Treatment Strategies for the Prevalence of Obesity in Austria Modelled with System Dynamics

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Abstract. In Austria, especially among children, the prevalence of obesity has increased since 1999. Therefore, a System Dynamics model, simulating the prevalence of obesity, has been developed. The modular architecture allows separate analysis of the model parts and also adding and connecting different modules, like for example a cost module or a module for an obesity-related disease to the existing population model and the disease model of obesity. Three interventions, treating obesity with an increase in caloric expenditure and/or a reduction of caloric intake, are tested, analysed and compared. The results show that a reduction of caloric intake reduces the prevalence of obesity and overweight until 2050 (intervention 1). Furthermore, a reduction for the doubled amount of kilocalories per day doesn't have the doubled effect on the reduction of the prevalence (intervention 2 compared to 1). Last, but not least, intervention 3 compared to intervention 2 shows that physical activity together with a reduction of caloric intake reduces the prevalence much more than a single reduction of caloric intake in the amount of kilocalories due to physical activity and eating less together.

Introduction

Obesity is a health concern of paramount importance. Especially in Austria more than one third of the population is obese or overweight and not only the adult population suffers from this disease, but also children [1]. It has long list of co-morbidities such as orthopeadic problem or cancer. Obesity is measured by the *Body Mass Index* (BMI) that is calculated by dividing body mass in kilogram through the square of body height in centimetre, as seen in equation (1).

$$BMI = \frac{body\ mass\ [kg]}{body\ height^2\ [cm]} \tag{1}$$

The main classification for adults according to the World Health Organization [2] is shown in Table 1.

Classification	BMI
underweight	< 18,50
normal weight	18,50 – 24,99
overweight	25,00 – 29,99
obese	≥ 30,00

Table 1. Classification of obesity and overweight according to the WHO [2] measured through the BMI.

For children the percentiles of the reference curves according to Kromeyer-Hauschild [3] are used and therfore the classification is slightly different.

The model's architecture is modular consisting of a population module that simulates the demographic development and of a disease module simulating the prevalence of the disease over time. Furthermore a connection in eating and physical activity habits of children to that of their parents is implemented as well [4].

There are three interventions that are compared:

1. A **reduction of the caloric intake of 80 kcal/day** in the adult obese and overweight population
2. A **reduction of the caloric intake of 160 kcal/day** in the adult obese and overweight population
3. A **reduction of the caloric intake of 80 kcal/day** and an additional increase in physical activity by an **increase of caloric expenditure of 80 kcal/day** in the adult obese and overweight population

It is researched if a reduction of the doubled amount of caloric intake reduces the prevalence of obesity twice as much (comparison of 1 and 2). Also the effect of physical activity in comparison to the effect of a reduction of caloric intake is evaluated (2 compared to 3).

1 Method: System Dynamics

System dynamics was originally developed in 1951 by J. Forrester [5] who used this top-down modelling technique for complex management systems. He furthermore applied this technique to social systems and used it to simulate complex causal links.

It is a graphic notation and is based on differential equations. Basically, the population is divided into *stocks* that are connected by *flows*. Each stock represents a differential equation in accordance to time *t*. These flows, going in or out of the stock are regulated through a valve that can be dependent on *parameters* or stocks. Additionally *auxiliaries* can be used to represent algebraic equations. A very simple stock and flow diagram can be seen in Figure 1.

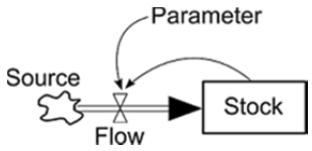

Figure 1. A simple stock and flow diagram. The flow is dependent on the stock and a parameter, represented by the smaller arrows.

Sources are the boundaries of the system. This small example can also be written as the differential equation as seen in equation (2) and (3).

$$\dot{Stock}(t) = Flow\ (t) \tag{2}$$

$$Flow(t) = Parameter\ \cdot Stock(t) \tag{3}$$

These differential equations can also be written as integrals and each stock represents the state of the system. The example in Figure1 can be interpreted as the stock being a population, where in accordance to the fertility rate (being the parameter) births (being the flow) increase the population. The births are calculated by multiplying the fertility rate with the stock.

