenditure, unemployment, urbanization, alcohol consumption and calorie intake. The paper employs bounds testing cointegration procedure and augmented causality tests. The empirical results suggest the existence of cointegration amongst the variables. Augmented Granger causality tests indicate the existence of a long-run causality as well as three different pairs of short-run causalities. The study draws some important policy recommendations.

Keywords: obesity, cointegration, causality, time series, Finland
JEL Classifications: C22, I1, H51

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1. Introduction

Obesity rates in Finland have more than doubled in the past three decades. Health Statistics of OECD (2010) show that the percentage of self reported adult obese people rose substantially from 6.6% in 1978 to 15.9% in 2009. In regards to comparable obesity data provided by Health Statistics of OECD (2010), Finland ranked 17th out of 30 OECD countries with 14.3% in 2005. In the same period, Japan had the lowest estimate of obesity (3.9%) whereas the highest rate was recorded in the USA (34.3%). Koskinen et al. (2006) states that in the early 2000s one in five adults in Finland were obese. Lahti-Koski (2001) estimates the trends and determinants of adult obesity in Finland using the survey data running from 1972 to 1997. The study of Lahti-Koski associates obesity to the several socio-economic factors such gender, ethnicity, marital status, dietary intake, physical activity, education, alcohol consumption and smoking. The results conclude that the mean BMI increased for both gender during the estimation period.

Obesity has been linked to increased incidence of several chronic diseases, like diabetes and heart disease, and to lower life expectancy. A study of World Health Organization (WHO) in 1997 classified obesity as a global epidemic. WHO (2011) estimates that more than 1.5 billion adults, aged 20 and older, were overweight in 2008. According to the WHO (2011), overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health. Body mass index (BMI) is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. It is defined as a person's weight in kilograms divided by the square of his height in meters ($\text{kg/m}^2$). A BMI value of between 20 and 22 is considered to be ideal for adults regardless of gender in the sense that mortality and morbidity risks are minimized in this range. Adults with BMI over 25 are overweight and
adults with BMI over 30 are obese. Worldwide obesity has more than doubled since 1980. In 2008, more than one in ten of the world’s adult population was obese.

Bhattacharya and Sood (2011) discuss that obesity is a complex social problem, interlinked with a variety of issues, including health care research and development, prices for food and exercise, agricultural sports, social security, peer effects. The prevalence of obesity is the result of several socio-economic changes that have altered the lifestyle choices of people. The WHO (2011) reports further that the fundamental cause of obesity and overweight is an energy imbalance between calories consumed and calories expended. Overweight and obesity are the fifth leading risk for global deaths. At least 2.8 million adults die each year as a result of being overweight or obese. In addition, 44% of the diabetes burden, 23% of the ischaemic heart disease burden and between 7% and 41% of certain cancer burdens are attributable to overweight and obesity. The report of the WHO (2002) reveals that rising BMI steadily increases the risks of type 2 diabetes, hypertension, cardiovascular disease, and some cancers. Overweight and obesity, as well as their related noncommunicable diseases, are largely preventable. Supportive environments and communities are fundamental in shaping people’s choices, making the healthier choice of foods and regular physical activity the easiest choice, and therefore preventing obesity. Finkelstein et al. (2005) points out that obesity is not only a health but also an economic phenomenon, and it entails important economic costs.

Preventing the obesity has become an increasingly urgent public health priority for national government but the effectiveness of the policies designed for preventing the obesity is not yet completely clear. The extremely high costs of obesity make it an important subject for economists and other social scientists. The epidemic of obesity absorbs increasingly greater healthcare budgets and attracts increasing concern for governments. Finkelstein et al. (2003) reports that obesity accounts for between 2% and 9% of the total health budgets in high-income countries. The true costs are greater, as not all obesity related conditions are included
in these calculations. Brunello et al. (2008) points out that obesity may have other negative economic consequences including work absenteeism, higher unemployment and disability payments, earlier retirement and lower wages in the case of European and the USA data. Fry and Finley (2005) reveals the combined direct and indirect costs of obesity in 2002 were estimated as €33 billion/year for all members of European Union. A most recent study carried out by Wang et al. (2011) suggests that the combined medical costs associated with obesity are estimated to increase by $48-66 billion/year in the USA and by £ 1.9-2 billion/year in the UK by 2030. Hence, effective policies to promote healthier weight also have economic benefits.

