Macroeconomic Effects of Obesity:
An Application to the UK *

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Abstract

This paper studies the presence and magnitude of externalities associated with obesity. We argue that restricting the analysis of the economic costs on health care spending ignores the effects of the obesity epidemic on net social security benefits caused by the higher level of mortality among obese individuals. In order to estimate the size of this externality, we develop an overlapping generations model with rational choice with respect to food consumption and weight as in Lakdawalla and Philipson (2009), endogenizing life expectancy, labour productivity and health care costs. Our results show that the life-time net contributions of the top 30% of the BMI distribution are negative but quantitatively small, despite the fact that the model generates substantial wealth and income inequality, consistent with the observed socio-economic gradient of obesity (Baum and Ruhm, 2009), which results in lower life-time contributions. Furthermore, we perform two policy experiment (i) eliminating childhood obesity and (ii) eliminating the VAT exemption of food consumption, which result in significant welfare gains, with the former eliminating the obesity externality.

JEL-Classification:E21, H31, H51, I00
Keywords: Overlapping Generations, Obesity, Health spending, Human Capital, Longevity

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1 Introduction

The rising prevalence of obesity during the last decades both in developed and developing countries has raised concern among policy makers and health practitioners. Obesity has direct linkages with several diseases such as Diabetes Mellitus, Cardiovascular Disease and several types of cancer (see inter alia Must et al. (1999), Ng et al. (2014)) and has significant effects of life expectancy (Fontaine et al., 2003). Furthermore, obesity has direct and indirect economic costs which burden the health care systems (see inter alia Allender and Rayner (2007), Wang et al. (2011), Scarborough et al. (2011)) and the individuals themselves through lower wages (Dackehag, Gerdtham and Nordin, 2014) and lost working hours (Trogdon et al., 2008). In an effort to tackle the obesity epidemic, governments have considered or implemented various measures such as labeling of food products (Trogdon et al., 2008) and taxes on ‘unhealthy’ foods or ‘healthy food subsidies’ (see Yaniv, Rosin and Tobol (2009)). However, weight is a matter of personal choice (Philipson and Posner, 2003) and unless overweight and obese individuals do not internalize their choices, government intervention can reduce welfare.

The contribution of this paper is twofold. First, in contrast to the literature we estimate the net fiscal burden of obesity taking into account not only the excess health care spending caused by overweight and obese individuals, but also the social security benefits and the life-time contributions through taxation. This is crucial, since on the one hand obese individuals face higher medical costs and are expected to pay less contributions throughout their life-time\(^1\), but on the other hand their life expectancy is lower, resulting in fewer periods of health care spending and social security benefits. In countries such the UK, where the health care system is dominated by a single-payer, publicly funded National Health Service (NHS) focusing solely on the health care cost of obesity does not account for the overall fiscal impact of excess weight\(^2\).

Second, this paper assesses the impact of government intervention in a general equilibrium model where (i) food consumption and weight, (ii) labour productivity and (iii) life expectancy are endogenous. In contrast to other health hazards like smoking, obesity is caused by relative overconsumption\(^3\) of a basic human need, food. Hence, untargeted fis-

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\(^1\)With proportional income, consumption and capital income taxes, individuals with lower labour productivity are expected to pay less taxes ceteris paribus

\(^2\)In countries such as the US where private insurance is more common, the effect of obesity on health care costs is assessed through health insurance premiums (Bhattacharya and Sood, 2006).

\(^3\)Excess weight is caused by calorific imbalance, where individuals consume more calories than they
cal policies such as a soda tax, affect all individuals and the welfare and economic benefits of reducing the prevalence of obesity become unclear. Furthermore, tackling the obesity epidemic has direct and indirect macroeconomic consequences through labour productivity and life expectancy, which subsequently affects the spending and saving decisions of the household through the effective discount rate.

Our results show that only the top 30% of the BMI distribution have negative, albeit small, net contributions\(^4\). In fact, individuals at the top 5% of the BMI distribution have a life-time net deficit between 4.42% and 5.81% of annualized per capita GDP ($1933 to $2540 in 2015), depending on the burden of obesity on health care spending. In our model, we observe a relatively low impact of obesity on government spending even though obese individuals pay less taxes throughout their lifetimes and have higher medical costs that increase non-linearly with BMI. This is the outcome of higher mortality rate among obese individuals which results in fewer periods of pensions payments and medical services.

In addition, the benchmark model generates substantial income and wealth inequality which is consistent with the observed socio-economic gradient of obesity\(^5\) (Baum and Ruhm, 2009). There are two sources for the observed inequality in our model, (i) lower productivity and (ii) lower life expectancy which affects the effective discount of the agents. In our counterfactual simulations where we shut-down the productivity channel, more than 65% of the increase in inequality can be attributed to the higher effective discount rate. The endogenous effective discount rate caused by higher mortality has multiple consequences for the individual; (i) lower asset accumulation, (ii) discounting the adverse effects of obesity on future labour productivity, (iii) future medical spending and (iv) future mortality risk.

Furthermore, we assess the welfare and economic implications of two policy interventions; (i) eliminating childhood obesity and (ii) eliminating the VAT exemption of specific categories of food consumption. The welfare and economic impact of eliminating childhood obesity are substantial, with an increase in welfare of 10.5% of equivalent consumption, an increase in GDP by 2.15% and a reduction of the NHS budget by $1.48 billion, while the

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\(^4\)In this paper, we define net contributions as the difference between life-time contributions through taxation and life-time benefits through health care spending subsidies and pensions.

\(^5\)Introducing obesity, increases the wealth Gini coefficient by 1.36 percentage points and the net income Gini coefficient by 0.55 percentage points.
obesity externality is effectively eliminated\textsuperscript{6}. With respect to the VAT reform, the results are more modest with respect to the magnitude but remain significant. The aggregate welfare gains are 0.26\% of equivalent consumption, however the policy is not Pareto optimal. The bottom 20\% of the BMI distribution losses up to 0.41\% of equivalent consumption, while the rest of the BMI distribution experience welfare gains up to 0.6\% of equivalent consumption.

Our study is related to two strands in the literature, the general equilibrium literature on obesity (see inter alia Lakdawalla, Philipson and Bhattacharya (2005) and Dioikitopoulos, Katsaiti and Shaw (2012)) and the empirical literature that studies the economic cost of obesity (see inter alia (Bhattacharya and Bundorf, 2009), Trogdon et al. (2008) and (Scarborough et al., 2011)). However, to the best of our knowledge, the literature hasn’t focused on the fiscal implications of obesity in a general equilibrium model. The general equilibrium literature attempts to shed light on the mechanisms that increased the prevalence of obesity over the last decades in both developed and developing countries. We follow the methodology of this literature in order to answer a different question in a more quantitative setting.

The rest of the paper is organized as follows. In the next section we set up the economic environment under which we are going to study the presence and magnitude of externalities caused by obesity. In section three, we describe the functional forms and the estimation of the parameters relevant for our model. In section four and five, we present the quantitative results and analyze the economic implications of policy interventions respectively. We conclude with section five.

\section{The Economic Environment}

Consider an economy inhabited by overlapping generations of agents who survive for a maximum of $J$ periods. Agents derive utility from general consumption, the consumption of food, leisure and their body mass index (BMI)\textsuperscript{7}. The latter is determined by net calorific balance- the consumption of food and energy expenditure during labour and leisure and affects the level of utility non-monotonically as in Philipson and Posner (2003) and Lak-

\textsuperscript{6}Eliminating childhood obesity does not imply that the all the agents have a healthy BMI in our simulations. In our quantitative exercise, by eliminating childhood obesity all individuals enter the economy at a healthy BMI and the subsequent evolution of their BMI depends on their food preferences and the strenuousness of their occupation.

\textsuperscript{7}The level of BMI is determined by weight (in kg) over height squared (in meters)
dawalla, Philipson and Bhattacharya (2005). In addition, BMI affects the life expectancy, labour productivity and health care costs of the agent. There is a representative firm which produces a single composite good utilizing capital and labour. Finally, the government runs a balanced budget every period, taxing capital income, labour income, general consumption, food consumption and provides health care subsidies for all cohorts and a Pay-As-You-Go social security scheme for cohorts $j > J_R$.

