The global health impacts of future food scenarios

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Abstract

The global food system is highly complex. It is both influenced by and has an influence on health, economic development and the environment. It will face considerable strain over the coming decades as income growth, urbanisation and globalisation lead to shifts towards Western dietary patterns (e.g. high in meat and processed foods) across the developing world. Under these conditions, the challenge is to find a sustainable method to feed the world's increasing population, whilst considering the trade-offs and synergies between health, environmental sustainability and economic development.

One approach is to build global health, environmental and development 'modules' that can be coupled to the GTAP database and computable general equilibrium models to estimate the impact of food trade scenarios on these domains. Here we present a multi-state Markov health module that represents the transition of a population cohort in yearly increments. The cohort is divided into four different weight categories: underweight, normal, overweight, obese. The state transition parameters are based on weight-specific risk parameters of all-cause mortality and mortality related to the consumption of specific food categories. The model allows for the calculation of life expectancy and changes in life expectancy resulting from changes in food consumption and a cohort's weight distribution. The model is calibrated with data from the World Health Organisation (WHO) on age-specific mortality rates and body-weight distributions for 194 countries. Relative risk parameters of food consumption and weight category are inferred from meta-analyses of prospective cohort studies.

We outline how the health module can be coupled to economic trade models in order to analyse the global health impacts of future food scenarios. For that purpose, we apply the health module to the global food scenarios for the year 2050 that have been produced by the International Food Policy Research Institute (IFPRI). The health module's output estimates the changes in life expectancy by region for the different food scenarios. The output complements the results reported by economic trade models which commonly focus on sectoral production and consumption-based welfare. We close by highlighting the possibilities of establishing a dynamic link between the health module and economic trade modules that would allow for feedbacks between the two model frameworks.

1. Introduction to the global food system

The global food system is highly complex. It is both influenced by and has an influence on health, economic development and the environment. It will face considerable strain over the coming decades as income growth, urbanisation and globalisation lead to shifts towards Western dietary patterns (e.g. high in meat and processed foods) across the developing world. Under these conditions, the challenge is to find a sustainable method to feed the world's increasing population, whilst considering the trade-offs and synergies between health, environmental sustainability and economic development.

One approach is to build global health, environmental and development 'modules' that can be coupled to the GTAP database and computable general equilibrium models to estimate the impact of food trade scenarios on these domains. Here we present a multi-state Markov health module that represents the transition of a population cohort in yearly increments. The aim of the model is to produce comparable health outcomes for most countries worldwide, using only data inputs that are either readily available for all countries or produced by a global food trade model. Although national Markov-type health models are used regularly (e.g., Scarborough et al., 2011, 2012), no such model us currently available with global coverage.

2. Description of the global health module

Markov models have become a standard tool in decision-analytical modelling of health-care interventions and chronic diseases (Beck and Pauker, 1983; Sonnenberg and Beck, 1993; Briggs and Sculpher, 1998; Textbook by Briggs et al., 2007). They are used for describing stochastic processes, which is particularly useful in the medical field for describing the progression of chronic diseases. The general modelling approach is to divide the process to be modelled into distinct, mutually exclusive states, and to assign transition probabilities to possible movements between those states that can occur during each time interval or model cycle (Beck and Pauker, 1983; Briggs and Sculpher, 1998).[1]

Here we build a population-based and time-dependent Markov model to represent the life course of a population cohort for each WHO member state. Besides age-specific rates of all-cause mortality, we introduce weight and diet-specific risk parameters and specific causes of death associated with those. This allows us to analyse the impacts of changes in diets and weight distribution on the disease progression in each region.

[1] In each cycle, the conditional probability distribution of future states depends only on the present state, something referred to as Markovian assumption (Markov, 1954). This 'memoryless' feature of Markov models can be circumvented in practice by using a combination of distinct states to model particular histories and by using time-dependent transition probabilities (Briggs and Sculpher, 1998).

2.1. Analytical model representation

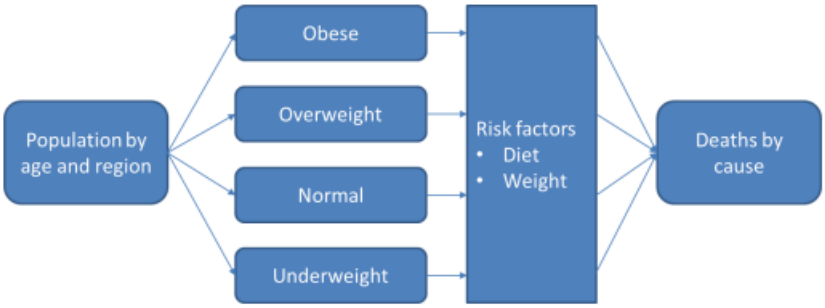

Figure 1. Schematic of modelling approach.

Figure 1 shows a schematic of our modelling approach. Using data from the UN Population division, we first divide each country's population into specific age groups of five-year intervals. We then use WHO data on mean body mass index (BMI) and overweight to calculate the body mass distribution in each region (the concrete method is outlined below) and further divide each population and age cohort into four specific weight categories: underweight, normal weight, overweight, and obese. Based on meta-analysis of prospective cohort studies, we assign weight and diet-specific risk parameters to each model state. Finally, we adopt age-specific mortality rates from the WHO, and we use data from the Global Burden of Disease study to separate the diseases and mortality rates associated with the weight and diet-specific risk parameters from all-cause mortality.