The empirical studies aiming at determining the causes of obesity is limited and they are based on survey, cross-section or panel data. As far as this study is concerned, there exists no previous study that estimates empirically the determinants of obesity on the basis of time series data and cointegration framework. The obesity issue is still open to debate and further research. Thus, this paper aims at contributing to the literature using a time series approach to identify the causes of obesity in Finland.

The remainder of this paper is organized as follows. Section 2 presents a brief literature review. Section 3 describes the study’s model and methodology. Section 4 discusses the empirical results, and finally Section 5 concludes.

2. A brief literature review

Obesity is a complex phenomenon but economics is at the heart of the obesity epidemic. The literature on obesity is large and covers many discipline, but there is little agreement about causes. The fundamental reason for being overweight is basically related to overeating, in
In other words, calorie intake exceeds calorie consumption. The literature identifies several socio-economic factors for a positive balance of calories. Finkelstein and Strombotne (2010) point out that economic forces have made it easier and cheaper to consume high-energy, tasty, affordable foods and have allowed us to be increasingly sedentary at work, at home, and in between. Moreover, medical advances have lowered the health costs that result from excess weight and have decreased the motivation to diet and exercise. As discussed in Popkin (1999), rising urbanization is associated with increased opportunities for eating and reduced opportunities for physical activity. Moreover, people in rural areas typically have higher levels of physical activity due to the focus on agricultural work. Phillipson (2001) identifies technological change another responsible factor for the obesity epidemic largely because of its effect reducing energy expenditure in the workplace. Sedentary technological change has lowered both the real price of food and physical expenditures of calories per hour worked in both market and household production. As a result, the cost of consuming calories decreased and the cost of expending calories increased, thus contributing to rise in obesity in two ways.

Unemployment is another contributor to the obesity epidemic as people are out of job, the energy expenditure or the amount of physical activity is substantially reduced leading to a positive balance of calories. The increase in obesity has given rise to demands for public intervention, mainly in the form of education and health programs, to reduce obesity through better diet and more exercise. It is not plausible to assume that individuals would make rational choices in the free market economy against the costs of obesity since the market forces promoting food consumption are extremely powerful to influence an individual’s decision adversely. Consequently, rising public health expenditures decreases significantly the risk of obesity and this reduction bring about more economic benefits in terms of higher level labour force participation and increase in income taxes, decrease in absenteeism, etc., in the long-run.
The phenomenon of obesity has been examined mainly from medical aspects. However, economic considerations of the causes and effects of obesity have proliferated in the last decade. Examples of these studies as follows: Phillipson and Posner (1999), Phillipson (2001), Cutler et al. (2003), Chou et al. (2004), Heineck (2006), Lakdawalla et al. (2005), Knai et al. (2007), Johansson et al. (2009), Lindeboom et al. (2010), and Andreyeva et al. (2011). Rosin (2008) provides an extensive survey on the economic causes of obesity. Similarly, Ball and Crawford (2005) surveys the socioeconomic aspects of obesity in adults. The economic literature has mainly focused on the factors that contribute to obesity in order to address the questions why people overeat, and what has upset the balance between energy intake and energy expenditures in the last three decades. The consequences of obesity and different strategies to alleviate the global increase in obesity rates are also examined in the literature. Rosin (2008) systematically identifies the empirical models and research that deal with possible contributors of obesity. There are basically three major factors: biological (for example, genetic susceptibility), behavioural (for example, addiction such eating or drinking alcohol) and environmental (for example, urbanization and technological change).

