REVIEW

# Microsimulation Modeling in Food Policy: A Scoping Review of Methodological Aspects

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**ABSTRACT**

Food policies for the prevention and management of diet-related noncommunicable diseases (NCDs) have been increasingly relying on microsimulation models (MSMs) to assess effectiveness. Given the increased uptake of MSMs, this review aims to provide an overview of the characteristics of MSMs that link diets with NCDs. A comprehensive review was conducted in PubMed and Web of Knowledge. Inclusion criteria were: *1*) findings from an MSM; *2*) diets, foods, or nutrients as the main exposure of interest; and *3*) NCDs, such as overweight/obesity, type 2 diabetes, coronary heart disease, stroke, or cancer, as the disease outcome for impact assessment. This review included information from 33 studies using MSM in analyzing diet and diverse food policies on NCDs. Hereby, most models employed stochastic, discrete-time, dynamic microsimulation techniques to calculate anticipated (cost-)effectiveness of strategies based on food pricing, food reformulation, or dietary (lifestyle) interventions. Currently available models differ in the methodology used for quantifying the effect of the dietary changes on disease, and in the method for modeling the disease incidence and mortality. However, all studies provided evidence that the models were sufficiently capturing the close-to-reality situation by justifying their choice of model parameters and validating externally their modeled disease incidence and mortality with observed or predicted event data. With the increasing use of various MSMs, between-model comparisons, facilitated by open access models and good reporting practices, would be important for judging a model's accuracy, leading to continued improvement in the methodologies for developing and applying MSMs and, subsequently, a better understanding of the results by policymakers. *Adv Nutr* 2022;13:621–632.

**Statement of Significance**: Given the advancement in the application of microsimulation modeling in evaluating food policies and measuring diet-related disease burdens, the present scoping review serves as an exercise to inform future modeling, hereby highlighting the need for transparency in model development, application, and dissemination to advance and safeguard accuracy and relevance in modeling efforts.

Keywords: food policies, public health, chronic diseases, health decision-making, microsimulation modeling

## Introduction

Chronic non-communicable diseases (NCDs) are the leading cause of mortality and morbidity globally (1), with much of this burden attributable to suboptimal diets (2, 3). In 2019, 8 million global deaths were estimated to be attributable to poor diet, with cardiovascular diseases (CVDs) as the leading cause of death, followed by cancers and type 2 diabetes (T2DM) (3). Further, these diet-related diseases disproportionately affect socio-economically disadvantaged population subgroups, with health disparities increasing over time (1). Improving diet for the prevention and management of NCDs features high on the global agenda (4, 5), highlighting the need for thorough decision-making tools to inform effective food policies.

Policy modeling has been used extensively in public health to identify potentially impactful strategies informed from different sources of population data (6). Similarly, the development of effective strategies for improving diet can be guided by health decision-modeling tools, as such techniques

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Address correspondence to EM (e-mail: ellymertens@itg.be).
Abbreviations used: CHD, coronary heart disease; CRA, comparative risk assessment; CVD, cardiovascular disease; DSA, deterministic sensitivity analysis; MI, myocardial infarction; NCD, noncommunicable disease; PSA, probabilistic sensitivity analysis; T2DM, type 2 diabetes mellitus.

 

are able to estimate the impact of a potential dietary improvement on reducing the burden of chronic NCDs in a particular population group. The most used epidemiological model structures for the evaluation of health policies include comparative risk assessments (CRAs) (7, 8) and state-transition models, based on either the cohort or individual (6). A state-transition model simulates consecutive transitions between predefined health states and the likelihood of an event happening at a specific time interval. Whether individual trajectories rather than the deterministic mean response of a homogeneous cohort are of interest determines whether a cohort-based or an individual-based model is more appropriate (9–11). A cohort-based model assesses populations or cohorts who share the same characteristics, while an individual-based or microsimulation model (MSM) simulates for each individual his/her potential disease history based on disease probabilities that fit his/her individual risk profile. Aggregated individuals' disease histories provide population-level estimates on disease outcomes with associated measures of uncertainty due to the inclusion of stochastic variation. In this way, MSMs allow for incorporating baseline variability in individuals' characteristics (6, 11, 12), a feature that is especially relevant when analyzing food policies. This is because dietary habits vary largely within populations (13), contributing to population's heterogeneity in disease histories, and thus making an MSM a key priority tool for informed decision-making on diet and health. In addition, MSMs have the advantage of proactively evaluating, for each individual, a potential outcome of interest prior to actual implementation of a food policy, as a way of ex-ante evaluating population health strategies; this is a theoretical analogue to a randomized controlled trial, with treatment and control being applied to the same hypothetical population.

MSMs for diet and NCDs provide policy-relevant output by forecasting the disease incidence and mortality under the current dietary practices compared with a counterfactual food policy scenario. This allows for the identification of effective dietary strategies to improve health, including their (cost-)effectiveness (6) and drivers of health inequalities (14). Because of these promising features, there is growing interest in the development of MSMs for food policies and for measuring diet-related disease burdens. The aim of this review is to provide an overview of the published studies using an MSM that links diet and/or food policies with NCDs. Due to the complexity of the model development, we aim to review the different approaches taken, including the model framework, key inputs, assumptions, and outputs, as well as the assessment of the model's validity, scenario sensitivity, and uncertainty.