2.1 Demographics

Time is discrete and the model is populated by $J$ overlapping generations. At the beginning of each period $t$, a measure of agents is born, whose mass grows at a constant rate $n$. Agents are ex-ante heterogeneous over two dimensions; preferences with respect to food and initial BMI and the strenuousness of their occupation. The distribution of each type of agent $i$ is predetermined and calibrated to match the UK data. The agents do not survive with certainty to the next period, but face a probability of survival $p_{j,t}^i \left( W_{j,t}^i \right) < 1$, which depends on their age $j$ and their BMI $W_{j,t}^i$. At age $j = J$, agents have a probability of survival $p_{j,t}^J = 0$. There is no bequest motive, however deceased agents leave unintended bequests that are seized by the government and transferred back to the agents via a lumpsum payment. Retirement is compulsory at age $J_R$ and agents receive pension benefits $b_t$ that are a fraction of the average labour income in the economy, irrespectively of the idiosyncratic labour income earnings history of the agent.

2.2 Agents

Agents are endowed with one unit of time which they allocate between labour and leisure and enter the economy without assets. They derive utility from general consumption, the consumption of food, leisure and their BMI. Their life-time utility of an agent of type $i$ is denoted as:

$$U_i = \sum_{j=1}^J \beta^{j-1} p_{j,t}^i \left( W_{j,t}^i \right) u_{j,t}^i \left( c_{j,t}^i, f_{j,t}^i, h_{j,t}^i, \Omega_{j,t}^i \left( W_{j,t}^i \right) \right)$$ (1)

with:

$$p_{j,t}^i \left( W_{j,t}^i \right) = \prod_{k=1}^{j-1} \left[ p_{k,t}^i \left( W_{k,t}^i \right) \right] p_{j,t}^i \left( W_{j,t}^i \right)$$ (2)

8For sensitivity analysis I set the preference uniform across all agents, differentiating solely with respect to their initial level of BMI.
where \( j \) denotes age, \( c^{i,j}_{j,t}, h^{i,j}_{j,t} \) and \( f^{i,j}_{j,t} \) are general consumption, labour supply and food consumption in period \( t \) for an agent of type \( i \) of age \( j \) respectively, \( \beta \) is the discount factor, while \( p^{i,j} (W^{i}_{j,t}) \) denotes the probability of surviving to age cohort \( j \), conditional on surviving until \( j - 1 \) and \( P^{i}_{j,t}(W^{i}_{j,t}) \) the unconditional probability of being alive at age \( j \). \( \Omega^{i}_{j,t}(W^{i}_{j,t}) \) denotes the utility derived from weight which is assumed to be inverted U-shaped since the agents have an ideal level of BMI. The households face the following budget constraint:

\[
(1 - \tau^w) \left( h^{i,j}_{j,t} w_t \varepsilon(j, W^{i}_{j,t}) \right) + (1 + r(1 - \tau^a)) a^{i}_{j-1,t-1} + b_t + beq_t = (1 + \tau^c) c^{i,j}_{j,t} + (1 + \tau^f) f^{i,j}_{j,t} (1 + \tau^m) m^{i,j}_{j,t} (W^{i}_{j,t}) + a^{i,j}_{j,t}
\]

(3)

where \( \tau^w, \tau^m, \tau^c, \tau^f \) and \( \tau^a \) denote labour income and health care spending, composite good consumption, food consumption and capital income tax rates respectively. Agents receive retirement benefits \( b_t \) after the the compulsory retirement age \( J_R \), which is a fraction of the average labour income during the working periods. The wage rate and interest rates are denoted as \( w_t \) and \( r_t \) respective and \( a_{j,t} \) denotes savings. Agents have an age and weight specific level of productivity \( \varepsilon_t(j, W) \), which consists of an exogenous age-specific productivity profile and an endogenous component which depends on weight. Furthermore, agents face medical spending \( m^{i,j}_{j,t} \) that needs to be paid in order to survive to the next period and it depends non-linearly on BMI.

2.3 Firms

There are two sectors in the economy, the food sector and the sector that produces the rest of the goods and services. However, we assume that both sectors share the same productivity and since capital and labour can move freely between the two sectors, the economy collapses into a one-sector economy. Hence, there is one representative firm, hiring labour and capital in order to produce a single composite good whose technology is described by a Cobb-Douglas production function. The aggregate resource constraint of the economy is given by:

\[
C_t + K_t + M_t + F_t + G_t - (1 - \delta) K_{t-1} = AK_{t-1}^{\alpha} L_t^{1-\alpha}
\]

(4)

where \( C_t, K_t, L_t, M_t, F_t \) denote the aggregate levels of consumption, capital stock, effect-
tive labour supply, medical spending and food consumption respectively. In addition, \( A \) denotes the total factor productivity\(^9\) and \( G_t \) denotes government consumption.

### 2.4 The Government

The government runs a balanced budget every period, collecting taxes from composite consumption, food consumption, labour income and capital income in order to finance an exogenous sequence of government spending \( G_t \), pension payments and subsidize health care spending as in:

\[
\tau^c C_t + \tau^f F_t + \tau^m M_t + \tau^a K_{t+1} + \tau^w wL = G_t + B_t \quad \forall t
\]

with \( B_t \) denoting aggregate pension benefits respectively.

### 2.5 Competitive Equilibrium

Here I present the formal definition of the competitive equilibrium. The agents’ state variables are assets \( a \), BMI \( w \), their type \( i \) and age \( j \). Let \( \Psi_t \) denote the mass of the total population, which is normalized to one and \( \psi_t(i,j) \) the mass of agents of type \( i \) and age \( j \) with a law of motion:

\[
\psi_t(i,j) = \frac{p_t(i,j) \psi_{t-1}(i-1,j-1)}{1+n}
\]

\( \text{Definition 1. (Competitive Equilibrium): Given fiscal policy } \pi: \{\tau^c_t\}_{t=0}^{\infty}, \{\tau^f_t\}_{t=0}^{\infty}, \{\tau^m_t\}_{t=0}^{\infty}, \{\tau^a_t\}_{t=0}^{\infty}, \{\chi_t\}_{t=0}^{\infty}, \{G_t\}_{t=0}^{\infty} \text{ a competitive equilibrium for this economy is the sequence of individual allocations } \left\{ \left\{ \left\{ c_{i,j,t}^i, f_{j,t}^i, h_{j,t}^i, m_{j,t}^i, a_{j,t}^i \right\}_{i=1}^{I} \right\}_{t=0}^{\infty}, \text{ production factors } \{K_t, L_t\}_{t=0}^{\infty} \text{ and relative prices } \{r_t, w_t\}_{t=0}^{\infty}, \text{ such that:} \right. \]

1. Households maximize life-time utility (1) subject to their budget constraint (3) for all \( t \)

2. \( c_{i,j,t}^i \geq 0 \quad f_{j,t}^i \geq 0 \quad a_{j,t}^i \geq 0 \quad \forall t, \)

3. \( 0 \leq h_{j,t}^i \leq 1, \quad b_t = 0, \quad \text{for } j < J_R \quad \forall t, \)

\( ^9 \text{Due to the agent’s preference specification, the economy is not on the balanced growth path and } A \text{ is time invariant (King, Plosser and Rebelo, 1988).} \)
4. 
\[ h_{j,t}^i = 0, \quad b_t = b, \quad \text{for} \quad j \geq J_R \quad \forall t, \]

5. Prices \( w_t \) and \( r_t \) satisfy:
\[ w_t = (1 - a) \frac{Y_t}{L_t} \quad \forall t, \quad (7) \]
\[ r_t = a \frac{Y_t}{K_{t-1}} - \delta \quad \forall t, \quad (8) \]

6. Aggregate pension benefits and bequests are given as:
\[ B_t = \sum_{i=1}^{I} \sum_{j=1}^{J} \psi_t(i,j) b_t \quad \forall t \]
\[ \text{beq}_t = \sum_{i=1}^{I} \sum_{j=1}^{J} \psi_t(i,j) \left( 1 - p_{j,t}^i \right) a_{j,t}^i (1 + r_t (1 - \tau^a)) \quad \forall t \quad (10) \]

7. Aggregate general consumption, food consumption and medical spending are given as:
\[ C_t = \sum_{i=1}^{I} \sum_{j=1}^{J} \psi_t(i,j) c_{j,t}^i \quad \forall t \]
\[ F_t = \sum_{i=1}^{I} \sum_{j=1}^{J} \psi_t(i,j) f_{j,t}^i \quad \forall t \]
\[ M_t = \sum_{i=1}^{I} \sum_{j=1}^{J} \psi_t(i,j) m_{j,t}^i \quad \forall t \]