The dynamic parameters of the state transition model are population, risk-specific death rates, and deaths. Table 1 details their specification. We use the following subscripts to denote the dimensions of model parameters: *r* for region-specific, *a* for age-specific, *w* for weight-specific, *c* for cause-of-death-specific, and *f* for food-specific parameter dimensions.

Table 1. Model equations.

- **Initial states:**

$$POP^{0}_{r,a,w} = popdata_{r,a}\ BMIdistr_{r,a,w};\quad DEAD^{0}_{r,a,c} = 0$$

$$DR^{0}_{r,a,w,c} = \prod_f cr^{cc_{r,f}}_{r,f,c}\ \frac{wr_{r,a,w,c}\ \sum_w POP^{0}_{r,a,w}}{\sum_w POP^{0}_{r,a,w}\ wr_{r,a,w,c}}\ dr_{r,a=0,c}$$

- **State transitions** $(i \to i+1; a \to a+1)$**:**

$$POP^{i+1}_{r,a+1,w} = (1 - \sum_c DR^{i}_{r,a,w,c})\ POP^{i}_{r,a,w}$$

$$DR^{i+1}_{r,a+1,w,c} = \prod_f cr^{cc_{r,f}}_{r,f,c}\ \frac{wr_{r,a+1,w,c}\ \sum_w POP^{i+1}_{r,a+1,w}}{\sum_w POP^{i+1}_{r,a+1,w}\ wr_{r,a+1,w,c}}\ dr_{r,a+1,c}$$

$$DEAD^{i+1}_{r,a,c} = DEAD^{i}_{r,a,c} + \sum_w DR^{i}_{r,a,w,c}\ POP^{i}_{r,a,w}$$

- **Outcomes:**

$$YLL_r = \sum_{a,i,c}\ (DEAD^{i,scen1}_{r,a,c} - DEAD^{i,scen2}_{r,a,c})$$

$$LEXP_{r,a} = a + 0.5 + \sum_{a>a-1,g} POP_{r,a,w} / \sum_w POP_{r,a,w}$$

The population of region *r*, age *a*, and weight *w* is computed by multiplying the region and age-specific population data ($popdata_{r,a}$) by the BMI distribution in each region ($BMIdistr_{r,w}$) (Eq.1). The death rate at iteration *i=0* is composed of age and cause-specific

mortality rates ($dr_{r,a=0,c}$) multiplied by food and cause-specific consumption risk factors (*cr*) that are exponentiated by consumption changes (*cc*), and by weight risk factors (*wr*) normalized by population in each weight category (Eq.2). The model is initialized with zero deaths in its first iteration (*i=0*) (Eq.3).

In each following iteration ($i \rightarrow i+1$), people age ($a \rightarrow a+1$. The evolution of the dynamic parameters is described by eqs 4-6. The population alive decreases by the number of people who die ($\sum_w DR^i_{r,a,w,c} POP^i_{r,a,w}$) (Eq.4); the number of deaths are composed of the people who die in the previous period and of all previous deaths (Eq.6); and the death rate in each iteration and age group depends on the population and risk-specific parameters in the same iteration and age group.

2.2. Data

We use a variety of publically available data sources to calibrate the model and set the levels of its exogenous parameters. The exogenous parameters of our health model are initial population numbers, weight distributions, cause-specific death rates, and weight and diet-specific risk parameters. Several assumptions had to be made to harmonize the data sources. Those assumptions and the data sources are described below.

Population data

We adopt region and age-specific population data for the year 2010 from the United Nations Population Division. The data is available separately for male, female, and both sexes, and it is provided in five-year age increments. We adopt the data for both sexes and interpolate the five-year data to fit a one-year cycle length by assuming an even distribution across each five-year interval.

Weight and diet-specific risk factors

Table 2. Causes of death associated with risk parameters.

Cause of death	Global death ratio (%)	Weight-sensitive	Diet-sensitive
Ischaemic heart disease	12.75%	x	x
Stroke	10.81%	x	x
Respiratory disease	6.26%	x	
Other vascular disease	3.12%	x	
Lung and upper aerodig cancer	2.44%	x	x
Diabetes	2.21%	x	x
Liver and gallbladder cancer	1.50%	x	
Stomach cancer	1.33%		x
Colorectal cancer	1.14%	x	x
Breast cancer	0.85%	x	
Oesophagus cancer	0.73%		x
Mouth and oropharynx cancers	0.49%		x
Prostate cancer	0.48%	x	
Pancreatic cancer	0.47%	x	x
Ovarian cancer	0.25%	x	x
Total	44.84%		

We identified a range of weight and diet-specific risk factors for specific causes of deaths from reviews and meta-analyses of prospective cohort studies. Table 2 provides an overview of those weight and diet-sensitive causes of deaths and their relative contributions to all global deaths. According to the Global Burden of Disease study, coronary (ischaemic) heart disease caused about 13% of deaths worldwide in 2008, a selection of various types of cancer caused 12%, stroke 11%, and respiratory disease 6%. Our disease-specific coverage allows us to account for the weight and diet-sensitivity of 45% of all deaths.