The empirical studies on obesity and overweight are mainly based on survey, household and cross-section rather than time series data. Examples of empirical studies include Chou et al. (2004) for the USA; Loureiro and Nayga (2004) for 32 OECD countries; Andreyeva et al. (2007) for 11 European countries; Brown and Siahpush (2007) for Australia; Huffman and Rizov (2007) for Russia; Bleich et al. (2008) for 7 OECD countries including Finland; Maenning et al. (2008) for Germany; Pieroni and Salmasi (2010) for the UK; Offer et al. (2010) for 11 European countries including Finland. The previous empirical studies relate obesity to a wide range of socio-economic variables ranging from calorie intake to urbanization but the results are not conclusive and comparable due to different data.
frequencies and estimation procedures. The results vary due to the selection of variables and data frequency.

3. Model and econometric methodology

Following the empirical literature on obesity, this study adopts the following long-run relationship between obesity, public health expenditures, unemployment, urbanization, alcohol consumption and calorie intake in double linear logarithmic form as:

\[ o_t = \alpha_0 + \alpha_1 h_t + \alpha_2 u_t + \alpha_3 b_t + \alpha_4 a_t + \alpha_5 c_t + \varepsilon_t, \]  

(1)

where \( o_t \) is the obesity rate; \( h_t \) is the real public health expenditures per capita; \( u_t \) is the unemployment rate; \( b_t \) is the urbanization rate; \( a_t \) is the alcohol consumption per capita litre, \( c_t \) is the calorie intake, and \( \varepsilon_t \) is the classical error term. Considering the empirical literature survey on obesity, the sign expectations for the parameters in equation (1) are as follows: \( \alpha_1 < 0, \alpha_2 > 0, \alpha_3 > 0, \alpha_4 > 0, \text{ and } \alpha_5 > 0. \)

Recent advances in econometric literature dictate that the long-run relation in equation (1) should incorporate the short-run dynamic adjustment process. It is possible to achieve this aim by expressing equation (1) in an error-correction model as suggested in Pesaran et al. (2001).

\[ \Delta o_t = \beta_0 + \sum_{i=1}^n \beta_{1i} \Delta o_{t-i} + \sum_{i=0}^n \beta_{2i} \Delta h_{t-i} + \sum_{i=0}^n \beta_{3i} \Delta u_{t-i} + \sum_{i=0}^n \beta_{4i} \Delta b_{t-i} + \sum_{i=0}^n \beta_{5i} \Delta a_{t-i} + \sum_{i=0}^n \beta_{6i} \Delta c_{t-i} + \sum_{i=0}^n \beta_{7i} \Delta \varepsilon_{t-i} + \sum_{i=0}^n \beta_{8i} \Delta \varepsilon_{t-i} + \sum_{i=0}^n \beta_{9i} \Delta \varepsilon_{t-i} \]  

(2)

This approach, also known as autoregressive-distributed lag (ARDL)\(^1\), provides the short-run and long-run estimates simultaneously. Short-run effects are reflected by the estimates of the

\(^1\) The ARDL approach to cointegration is extensively applied in empirical research, see for example: Halicioglu (2012), Andres, Halicioglu and Yamamura (2011), Andres and Halicioglu (2010), Altinanahtar and Halicioglu (2009), Halicioglu (2008, 2007), and Halicioglu and Ugur (2005).
coefficients attached to all first-differenced variables. The long-run effects of the explanatory variables on the dependent variable are obtained by the estimates of $\beta_7$ to $\beta_{12}$ that are normalized on $\beta_7$. The inclusion of the lagged-level variables in equation (2) is verified through the bounds testing procedure, which is based on the Fisher (F) or Wald (W)-statistics. This procedure is considered as the pre-testing stage of the ARDL cointegration method. Accordingly, a joint significance test that implies no cointegration hypothesis, ($H_0$: all $\beta_i$ to $\beta_{12} = 0$), against the alternative hypothesis, ($H_1$: at least one $\beta_i$ to $\beta_{12} \neq 0$) should be performed for equation (2). The F/W test used for this procedure has a non-standard distribution. Thus, Pesaran et al. (2001) compute two sets of critical values for a given significance level with and without a time trend. One set assumes that all variables are $I(0)$ and the other set assumes they are all $I(1)$. If the computed F/W-statistic exceeds the upper critical bounds value, then the $H_0$ is rejected, implying cointegration. In order to determine whether the adjustment of variables is toward their long-run equilibrium values, estimates of $\beta_7$ to $\beta_{12}$ are used to construct an error-correction term (EC). Then lagged-level variables in equation (2) are replaced by EC$_{t-1}$ forming a modified version of equation (2) as follows:

