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Projected Trends in the Incidence and Mortality of Uterine Cancer in the United States

Jason D. Wright[1,2,3,*], **Matthew T. Prest**[*,1,4], **Jennifer S. Ferris**[1,4], **Ling Chen**[1], **Xiao Xu**[1,2], **Kevin J. Rouse**[1,4], **Alexander Melamed**[5], **Chin Hur**[2,3,4], **Brandy M. Heckman-Stoddard**[6], **Goli Samimi**[6], **Nina A. Bickell**[7], **Tracy M. Layne**[8], **Evan R. Myers**[9], **Laura J. Havrilesky**[9], **Stephanie V. Blank**[8], **Natasha K. Stout**[6], **William D. Hazelton**[10], **Chung Yin Kong**[7], **Elena B. Elkin**[2,11]

[1]Department of Obstetrics and Gynecology, Columbia University College of Physicians and Surgeons, New York, NY, USA

[2]Herbert Irving Comprehensive Cancer Center, New York, NY, USA

[3]New York-Presbyterian Hospital, New York, NY, USA

[4]Department of Medicine, Columbia University College of Physicians and Surgeons, New York, NY, USA

[5]Department of Obstetrics and Gynecology, Massachusetts General Hospital, Boston, MA, USA

[6]National Cancer Institute. Bethesda, MD, USA

[7]Division of General Internal Medicine, Icahn School of Medicine at Mount Sinai, New York, NY, USA

[8]Department of Obstetrics, Gynecology, and Reproductive Science, and Population Health

[9]Department of Obstetrics and Gynecology, Duke University Medical Center, Durham, NC, USA Science and Policy, Icahn School of Medicine at Mount Sinai, New York, NY, USA

[10]Herbold Computational Biology Program, Division of Public Health Sciences, Fred Hutchinson Cancer Center, Seattle, WA, USA

[11]Department of Epidemiology, Joseph L. Mailman School of Public Health, Columbia University, New York, NY, USA

Abstract

Background: To develop a natural history model for uterine cancer calibrated to population-based incidence and mortality data to project future trends in the disease through 2050.

Methods: We developed a state-transition microsimulation model of uterine cancer. The model begins at 18 years of age and simulates Black and White patients, includes transition states for

Corresponding Author: Jason D. Wright, M.D., Division of Gynecologic Oncology, Department of Obstetrics and Gynecology, Columbia University College of Physicians and Surgeons, 161 Fort Washington Ave, 4th Floor, New York, NY 10032, Fax: (212) 305-3412, jw2459@columbia.edu.

*JDW and MTP contributed equally

precursor lesions, and separately models endometrioid and non-endometrioid tumors. The model was calibrated to population-based incidence and mortality data using parameter extrapolation.

Results: The model closely fit population-based incidence and mortality data of uterine cancer. From 2020-2050, the incidence of uterine cancer is projected to increase in White women to 74.2 cases per 100,000 (compared to 57.7 cases per 100,000 in 2018) and to rise to 86.9 per 100,00 (compared to 56.8 cases per 100,000 in 2018) in Black women. Among White women, incidence-based mortality will increase from 6.1 per 100,000 in 2018 to 11.2 per 100,000 in 2050 while incidence-based mortality in Black women will increase from 14.1 per 100,000 to 27.9 per 100,000. While endometrioid tumors are expected to increase considerably in both White and Black women, White women will experience only a slight increase in non-endometrioid tumors while the incidence of these tumors will rise substantially in Black women.

Conclusion: The incidence and mortality of uterine cancer is projected to increase substantially over the next three decades. Black women will experience a disproportionate increase in the disease.

Impact: Projecting the incidence and mortality of uterine cancer can facilitate future cancer control efforts.

Introduction

Uterine cancer is the most common gynecologic malignancy in the United States, with an estimated 67,880 cases and 13,250 deaths projected in 2024.(1) The last thirty years has seen a dramatic rise in both the incidence and death rate from uterine cancer.(2) A recent report from the National Cancer Institute noted that there was an average annual percentage increase of 0.8% for uterine cancer cases from 2014 to 2018. From 2015-2019, mortality increased 1.9% annually, the highest for any malignancy in women.(3)

The increase in the incidence and mortality of uterine cancer is likely multifactorial. Uterine cancer is predominately a disease of older women and the aging of the population in the U.S. has undoubtedly contributed to these trends.(4–6) Likewise, the rising rate of overweight and obesity has influenced the changing trends in uterine cancer.(7) Adipocytes produce estrogen which can stimulate the endometrium making obesity one of the strongest risk factors for uterine cancer.(8) However, the aging of the population and rising prevalence of obesity likely only explain a part of the changing trends in uterine cancer incidence and mortality.

Planning for cancer control and prevention activities requires an understanding of the projected burden of a given cancer. At present, there is no routine screening or prevention for uterine cancer. Uterine cancer is most commonly detected in women who present with symptoms, most frequently abnormal bleeding. The changing risk profile for uterine cancer in the U.S. has challenged the ability to accurately forecast the burden of the disease. The objective of our study was to estimate the projected incidence and mortality of uterine cancer in the United States. To accomplish this, we developed a natural history model for uterine cancer calibrated to the population-based incidence and mortality of the disease. We then utilized the model to project future trends in the incidence and mortality of uterine cancer through 2050.