We adopt the diet-specific risk factors related to cancer from the Second Expert Report "Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective" published in 2007 by the World Cancer Research Fund together with the American Institute for Cancer Research. The report is based on reviews and meta-analysis of over 7,000 scientific studies published on cancer prevention. It is the outcome of a 5-year project which involved a panel of 21 leading scientists (experts) and 9 research centres around the world. The relative risks related to ischaemic heart disease and stroke are adopted from dedicated meta-analyses of the existing literature (Dauchet et al., 2005, 2006; Micha et al., 2010). Table 3 provides an overview of the values adopted.

Table 3. Diet-specific risk factors

Cause of death	F-V	Meat
Ischaemic heart disease	0.96	1.27
Stroke	0.95	1.24
Diabetes	x	1.12
Mouth and oropharynx cancers	0.72	x
Oesophagus cancer	0.72	x
Stomach cancer	0.69	1.13
Lung and upper aerodig cancer	0.74	1.60
Pancreatic cancer	0.88	1.11
Colorectal cancer	0.84	1.25
Ovarian cancer	0.64	x

There are various estimates of weight-related mortality (e.g., Flegal et al., 2007; Berrington de Gonzalez et al., 2010; PSC, 2009). Some general problems of studies analysing the relationship between weight and mortality are that it is hard to isolate causal relationships due to confounding factors (e.g. smoking is associated with less body weight, but with higher mortality), there is little data for studying the association of underweight and mortality, and the choice of weight categories does not always overlap across studies.

We adopt the weight-specific risk factors from a large collaboration of prospective cohort studies which covers 900,000 adults in different regions of the world (Prospective Studies Collaboration, 2009). The benefit of that study over others is that pooled data from several cohort studies, isolated the weight-sensitive relative risks of dying from specific causes, and agreed on likely causal risk parameters. The assumed causal risk parameters are shown in Table 3. We adopt those data in a conservative manner by assigning the relative risks for

obesity category 1 to all obesity and the most beneficial category to our normal category. Since no assumptions have been made regarding the underweight category, we assume unchanged relative risks for that category. However, we analyse the sensitivity to those assumptions in dedicated sensitivity analyses.

Table 3. Weight-specific risk factors

Cause of death	Age at risk (years)	Relative risk for baseline BMI range				
		22.5-25 normal	25-30 overweight	30-35 obese	35-40 obese II	40-50 obese III
Ischaemic heart disease	35-59	0.73	1.00	1.48	2.25	3.70
Ischaemic heart disease	60-69	0.83	1.00	1.47	1.94	2.92
Ischaemic heart disease	70-79	0.84	1.00	1.40	1.70	2.36
Stroke	35-59	0.95	1.00	1.72	3.10	6.19
Stroke	60-69	0.82	1.00	1.52	2.20	3.57
Stroke	70-79	0.95	1.00	1.35	1.75	2.47
Other vascular disease	35-59	0.89	1.00	1.83	2.81	5.30
Other vascular disease	60-69	0.82	1.00	1.57	2.52	4.45
Other vascular disease	70-79	0.82	1.00	1.28	1.88	2.78
Diabetes	35-79	0.58	1.00	2.57	4.90	13.05
Liver and gallbladder cancer	35-79	0.74	1.00	2.05	2.41	4.19
Colorectal cancer, male	35-79	0.83	1.00	1.23	1.53	2.00
Colorectal cancer, female	35-79	0.91	1.00	1.21	1.24	1.33
Female breast cancer	35-59	1.00	1.00	1.00	1.00	1.00
Female breast cancer	60-79	0.75	1.00	1.22	1.27	1.58
Ovarian cancer	35-79	0.87	1.00	1.01	1.31	1.50
Prostate cancer	35-79	0.89	1.00	1.12	1.23	1.39
Pancreatic cancer	35-79	0.85	1.00	1.12	1.17	1.28
Lung and upper aerodig cancer	35-79	1.11	1.00	0.96	0.92	0.88
Respiratory disease	35-79	1.09	1.00	1.10	1.51	1.95

Weight distribution

There is no complete data on a country's weight distribution. We therefore calculate region-specific weight distributions based on WHO data for the year 2008 on mean BMI, percentage overweight (BMI > 25), and percentage obese (BMI > 30) assuming weight is distributed according to a log-normal distribution. This assumption is supported by studies of Penman and Johnson (2006), Swinburn and Egger (2004), and Murphy (1979).[2] We use two of the data points to fit to a log-normal distribution, and the third point to assess the accuracy of fit. Using percentage overweight and obese as fit parameters yields a deviation of mean BMI of about 20%, whilst using percentage overweight and mean BMI as fit parameters yields a deviation of percentage obese of 40% on average. Given the sensitivity of weight and diet-specific risk parameters to the categories of overweight and obesity, we choose the former constellation of fit parameters in our main parameterization. We assign the country-specific weight distribution obtained in that way to each age category within a country except for

[2] Penman and Johnson (2006) state that "the growth of living tissues likely proceeds by multiplicative effects, and measures of body size (such as BMI) are [,,,] likely to follow a skewed, possibly log-normal, distribution (Murphy, 1979; Swinburn and Egger, 2004).

children aged less than five for which we adopt WHO data on underweight in children aged less than five years.[3]