$$\Delta o_t = \beta_0 + \sum_{i=1}^{p} \beta_{1i} \Delta o_{t-i} + \sum_{i=0}^{p} \beta_{2i} \Delta u_{t-i} + \sum_{i=0}^{p} \beta_{3i} \Delta h_{t-i} + \sum_{i=0}^{p} \beta_{4i} \Delta \lambda_{t-i} + \sum_{i=0}^{p} \beta_{5i} \Delta a_{t-i} + \sum_{i=0}^{p} \beta_{6i} \Delta c_{t-i} + \lambda EC_{t-1} + \sigma_t$$ (3)

Equation (3) is re-estimated one more time using the same lags previously. A negative and statistically significant estimation of $\lambda$ not only represents the speed of adjustment but also provides an alternative means of supporting cointegration between the variables. Pesaran et al. (2001) cointegration approach has some methodological advantages in comparison to other single cointegration procedures. Reasons for the ARDL are: i) endogeneity problems and inability to test hypotheses on the estimated coefficients in the long-run associated with the
Engle-Granger (1987) method are avoided, all variables are considered to be potentially endogenous; ii) the long and short-run coefficients of the model in question are estimated simultaneously; iii) the ARDL approach to testing for the existence of a long-run relationship between the variables in levels is applicable irrespective of whether the underlying regressors are purely stationary $I(0)$, purely non-stationary $I(1)$, or mutually cointegrated; iv) the small sample properties of the bounds testing approach are far superior to that of multivariate cointegration, as argued in Narayan (2005).

The Granger representation theorem suggests that there will be Granger causality in at least one direction if there exists a cointegration relationship among the variables in equation (1), providing that they are $I(1)$. Engle and Granger (1987) cautions that the Granger causality test, which is conducted in the first-differenced variables by means of a VAR, will be misleading in the presence of cointegration. Therefore, an inclusion of an additional variable to the VAR system, such as the error correction term would help us to capture the long-run relationship. To this end, an augmented form of the Granger causality test involving the error correction term is formulated in a multivariate $p$th order vector error correction model.

$$
(1-L) \begin{bmatrix}
  o_t \\
  h_t \\
  b_t \\
  a_t \\
  c_t
\end{bmatrix} + \sum_{i=1}^{r} (1-L) \begin{bmatrix}
  \phi_{1i} & \ldots & \phi_{5i} \\
  \phi_{1i} & \ldots & \phi_{5i} \\
  \phi_{3i} & \ldots & \phi_{5i} \\
  \phi_{4i} & \ldots & \phi_{5i} \\
  \phi_{5i} & \ldots & \phi_{5i}
\end{bmatrix} \begin{bmatrix}
  a_{t-i} \\
  h_{t-i} \\
  b_{t-i} \\
  a_{t-i} \\
  c_{t-i}
\end{bmatrix} + \begin{bmatrix}
  \delta_{1t} \\
  \delta_{2t} \\
  \delta_{3t} \\
  \delta_{4t} \\
  \delta_{5t} \\
  \delta_{6t}
\end{bmatrix} + \begin{bmatrix}
  \omega_{1t} \\
  \omega_{2t} \\
  \omega_{3t} \\
  \omega_{4t} \\
  \omega_{5t} \\
  \omega_{6t}
\end{bmatrix} (4)
$$

$L$ is the lag operator. $EC_{t-1}$ is the lagged error correction term, which is obtained from the long-run relationship described in equation (1), and it is not included in equation (4) if one finds no cointegration amongst the vector in question. The Granger causality test may be applied to equation (4) as follows: i) by checking statistical significance of the lagged differences of the variables for each vector; this is a measure of short-run causality; and ii) by
examining statistical significance of the error-correction term for the vector that there exists a long-run relationship. As a passing note, one should reveal that equation (3) and (4) do not represent competing error-correction models because equation (3) may result in different lag structures on each regressors at the actual estimation stage; see Pesaran et al. (2001) for details and its mathematical derivation. All error-correction vectors in equation (4) are estimated with the same lag structure that is determined in unrestricted VAR framework.