## Methods

### Search strategies and data extraction

For this Scoping review, a literature search was performed in PubMed and Web of Science in January 2021 to identify relevant articles using the following search terms: ("diet∗" OR "fat" OR "sugar∗" OR "fruit" OR "vegetable" OR "meat" OR "sodium" OR "salt" OR "grains" OR "fibre" OR "energy" OR "portion size") AND ("disease" OR "burden") AND (("microsimulation" OR "micro-simulation" OR "state-transition model" OR "Markov model" OR ("stochastic" AND "individual∗ model")) without time restrictions. Articles included in the present review met the eligibility criteria: *1*) findings from an MSM; *2*) diets, foods or nutrients, or food policies as the main exposure of interest; and *3*) NCDs, such as overweight/obesity, T2DM, myocardial infarction (MI) and coronary heart disease (CHD), cerebrovascular disease (stroke), or cancer as the disease outcome for the burden of a food policy assessment. Searches were restricted to English-language publications and conference abstracts were not included. The selection of articles that met the inclusion criteria was based on information available in the manuscript.

**Figure 1** presents the PRISMA flow diagram (Preferred Reporting Items for Systematic Review and Meta-Analyses) (15). The initial search yielded 269 articles and, after removing duplicates, 179 abstracts were screened, yielding 69 abstracts retrieved for a full-text review. After exclusion of 36 full-text articles (with reasons as mentioned in Figure 1), 33 articles were included in the present review.

Information was extracted from the full-text articles, their supplementary materials, and their reference lists in relation to the application of the MSM for evaluating food policies, as well as its development and assessment. Extracted information included:

*1*) Publication details: authors, year, country, and acronym/name of the MSM, when available;
*2*) Population details: demographics of the starting cohort, including country and age; number of individuals; open compared with closed cohort; and time horizon and cycle length of follow-up;
*3*) Details of the primary objective of the MSM: the food policy scenarios or dietary factors under study, NCDs of interest, and outcome measure of the main analyses;
*4*) Model development details: model type, approaches to formulate the starting cohort, approaches to estimate individual disease risks, approaches to quantify dietary impacts on the disease process, and model implementation software; and
*5*) Model assessment details: model validation, including face validity, internal and external validation, scenario sensitivity analyses, and uncertainty analyses.

Definitions of the criteria used in this review are presented in **Box 1**.

## Results

The last decade has seen a growing trend towards the use of MSMs in the field of diet and food policies in relation to the NCD burden. This review identified 33 studies, mostly from the United States (23 studies), using an MSM in analyzing diet or diverse food policies on NCDs (**Table 1**).

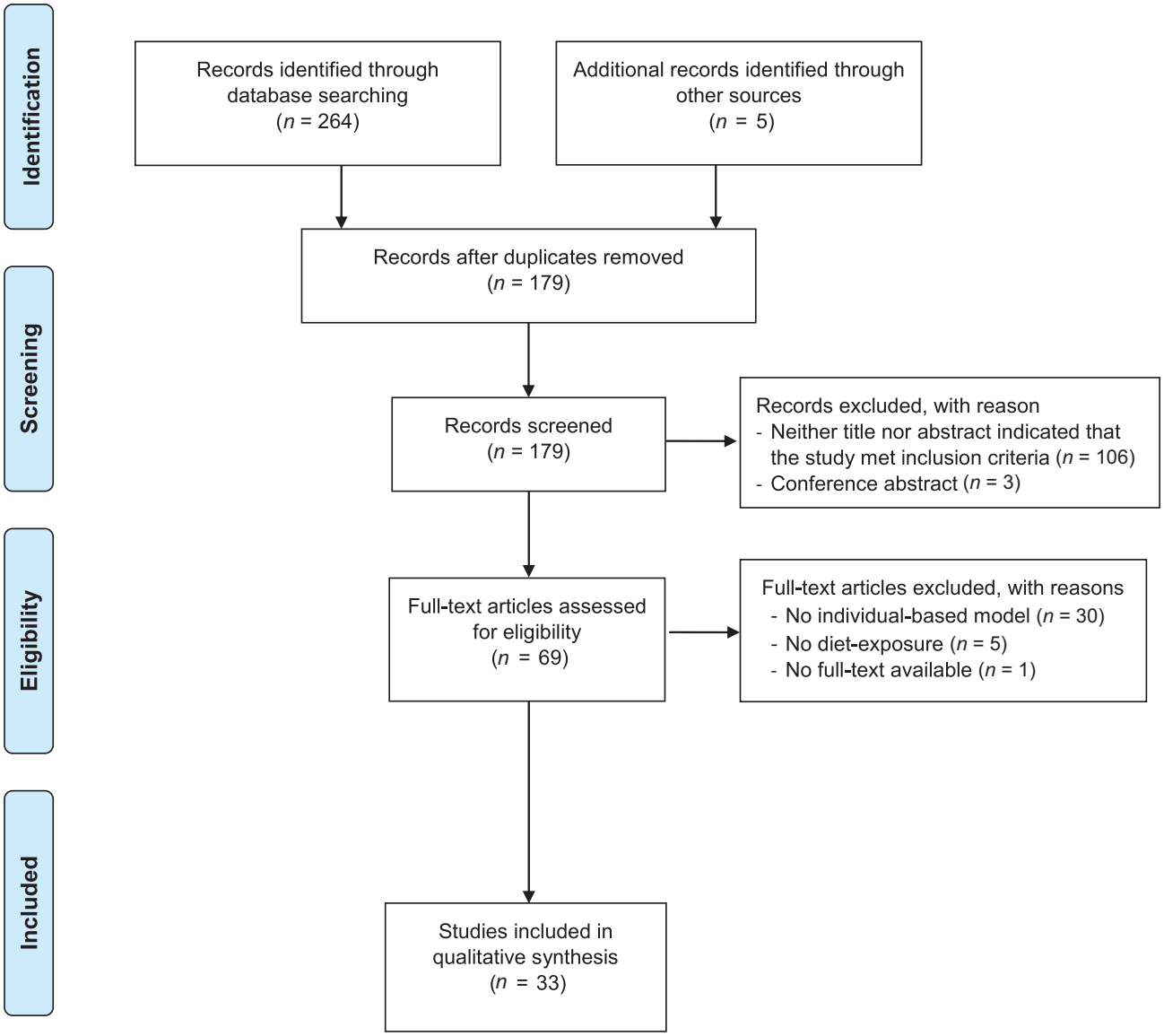


**FIGURE 1** Flowchart of the literature review (PRISMA flow diagram). Abbreviations: PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses.