Mortality rates

We adopt age-specific all-cause death rates from the WHO for the year 2011 (other years available are 1990 and 2000) and use data from the Global Burden of Disease (GBD) study to separate disease-specific mortality from all-cause mortality. We separate out only those specific diseases that are sensitive to weight and diet conditions as discussed above. The GBP data lists deaths by cause and (coarse) age class (0-14, 15-59, 60+) for the year 2008. The cause-specific deaths rates are obtained by computing age-specific death-from-disease ratios and multiplying those by the WHO all-cause death rates, identifying the complement as death rate from all other causes:

$$dr_{r,a,c} = \frac{deaths_{r,a,c}}{deaths_{r,a,total}} dr_{r,a}$$

$$dr_{r,a,c=other} = (1 - \sum_{c \neq other} \frac{deaths_{r,a,c}}{deaths_{r,a,total}}) dr_{r,a}$$

3. Model scenarios and results

We illustrate the workings of the model by devising different implementations. First, we run the model as a cohort model, i.e., we populate the model with only one cohort aged zero and follow its transition from birth to death. This allows us to calculate life expectancies for that cohort. Second, we run the model as a population model, i.e., we calibrate all age classes to actual population data right from the start. We cannot follow one cohort in this setup and therefore are not able to calculate life expectancies. But instead we can calculate the years of life lost due between different policy interventions (see Table 1 – Outcomes).

3.1. Model scenarios

Table 4. Model scenarios

Scenario analysis (for illustration):

- CON: Increase in fruit and veg consumption, decrease in meat consumption; one serving per day each.
- DEV: 20% decrease in prevalence of underweight; move into normal weight category.
- OVW: 20% increase in prevalence of overweight; move from normal weight category.

[3] Children malnutrition still constitutes a major problem in several developing countries. We adopt WHO data on underweight in children aged less than five years to capture the dynamic feedbacks between children malnutrition and the associated increased risk of deaths. We adopt a relative risk of dying for underweight infants of 4.24 based on Fishman et al. (2004).

We implement three stylized policy scenarios to illustrate the model response – see Table 4. Those are a consumption scenario (CON) which increases fruit and vegetable consumption by one serving per day and decreases meat consumption by one serving per day; a development scenario (DEV) in which the prevalence of underweight is reduced by 20% and the change absorbed by the normal-weight category; and an overweight scenario (OVW) in which the prevalence of overweight is increased by 20% at the cost of the normal-weight category.

3.2. Model results

Figures 2 shows the transition diagram for the global cohort model in the benchmark scenario (without policies). Initially, at iteration zero, the population is divided into underweight, normal, overweight, and obese individuals. As iterations progress, people in each category die until the complete population is dead at iteration 100. Figure 3 provides additional detail by disaggregating the deaths in each iteration by cause.

The data from such transition diagrams can be used to calculate life expectancy at birth (or at other ages) by adding the current life year (corrected by one half) to the expected life years weighted by all life years (see Table 1 – Outcomes). Figures 4 and 5 show the life expectancies at birth and the change in those for the different policy scenarios in different model regions. The model regions correspond to those of the WHO.

Figures 6 to 9 illustrate how the model output changes when implemented as a population model. The output variable then becomes life of years lost which are shown in Figure 6 for each policy scenario. The transition diagram becomes more linear as individuals who are older at iteration zero die earlier. Similar changes can be observed when disaggregating the cause of death in Figure 8. Because age classes are completely populated from the start in the population model, one can also look at the causes of death by age instead of by iteration – those are depicted in Figure 9.

4. Outlook

The next working steps (which are hopefully completed by presentation date) are (i) to iron out the kinks of the model by comprehensive parameter testing and sensitivity analyses; and (ii) to analyse “realistic” food scenarios by coupling the health model to the International Model for Policy analysis of Agricultural Commodities and Trade (IMPACT). The coupling will take place via changes in population, food consumption, and weight distribution (mediated by changes in daily caloric availability).