4. Results

Annual data over the period 1978-2007 were used to estimate equation (2) and (3) by the ARDL cointegration procedure of Pesaran et al. (2001). Variable definition and sources of data are cited in the Appendix.

To implement the Pesaran et al. (2001) procedure, one has to ensure that none of the explanatory variables in equation (1) is above I(1). In the presence of I(2) series, the critical values computed by the Pesaran et al. (2001) cointegration procedure are not valid. Three tests were used to test unit roots in the variables: Augmented Dickey-Fuller (henceforth, ADF) (1979, 1981), Phillips-Perron (henceforth, PP) (1988), and Elliott-Rothenberg-Stock (henceforth, ERS) (1996). Unit root tests results are displayed in Table 1. Table 1 illustrates that none of the variables included in equation (1) are beyond I(1). Consequently, the results warrant implementing the Pesaran et al. (2001) procedure. Visual inspections of the variables in logarithm show no structural breaks.
Table 1. Unit root results.

<table>
<thead>
<tr>
<th>Variables</th>
<th>ADF</th>
<th>PP</th>
<th>ERS</th>
</tr>
</thead>
<tbody>
<tr>
<td>( o_t )</td>
<td>2.85</td>
<td>2.56</td>
<td>2.92</td>
</tr>
<tr>
<td>( h_t )</td>
<td>3.52</td>
<td>2.43</td>
<td>2.06</td>
</tr>
<tr>
<td>( u_t )</td>
<td>3.08</td>
<td>2.25</td>
<td>3.25</td>
</tr>
<tr>
<td>( b_t )</td>
<td>2.55</td>
<td>2.33</td>
<td>2.41</td>
</tr>
<tr>
<td>( a_t )</td>
<td>2.22</td>
<td>1.86</td>
<td>2.79</td>
</tr>
<tr>
<td>( c_t )</td>
<td>3.14</td>
<td>3.24</td>
<td>2.53</td>
</tr>
<tr>
<td>( \Delta o_t )</td>
<td>4.03*</td>
<td>6.57*</td>
<td>3.82*</td>
</tr>
<tr>
<td>( \Delta h_t )</td>
<td>5.30*</td>
<td>3.90*</td>
<td>2.74*</td>
</tr>
<tr>
<td>( \Delta u_t )</td>
<td>3.84*</td>
<td>3.23*</td>
<td>3.46*</td>
</tr>
<tr>
<td>( \Delta b_t )</td>
<td>3.67*</td>
<td>3.63*</td>
<td>3.35*</td>
</tr>
<tr>
<td>( \Delta a_t )</td>
<td>2.91*</td>
<td>4.20*</td>
<td>2.96*</td>
</tr>
<tr>
<td>( \Delta c_t )</td>
<td>4.12*</td>
<td>6.57*</td>
<td>3.48*</td>
</tr>
</tbody>
</table>

Notes: The sample level unit root regressions include a constant and a trend. The differenced level unit root regressions are with a constant and without a trend. All test statistics are expressed in absolute terms for convenience. Rejection of unit root hypothesis is indicated with an asterisk. \( \Delta \) stands for first difference.

In order to test the existence of a long-run cointegrating relationship amongst the variables of equation (1), a two-step procedure to estimate the ARDL representation model was carried out. First, the selection of the optimal lag length on the first-differenced variables in equation (2), unrestricted Vector Auto Regression (VAR) was employed by means of Akaike Information criterion. The results suggest the optimal lag length as 2, but this stage of the results is not presented here to conserve space. Second, a bound F/W-test was applied to equation (2) in order to determine whether the dependent and independent variables are cointegrated. The results of the bounds F/W-testing are reported in Table 2. It can be seen from Table 2 that the computed F/W statistics are above the upper bound values thus implying cointegration relation.
Table 2. The results of F and W tests for cointegration.