8. Markets clear:
\[ K_{t-1} (1 + n) = \sum_{i=1}^{I} \sum_{j=1}^{J} \psi_t(i,j) a_{j,t}^i \quad \forall t, \quad (14) \]
\[ \hat{L}_t = \sum_{i=1}^{I} \sum_{j=1}^{J} \psi_t(i,j) h_{j,t}^i c_{j,t}^i (j, W) \quad \forall t \quad (15) \]

9. The government budget constraint (5) is satisfied for all \( t \)

10. The resource constraint (4) holds for all \( t \)

3 Calibration and Functional Forms

3.1 Demographics

Agents are born at the age of 20 \((j = 1)\), they retire at the age of 65 \((J_R = 9)\) and survive at most at the age of 95 \((J = 15)\). Thus each period in our model last 5 years. Population
grows at a rate of \( n = (1 + 0.006)^5 - 1 = 0.03 \). The conditional probability of survival is denoted as:

\[
p_{j,t}^{i} (W_{j,t}^{i}) = 1 - \rho (W_{j,t}^{i}) \Phi (j, t) \tag{16}
\]

where \( \rho_{j,t}^{i} (W_{j,t}^{i}) \) denotes the hazard risk associated with weight and \( \Phi (j, t) \) is the age-specific, conditional probability of death. In a nutshell, we assume that agents face a baseline probability of death which is increasing with age and deviations from the medically optimal BMI amplify the probability of death. The risk hazard is increased both for underweight and overweight agents and thus it is assumed to be U-shaped as in\(^{10}\):

\[
\rho_{j,t}^{i} (W_{j,t}^{i}) = \rho_0 + \rho_1 W_{j,t}^{i} + \rho_2 (W_{j,t}^{i})^2 + \rho_3 (W_{j,t}^{i})^3 \tag{17}
\]

We estimate the non-monotonic relation between BMI and hazard risk using OLS in order to fit the data from de Gonzalez et al. (2010). Our results suggest that \( \rho_0 = 7.72, \rho_1 = -0.64, \rho_2 = 0.02 \) and \( \rho_4 = 0.0001. \)

**Figure 1: Relation between BMI and mortality hazard risk**

![Figure 1](image)

**Notes:** Data are obtained from de Gonzalez et al. (2010). I estimate a non-linear equation using OLS in order to fit the hazard curve.

The exogenous probability of death is taken from the Human Mortality Database with the latest available data being for the year 2014. Since the data refer to the population as a whole with an average BMI above the healthy levels, we adjust the exogenous probability of death in order to derive the age-specific mortality risk of a healthy BMI agent. We estimate

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\(^{10}\)I postulate a cubic relation between BMI and hazard risk because the quadratic equation does not provide a good enough fit for such a fundamental variable in our model. Since the level of BMI does not deviate to values that would result in decreasing risk for extremely low and high values of BMI, I make use of this formulation.
the average BMI per age group using data from the Health Survey for England in year 2014 and using the hazard risk as estimated above, I adjust the age specific mortality risk. This ensures that the average mortality risk derived from our simulations does not double count the hazard risk associated with excess weight, which would inflate the probability of death.

3.2 Preferences

Agents derive instantaneous utility from general consumption, food consumption, leisure and BMI:

$$u_{i,t} = \frac{c_{i,t}^{1-\gamma}}{1-\gamma} + \varphi i f_{i,t}^{1-\sigma} + \psi \frac{(1 - h_{i,t})^{1-\eta}}{1-\eta} + \Omega(W_{j,t})$$ (18)

We set $\gamma = 2, \eta = 3$ (Conesa, Kitao and Krueger, 2009), $\sigma = 2$ and I calibrate the relative weights of food and leisure in order to match the average share of food consumption as a percentage of GDP and a level of average labour supply equal to 0.3 respectively. In our model agents are ex-ante heterogeneous with respect to food preference, in order to reflect the genetic differences with respect to food satiation. This choice is strongly supported by medical research which suggest that genetic factors explain 40-70% of weight variation (see inter alia Waalen (2005), Elks et al. (2012); Haworth et al. (2008)). The utility derived from BMI is denoted as in Dioikitopoulos, Katsaiti and Shaw (2012):

$$\Omega_{i,t}^j (W_{j,t}^i) = \omega_0 + \omega_1 W_{j,t}^i + \omega_2 (W_{j,t}^i)^2$$ (19)

We estimate the non-monotonic relation between BMI and utility with OLS using data from the Health Survey for England, using different specifications and controls\textsuperscript{11}. For robustness, we choose two different proxies for felicity, the Warwick-Edinburgh Mental Wellbeing Scale (WEMWBS) and the 12-item General Health Questionnaire (GHQ-12) that assess the mental wellbeing of the respondents. We control for age, marital status, income quantile, education, illness status, smoking status, a dummy for children in the household and a dummy for the type of dwelling. Under all specification, the BMI coefficients are statistically significant and suggest that BMI affects mental wellbeing non-monotonically. In fact, the level of ideal BMI is 26.31 which marginally lies in the overweight category and wellbeing is diminishes for lower and higher values of BMI.

\textsuperscript{11}For details consult Appendix B
3.3 The evolution of BMI

The level of BMI depends on the previous period BMI and the net calorific balance, meaning that if agents expend as many calories as they consume their BMI does not change. We use the following specification as in Griffith, Lluberas and Lührmann (2016):

\[ W_{i,j,t} = W_{i,j,t-1} + \xi f^i_{j,t} - R_{j,t} \left( s^i_{j,t} h^i_{j,t} + \bar{s}_t \left( 1 - h^i_{j,t} \right) \right) \]  

(20)

with \( W_{i,j,t-1} \) being the weight of the previous period, \( \xi \) transforms food consumption into calories, \( R_{j,t} \) denotes the age-specific basal metabolic rate (BMR)\(^{12} \), \( s^i_{j,t} \) denotes the degree of strenuousness of working, which is sector specific and \( \bar{s}_t \) denotes the strenuousness of leisure time which is constant for all agents.

We estimate the age-specific BMR using the Mifflin-St. Jeor equation (Mifflin et al., 1990), which depends of gender, height, weight and age. This ensures that the average BMR per age reflects the characteristics of our dataset, and the UK distribution of weight, height and gender in the population.

**Figure 2: Age-Specific Basal Metabolic Rate**

![Figure 2: Age-Specific Basal Metabolic Rate](image)

**Notes:** I estimate the age-specific Basal Metabolic Rate using the Mifflin et al. (1990) from the pooled data of the Health Survey for England, 2011-2014. The equation takes into account the gender, reported height and weight and age of the respondent.

We find that even though we make the simplifying assumption that the level of BMR is exogenous in our model and does not depend on the BMI of the agents in our model,

\(^{12}\)BMR is the minimum daily expenditure of a person that spends 24 hours at rest
we obtain reasonable results with respect to the distribution of BMI\textsuperscript{13}. The reason behind this result is that the estimated BMR takes into account the fact that weight increases as individuals age in the UK and the fall of the level of BMR is not as sharp compared to an individual with constant weight.

The parameters \( s_i \) and \( \bar{s}_t \) denote the strenuousness of working and leisure respectively and are estimated in terms of Metabolically Equivalent Task (MET), which is the ratio of the energy expended during a given task over the BMR. In order to estimate the distribution and the level of strenuousness of the working population in the UK we do the following; first, we calculate the percentage of the workforce working in each occupation using the Standard Occupational Classification (SOC2010) and then we assign MET values for each occupation using data from Tudor-Locke et al. (2011).

![Table 1: Strenuousness](image)

<table>
<thead>
<tr>
<th>Level of Strenuousness</th>
<th>% of Workforce</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedentary</td>
<td>52.02</td>
</tr>
<tr>
<td>Moderate</td>
<td>28.72</td>
</tr>
<tr>
<td>Strenuous</td>
<td>19.26</td>
</tr>
</tbody>
</table>

Notes: The percentage of workforce employed in each level of strenuousness are derived from Table 5 in Appendix B. Sedentary occupations are considered the ones with a level of MET of 1-2, moderate with a level of 2-3 and strenuous with above 3.