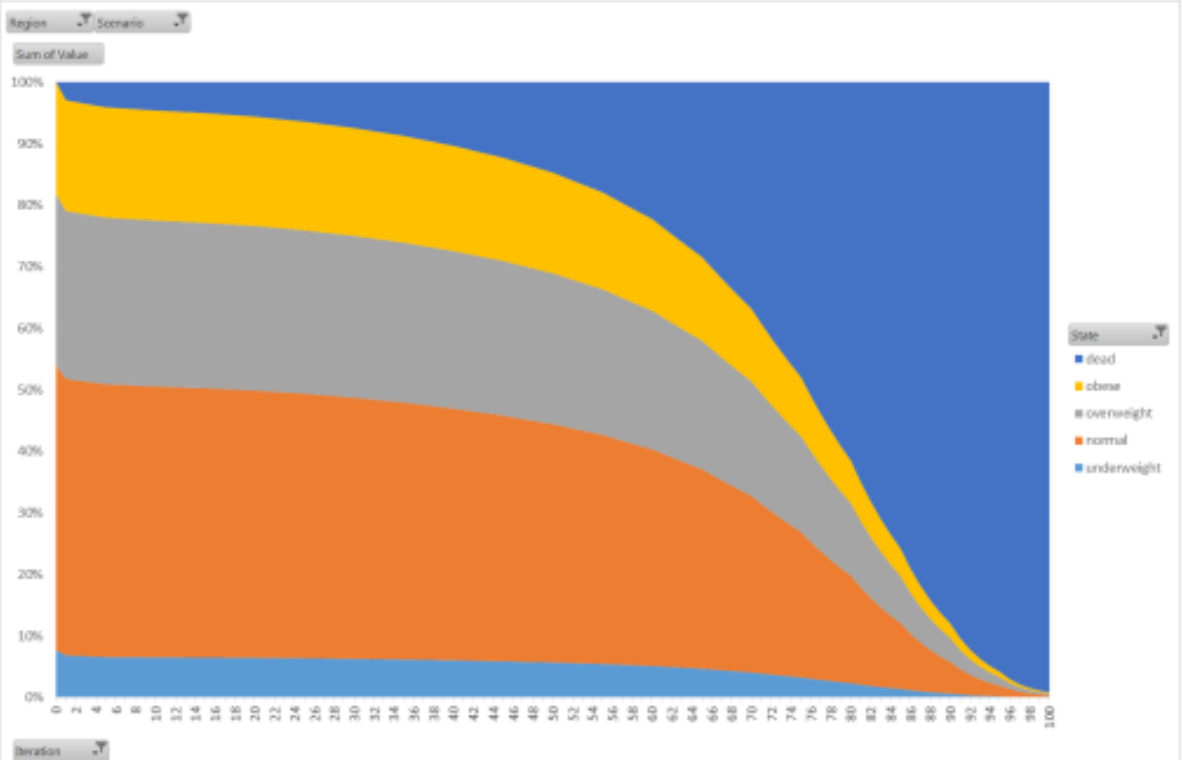

Figure 2. Transition diagram in benchmark (no-policy) scenario.

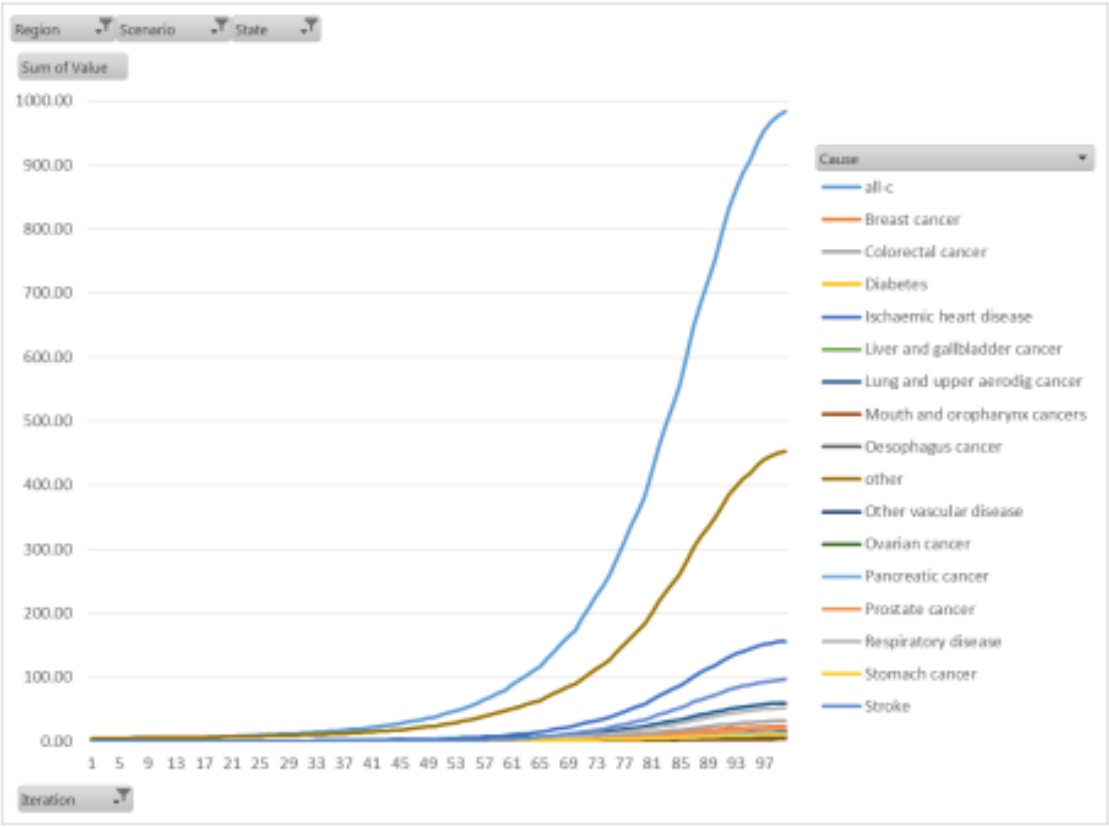

Figure 3. Deaths by cause and iteration.