<table>
<thead>
<tr>
<th>The assumed long-run relationship: $F/W(h,u,b,a,c)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>F-statistic</td>
</tr>
<tr>
<td>5.6270</td>
</tr>
<tr>
<td>W-statistic</td>
</tr>
</tbody>
</table>

If the test statistic lies between the bounds, the test is inconclusive. If it is above the upper bound (UB), the null hypothesis of no level effect is rejected. If it is below the lower bound (LB), the null hypothesis of no level effect cannot be rejected.

The ARDL cointegration equation was estimated to obtain the long-run and short-run coefficients simultaneously. The results are presented in Table 3. The long results are displayed in Panel A of Table 3. The error correction representation from the estimation of equation (3) is revealed in Panel B of Table 3. The overall regression diagnostics reported in Panel C of Table 3 illustrates that the econometric results are satisfactory to infer from them. Amongst explanatory variables, the urbanization rate seems to be highest impact on the obesity rate indicating that a 1% rise in urbanization rate increases the obesity rate by 4.61% in the long-run. The policy implication of this result suggest that obesity is a largely associated with urbanization. In the urban areas, there are a wide range of convenience and fast-food shops with low prices and high energy density. People tend to work in less physically demanded jobs in the urban areas. Thus, the combination of these adverse affects is substantial on obesity. This finding is consistent with the study of Loureiro and Nagya (2004), which suggests empirically that the urbanization is the major factor in rising obesity in the OECD countries including Finland. The elasticity of obesity rate with respect to alcohol consumption is 3.19 implying that a 1% increase in alcohol consumption rises the obesity rate by 3.19%. Alcohol is a significant source of calories and drinking may stimulate eating, particularly in social settings. Rohrer et al. (2005) reveals that in comparison to non-drinkers, people who consumed alcohol 3 or more days per month had a lower odds of being obese. As for the elasticity of the obesity with respect to per capita real health expenditures, it is
estimated as -1.40 showing that a 1% rise in the per capita real health expenditures reduces the obesity rate by 1.40%. The public health expenditures aims at preventing obesity should make it easier and cheaper to engage in a healthy diet and regular physical activity. These policy measurements may be complemented appropriate public training and facilities on obesity. Even though the calorie intake variable is statistically not significant, it carries the expected sign. However, there are ample empirical evidence that calorie intake is a significant contributor to obesity, see for example Rosin (2008). Finally, the impact of unemployment rate on the obesity rate is seems to be relatively small in comparison to other explanatory variables since 1% increase in unemployment rate results in only 0.38% rise in the obesity rate. This finding confirms the study of Laitinen et al. (2002) demonstrating that a long history of unemployment is associated with increased risk of obesity amongst the young Finnish adults. The speed of adjustment parameter is – 0.86, indicating that when the aggregate obesity equation is above or below its equilibrium level, it adjusts by 86% within the first year. The full convergence to its equilibrium level takes a little more than one year.
Table 3. ARDL cointegration results.

### Panel A. Long-run results.

<table>
<thead>
<tr>
<th>Dependent variable o₁</th>
<th>Regressor</th>
<th>Coefficient</th>
<th>T-ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>h₁</td>
<td>-1.4061∗∗</td>
<td>-2.6773</td>
<td></td>
</tr>
<tr>
<td>u₁</td>
<td>0.3805∗</td>
<td>3.8476</td>
<td></td>
</tr>
<tr>
<td>b₁</td>
<td>4.6163∗</td>
<td>5.0502</td>
<td></td>
</tr>
<tr>
<td>a₁</td>
<td>3.1947∗</td>
<td>3.9766</td>
<td></td>
</tr>
<tr>
<td>c₁</td>
<td>0.7310</td>
<td>1.1836</td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>-22.848∗</td>
<td>5.5985</td>
<td></td>
</tr>
</tbody>
</table>