Finally, we reduce the level of strenuousness in three categories; sedentary, moderate and strenuous, whose values and distribution are used in our parameterization\textsuperscript{14}. The value of MET during leisure \( \bar{s}_t \) is constant for all agents and is the average of the strenuousness of leisure estimated by Griffith, Lluberas and Lührmann (2016), taking into account sleep, home work and other activities.

3.4 Labour Productivity

The productivity of agents is determined by an exogenous age-specific profile and an endogenous component which depends on BMI.

\textsuperscript{13} Although all agents share the same path of BMR as they age irrespectively of weight, I don’t observe extreme values of BMI which would suggest that the level of BMR is too low for these individuals. For the bottom of the BMI distribution, I observe an increase of BMI over time, which also suggest that the assigned BMR is not too high given their weight.

\textsuperscript{14} The results are close to the estimation of Griffith, Lluberas and Lührmann (2016) which refers to the year 2009 using the a different occupational classification.
The exogenous component $\pi_t(j)$ is calibrated to match the hump-shaped labour supply and labour income on the households, while I calibrate the relation between BMI and productivity $e^{\xi W_{i,j}}$. The estimation of the relation between labour productivity and obesity poses a series of challenges because of reverse causality and cofounding factors and the results in the literature are mixed. Since labour productivity plays an important role for our results, we conduct extensive sensitivity analysis in order to estimate the effect of different parameterization on our steady state results.

Dackehag, Gerdtham and Nordin (2014) use Swedish data in order to estimate the impact of obesity on labour income. Using OLS the results suggest that men lose approximately 6% of labour income compared to normal weight individuals, while the Fixed Effects regression increases the estimate to 9%. Both results are statistically insignificant for women. Nustad and Black (2011) study the loss in productivity directly, using the Work Limitations Questionnaire in US manufacturing workers and estimate that the loss of productivity is 1.18% compared to normal weight individuals. However, another source of lost productivity beyond presenteeism, is absenteeism\textsuperscript{15}. Harvey et al. (2010) using data from the London Underground find that normal weight individuals lose on average 6 days per calendar year, while obese individuals lose 9.5-11 days per calendar year, hence 1.6-2.2% more working days\textsuperscript{16,17}.

In our benchmark model, we calibrate $\xi$ such that a healthy BMI individual is 5% more productive than an obese individual, while in our counterfactual simulation we assume that BMI does not affect productivity.

### 3.5 Government Policies and Health Care Spending

The labour income tax rate is determined endogenously in order to balance the government budget, while consumption, food consumption, capital income and health care spending subsidies are calibrated or estimated. Food consumption is either taxed with a VAT of 0% or 20% and thus we need to estimate the effective tax rate on food consumption. Typically

\textsuperscript{15}Presenteeism is defined as the loss in productivity while on the job and absenteeism is defined as lost working days due to illness.

\textsuperscript{16}According to the ONS statistics the calendar year has 226 working days for the average employee.

\textsuperscript{17}It is well documented that public sector employees take more days of sick leave compared to their private sector counterparts ONS (2017) and these estimates are follow closely the rate of absence in the public sector.
food prepared at home has a 0%\textsuperscript{18} and eating out, soft drinks, confectionary and alcohol are taxed at a rate of 20%. From Griffith, Lluberas and Lührmann (2016) I apply the shares of expenditure to each category in order to estimate an effective VAT of 9.7%. For general consumption, I apply the standard VAT of 20% since most of the excluded categories refer to food consumption. Capital income is taxed at an effective rate of 46% (Trabandt and Uhlig, 2011) and the government share of aggregate medical spending is 79\%\textsuperscript{19}. Finally, I set the pension replacement rate $\chi$ to 0.335, which is the average replacement ratio in the UK (OECD, 2017)

Health care spending has an exogenous component which is calibrated to match the age-specific average per capita health care spending of a healthy weight adult and a non-linear mark-up that is determined by the deviation from the healthy BMI. Thus health care spending $m_{j,t}$ is denoted as:

$$m_{j,t}^i = \mu_0 (j) + \mu_1 (W_{j,t}^i - \bar{W})^2$$ (22)

We assume a non-linear relation between BMI (Andreyeva, Sturm and Ringel, 2004) and medical spending to reflect the non-linear relation between BMI and mortality. Since the majority of medical spending takes place at the last two years of life (Zweifel, Felder and Meiers, 1999), premature death caused by excess weight is reflected in the medical spending equation.

In order to disentangle the effects of excess weight on health care spending we apply the methodology of Scarborough et al. (2011) on the 2016 NHS budget. In a nutshell, we estimate the burden of specific diseases associated with obesity on the NHS budget and using the Population Attributable Fractions (PAF), we estimate what percentage of these cost are caused by excess weight. For example diabetes mellitus, a common disease associated with excess weight, cost each year 1.9\% of the NHS budget, of which 79\% is attributed to overweight or obese patients.

\textsuperscript{18}This includes fruits and vegetables, meat and poultry but also chilled/frozen ready meals and convenience foods
\textsuperscript{19}This is the average share of overall health care spending that is financed by the central government. Since the UK health care system is dominated by the single-payer, government financed centralized National Health System we can abstract from the effects of private health insurance. I will consider the share of health care spending that is not covered by the government as out-of-pocket payments, which includes co-payments and over-the-counter medicine.
Table 2: Economic Costs of Overweight/Obesity, Poor Diet, Physical Inactivity to the NHS

<table>
<thead>
<tr>
<th>Condition</th>
<th>% of Total NHS Costs</th>
<th>PAF</th>
<th>Total NHS Costs (in bn £)</th>
<th>% of Total Budget</th>
<th>Per Capita (in £)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overweight and obesity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>2.9</td>
<td>34</td>
<td>1.1832</td>
<td>0.986</td>
<td>18.2</td>
</tr>
<tr>
<td>Ischaemic stroke</td>
<td>1.2</td>
<td>34</td>
<td>0.4896</td>
<td>0.408</td>
<td>7.5</td>
</tr>
<tr>
<td>Breast cancer</td>
<td>0.6</td>
<td>12</td>
<td>0.0864</td>
<td>0.072</td>
<td>1.3</td>
</tr>
<tr>
<td>Colon/rectum cancer</td>
<td>0.5</td>
<td>16</td>
<td>0.09</td>
<td>0.075</td>
<td>1.4</td>
</tr>
<tr>
<td>Hypertensive disease</td>
<td>4.5</td>
<td>58</td>
<td>3.132</td>
<td>2.61</td>
<td>48.1</td>
</tr>
<tr>
<td>Corpus uteri cancer</td>
<td>0.2</td>
<td>49</td>
<td>0.1176</td>
<td>0.098</td>
<td>1.8</td>
</tr>
<tr>
<td>Osteoarthritis</td>
<td>5.0</td>
<td>21</td>
<td>1.26</td>
<td>1.05</td>
<td>19.4</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.3</td>
<td>79</td>
<td>1.2324</td>
<td>1.027</td>
<td>18.9</td>
</tr>
<tr>
<td>Total</td>
<td>11.6</td>
<td></td>
<td>7.5912</td>
<td>6.326</td>
<td>116.6</td>
</tr>
<tr>
<td>Poor diet</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CVD</td>
<td>9.2</td>
<td>22</td>
<td>3.6432</td>
<td>3.036</td>
<td>56</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>2.8</td>
<td>33</td>
<td>1.1088</td>
<td>0.924</td>
<td>17</td>
</tr>
<tr>
<td>Cancer</td>
<td>6.2</td>
<td>33</td>
<td>2.4552</td>
<td>2.046</td>
<td>37.7</td>
</tr>
<tr>
<td>Dental caries</td>
<td>3.4</td>
<td>33</td>
<td>1.3464</td>
<td>1.122</td>
<td>20.7</td>
</tr>
<tr>
<td>Total</td>
<td>21.6</td>
<td></td>
<td>8.5536</td>
<td>7.128</td>
<td>131.4</td>
</tr>
<tr>
<td>Physical inactivity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>2.9</td>
<td>23</td>
<td>0.8004</td>
<td>0.667</td>
<td>12.3</td>
</tr>
<tr>
<td>Ischaemic stroke</td>
<td>1.2</td>
<td>12</td>
<td>0.1728</td>
<td>0.144</td>
<td>2.7</td>
</tr>
<tr>
<td>Breast cancer</td>
<td>0.6</td>
<td>11</td>
<td>0.0792</td>
<td>0.066</td>
<td>1.2</td>
</tr>
<tr>
<td>Colon/rectum cancer</td>
<td>0.5</td>
<td>16</td>
<td>0.096</td>
<td>0.08</td>
<td>1.5</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1.3</td>
<td>15</td>
<td>0.234</td>
<td>0.195</td>
<td>3.6</td>
</tr>
<tr>
<td>Total</td>
<td>6.5</td>
<td></td>
<td>1.3824</td>
<td>1.152</td>
<td>21.2</td>
</tr>
</tbody>
</table>

Notes: Data for the percentage of total NHS budget dedicated to each condition and population attributable fractions (PAF) are taken from Scarborough et al. (2011). The NHS budget in 2016 was £120 billion.