### Objectives of MSMs

The purpose of most MSMs was to evaluate food policies, except for 2 studies that were instead modeling optimal dietary intakes (16, 17). The food policy strategies most frequently considered were taxes on sugar-sweetened beverages [10 studies; (18–27)], subsidies on fruits and vegetables [7 studies; (21, 24–26, 28–30)], sodium/salt reduction policies [6 studies; (21, 31–36)], and reformulation and labeling policies [12 studies; (21, 23, 31–40); Table 1]. The impacts of these food policies on dietary consumption were obtained from previous studies on price elasticities for taxes and subsidies (17 studies), time-trend series or published effect sizes for reformulation (9 studies), labeling (4 studies), and dietary interventions (5 studies; **Supplemental Table 1**). Once the change in diet induced by the food policy was introduced, the subsequent effect on the natural history of disease was directly and/or indirectly quantified (via changing biological risk factors, which in turn influence the NCD risk), using published estimates of the etiological effects of diet on the NCD risk by age and sex. In some studies, the impact of the food policy on the disease outcome was assumed to occur a few years after implementation of the intervention; in particular, a time delay for weight changes after a caloric intake change (18, 19, 22, 23, 25, 27), a 5-year time lag for CVD (21, 32, 34, 35, 41), and an 8-year time lag for gastric cancer (34, 35) have been used. Similarly, the impact of the food policy was assumed to fade out over the years in 6 food policy scenarios (22, 39, 42, 43).

In most studies, overweight/obesity, T2DM, MI, CHD, and stroke were the diseases of interest, because of their high disease burdens in the populations being modeled

**BOX 1**
**Definitions for the methodological aspects discussed in the review**

| | Definition | References |
|---|---|---|
| Individual-level state-transition models | For each individual, his/her disease history is simulated by applying a set of randomly treated transition processes that operate in discrete time intervals, often annually. | (10–12) |
| Base-case scenario | A natural disease history model that describes the course of the disease process from onset to progression to death in a biologically meaningful and representative way whilst being mathematically as simple as possible and using estimable model parameters. | (12) |
| Food policy scenario | A counterfactual scenario that can potentially modify an individual's diet and alter the natural disease history. | (12) |
| Starting cohort | A theoretical group of individuals defined by a set of demographic and clinical characteristics relevant for the course of the disease process modeled. In an individual-level state-transition model, cohort members might be heterogenous in their demographic and clinical characteristics. Often the cohort is a synthetic population—i.e., the characteristics of the population match the various statistical distributions of the real population—and is therefore a close-to-reality population to be used in modeling. | (10) |
| States | Relevant states in a simple state-transition model include: "health," "disease," and "death," with disease being an intermediate state between health and death, and death being an absorbing state. States are collectively exhaustive and mutually exclusive: i.e., an individual can only be in exactly 1 state at each model cycle. Further specifications of distinct states, including the number of distinct states, depend on the disease process, the research question, and data availability, as well as how demographic and clinical characteristics are attributed to states. | (10–12) |
| Transition probabilities | Individuals are allowed to move between states, with their probabilities of moving depending on demographic and clinical characteristics and the current state, and possibly also accounting for previous states' histories. | |
| Time horizon | The follow-up time of the cohort, related to the number of cycles. | |
| Cycle length | The time period between the potential transitions to distinct health states, and the duration of experiencing particular events. | |
| Validation analyses | A kind of model assessment that refers to the consistency of the model with observed/predicted data. Validation for decision modeling includes face validity (plausibility), internal validity (verification), cross validity (between-model comparison, external consistency), external historical validity, and external predictive validity. | (12) |
| Scenario sensitivity analyses | A kind of model assessment that refers to the explorations of model results under various scenarios, often varying model parameters that are inestimable or poorly estimable. | (12) |
| Uncertainty analyses | A kind of model assessment that refers to the variability/uncertainty inherent to the modeling, aimed at better informing the decision by assessing confidence in a chosen modeling strategy and/or determining the need for additional information. Uncertainty for decision modeling includes stochastic uncertainty, parameter uncertainty, heterogeneity, and structural uncertainty. | (12, 51) |

(Table 1), and based on the available evidence of their relationships with diet (Supplemental Table 1). However, the outcome measure of the main analyses was often health-care costs (23 studies), either operationalized as a cost-effectiveness analysis (i.e., costs/disease or quality-adjusted life years; 18 studies) or as medical expenditures (6 studies), followed by the number of (new) cases and deaths (10 studies) and the number of cases/deaths prevented/postponed (8 studies).

## Methodological approaches for the development of an MSM

The reviewed MSMs differed in their model type and the methodologies used to formulate the theoretical starting cohort of individuals that resembles the reality of the population under study and to define the natural history of the disease, using individual risks and associated transition rules for disease incidence and mortality (**Table 2**).