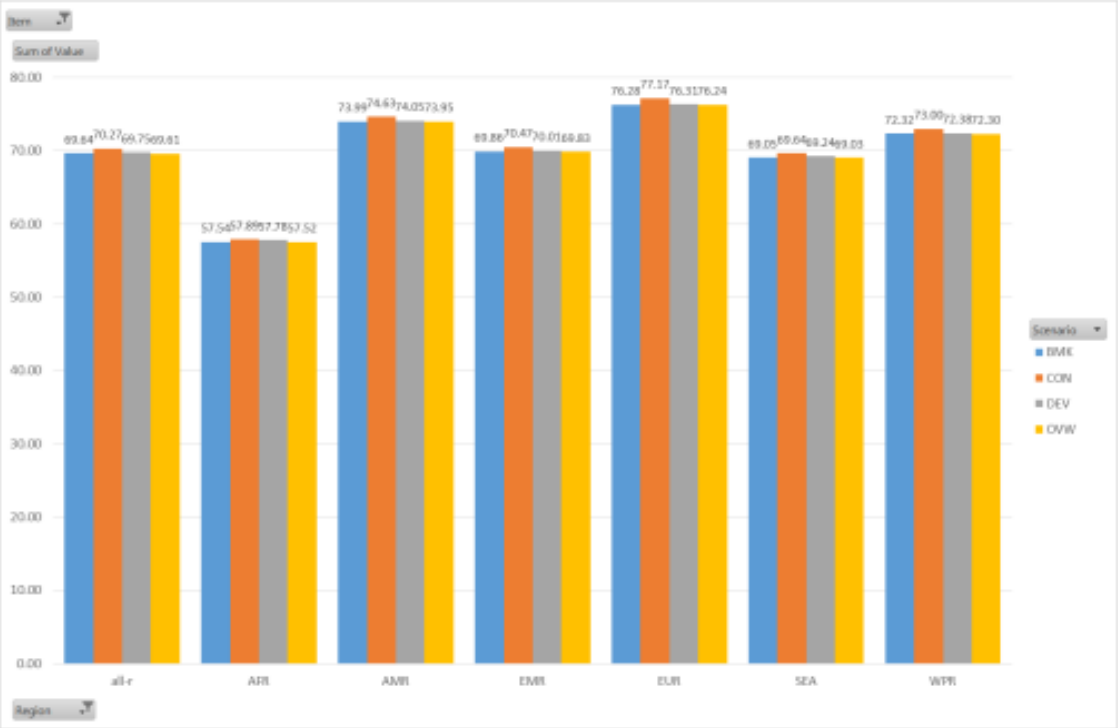

Figure 4. Life expectancies in model scenarios.

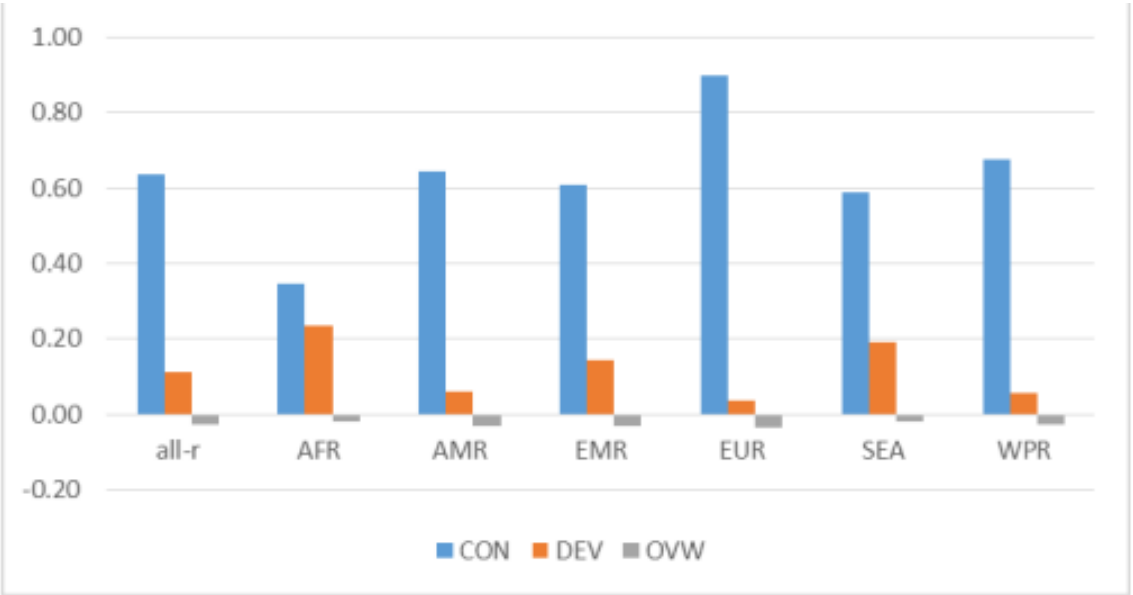

Figure 5. Difference in life expectancies between the model scenarios and the no-policy benchmark