These estimates provide a lower bound since they only include direct cost of diseases that are directly associated with excess BMI. We also estimate the upper bound with respect to the health care costs of excess weight, taking into account the health care cost of poor diet and inactivity. Since not all overweight agents consume a poor diet and have low levels of physical activity and vice versa, the true health care cost of excess BMI lies between the two bounds. We find that accounting only for the direct cost of obesity, the NHS spends 6.3% of its budget to treat obesity-related diseases, while taking into account poor diet and physical inactivity the estimate for the cost of obesity rises to 14.56% of the NHS budget. We make the simplifying assumption that these shares also apply to the aggregate health care spending; private and public combined. Hence, the lower and upper bounds for the aggregate health care spending caused by excess weight are 0.62% and 1.43% of GDP respectively, given the level of aggregate health care spending of 9.8% in 2016.

For the exogenous component of medical spending we use the data from Kelly, Stoye and Vera-Hernández (2015) who estimate the age-specific cost of hospital services in the UK. Assuming that the fraction of health care spending allocated to hospital services does not
vary with age, we scale these estimates such that the aggregate exogenous component amounts to 9.18% and 8.37% of GDP for the lower and upper bounds with respect to excess weight costs respectively.

**Figure 3: Per Capita Hospital Costs per Age**

![Per Capita Hospital Costs per Age](chart.png)

*Notes:* Data are obtained from Kelly, Stoye and Vera-Hernández (2015). The hospital costs are estimated as the average between men and women with equal weights.

In a nutshell, given that the aggregate health care spending was 9.8% of GDP in the UK in 2016, we assume that 6.3% was caused by excess weight and the rest is the exogenous component for a healthy BMI individual, respecting the age-specific profile of health care spending.

## 4 Steady State Results

The model succeeds in producing key aggregate variables that are close to the actual UK economy. Aggregate health care spending and pensions provided by the government amount to 9.8% and 4.7% of GDP respectively while government consumption is calibrated to match the UK average of 19.4% (Table 3) and the average ratio of food over consumption of approximately 12%.
Table 3: Benchmark Model Statistics

<table>
<thead>
<tr>
<th>Name</th>
<th>Model</th>
<th>Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Health Care Spending</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aggr. Health Care Spending (% GDP) in 2015</td>
<td>9.8 %</td>
<td>9.8%</td>
</tr>
<tr>
<td>Private Share (% GDP) in 2015</td>
<td>2.0%</td>
<td>2.0%</td>
</tr>
<tr>
<td>Government</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Public Pensions (% GDP) in 2015</td>
<td>4.7%</td>
<td>4.7%</td>
</tr>
<tr>
<td>NHS Spending (% GDP) in 2015</td>
<td>7.7%</td>
<td>7.7%</td>
</tr>
<tr>
<td>Government Consumption (% GDP) in 2015</td>
<td>19.4%</td>
<td>19.4%</td>
</tr>
<tr>
<td>Labour Income Tax</td>
<td>20.3%</td>
<td>28%</td>
</tr>
</tbody>
</table>

Furthermore, our simulation results approximate the distribution of obese and overweight individuals in the economy taking into account food preferences, job strenuousness and mortality risk.

Figure 4: Percentage of Obese and Overweight by Age Group

![Figure 4: Percentage of Obese and Overweight by Age Group](image)

Notes: Data are obtained from the Health Survey for England (2011-14). The simulation results are the weighted average BMI of each age group.

These results are important for the quantitative exercise of estimating the net benefits of each percentile of the BMI distribution.

On the disaggregated level, we face a fundamental issue that this paper attempts to address. Agents switch between BMI classifications (i.e. underweight, normal, overweight and obese) throughout their lifetime, with a general trend of increasing BMI (Meeuwsen, Horgan and Elia, 2010) that on an individual level can be reversed because of life-style choices and health shocks such as terminal diseases (Harrington, Gibson and Cottrell, 2009). In order to simplify the analysis, we ignore life-style changes and shocks that result
in a reduction of BMI. With respect to the former, we consider this simplification reasonable since even though individuals can be successful in losing weight, out of the overweight or obese individuals that decide and accomplish to lose at least 10% of their weight, only 20% maintain the weight loss after one year, with the fraction further reduced for more extended periods (Wing and Phelan, 2005) and thus on the aggregate level the effect is insignificant. With respect to the latter, we leave the incorporation of health shocks associated with obesity for future research.

In this paper, we classify the agents based on their initial BMI which is calibrated to match the UK deciles at the age of 20 to 25. This exercise is useful for two reasons; first, we can study the agents’ life-cycle profiles with respect to asset accumulation, labour supply and their net contributions\textsuperscript{20}. Classifying the agents solely based on their current BMI and estimating the net cost of obesity is misleading because agents are more likely to be obese as they age, ignoring their life-time contributions and overestimating the medical cost of obesity. Second, we can assess the significance of initial BMI and path dependence with respect to weight when analyzing the effectiveness of policy interventions. In fact, according to our simulation results even though all agents gain weight over time at different paces depending on their occupation, food preferences and initial BMI, agents remain to their respective decile throughout their lifetime. This is the outcome of two factors, (i) the absence of shocks and (ii) the fact that losing weight is not only costly but increasingly so, since the basal metabolic rate falls over time. Hence, even obese individuals that are above their preferred level of BMI and suffer losses in productivity and life expectancy do not lose weight even in strenuous occupations because they would have to reduce their food consumption at an increasing rate as they age\textsuperscript{21,22}.

For the rest of the analysis, I will focus on three groups for clarity of exposition; the bottom 10%, the median and the top 5% of the BMI distribution. Our results suggest that the level of BMI has significant effects on the life-time earnings of the individuals both from labour income and capital income. Obese agents hold less assets and receive less labour income, even though they supply more labour (Figures 5-7).

\textsuperscript{20}We define as net life-time contributions the sum of all tax payments minus the total benefits.

\textsuperscript{21}This result is robust even if agents have uniform preferences with respect to food and they are heterogeneous only with respect to initial BMI and strenuousness of occupation.

\textsuperscript{22}Recall from the previous section that the estimation of BMR for the UK economy is not the estimated BMR of a healthy weight individual over time, but takes into account the average weight per age observed in the UK which tends to increase BMR. Hence the overall fall in BMR is not as pronounced in order to be the sole reason for the observed trends.
Obese agents accumulate less assets because of lower life expectancy (Table 6) which tends to increase the effective discount rate of the households. Furthermore, agents earn a lower wage on average during their lifetime compared to normal weight agents, caused by lower labour productivity and hours lost due to illness; this has a negative effect on both wealth and labour income earnings, making obese agents poorer on average. The latter is true even though obese agents tend to supply more labour because of the income effect.

Notes: Simulation Results for the bottom 10%, the median and the top 10% of the BMI distribution.

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23 See Appendix A
4.1 BMI and Labour Supply

In a nutshell, obese agents react to lower income and by extension consumption, by increasing the hours of work. It should be noted that we solve for the rational expectations solution, where agents are aware of the effect of labour supply on their level of BMI since even sedentary occupations are more strenuous than leisure. Hence, the relatively higher labour supply of obese agents could be explained as an attempt to reduce the level of BMI without reducing food consumption. However, in our sensitivity analysis under which agents do not take into account the effect of labour on BMI when making the labour supply decisions, we find that our results are robust and the main driver of the observed labour supply differentials is the income effect\(^{24}\). This result is consistent with Lakdawalla and Philipson (2007) who find that obese individuals do not self-select to strenuous occupations in order to reduce their BMI.