### *Model type.*

The MSMs identified were based on state-transition modeling techniques, most employing dynamic, stochastic, discrete-time microsimulation techniques; that is, for each individual, a disease history is simulated by applying a set of randomly treated transition processes that operate in discrete time intervals, often annually (Table 2). In only 4 studies, a compromise was made between the model flexibility and execution time by applying a combination of macro- and micro-simulation approaches. With such partial MSMs, the

**TABLE 1** Chronological overview of the use of microsimulation models in assessing the impact of diet and food policies in relation to NCD burden

| Author, year, reference | Countries | Food policy scenarios | NCDs of interest | Outcome measure of main analysis |
|---|---|---|---|---|
| Bertram et al., 2010 (42) | AU | Lifestyle intervention (diet and exercise) after T2DM screening | T2DM, CHD, stroke, ESRD | CEA; i.e., costs/DALY |
| Basu et al., 2013 (41) | IN | Tax (palm oil) | MI ischemic stroke | CVD deaths |
| Basu et al., 2013 (24) | US | Tax (SSBs) and subsidies (F&V) through SNAP | T2DM, MI, stroke | CEA; i.e., costs/QALYs |
| Basu et al., 2014 (25) | US | Tax (SSBs) and subsidies (F&V) through SNAP | Overweight/obesity, T2DM | Obesity prevalence, T2DM incidence |
| Basu et al., 2014 (18) | US | Tax (SSBs) | Overweight/obesity, T2DM | Overweight/obesity prevalence |
| Dall et al., 2015 (47) | US | Diabetes Prevention Program based on a lifestyle (diet and exercise) intervention | Overweight/obesity, T2DM, CVD events | Medical expenditures |
| Gortmaker et al., 2015 (19) | US | Obesity reduction policy[1] | Childhood obesity | CEA; i.e., costs/BMI unit reduction |
| Choi et al., 2016 (31) | US | National Salt Reduction Initiative | Hypertension MI, Stroke | CVD incidence and mortality |
| Kypridemos et al., 2016 (21) | UK | Population-wide combinations of dietary interventions[2] | CHD, stroke | Cases/deaths prevented/postponed SII[3] |
| Breeze et al., 2017 (22) | UK | Tax (SSBs), retail policy (F&V), worksite healthy eating promotion, community education programs | T2DM and related complications/disorders | CEA; i.e., costs/QALY |
| Choi et al., 2017 (30) | US | Subsidies (F&V) through SNAP | Obesity, T2DM, MI, stroke | CEA; i.e., costs/QALY |
| Kypridemos et al., 2017 (35) | UK | Salt reduction policy through reformulation, taxes, public awareness campaigns, food labeling | CHD, stroke, GCa | Cases/deaths prevented/postponed SII[3] |
| Pitt and Bendavid, 2017 (43) | US | Price change of meat and seafood | Obesity | Obesity prevalence, QALY |
| Vreman et al., 2017 (38) | US | Added sugar reduction policy (unspecified) | NAFLD, overweight, T2DM, CHD | Population disease prevalence DALYs, direct medical costs |
| Basu et al., 2018 (46) | SY, JO, LB, GZ | Food aid delivery | T2DM, MI, stroke | Population disease incidence CEA; i.e., costs/DALYs |
| Javanbakht et al., 2018 (17) | IR | Optimal intake of dairy | T2DM, CHD, stroke | Avoidable T2DM/CVD health-care costs |
| Mozaffarian et al., 2018 (26) | US | Tax (SSBs) and subsidies (F&V) through SNAP | T2DM, CHD, stroke | Cases/deaths prevented/postponed CEA; i.e., costs/QALYs |
| Pearson-Stuttard et al., 2018 (32) | US | Salt reduction policy through reformulation | CHD, stroke | Cases/deaths prevented/postponed CEA; i.e., costs/QALYs |
| Collins et al., 2019 (33) | US | Salt reduction policy through reformulation | CHD, stroke | Cases/deaths prevented/postponed CEA; i.e., costs/QALYs |
| Grummon et al., 2019 (40) | US | Health warning policy (SSB) | Obesity | Obesity prevalence |
| Huang et al., 2019 (37) | US | Added sugar reduction policy through labeling and reformulation | T2DM, CHD, stroke | T2DM/CVD deaths prevented CEA; i.e., costs/QALYs |
| Jardim et al., 2019 (16) | US | Optimal intake of 10 dietary factors | T2DM, MI, angina, stroke | Annual T2DM/CVD costs related to suboptimal intake |
| Laverty et al., 2019 (34) | UK | Salt reduction policy through reformulation | CVD, GCa | CVD/GCa deaths, health costs, equity impacts |
| Lee et al., 2019 (28) | US | Subsidy (Healthy foods) | T2DM, CHD, stroke | CVD/T2DM cases prevented CEA; i.e., costs/QALY |
| Long et al., 2019 (27) | US | Tax (SSB) through SNAP | (Childhood) obesity | CEA; i.e., costs/QALY |
| Wilde et al., 2019 (20) | US | Tax (SSBs) | CHD, stroke | CVD cases prevented/postponed CEA; i.e., costs/QALY |

(*Continued*)

**TABLE 1** (*Continued*)

| Author, year, reference | Countries | Food policy scenarios | NCDs of interest | Outcome measure of main analysis |
|---|---|---|---|---|
| Basu et al., 2020 (45) | US | Sales ban (SSBs) | Overweight/obesity, T2DM, CHD, stroke, chronic kidney disease | CEA; i.e., costs/QALY |
| Basu et al., 2020 (44) | US | Purchases of farm's produce through Community-Supported Agriculture | T2DM, MI, stroke | CEA; i.e., costs/DALY |
| Broeks et al., 2020 (29) | NL | Tax (meat) and subsidies (F&V) | T2DM, stroke, CHD, lung, colorectal cancer | CEA; i.e., costs/QALY |
| Dehmer et al., 2020 (36) | US | Salt reduction policy through reformulation | Hypertension, MI, stroke | Averted medical costs by payer productivity gains |
| Lee et al., 2020 (23) | US | Tax (SSBs and sugar content) | T2DM, CHD, stroke | CEA; i.e., costs/QALY |
| Liu et al., 2020 (39) | US | Menu calorie labeling | T2DM, CHD, stroke | CEA; i.e., costs/QALY |
| Choi et al., 2021 (48) | US | SSB purchase restrictions in SNAP | Childhood obesity | Obesity prevalence |