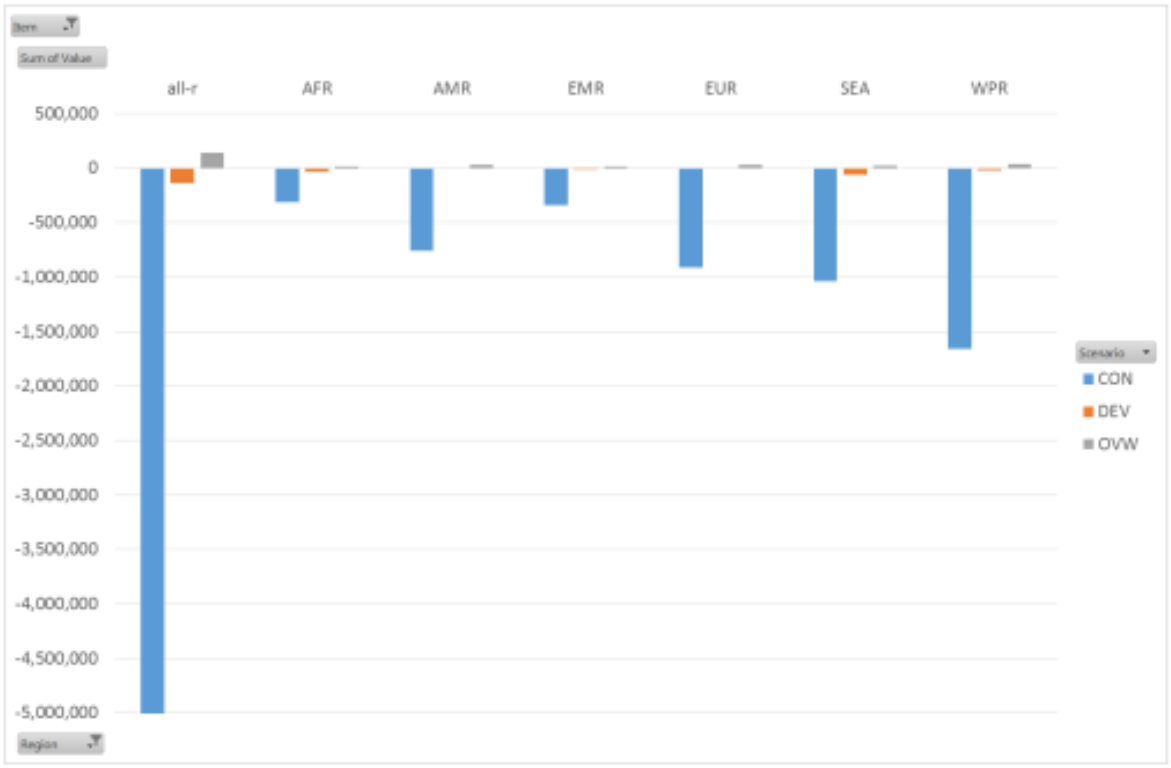

Figure 6. Years of life lost in the different policy scenarios in population model

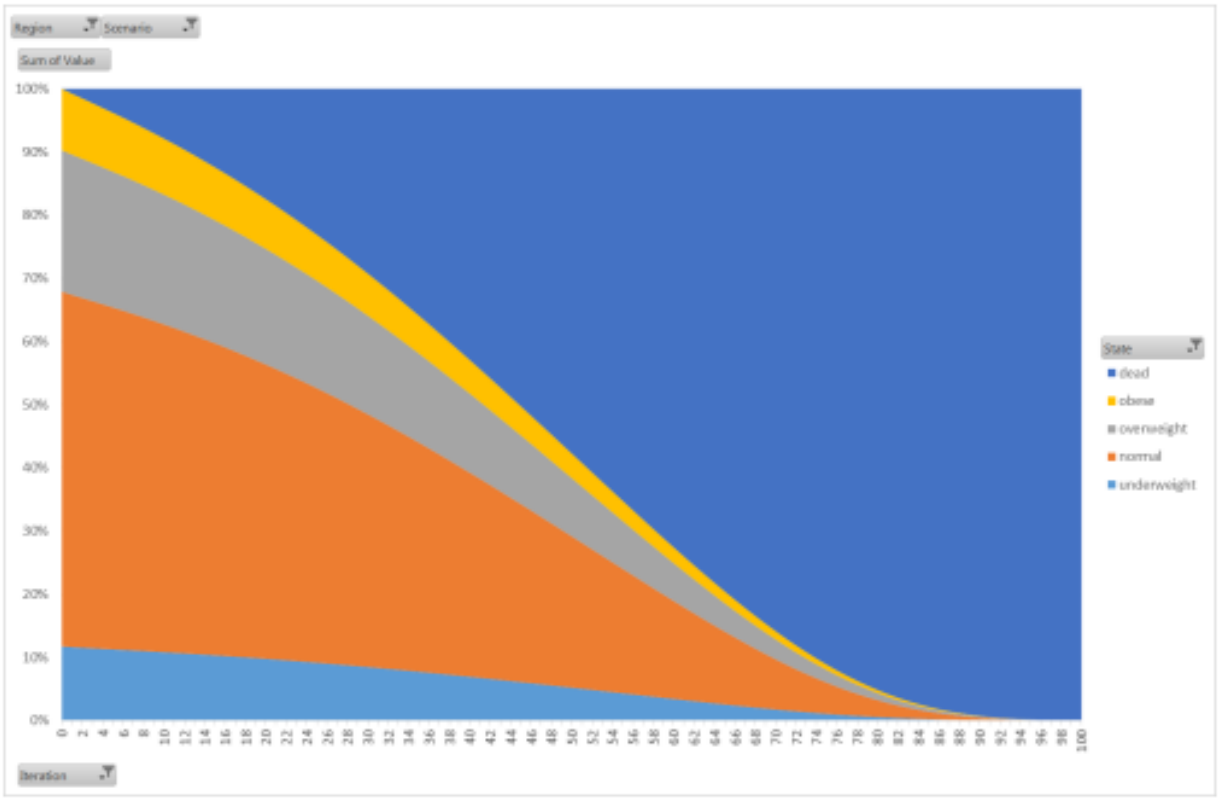

Figure 7. Transition diagram in population model.