2 Architecture of the Model

The model is set up **modular**. This means that a *population model* is used for the simulation of the demographic development of the population. A *disease model* simulates the development of obesity over time until 2050. These two parts are connected by a special defined *interface*. This modular setup is very useful, because error analysis is much easier. Other required model parts for cost calculation or co-morbidities can be connected through special defined interfaces.

2.1 Population model

The population is divided into 192 stocks for 96 age classes (0-94, 95+) and two sexes (male, female). Each stock can be changed by flows for *aging*, *death* and *migration*. The stock of the age class 0 additionally can be changed by flows of *births*, as seen in Figure 2.

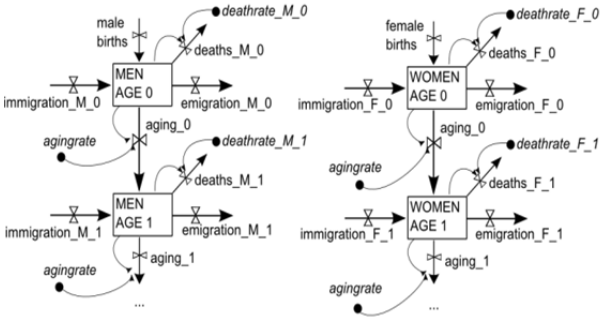

Figure 2. Structure of the population model.

The deaths are dependent on the death rate and the connected stock, which can be seen by the small arrows pointing at the valve of deaths. Migration, on the other side, is not dependent on the size of the population and is given as a net in- respectively out-flow. The births are calculated by multiplying the one-year age specific fertility rates with the stocks of fertile women (aged 15-45). This calculation is not shown in this figure, except for the already calculated total *births (male births, female births*).

The data is provided by Statistics Austria [6]. For death rates *r* the death probabilities *p* from death tables (regular tables for 1999 until 2010 and prognostic tables for the years 2010 until 2050) are used and transformed into rates as seen in equation (4).

$$r = -\ln(1-p) \tag{4}$$

The birth rates are given as one-year *age specific fertility rates*, being the number of total life-births per 1.000 women for the years 1999 until 2010.

For the forecast years 2011 until 2050 there are only the estimated *total fertility* rate, being the number of children a women would birth in her life, if there are the same fertility conditions in her future life as they are now, and the estimated *average fertility age* available. These are used to calculate an estimation of the one-year age specific fertility rates for the forecast years. Data for migration is also provided and estimated by Statistics Austria.

2.2 Disease model

The demographic development is calculated in the population model. Therefore the development of the disease itself is calculated in the disease model. The population is split into ten age classes, sex (because there are differences in the basal metabolic rates for men and women) and the four BMI categories representing the severity of obesity. This means that people can only change between stocks within one age class and sex of adjacent BMI categories as seen in Figure 3.

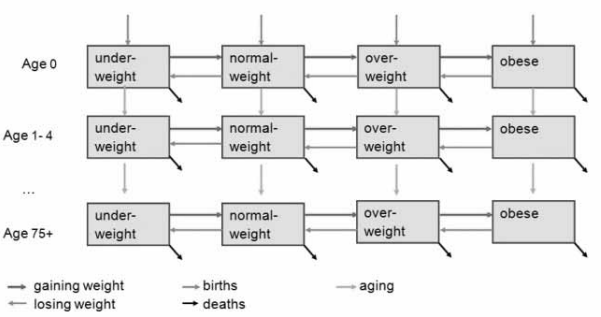

Figure 3. Flows between adjacent BMI categories in the disease model.

The rates, regulating the amount of the flows, stay constant in the base run and change in 2010 if an intervention is tested. An intervention only calculates *caloric* changes. Therefore, the core part of the disease model is the translation of *changes in energy balance* within a stock to *changes of rates* between the BMI-categories between the stocks.