Abbreviations: AU, Australia; CEA, cost-effectiveness analysis; CHD, coronary heart disease; CVD, cardiovascular disease; DALY, disability-adjusted life-years; ESRD, end-stage renal disease; F&V, fruits and vegetables; GCa, gastric cancer; GZ, Gaza Strip; IN, India; IR, Ireland; JO, Jordan; LB, Lebanon; MI, myocardial infarction; NAFLD, nonalcoholic fatty liver disease; NCD, noncommunicable disease; NL, the Netherlands; QALY, quality-adjusted life-years; SII, Slope Index of Inequality; SNAP, Supplemental Nutrition Assistance Program; SSB, sugar-sweetened beverages; SY, Syria; T2DM, type 2 diabetes mellitus; UK, United Kingdom; US, United States

[1]Obesity reduction policies include SSBs excise taxes, elimination of the tax subsidy for advertising unhealthy food, restaurant menu calorie labeling, nutrition standards for school meals, nutrition standards for foods/beverages sold in schools, improved early care and education, and increased access to adolescent bariatric surgery.

[2]Population-wide dietary interventions include an SSB tax, salt policies, and F&V subsidies combined with taxing unhealthy foods. Their impacts on NCDs were modeled together and additionally included the impact of a smoking cessation intervention.

[3]SII included absolute inequity: that is, the impact of an intervention expressed in the amount of cases in the most deprived areas compared with the least deprived areas (magnitude of the difference), and the relative inequity accounts for a preexisting socio-economic gradient of disease burden, showing proportional differences.

risk factor history follows an MSM and the disease and mortality factors follow a cohort or Markov model, assigning probabilities of diseases and mortality that are used as averages over all (or a subgroup) of the simulated individuals.

***Formulation of the starting cohort.***

MSMs were initially populated by a sample of theoretical (synthetic) individuals using population distributions' parameters of demographics and risk factors (including diet) taken either from observational prospective cohort studies [as applied in (17, 41, 42)] or, more frequently, from population-representative health surveys, often combined with census statistics [as applied in (16, 18–40, 43–48)]. This sample of individuals—the starting cohort—was either drawn by taking a weighted sample of individuals included in the cohorts/surveys or was created by generating a "close-to-reality" synthetic population (Table 2). Most models were restricted to the adult population, but 6 studies also included children (**Supplemental Table 2**) (17, 27, 29, 30, 48, 49). In studies using an open cohort design (16, 18–21, 24, 27, 29, 32–39, 41, 43, 46), individuals can enter the cohort and leave the cohort (mortality), with rates of entry and exit based on population projections by census statistics to account for population ageing and demographic shifts over the years.

***Individual risks (and associated transition rules).***

In all studies, synthetic individuals entering the MSM acquired individualized risk factor trajectories, simulated using age and time trends from survey data, and that determined the associated individualized health transition rules. For all studies identified, a dynamic MSM based on discrete time was used; hence, individuals in the MSM were simulated to experience particular events in cycles with a length of either 1 day (40), 1 month (19, 24, 27), or, more commonly, 1 year (16–18, 20–23, 25, 26, 28–39, 41–48) (Supplemental Table 2). Subsequently, cycles were run for a predefined, fixed number of years, varying from 1 to 35 years (16–19, 21, 24–29, 31–41, 43–45, 47, 48), or for the lifetime of the individuals included (i.e., until death or the age of 100, whichever came first) (20, 22, 23, 26, 28, 30, 39, 42, 44–46).

The daily, monthly, or annually based risks and the associated transition probabilities for the onset of the NCDs of interest were estimated from either a multi-state life table approach, a hazard calculation approach, a risk score framework, or a CRA framework (Table 2). In a multi-state life table approach (17, 29, 38, 42, 47), the transition probabilities (for an individual to develop the disease before his/her next birthday) were derived from published age- and sex-specific incidence/prevalence rates. This approach is often applied for (mortality) events where no information on risk factors is available. In a hazard calculation model (17, 18, 24, 25, 41), the disease probabilities were calculated by multiplying the incidence rate by the ratio of an individual's hazard of an event to the typical hazard in the cohort that year. These 2 basic approaches are likely to result in conservative, lower-bound projections of the disease risk, as in the counterfactual food policy scenario they only consider