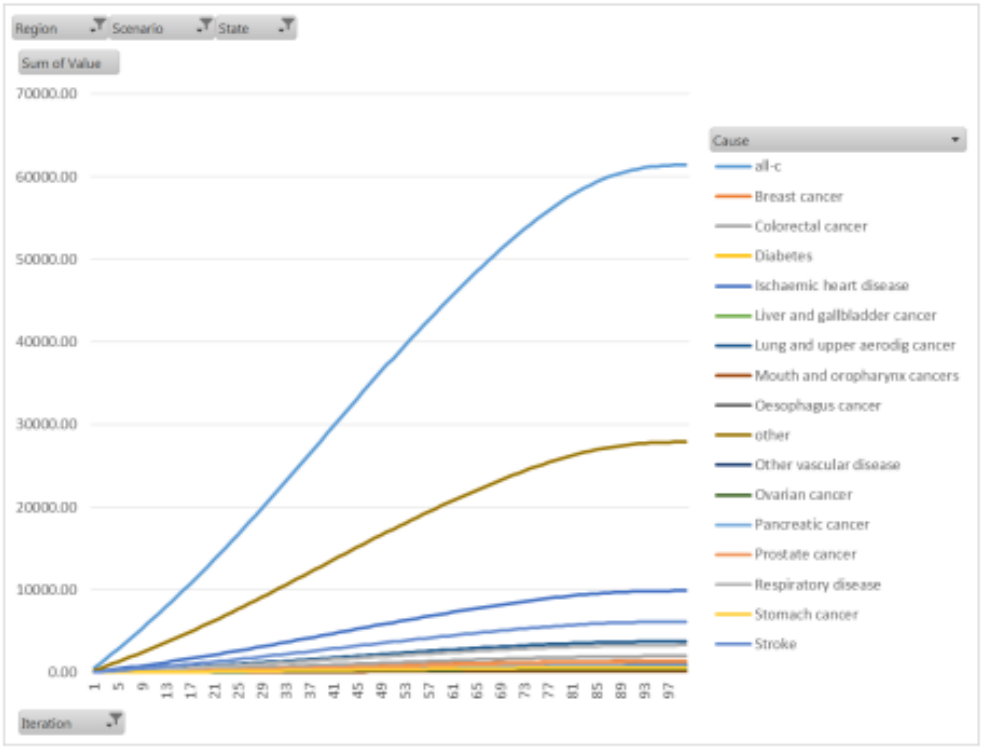

Figure 8. Causes of death per iteration in population model

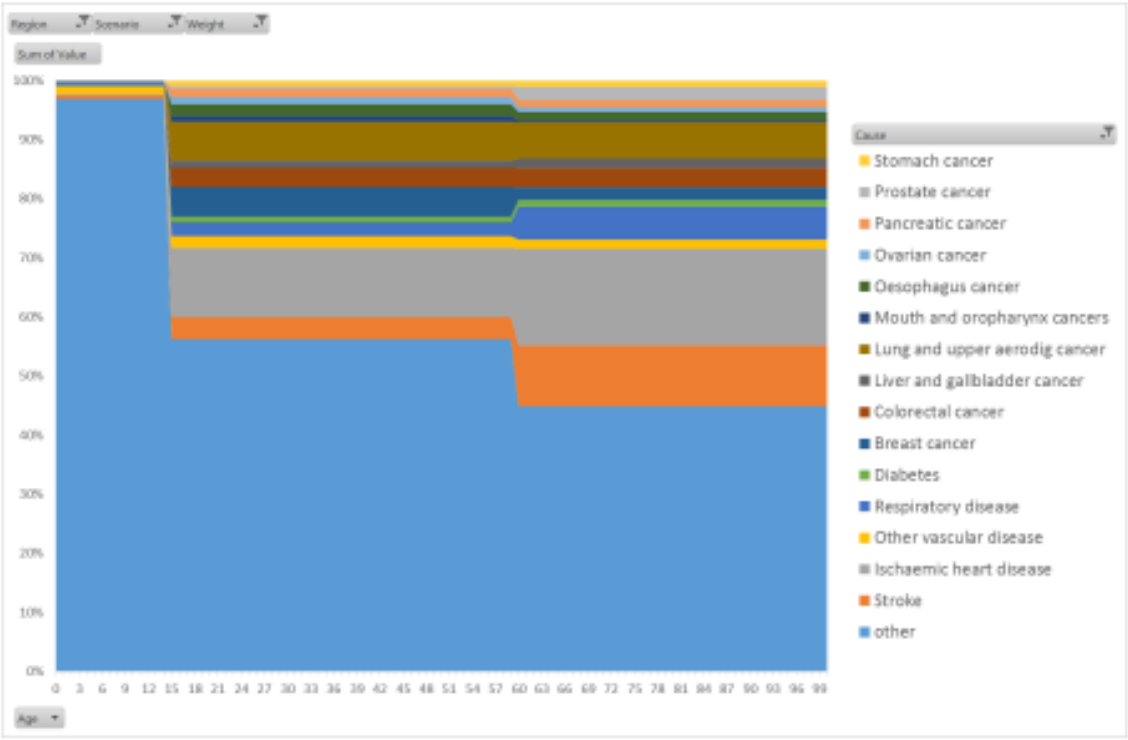

Figure 9. Causes of death by age in population model