The energy balance is calculated by energy intake minus energy expenditure. The Energy intake is calculated by the kilocalories taken in by eating food or drinking beverages. The energy expenditure EE is calculated as shown in equation (5).

$$EE = BMR + TEF + PA \tag{5}$$

The basal metabolic rate (BMR) depicts the energy the body needs to maintain the basal functions each day at indifference temperature of 28 degrees Celsius and with empty stomach. The estimation of the BMR requires age, weight, height and gender of the person. The Revised Harris-Benedict equations [7] shown in equation 6 and equation 7, are used.

The factors for weight and height are larger for men than for women resulting in a larger BMR for men than for women. The thermic effect of foods (TEF) is the energy that is used to process the food a person eats (usually 10% of the value of caloric intake). Physical activity (PA) also contributes to the energy expenditure.

$$\begin{aligned} BMR_{Men} = {} & 88{,}362\ kcal \\ & + \left(13{,}397\ \frac{kcal}{kg} \cdot \boldsymbol{weight}\ [kg]\right) \\ & + \left(4{,}799\ \frac{kcal}{cm} \cdot \boldsymbol{height}\ [cm]\right) \\ & - \left(5{,}677\ \frac{kcal}{year} \cdot \boldsymbol{age}\ [years]\right) \end{aligned} \tag{6}$$

$$\begin{aligned} BMR_{woman} = {} & 447{,}593 kcal \\ & + \left(9{,}247\ \frac{kcal}{kg} \cdot \boldsymbol{weight}\ [kg]\right) \\ & + \left(3{,}098\ \frac{kcal}{cm} \cdot \boldsymbol{height}\ [cm]\right) \\ & - \left(4{,}330\ \frac{kcal}{year} \cdot \boldsymbol{age}\ [years]\right) \end{aligned} \tag{7}$$

Each stock in the disease model has an average ener-gy balance, calculated as mentioned above. The energy balance in 1999, at simulation start, is given and then used to calculate the change of energy balance (ΔK) throughout the simulation, which is then transformed into a *change of rates* (ΔF) between the BMI-categories as seen in equation 8.

$$\Delta F = MAX\left\{0;\ 0{,}5 \cdot \frac{\Delta K}{(b_c - b_m) \cdot (h_m)^2 \cdot k} \cdot \frac{365}{12}\right\} \tag{8}$$

This equation was adapted from a paper by Homer et al. [8] and includes the BMI-cut-off points b_c to the adjacent BMI-category, the average BMI b_m in each category, the average height h_m in each sex, age class and BMI-category specific stock and furthermore, an empiric estimation of the average kilocalories per kilogram of weight-change k (8.050 kcal/kg) according to Forbes [9].

There is a correlation between energy intake and energy expenditure from parents to that of their children. Therefore, a weighted average of energy expenditure, respectively energy intake, over all parentsages (19-64 years) and severity degrees is calculated. The percentage of the caloric change during simulation to the initial value in 1999 is then translated to a change in the energy expenditure, respectively energy intake, within the age classes of the children (0-18 years). For example, if the energy intake of parents increases for 2% then the energy intake of children will increase for 2% as well. This average over all age classes and BMI-categories is used, because there is no data available on how many adults, categorized by their BMI, actually do have children at a specific age.

The weighting of this average value is done in a manner that 'younger' parents with probably 'younger' children have a greater influence on their children than 'older' parents do have on their 'older' children. Furthermore, the percentage of, for example, overweight parents having overweight children is not known as well.

There are two kinds of rates between the BMI-categories, the *upflow rates*, calculating the change of stocks from a lower BMI-category to a higher BMI-category, and the *downflow rates*, calculating the change of stocks from a higher BMI-category to a lower BMI-category (see Figure 3). During the simulation these rates are changed in a manner that was mentioned before by matching the change of energy balance to a change of rates. This means that an initial value of the rates has to be provided. There was no data on changes of weight within a year for the Austrian population available that would describe weight gaining or weight losing. Therefore the downflow rates are estimated out of data from the National Health and Nutrition Survey (NHANES) [10] from the USA. This dataset includes person specific data for age, weight, height, sex and, most important, for the estimated weight one year ago. Out of this data the BMI and the BMI one year ago can be calculated and used for an estimation of the rates for a downwards BMI-category change. The upflow rates in the model were then calibrated. A dataset depicting the state of the system, meaning the distribution of BMI-category within the population in 2006/2007 [11], was used for minimizing the objective function, the difference between the simulated prevalence of obesity in Austria and the actual prevalence in 2006/2007.