### Panel B. Error correction representation results.

<table>
<thead>
<tr>
<th>Dependent variable Δo₁</th>
<th>Regressor</th>
<th>Coefficient</th>
<th>T-ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δh₁</td>
<td>0.4987∗</td>
<td></td>
<td>3.1960</td>
</tr>
<tr>
<td>Δu₁</td>
<td>0.8167</td>
<td>0.5306</td>
<td></td>
</tr>
<tr>
<td>Δb₁</td>
<td>27.185∗</td>
<td>2.1953</td>
<td></td>
</tr>
<tr>
<td>Δa₁</td>
<td>2.1332∗</td>
<td>3.6830</td>
<td></td>
</tr>
<tr>
<td>Δc₁</td>
<td>-0.4484</td>
<td>0.8643</td>
<td></td>
</tr>
<tr>
<td>EC₁₋₁</td>
<td>-0.8625∗</td>
<td>4.9009</td>
<td></td>
</tr>
</tbody>
</table>

### Panel C. Diagnostic tests.

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Critical Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>R²</td>
<td>0.64</td>
<td></td>
</tr>
<tr>
<td>F-statistic</td>
<td>6.39∗</td>
<td></td>
</tr>
<tr>
<td>χ²(1)</td>
<td>0.16</td>
<td>3.15</td>
</tr>
<tr>
<td>SCχ²</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>DW-statistic</td>
<td>2.05</td>
<td></td>
</tr>
<tr>
<td>χ²(2)</td>
<td>0.96</td>
<td>0.38</td>
</tr>
</tbody>
</table>

T-ratios are in absolute values. χ²(1), χ²(2), χ²(χ²), and χ²(χ²) are Lagrange multiplier statistics for tests of residual correlation, functional form mis-specification, non-normal errors and heteroskedasticity, respectively. These statistics are distributed as Chi-squared variates with degrees of freedom in parentheses. The critical values for χ²(1) = 3.84 and χ²(2) = 5.99 are at 5% significance level.

The results of Granger causality tests presented in Table 4. As it can be seen from Table 4, in the long-run causality runs from public health expenditure, unemployment, urbanization, alcohol consumption, and calorie intake to obesity since the estimated lagged error correction term is statistically significant and negative. Considering the pairs of causalities between obesity and explanatory variables, the results identifies only one bilateral causality relationship between urbanization and unemployment rates. There are also three unilateral causality relationships in the short-run. The first unilateral causality runs from health expenditures to unemployment rate. Unemployment rate seems to be Granger-cause alcohol consumption unilaterally. There is also a unilateral causality running from alcohol consumption to obesity.
Table 4. Results of Granger causality.

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>$\Delta o_t$</th>
<th>$\Delta h_t$</th>
<th>$\Delta u_t$</th>
<th>$\Delta b_t$</th>
<th>$\Delta a_t$</th>
<th>$\Delta c_t$</th>
<th>$EC_{t-1}$</th>
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<td></td>
<td>-</td>
<td>0.14</td>
<td>1.36</td>
<td>1.66</td>
<td>2.42</td>
<td>2.24</td>
<td>-0.79</td>
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<td></td>
<td>(0.86)</td>
<td>(0.28)</td>
<td>(0.22)</td>
<td>(0.10)</td>
<td>(0.13)</td>
<td>(3.32)</td>
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<tr>
<td>$\Delta h_t$</td>
<td>0.16</td>
<td>-</td>
<td>1.64</td>
<td>0.07</td>
<td>0.42</td>
<td>0.04</td>
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<tr>
<td></td>
<td>(0.84)</td>
<td>(0.22)</td>
<td>(0.92)</td>
<td>(0.66)</td>
<td>(0.95)</td>
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<tr>
<td>$\Delta u_t$</td>
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<td>8.66</td>
<td>-</td>
<td>5.54</td>
<td>2.52</td>
<td>1.53</td>
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<tr>
<td></td>
<td>(0.37)</td>
<td>(0.03)</td>
<td>(0.01)</td>
<td>(0.11)</td>
<td>(0.24)</td>
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<tr>
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<td>0.76</td>
<td>5.79</td>
<td>-</td>
<td>0.90</td>
<td>1.22</td>
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<td></td>
<td>(0.22)</td>
<td>(0.48)</td>
<td>(0.01)</td>
<td>(0.42)</td>
<td>(0.32)</td>
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</tr>
<tr>
<td>$\Delta a_t$</td>
<td>2.52</td>
<td>1.54</td>
<td>2.92</td>
<td>0.45</td>
<td>-</td>
<td>1.23</td>
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<td>(0.11)</td>
<td>(0.24)</td>
<td>(0.08)</td>
<td>(0.64)</td>
<td>(0.31)</td>
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<td>$\Delta c_t$</td>
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<td>1.10</td>
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<td>0.75</td>
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<td>(0.14)</td>
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<td>(0.22)</td>
<td>(0.35)</td>
<td>(0.48)</td>
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</table>