The relation between BMI and working hours has been studied extensively with a suggested positive correlation, however the direction of causality is assumed to be the reverse compared to our results. Au, Hauck and Hollingsworth (2013) surveyed metro transit workers with respect to their BMI, working hours and eating habits. They find a positive correlation between BMI and working hours, however the relation between eating habits and working hours is unclear. Working more than 50 hours per week is positively correlated with higher frequency of purchases from vending machines, however working hours were uncorrelated with consuming fast food, sweetened beverages and sweets or the perception of ease of eating healthily. On the contrary, the frequency of eating foods and vegetables was positively correlated with working hours. On the other hand, the results of Escoto et al. (2010) suggest that working hours are associated with a greater pace of weight gain, studying a sample of Australian women.

Although our simulation results are consistent with the literature with respect to the correlation between BMI and working hours, addressing the direction of causality is beyond the scope of this paper.

\(^{24}\)Increasing labour supply in order to lose weight is ineffective for two reasons in our model. First, agents would have to gain direct and indirect utility (from a lower BMI and higher productivity and life expectancy respectively) which is high enough to compensate for the disutility from the increase in labour supply. Secondly, even though working is more strenuous than leisure, agents still expend calories in their leisure time and the difference is not as pronounced to justify working as a weight loss strategy, especially for the majority of the population that is employed in sedentary occupations.
4.2 BMI and Inequality

In order to assess the magnitude of income and wealth inequality caused by obesity, I calculate the respective Gini coefficients. However, since the model has a life-cycle aspect which is well known to cause income and wealth inequality by its nature, we estimate the counterfactual Gini coefficients of the same model without any intragenerational heterogeneity. In a nutshell, we estimate the Gini coefficients of a model where agents start with the same level of BMI, have the same preferences over food consumption and their occupation is equally strenuous for all agents. Considering the absence of idiosyncratic shocks and liquidity constraints, the counterfactual model generates substantial wealth inequality with a Gini coefficient of 50.47, compared to 73.2 in the data, while the Gini coefficient for net income is 21.9 for the counterfactual and 31.6 in the data. Both measures of inequality increase substantially once we take into account the intragenerational heterogeneity gener-
ated in our model. Wealth inequality increases by 2.5 and net income inequality by 2.0, to 52.99 and 23.96 respectively (Table 4).

Table 4: Gini Coefficients

<table>
<thead>
<tr>
<th></th>
<th>Wealth</th>
<th>Labour Income</th>
<th>Net Income</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benchmark</td>
<td>52.99</td>
<td>8.71</td>
<td>23.96</td>
</tr>
<tr>
<td>Exogenous Productivity</td>
<td>52.51</td>
<td>8.37</td>
<td>23.49</td>
</tr>
<tr>
<td>No Heterogeneity</td>
<td>51.63</td>
<td>7.95</td>
<td>23.41</td>
</tr>
<tr>
<td>Data</td>
<td>73.2</td>
<td>N/A</td>
<td>34</td>
</tr>
</tbody>
</table>

The socio-economic gradient of obesity in developed countries is well-documented (Baum and Ruhm, 2009). Higher discounting (Courtemanche, Heutel and Mcalvanah, 2015), hyperbolic discounting (Ikeda, Kang and Ohtake, 2010), discrimination (Dackehag, Gerdtham and Nordin, 2014) and confounding factors such as genetics have been suggested in order to shed light on the relationship between socio-economic status and obesity. In our model this relationship is straightforward; a higher level of BMI is causing a significant reduction in labour and capital income via lower productivity and a higher effective discount rate. The key difference with the results in the literature is that in order to generate the observe relation between income and BMI, no additional assumptions need to be made apart from differences in the initial BMI. The higher discount rate of obese agents is determined endogenously and the sole driver is the lower life expectancy.

Furthermore, Lakdawalla and Philipson (2009) postulate a non-monotonic relationship between income and BMI with income affecting the level of BMI. In their model, consumption and the utility derived from the level of weight are complements which can result to the cross derivative of the utility function with respect to consumption and the level of BMI to have a non-constant sign. This is not the case in our model since the the utility function is separable in all of its arguments and the cross derivatives are zero. We leave the investigation of the bi-directional relationship between income and BMI for future research.

4.3 Net Life-Time Contributions

We define the net life-time contributions as the sum of all taxes paid throughout the individuals’ life-time minus the life-time benefits, namely pensions, health care spending provided by the NHS and per capita government consumption. In order to make the exercise

\[\text{In our sensitivity analysis, we remove the heterogeneity in preferences and occupation and even though the magnitude of our results changes, the key dynamics do not.}\]
tractable, we define the percentiles according to the initial level of BMI, hence an individual that starts at the bottom 10% of the BMI distribution will belong to this percentile for this accounting exercise. As stated above, although individuals may switch between BMI classifications, they do not switch between the percentiles of the BMI distribution in our model. Furthermore, we take into account the population mass of each percentile over time and thus we account for life-expectancy differentials. Hence, individuals of type $i$ will have life-time net benefits of:

$$\text{NET}_i^t = \sum_{j=1}^{l} \psi_{i,j} \left( \tau^v v_{i,t}^j + \tau^d d_{i,t}^j + \tau^c c_{i,t}^j + \tau^f f_{i,t}^j \right) - \sum_{j=1}^{l} \psi_{i,j} \left( \tau^m m_{i,t}^j + b_t + g_t \right)$$

Figure 8 demonstrates the life-time net contributions for each percentile. The merit of this approach is that we can clearly assess the magnitude of the effect of obesity on the net contributions of the agents since the net contributions of all agents as a percent of annualized GDP need to add up one.

**Figure 8: Life-Time Net Contributions for Each Decile as a Percentage of Per Capita GDP**

Notes: The net contribution of each decile of the BMI distribution is calculated as the life-time tax payment that finance pensions and health care subsidies minus the life-time pension receipts and subsidized medical spending. The results take into account the distinct mortality risk of each decile and are expressed in terms of annual per capita income.

We observe that net life-time contributions fall monotonically with BMI, with the lower percentiles having positive and higher percentiles negative net contributions. The cut-off point is the bottom 60%, for which the net life-time contributions are essentially zero and they receive in benefits as much as they contribute throughout their life-times. However, the differences between the percentiles are quantitatively small, with the top 95% of the BMI distribution having negative net contributions of 4.42% of annualized GDP per capita.
Figure 9 demonstrates the life time contributions in greater detail. Agents have relatively low net contributions at the beginning of their life-time with higher percentiles of the BMI distribution having negative net contributions caused by excess medical spending. As agents age and before they reach the retirement age, their net contribution is increasing since agents accumulate more assets and their labour productivity is increasing. Once agents reach the age of retirement, they stop paying labour income taxes, start decumulating assets and face higher medical spending due to the higher mortality risk caused by age and BMI, while receiving pensions. Naturally, as agents age their net contributions fall into negative territory, however, taking into account the population mass of each percentile of the BMI distribution, we observe that the burden of the obese is less than the healthy BMI percentiles at older ages.

5 Policy Experiments

We perform two distinct policy experiments in order to assess the response of the agents and the fiscal impact of obesity; (i) eliminate childhood obesity and (ii) eliminate the VAT exemption on food consumption. The welfare consequences of these policies are quantitatively large, especially for the elimination of childhood obesity, which has significant implications for government policy.
5.1 Eliminating Childhood Obesity

In order to assess what is the economic impact of eliminating childhood obesity, we drop the intragenerational heterogeneity with respect to the initial level of BMI, keeping the rest of the specification constant. In a nutshell, agents continue to have different preferences with respect to food consumption and supply labour in occupations with different levels of strenuousness, however the BMI that agents enter the economy is uniform and equal to the median BMI of the UK population at ages 20-25, which lies in the healthy classification. Hence, agents continue to be heterogenous with respect to their evolution of BMI over their life-time, with some agents becoming obese, however all agents enter the economy with a healthy BMI.

The welfare implications of eliminating childhood obesity are substantial. On the aggregate level the increase in welfare is equivalent to an increase in consumption by 10.5%. All agents experience an increase in welfare, with the median agents whose characteristics did not change compared to the benchmark case earning the equivalent of 0.3% of consumption. The reason behind this result is the general equilibrium effect of reducing the prevalence of obesity. First, the NHS saves almost 1% of its budget ($1.48 bn) when we consider the lower bound of the effects of obesity on health care spending. Secondly, GDP increases by 2.15% compared to the benchmark case, due to an increase in labour productivity and savings, with the latter being affected by the lower effective discount rate.