2.3 Interface

The population model and the disease model are connected by an interface that calculates the changes of the stocks due to births, aging, migration and deaths in the population model and transfers them to the stocks of the disease model. The structure of the population model differs from the structure of the disease model. The first one has 96 age classes, 2 sexes and no difference in BMI-category, whereas the latter one has only 10 age classes, 2 sexes, but 4 BMI-categories. Therefore, the changes of the stocks in the population model are aggregated for each sex according to the age classes in the disease model. Then they are split up into the stocks of the BMI-categories in the disease model within this age class and sex.

This is done in a manner that the distribution of the BMI-categories of the changes from the interface show the same distribution of BMI-categories as in the disease model: If there are 50% people normal weight and 20% overweight in the disease model at this time, then the changes of the stocks of the population model are split up and 70% are transferred to the stock of normal weight people and 20% are transferred to the stock of overweight people.

In this model there is no connection from the disease model back to the population model, since no obesity-related deaths are simulated. The reason for that is that there are no direct obesity related deaths. Usually no one dies because of overweight, but sometimes because of obesity *associated* diseases, like problems with the heart. The modular setup allows coupling other model parts simulating co-morbidities. If such a model part, simulating an obesity associated disease, is connected, it is possible to redefine the interface or to define an interface from that model part to the population model and then allow informationflows to the population model and also simulate disease related deaths.

3 Results

The base run shows an increase in the prevalence of obesity and overweight in 2050. According to the simulation there are 338.110 obese male aged 19-64 in 2050, whereas in 1999 there were 217.983 obese male people. Within the female adult population the number increased from 198.933 in 1999 to 326.361 in 2050. Of course this result includes the demographic development as well, but there is still an increase of the prevalence of obesity within the relative numbers.

3.1 Intervention 1: Reduction of caloric intake for 80 kcal per day

If there is a reduction of the caloric intake for 80 kilocalories per day starting in 2010 within the adult obese and overweight population, there is a reduction of the prevalence of obesity and overweight in 2050. The comparison of the base run to intervention 1 is shown in Figure 4 for the obese male and female population together. The results are depicted for some age classes and the vertical axis shows the absolute number of obese people in 2050.

3.2 Intervention 2: Reduction of caloric intake for 160 kcal per day (doubled amount)

In intervention 2 the caloric intake is reduced for 160 kilocalories per day in the obese and overweight adult population. Due to the fact that a dependency between adult eating behaviour and that of children is implemented, a decrease of the caloric intake in adults also reduces the caloric intake within children. The prevalence of obesity and overweight reduces as well as in intervention 1, but the effect of the reduction compared to the effort of reducing the caloric intake for twice as much, is for intervention 2 smaller than for intervention 1, as seen in Table 2.

	Intervention 1 vs. base run	Intervention 2 vs. base run
Children 2 - 18 years	-32%	-44%
Adults 19 - 64 years	-18%	-31%

Table 2. Comparison of the reduction of the prevalence of obesity due to intervention 1 to that due to intervention 2.

In intervention 1 the prevalence of obesity was re-duced for 32% within children, whereas in intervention 2 only a reduction of 44% can be shown, both compared to the base run. This effect is not twice as much as the effort of reducing the doubled amount of caloric intake.

This means that the effect of a decrease of the prevalence of obesity, and it is the same for overweight, reduces relatively to the effort.