Causality inference: $b \leftrightarrow u$, $h \rightarrow u$, $u \rightarrow a$, $a \rightarrow o$.

* and ** indicate 5% and 10% significance levels, respectively. The probability values are in brackets. The optimal lag length is 1 and is based on SBC (Schwarz Bayesian Criterion).

5. Conclusions

This study has attempted to identify the causes of increasing obesity prevalence amongst the Finish adults using time series cointegration procedure. The results illustrate that there exists a long-run association between obesity, public health expenditures, unemployment, urbanization, alcohol consumption and calorie intake. The econometric results reveal that the main factor of the obesity in Finland is urbanization, as it provides an increasing availability of fast-and convenience foods with high-energy density, and the sedentary jobs with little incentive to exercise. Alcohol consumption seems to be another major contributor to obesity whereas the contribution of unemployment to obesity is relatively minimal. The results also demonstrate that the public health expenditures appear to have a substantial impact to alleviate the obesity epidemic. As for the Granger causality tests, there exists a long-run causality running from all of explanatory factors to obesity. In the short-run, there is a unilateral causality from alcohol consumption to obesity which requires a public policy intervention to combat it.
Regarding the social and economic costs of obesity, the government policies should be based on tackling the causes of obesity in Finland. It is unreasonable to expect that people will alter their life style and eating habits easily when so many forces in the social, cultural and physical environment conspire against such change. As argued in Sassi (2010) the fiscal policies designed to combat the obesity epidemic are likely to generate larger savings in health expenditure than cost of delivery. Therefore, a strong and committed public intervention is the paramount in dealing with the obesity epidemic. The treatment of obesity in the short-run is difficult but in the long–run, it can be prevented by designing and implementing appropriate public health and education policies. These policy measurements should include providing living environments that promote physical activity at schools, in the workplace and in other everyday settings, and leisure-time physical activity and healthy diets should be encouraged. Some of these policy measurements may specifically include mandatory nutritional information at fast food restaurants, banning junk food and beverages advertising, communities designed for walking and biking, increased hours for physical education at schools. Regulating the food/beverages consumption on the basis of incentives and taxation with a view to reduce obesity rates require an extensive work and a long-term policy planning and coordination. Consequently, a new organizational board/body may be set up particularly for this purpose to plan and coordinate the fight against the obesity epidemic.
Appendix

Data definition and sources

Data are collected from four different sources, namely: Food Balance Sheets of Food and Agriculture Organization of the United Nations (FAO), Health Statistics (HS) of OECD, Main Economic Indicators (MEI) of OECD and World Development Indicators (WDI) of World Bank.

\(o\) : is the obesity rate in logarithm. It refers to the percentage of adult people with a BMI>30kg/m\(^2\) based on self reports. Source: HS.

\(h\) : is the per capita real public health expenditures as of gross domestic product (GDP) in logarithm. Source: HS.

\(u\) : is the unemployment rate in logarithm. Source: MEI.

\(b\) : is the urbanization rate in logarithm. Source: WDI.

\(a\) : is the alcohol consumption for aged 15 over per litre. Source: HS.

\(c\) : is the daily per capita (kilo) calorie availability in logarithm. Source: FAO.
References