On an individual level, the effects of eliminating childhood obesity are heterogenous with higher percentiles of the BMI distribution benefiting the most. In fact, the top 5% of the BMI distribution experiences an increase in welfare which is equivalent to 47.5% of consumption. These agents experience significant welfare gains for five reasons; (i) lower medical spending, (ii) higher labour productivity, (iii) higher asset accumulation (iv) higher instantaneous utility since they are closer to their ideal weight and (v) more periods that they can derive utility from, caused by higher life expectancy. Agents at the top 5% of the BMI distribution at the age of 65 are expected to live 6 years longer than the benchmark case.

Furthermore, eliminating childhood obesity essentially eliminates the obesity externality, even though the prevalence of obesity is not eliminated. In fact, the net contributions for the higher percentiles of the BMI distribution are positive, while the net contributions of the lower percentiles are negative. However, the magnitude of both the positive and negative
net contributions is reduced even further.

**Figure 10: Life-Time Net Contributions After Eliminating Childhood Obesity**

![Net Contributions Graph]

*Notes:* The net contribution of each decile of the BMI distribution is calculated as the life-time tax payment that finance pensions and health care subsidies minus the life-time pension receipts and subsidized medical spending. The results take into account the distinct mortality risk of each decile and are expressed in terms of annual per capita income.

The reason for the negative net contributions of the lower percentiles lies in their life expectancy differentials that results in more periods of pension payments. Higher percentiles still face higher medical spending and lower life expectancy compared to lower percentiles, however the reduction in medical spending is more than enough to compensate for the increase in pension payments towards this demographic and in fact, eliminate the obesity externality.

### 5.2 Eliminating the VAT Exemption on Food Consumption

Since we do not distinguish between food categories, we cannot assess the macroeconomic affects of taxes on specific goods, such as the soda or fat tax. However, we can assess what is the effect of eliminating the VAT exemption on specific food products, as described in the previous section. Given the share of food consumption towards goods that are exempt from VAT and food consumption that faces a VAT of 20%, we estimated that the effective VAT on food consumption is 9.7%. For this exercise, we consider the effect of taxing all food consumption with a VAT of 20%, while the rest of the parameterization remains constant.

On the aggregate level, eliminating the exemptions on food consumption has a positive welfare effect equivalent to 0.26-0.27% of consumption depending on whether we consider
the lower of upper bound of effect of BMI on health care spending. However, the improve-
ment is not Pareto optimal as the case of eliminating childhood obesity and the aggregate
result conceals a lot of variation between demographics. We obtain the counterintuitive
result of a reduction in welfare for the bottom 20% of the BMI distribution and an increase
in welfare for the rest of the percentiles. Quantitatively, the magnitude of the welfare gains
and losses is not as great as the case of eliminating childhood obesity, however the losses
for the bottom 10% are 0.41% of consumption and the gains for the top 5% are equivalent
to 0.6% of consumption.

Higher percentiles of the BMI distribution experience welfare gains for two reasons; first,
an increase in the VAT of food consumptions distorts their behaviour, leading to a slower
pace of weight gain with subsequent effects on labour productivity, asset accumulation and
utility from the level of BMI. Secondly, an increase in the VAT of food consumption reduces
the labour income tax. Recall that the labour income tax acts as a residual that balances
the government budget. In a nutshell, an increase in the VAT is equivalent to transferring
a part of revenue resources from labour income taxation to food consumption taxation.
This has significant implication on the the financing of the government budget because of
the life expectancy differentials. Until the age of 65, the mortality rate is low for all de-
mographics and hence both obese and non-obese finance government spending through
labour income taxes, however at higher ages both the mortality rate and the mortality dif-
ferentials increase, less obese individuals are contributing through capital income, general
consumption and food consumption taxes. By transferring the burden of financing the
government budget from a tax that has to be paid until the age of 65 to a tax that is paid
throughout the whole life-time, the government puts additional pressure on percentiles
that have higher life-expectancy.
Figure 11: Life-Time Net Contributions After Eliminating the VAT Exemption

Notes: The net contribution of each decile of the BMI distribution is calculated as the life-time tax payment that finance pensions and health care subsidies minus the life-time pension receipts and subsidized medical spending. The results take into account the distinct mortality risk of each decile and are expressed in terms of annual per capita income.

Since the magnitude of the VAT change is not large, the quantitative effect of this policy on a macroeconomic level are expected to be small. Life expectancy for the higher percentiles increases by approximately one month for all age groups, while the NHS saves a 0.16% of its budget ($0.26 bn). With respect to the response of labour supply with an increase in VAT and a decrease the labour income tax, we observe a consistent pattern across all BMI percentiles. Labour supply decreases by as much as 0.29% at the age of 20 and increases as much as 0.37% at the age of 65. We find that the income effect dominates the substitution effect earlier in the life-cycle, but labour supply increases at older ages since agents are more productive compared to the benchmark case. GDP increases by 0.27% after the elimination of VAT exemption, which affects the labour supply decisions of the agents. However, the increase in GDP is not attributed to changes in labour supply, since the aggregate hours of work do not change in our model, with the decrease during earlier periods being offset by the increase in later periods of life. The main drivers of the GDP increase are higher savings caused by the lower effective discount rate, higher labour productivity caused by a reduction in average BMI and lower health care spending. Finally, the net contributions of the percentile changes only slightly with the deficit of the top 5% of the BMI distribution being 4.39% instead of 4.42% of annualized per capita GDP (Figure 11).
6 Conclusions

This paper studied the fiscal and welfare implication of obesity in a model where (i) food consumption and weight (ii) labour productivity (iii) life expectancy and (iv) medical spending are endogenous. Our aim is to replicate key moments of the UK economy and assess the presence and magnitude of externalities associated with obesity. Subsequently, we perform policy experiments in order to quantify the economic and welfare implications of changes in government policy.

Our result suggest that the magnitude of the obesity externality is quantitatively small, up to 5.81% of annualized per capita GDP, despite the significant effects of obesity on income inequality and the subsequent effect on the level of contributions. Furthermore, our result suggest that government intervention with respect to childhood obesity and food taxation can have substantial welfare and economic implications, however the latter is not Pareto improving.

Given the significant welfare and general equilibrium effects associated with obesity in our model, future research should focus on sensitivity analysis of our results and extend our framework incorporating uncertainty and incomplete markets, heterogeneity with respect to food categories, and the choice of exercise in order to lose weight.
Here I present the first order conditions of the household with respect to general consumption, food consumption and labour supply in order to demonstrate the objectives of the agents. For clarity, I drop the time notation and I will denote $u_{j,c_j}$ as the partial derivative of the utility function at period $j$ with respect to consumption at period $j$. Let $\lambda_j$ denote the Lagrange multiplier at period $j$. For clarity of exposition I demonstrate a 3-period model. The first order condition of the household with respect to consumption in periods 1, 2 and 3 respectively are denoted as:

$$u_{1,c_1} - \lambda_1 (1 + \tau_c) = 0 \quad \forall t \tag{24}$$

$$\beta P_2 (W_{1,t}) u_{2,c_2} - \lambda_2 (1 + \tau_c) = 0 \quad \forall t \tag{25}$$

$$\beta^2 P_3 (W_2) u_{3,c_3} - \lambda_3 (1 + \tau_c) = 0 \quad \forall t \tag{26}$$

However, the decision of the agent with respect to food consumption and labour supply is not as straightforward. Agents decide their level of food consumption taking into account the direct effect of food consumption on the current period’s utility, the effect of food on BMI, and the subsequent indirect effect on utility, labour productivity, probability of survival and medical spending. Moreover, since the decision of food consumption today affects the level of BMI of the next period, agents take into account the effects of today’s food consumption on next period’s BMI. Hence, agents face different incentives throughout their lifetime; from the $J = 1$ until $J_R - 1$ agents take into account not only next period’s level of utility and medical spending, but also the effects of BMI on next period’s labour productivity. At age $J_R$, the agents’ last working period, there is no effect on next period’s productivity since the agent retires and she takes into account only the effects of BMI on utility, the probability of survival and medical spending. At age $J$, the agent dies with certainty and current decisions have no effect on future variables.