**TABLE 2** Microsimulation models for diet, food policies, and NCDs, and their modeling approaches for formulating the starting cohort and estimating individual disease risks

| | Approaches | Short explanation | References |
|---|---|---|---|
| Model type | Dynamic, discrete-time, stochastic microsimulation model | For each individual in the population, a set of randomly treated transition rules, determined by individual characteristics, are applied at each time step, leading to the possibility of transitioning to another health state (that are mutually exclusive competing and exhaustive) or death. | CVD PREDICT (16, 20, 23, 26, 28, 39)<br>US IMPACT Food Policy Model (32, 33, 37)<br>CHOICES model (19, 27)<br>$IMPACT_{NCD}$ model (21, 34, 35)<br>SPHR Diabetes Prevention Model (22)<br>ModelHealth CVD (36)<br>Unspecified (18, 24, 25, 30, 31, 40–46, 48) |
| | Partial micro-simulation | Markov-type state-transition model that combines microsimulation of risk factors with macrosimulation of disease and survival | DYNAMO-HIA model (29)<br>Disease Prevention Microsimulation Model (47)<br>Unspecified (17, 38) |
| Formulation of the starting cohort | Weighted sampling (with replacements) | Expansion of the survey sample by sampling individuals from the survey with replacements using sample weights; only possible if the survey reports all baseline variables needed | (16, 18, 20, 22–26, 28, 30, 31, 39, 41, 42, 44, 46, 47, 50) |
| | Generating a "close-to-reality" synthetic population | Expansion of the survey sample with other data sources, using statistical approaches such as synthetic reconstruction, model-based generations, combinatorial optimization, and/or (non-)parametric statistical matching | (19, 21, 32–37, 43) |
| | | Simulating an individual by sampling from cohort-specific joint probability distributions; guided by correlation matrix of risk factors | (17, 18, 24, 25, 30, 31, 38, 40, 41, 45, 46, 48) |
| Estimation of individual disease risk | From literature and/or published incidence/prevalence rates | Using multi-state life tables with 1-year intervals to estimate disease probability | (17, 29, 38, 42, 47) |
| | Hazard calculation approach | Calculating an individual's relative hazard of an event in relation to the typical hazard in the cohort that year, and multiplying this ratio by the cohort- and year-specific incidence rate to estimate his/her disease probability | (18, 24, 25, 41, 45) |
| | Risk score framework | Using risk functions with the specific risk exposures of an individual to estimate his/her disease probability | Framingham risk equations (16, 20, 22, 23, 26, 28, 30, 31, 36, 39, 47, 50), Globorisk (46), RECODe (44, 46), Pooled Cohort (44), QRISK2 (22), Leicester Risk Score (22), kcal to body weight (40, 43) |
| | Comparative Risk Assessment framework | Using population-attributable fractions to estimate disease incidence not attributable to modeled risk factors, and multiplying this not-attributable incidence by the relative risks of specific risk exposures of an individual to estimate his/her disease probability. | (21, 32–35, 37) |

ModelHealth CVD is a stochastic discrete-time model to estimate life-time incidence of CVD events and associated costs in a representative cross-section of US population.
Abbreviations: CHOICES model, Childhood Obesity Interventions Cost-Effectiveness Study project; CVD, cardiovascular disease; CVD PREDICT model, Cardiovascular Disease Policy Model for Risk, Events, Detection, Interventions, Costs and Trends; DYNAMO-HIA model, Dynamic Modelling for Health Impact Analysis; NCD, noncommunicable disease; QRISK2, a cardiovascular disease risk algorithm version 2; RECODe, Risk Equations for Complications Of type 2 Diabetes; SPHR, School for Public Health Research Diabetes Prevention Model.

the influence of the dietary exposures of interest relevant to the disease risk. More recent approaches, however, also consider a broader range of relevant risk factors. In a risk score framework, as applied in the Cardiovascular Disease Policy Model for Risk, Events, Detection, Interventions, Costs and Trends (CVD PREDICT) models (16, 20, 23, 26, 28, 39, 50); in some recent models of Basu and coworkers (30, 31, 44–46); and in other studies (19, 22, 27, 36, 38, 40, 42, 43, 47), the disease risk was calculated using calibrated risk scores, often Framingham risk equations, that translate the distributions of traditional risk factors into specific disease outcomes and are validated to empirical, historical disease trends. In contrast, in a CRA framework, as applied in $IMPACT_{NCD}$model (a dynamic, discrete-time, stochastic

microsimulation model) (21, 34, 35), the US IMPACT Food Policy model (32, 33, 37), and the Dynamic Modelling for Health Impact Analysis (DYNAMO-HIA) model (29), the disease risk was captured by all the well-accepted risk factors, with magnitudes of associations dependent on the prevalences of risk factors in the population. Hereby, these models take into account the distributional nature of the risk factors and their impacts on the population disease risks, hence providing more accurate estimates of disease risks. In an MSM context, both the risk score and CRA framework are highly dependent on the data available from nationally representative surveys in order to calculate an individual's disease risk. Nevertheless, independent of the approach used to model disease risks, the future projections rely on existing data and trends in the prevalence of risk factors, and hence are likely to overestimate disease events when risk factors and their corresponding clinical treatments improve over time.

The MSMs simulate whether an individual will transition to a new state or remain in the current state at the end of the cycle using stochastic transition rules; that is, the uncertainty of experiencing an event was incorporated, for example, by using Monte Carlo simulation, with sampling from a binomial (21, 34, 35) or a uniform distribution (21, 32–35, 37), possibly with the inclusion of common random numbers (16, 18, 20, 22, 23, 26, 28, 38, 39, 42, 50).

After modeling the base case scenario for disease incidence and mortality, a symmetric model with the same individuals was used to study the influence of a counterfactual food policy scenario by means of quantifying the impact of the food policy on dietary intakes and, subsequently, the impact of dietary change(s) on the disease/mortality risk (Supplemental Table 1), while ensuring that the disease process is represented consistently across the scenarios (10).

### Model assessment

When applying an MSM, evidence of model credibility was derived from examining validity, scenario sensitivity, and parameter uncertainty (**Table 3**).

Regarding model validation, only 3 studies included face validity (17, 22, 38), whereas most studies included internal and external validation. So far, a systematic comparison between models—that is, using 2 models for the same research question—has not yet been reported, although this between-model comparison would provide important insights in the variability due to the underlying model structure with assumptions. Internal validity checks included calibrating the starting cohort (31) and the modeled disease incidence and mortality rates (22, 30–32, 37, 46–48). External validity checks included comparing a model's output with either observed (16, 18, 20–24, 26, 28, 31–33, 36–39, 41, 43, 48, 50) and/or predicted data (16, 21, 32, 33, 37) on disease incidence and mortality rates.