3.3 Intervention 3: Reduction of caloric intake for 80 kcal per day and additional increase of physical activity for the amount of 80 kcal per day

In intervention 3 the caloric intake is reduced for 80 kilocalories per day for the obese and overweight population together with an increase of physical activity of the amount of 80 kilocalories of energy expenditure per day. This means that each day people have 160 kilocalories less than they have in the base run. This looks like it is the same reduction of calories as in intervention 2. A comparison of intervention 2 and 3 show that the prevalence of obesity in intervention 3 is reduced as well, but, as seen in Figure 5, additional physical activity instead of eating less has a larger effect on the prevalence of obesity. The prevalence of obesity in intervention 3 is slightly more reduced than the prevalence of obesity in intervention 2.

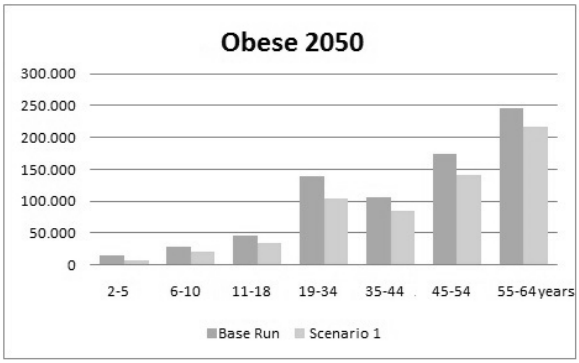

Figure 4. Prevalences of people with obesity in base run compared to intervention 1.

Figure 5. Prevalences of people with obesity in intervention 2 compared to intervention 3 for some age classes.

The reason why intervention 3 has a better effect is the thermic effect of foods: by eating more in inter-vention 3 the thermic effect of foods is higher than that in intervention 2 and therefore the energy balance is reduced slightly more by the thermic effect of foods, although the overall reduction of kilocalories, by physical activity or eating less, is in both interventions the same.

4 Validation

There are a lot of different methods that can be used to validate a model, but nevertheless, a model will never be validated for 100%. In this case there are two validation methods used, a *cross-model* validation for the population model itself and a *probabilistic sensitivity analysis* for the disease model.

4.1 Cross-model validation

The results of the population model are compared to the prognosis data of Statistics Austria and the difference in 2050 is less than 3.3% [4]. This means that the validation of the population model via another model is acceptable.

4.2 Probabilistic sensitivity analysis

A probabilistic sensitivity analysis (PSA) is done for the parameters of the up- and downflow rates which are the parameters with the biggest uncertainty due to the fact that they are partially calculated out of the NHANES study and partially calibrated. The PSA is done in the following way:

- For each of the 120 parameters a set of 400 probabilistic samples is calculated using the original estimateed value as mean of a normal distribution and 10% of the mean as standard deviation.
- For each of the 400 parameter sets a simulation run is done for base run, intervention 1, 2 and 3.
- The prevalences of obesity and overweight for each parameter set for all age classes and male and females are saved.
- Finally, a qualitative comparison of the results for each of the three interventions compared to the base run for each parameter set is done.

The results of the PSA [4] show that for each parameter set all interventions reduce the prevalence of obesity and overweight compared to the base run for every age class and sex. This means that the model output is not dependent on the parameters used in the PSA or that the range in which the parameters are varied is too small.

5 Conclusion

Each intervention leads to a decrease of the prevalence of obesity and overweight. It can be shown that a reduction of caloric intake is effective, but a further reduction is not that effective as the reduction at the beginning. Furthermore, due to the thermic effect of foods, a combined treatment with physical activity and a reduction of caloric intake is more effective than reducing intake.

Furthermore, the modular setup allows a high degree of reusability of model parts. For example the population model can be used in another model where the demographic development of a population is required. The parameterization can be changed to that of another population, if another population is researched. Obesity is a disease with a long list of co-morbidities, like diabetes or hypertension, and the modular setup allows an integration of other model parts that can be connected through another interface.

This also means that other influences can be modelled as well. A cost model, calculating the costs of the disease due to medication or surgery, can also be connected.

This model makes qualitative statements for treatment strategies of obesity and overweight and provides a high degree of reusability for model parts due to the modular setup.

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