Hence during the first period of life, the optimal decision with respect to food consumption is denoted as:
\[ u_{1, f_1} + \Omega_{1, W_1} W_{1, f_1} + \beta P_{1, W_1} W_{1, f_1} u_2 (c_2, f_2, h_2, \Omega_2) + \\
+ \beta P_1 (W_1) (\Omega_{2, f_1} W_{2, f_1}) + \\
+ \lambda_1 (1 - \tau^m) \omega h_1 \epsilon W_{1, f_1} + \lambda_2 (1 - \tau^w) \omega h_2 \epsilon W_{2, f_1} - \\
- \lambda_1 (1 + \tau^m) m_{1, W_1} W_{1, f_1} - \lambda_2 (1 + \tau^m) m_{2, W_2} W_{2, f_1} - \\
- \lambda_1 (1 + \tau^f) = 0 \tag{27} \]

At period 2, the agent expects that she will retire, and thus she doesn’t take into account the effect of food consumption on next period’s productivity:

\[ \beta P_1 (W_1) u_{1, f_1} + \beta P_1 (W_1) \Omega_{2, W_2} W_{2, f_2} + \beta^2 P_{2, W_3} W_{3, f_2} u_3 (c_3, f_3, h_3, \Omega_3) + \\
+ \beta^2 P_2 (W_2) (\Omega_{3, f_2} W_{3, f_2}) + \\
+ \lambda_2 (1 - \tau^w) \omega h_2 \epsilon W_{2, f_2} - \\
- \lambda_2 (1 + \tau^m) m_{2, W_2} W_{2, f_2} - \lambda_3 (1 + \tau^m) m_{3, W_3} W_{3, f_2} - \\
- \lambda_2 (1 + \tau^f) = 0 \tag{28} \]

Finally, during the last period of life, the agent takes into account only the effects of food consumption on current utility and BMI:

\[ \beta^2 P_2 (W_2) u_{3, f_3} + \beta^2 P_2 (W_2) \Omega_{3, W_3} W_{3, f_3} \\
- \lambda_3 (1 + \tau^m) m_{3, W_3} W_{3, f_3} - \lambda_2 (1 + \tau^f) = 0 \tag{29} \]

Equivalently, the labour supply decision of the agent takes into account the effects of labour supply on BMI and the indirect effects as discussed above. Thus, the first order condition with respect to labour supply for the first period is denoted as:

\[ u_{1, h_1} + \Omega_{1, W_1} W_{1, h_1} + \beta P_{1, W_1} W_{1, h_1} u_2 (c_2, f_2, h_2, \Omega_2) + \\
+ \beta P_1 (W_1) (\Omega_{2, W_2} W_{2, h_1}) + \\
+ \lambda_1 (1 - \tau^w) \omega h_1 \epsilon W_{1, h_1} + \lambda_1 (1 - \tau^w) \omega \epsilon (W_1) \\
+ \lambda_2 (1 - \tau^w) \omega h_2 \epsilon W_{2, h_1} - \\
- \lambda_1 (1 + \tau^m) m_{1, W_1} W_{1, h_1} - \lambda_2 (1 + \tau^m) m_{2, W_2} W_{2, h_1} = 0 \tag{30} \]

And for the second period:

\[ \beta P_1 (W_1) u_{2, h_1} + \beta P_1 (W_1) \Omega_{2, W_2} W_{2, h_2} + \beta^2 P_{2, W_3} W_{3, h_2} u_3 (c_3, f_3, h_3, \Omega_3) + \\
+ \beta^2 P_2 (W_2) (\Omega_{3, W_3} W_{3, h_2}) + \\
+ \lambda_2 (1 - \tau^w) \omega h_2 \epsilon W_{2, h_2} + \lambda_2 (1 - \tau^w) \omega \epsilon (W_2) \\
- \lambda_1 (1 + \tau^m) m_{2, W_2} W_{2, h_2} - \lambda_3 (1 + \tau^m) m_{3, W_3} W_{3, h_2} = 0 \tag{31} \]
## 8 Appendix B

### Table 7: Calibration Parameters

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<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<td>Retirement Age: $J_R$</td>
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<tr>
<td>Maximum Age: $J$</td>
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<td>By assumption</td>
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<td><strong>Preferences</strong></td>
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<td>CRRA Consumption: $\gamma$</td>
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<td>Conesa et al. (2009)</td>
</tr>
<tr>
<td>CRRA Food: $\sigma$</td>
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<td>By assumption</td>
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<td>CRRA Labour Supply: $\eta$</td>
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<td>Conesa et al. (2009)</td>
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<tr>
<td>Pref. Par. Food: $\phi$</td>
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<td>Capital Share: $\alpha$</td>
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<td>Depreciation: $\delta$</td>
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<td>Endog. Productivity: $\zeta$</td>
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<td><strong>Government</strong></td>
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<td>Capital Income Tax: $\tau^r$</td>
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<td>Food Consumption Tax: $\tau^f$</td>
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<td>Pension Replacement Rate: $\chi$</td>
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Table 5: Occupational Classification and Metabolic Equivalent Task

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<th>Standard Occupational Classification (SOC2010)</th>
<th>% of Workforce</th>
<th>MET</th>
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<td>Corporate managers and directors</td>
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<td>Other managers and proprietors</td>
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<td>1.73</td>
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<tr>
<td>Science, research, engineering and technology</td>
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<td>Health professionals</td>
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<td>Teaching and educational professionals</td>
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<tr>
<td>Business, media and public service professionals</td>
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<tr>
<td>Science, engineering and technology associate professionals</td>
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<td>Health and social care associate professionals</td>
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<td>Elementary administration and service occupations</td>
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Notes: The percentage of workforce employed in each Standard Occupational Classification are calculated from the pooled data of the Health Survey for England, 2011-2014. The values for the Metabolic Equivalent Task are assigned manually to the closest US classification as derived by...
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<th>(6)</th>
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<th>(8)</th>
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<tr>
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<td>1.253***</td>
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<td>(-3.22)</td>
</tr>
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<td>Med. Educ. Qual.</td>
<td>-1.284***</td>
<td>-1.296***</td>
<td>-1.166***</td>
<td>-1.168***</td>
<td>0.0220</td>
<td>0.0132</td>
<td>-0.00531</td>
<td>-0.00472</td>
</tr>
<tr>
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<td>(-9.78)</td>
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<td>(-8.87)</td>
<td>(-8.87)</td>
<td>(0.27)</td>
<td>(0.16)</td>
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<td>-1.889***</td>
<td>-1.669***</td>
<td>-1.665***</td>
<td>0.299**</td>
<td>0.289*</td>
<td>0.250*</td>
<td>0.248*</td>
</tr>
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<td>(-10.39)</td>
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<td>(-9.21)</td>
<td>(-9.18)</td>
<td>(2.58)</td>
<td>(2.50)</td>
<td>(2.15)</td>
<td>(2.13)</td>
</tr>
<tr>
<td>Children</td>
<td>-0.343*</td>
<td>-0.382**</td>
<td>-0.383**</td>
<td>-0.383**</td>
<td>-0.187*</td>
<td>-0.180*</td>
<td>-0.179*</td>
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<td></td>
<td>(-2.30)</td>
<td>(-2.58)</td>
<td>(-2.58)</td>
<td>(-2.58)</td>
<td>(-2.04)</td>
<td>(-1.96)</td>
<td>(-1.95)</td>
<td>(-1.95)</td>
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<tr>
<td>Smoker</td>
<td>-1.571***</td>
<td>-1.569***</td>
<td>-1.569***</td>
<td>-1.569***</td>
<td>0.285**</td>
<td>0.285**</td>
<td>0.285**</td>
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</tr>
<tr>
<td>Urban</td>
<td>-0.0698</td>
<td>(0.54)</td>
<td></td>
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<tr>
<td>Constant</td>
<td>47.76***</td>
<td>47.97***</td>
<td>48.97***</td>
<td>49.03***</td>
<td>3.403***</td>
<td>3.472***</td>
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<tr>
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<td>(41.64)</td>
<td>(41.70)</td>
<td>(42.50)</td>
<td>(42.37)</td>
<td>(4.71)</td>
<td>(4.80)</td>
<td>(4.48)</td>
<td>(4.46)</td>
</tr>
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</table>

Notes: Data are obtained from the Health Survey For England (2011-2014). The regressor for regression (1)-(4) is the WEMWB scale and for (5)-(8) the GHQ scale. Year dummies are included throughout.
References


ONS. 2017. “Sickness absence in the labour market.”