Scenario sensitivity analyses included modeling results under various scenarios using variations in some preselected model parameters (12), such as varying taxes and subsidy levels (18, 23, 24, 28, 30, 41, 44–46), price elasticities (20, 23, 29, 43), and consumption trends (18, 24, 30, 34, 40, 43, 45, 46). This often provided further understanding of the research question rather than assessment of the model performance.

Uncertainty analyses of the MSMs included only covered examining parameter uncertainty; that is, when the estimated input values that steer outcomes are themselves uncertain, because of measurement error, sampling error, variability, and proxy data. Examples of this in the MSM included the uncertainties inherited in cohort/survey data referring to the representativeness/accuracy of the estimates of population characteristics and dietary intakes, their accuracy for generating likely trajectories of future risk factors and disease prevalences based on observed trends, and the uncertainties in the estimations of effect estimates. Studies quantified their parameter uncertainty by $x$-times repeated model replication either in a deterministic sensitivity analysis (DSA)—also known as a 1-way sensitivity analysis—to answer "what-if" questions or, more frequently, a probability sensitivity analysis (PSA) (51). In a DSA, as applied in 2 studies (38, 41), parameter values are manually specified as multiple-point estimates successively to test the sensitivity of the model's results to a specific parameter or sets of parameters. In a PSA, as applied in most studies (17–35, 37–42, 44–46, 48), the parameter values are sampled from predefined probability distributions and varied simultaneously to fully evaluate the combination of uncertainty in all model inputs on the robustness of model results. The PSA has become the accepted standard for providing nuanced decision options that generate 95% CIs or IQRs around the mean or median. This is, however, not the same as knowing the impact of an input parameter taking a specific value on the outcome, which is often of interest for policy decision-makers.

## Discussion

### Methodological considerations

This review provided an overview of the structure and methodological features of existing MSMs for food policies tackling diet and NCDs, independently of the findings of the individual models. An MSM is a suitable approach for untangling the multifaceted diet-health associations and the influence that diet has in the accumulation of multiple risks for each individual, while accounting for the large random variation in diet between individuals and population subgroups contributing to heterogeneity in the disease burden.

Results of the models are inevitably influenced by the choices of data sources and uncertainties around the input data sources and assumptions inherent to the modeling. In order to model the impact of diet on the onset of one or more NCDs, the available MSMs incorporated a broad range of data inputs from various publicly available data sources (52). Briefly, models relied on using cohort/survey data for demographics, trends in prevalences of biological factors and dietary intakes, and disease incidence and mortality rates by age and sex, and using published literature data for well-accepted risk factor–health associations and, when using a

**TABLE 3** Model assessment, including model validity, scenario sensitivity and uncertainty analyses

| Model assessment | | Examples on how this is carried out |
|---|---|---|
| Model validation | Face validity | Manually checking each transition (17, 38)<br>Manually checking sampling values (22) |
| | Internal validation | Model calibration to national data (22, 30, 31, 47, 48)<br>Comparison of the synthetic population with the original sample of the Health Survey of England to internally validate the synthetic population and their risk factor trends (21)<br>Baseline hazard rate in the risk equations of disease incidence and mortality calibrated to observed rates in health audits (46)<br>Annual case fatality for CVD calibrated to forecasted mortality rates in a population attributable risk framework (32, 37) |
| | External validation | Comparison against<br>Historical/observed data (16, 18, 20–24, 26, 28, 31–33, 36–39, 41, 43, 48, 50)<br>Forecasted/predicted data (16, 21, 32, 33, 37) |
| Scenario sensitivity analysis | Modeling results under various scenarios in 1-way sensitivity analyses | Varying values of model parameters:<br>Tax/subsidy/funding/sales ban levels (18, 23, 24, 28, 30, 41, 44–46, 48)<br>Consumption trends, including purchases trends (18, 24, 30, 34, 40, 43, 45, 46, 48)<br>Diet-risk factor associations (31, 36, 43, 47, 48)<br>Options in intervention strategy (21)<br>Participation rate (22, 30, 44, 48)<br>Participation time length/intervention duration (22, 30, 48)<br>Intervention efficacy during and afterwards (22, 44, 47)<br>Discount rate and willingness to pay (22, 32, 33, 37)<br>Policy size effects of labeling and food reformulation (36, 37, 39)<br>Elasticities (20, 23, 29, 43)<br>Bias in dietary recall (46)<br>Additional disease outcome:<br>Lung cancer (30) |
| Uncertainty analysis | Parameter uncertainty analyses (second-order) | Deterministic sensitivity analysis (38, 41)<br>Probabilistic sensitivity analysis<br>*x*-times repeated model replications by Monte Carlo sampling from the distributions/uncertainty ranges of the input parameters<br>100 times in (29)<br>1000 times in (17, 19, 20, 23, 26–28, 39, 42, 48)<br>10,000 times in (18, 24, 25, 30, 31, 38, 40, 41, 44, 46)<br>Not specified in (45)<br>*x*-times repeated model replications by Monte Carlo sampling from the distributions of the input parameters, and from a different sample of the synthetic population<br>1000 times in (21, 22, 34, 35)<br>2000 times in (32, 33, 37)<br>Copula functions (24) |

Abbreviation: CVD, cardiovascular disease.

risk score framework, well-established risk prediction models. Risk prediction models, derived both from traditional statistical methods (53–55) and machine learning techniques (56, 57), are abundantly present in the literature, but often of unknown value in MSM development because of the absence of external validation, direct comparison with other models on the relative predictive performance, or because they are not yet tailored to local settings. Therefore, modeling the individual disease risk via a risk score framework could only be endorsed when validated risk prediction models for the specific disease of interest are available. Also, the modeling approach following a CRA framework is relying on not only the available evidence of well-accepted risk factor–health associations, but also the known disease incidence in the population or when this could be estimated by, for example, using multi-state life table models, such as the WHO disease modeling software (DISMOD II). Modeling results that give insights on the population disease burden, health-care costs, and/or cost-effectiveness of a food policy scenario through time are therefore highly dependent not only on the underlying modeling assumptions, but also on the data available from nationally representative surveys and census statistics.

With the increasing application of MSMs in public health food policy, it is important to judge a model's accuracy in making relevant predictions. In particular, the external historical and external predictive validity are the most aligned with the model's purpose of providing the decision-maker with insight into what would happen after implementation of certain food policy strategies. The external

validation involves simulating events that have occurred and examining to what extent results under the base-case scenario correspond with observed/predicted event data. When supporting decision-making, failure to predict future trends is, however, not necessarily a concern, as policy decisions are based on scenario comparisons cancelling out systematic errors in absolute predictions. Still, because of the increasing number of MSMs for diet and NCDs, between-model comparisons—which involve comparing a model with others and determining the extent to which they calculate similar results—become increasingly important for judging a model's accuracy (58). Indeed, underlying methodological assumptions might differ between MSMs evaluating health-care costs or cost-effectiveness and those estimating the disease burden. This is mainly because the former were developed for that particular purpose of evaluating a specific food policy scenario on a specific NCD outcome, while the latter were fitted to provide a more detailed simulation of individual risk factors and disease trajectories, including accounting for diverse individual features affecting health. This highlights the need for greater transparency in the model development, application, and dissemination to advance and safeguard accuracy and the relevance of modeling in informing public health.

In the counterfactual food policy scenario, individual risks and associated transition probabilities were adjusted for the effect estimates of the food policy impact on diet, and subsequently the disease incidence and mortality, directly and/or indirectly (i.e., mediated by changes in risk factors). In all studies, it was noted that etiological effects of dietary changes on the specific NCDs and risk factors were estimated from robust meta-analyses. However, it is important to consider that amongst the direct and/or mediated effects of these dietary changes on NCDs, these changes could also have an effect upon a wide range of health burdens that were not modeled [e.g., a beneficial effect on productivity (59) and cancer prevention (60)]. Moreover, targeting one food group/nutrient is likely to change dietary intakes of other food groups, resulting from compensatory or rewarding behavior, as accounted for in some studies (18, 24, 25, 30, 39, 41). Also, that is why instead of focusing on the specific food groups/nutrients targeted in the food policy strategy of interest, some studies accounted for diet as a whole to represent more likely, counterfactual dietary practices with their influence on the onset of NCDs: for example, using the (Alternative) Healthy Eating Index (24, 44, 45) or the Mediterranean Dietary Score (46). Impacts of food policy strategies on disease risks are, however, thought to be conservative, because of the use of dietary survey data that are prone to recall bias, socially desirable reports, and underreporting of unhealthy foods (61).

In conclusion, MSMs have been applied to study the impacts of food policy strategies on NCDs from 2010 onwards, with cost-effectiveness as a key outcome measure of interest, and most models have been developed for the US adult population. MSMs mimic individual health trajectories over the life course, incorporating heterogeneity in food policy effects. This allows for exploring the distributional nature of a policy's impact on the population's health over time, and thereby providing evidence to support timely implementation in a cost-effective way. The output of every model is, however, highly dependent on the best available evidence on population characteristics and effect estimates using publicly available data, and the set of assumptions regarding the life course of the individuals simulated. It is therefore important to accurately calibrate and validate the models to the population dynamics they are supposed to describe/simulate. In particular, the between-model comparisons become increasingly important for judging a model's accuracy as the number of MSMs increases. In line with this is the need for good reporting practices and model transparency: that is, the model developers should provide sufficient information enabling researchers to evaluate model performance before applying it for their purposes. This would lead to continued improvement in methodologies for developing and applying MSMs and, subsequently, a better understanding of the results by policymakers.

### Outlook

Incorporating a life-course approach and bringing additional inputs into the MSM, such as early-life determinants and current and potential future choices on key lifestyle factors, namely diet, physical activity, smoking, and alcohol consumption, is key for disentangling the influences of and the interplays between (early-life) lifestyle factors on the progression to NCDs (62), and thus for identifying effective early-life strategies to prevent and control NCDs in future generations of adults (63).

Extending the model to specific populations, including those in low- and middle-income countries where addressing the rising burden of NCDs is a public health priority (64), will allow for the evaluation of policy strategies in different population subgroups or geographic locations instead of running experimental trials in different resource settings. The MSM's ability in ex-ante evaluations of counterfactual scenarios is, however, limited by the strength of causal inferences available in the literature that were used to inform model inputs.

In addition, integrating interactions within and between individuals, populations, and the environment enables calculations of the probabilities of events occurring through the social and built environment, as applied in agent-based simulation models for understanding who to target and how best to target them (6, 65). The identification of cost-effective and feasible policy strategies to improve population health will be crucial to increase sensible use of resources, as well as potential economic gains from increased productivity and reduced health-care utilization.

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reviewed and completed the final manuscript; and all authors: read and approved the final manuscript.

## Data Availability

Data sharing is not applicable to this article as no data sets were generated or analyzed during the current study.

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