Methods

Modeling Approach

The Columbia University Uterine Cancer Model (UTMO) is a state-transition model of the natural history of uterine cancer developed as part of the National Cancer Institute's Cancer Intervention and Surveillance Modeling Network (CISNET) (Figure 1). State-transition microsimulation models simulate individual subject trajectories and allow variation in individual characteristics from a sampled population (Supplemental Table 1).(9) The model begins at 18 years of age and uses a one-month cycle length to dynamically account for short duration changes in risk factors and health states over time until 84 years of age (Table 1). The model simulates non-Hispanic White and non-Hispanic Black women separately as there are well-documented racial disparities in uterine cancer incidence, histologic type, and mortality. The model has four general states: healthy (unaffected), precursor lesion (endometrial intraepithelial neoplasia), cancer, and death. Individuals with uterine cancer may have two disease states. Undetected uterine cancer is based on the true presence or absence of cancer for a given tumor stage that has not yet been detected clinically or pathologically. Detected uterine cancer is also based on the true presence or absence of a cancer for a given tumor stage, but one that has been clinically identified based on a diagnostic test.(10)

The model includes two general histologic groupings of uterine cancer: endometrioid (EM) uterine cancer and non-endometrioid (non-EM) uterine cancer (Supplemental Table 2).(11) Endometrioid cancers are the most common histologic subtype of uterine cancer and encompass tumors that are generally estrogen driven and associated with a more favorable prognosis.(11) Non-endometrioid cancers include all other subtypes of uterine cancer exclusive of uterine sarcomas. The most common histologic variants in the non-endometrioid group are uterine serous carcinomas, carcinosarcomas, and clear cell carcinomas. In general, these tumors have a less favorable prognosis.(11) The model assigned each simulated individual with cancer to a mutually exclusive histologic subtype based on age, race, birth cohort, and stage-specific incidence rates derived from the Surveillance, Epidemiology, and End Results (SEER) 18 national cancer registry data (https://seer.cancer.gov/) (Supplemental Table 2). In that analysis, patients with ambiguous or not otherwise specified (NOS) histology codes were reclassified as either EM or non-EM, and missing American Joint Commission on Cancer (AJCC) stage values were imputed using multiple imputations. Methodology for imputation was based on an approach developed by the National Cancer Institute's SEER Registry. This methodology assumes that for cancer cases with similar demographic and tumor characteristics, treatment, and survival, the distribution of specific and non-specific histology is similar. We used multiple imputation through chained equations (MICE) with M=5 imputations to redistribute cases with non-specific histology (ICD-O-3 histology codes of 8000, 8140, 8255, 8323, 8481, 8560; mean 17.9% of cases per year) to endometrioid or non-endometrioid subtypes. A similar approach was used to impute AJCC stage I-IV for cases with missing AJCC stage (mean 7.3% of cases per year). The multiple imputation model included histologic group, AJCC stage, sociodemographic characteristics (age and race), year of diagnosis, receipt of cancer treatment (chemotherapy and radiation), vital status, cause of death, and survival

time. Other endometrial cancer histologic types and sarcomas account for <7% of cases and were not included in the model.

Uterine cancer can arise from one of two pathways, either through a precursor lesion, endometrial intraepithelial neoplasia (EIN), or de novo from a normal health state without uterine pathology for non-endometrioid cancers.(12) Classification of EIN is based on the 2014 WHO classification schema for endometrial hyperplasia. EIN is largely equivalent to atypical endometrial hyperplasia. Undetected EIN may persist in an undetected state, progress to detected EIN, regress, or progress directly to a cancer state without a clinically detectable EIN lesion. Detected EIN may persist or progress to cancer. As EIN is generally treated, individuals with detected EIN may have a hysterectomy prior to the development of cancer.

EIN, detected or undetected, may progress to either an EM or non-EM cancer. The majority of cancers that arise from EIN follow a pathway of progression to EM tumors.(13–15) However, a small number of non-EM tumors are also thought to arise from EIN lesions.(16–20) EIN progression to cancer occurs initially as an undetected tumor. EIN initially transitions to a stage I cancer which could then progress undetected to a higher stage or become clinically detected.

Hysterectomy is performed frequently in the US, most often for benign indications.(21–23) Because hysterectomy effectively removes one's risk of developing uterine cancer, UTMO incorporates the population-level prevalence of hysterectomy. Variation in the hysterectomy rate over time and by race is assigned to birth cohorts using data from the National Health and Nutrition Examination Survey (NHANES) (https://wwwn.cdc.gov/nchs/nhanes/). These data allowed capture of race- and age-specific hysterectomy and the variation of these rates over time. Individuals may transition from a healthy state to hysterectomy or from undetected or detected EIN to hysterectomy. Hysterectomy is the treatment of choice for the majority of women with uterine cancer and is incorporated into the treatment nodes of patients with detected uterine cancer. Individuals who transition to hysterectomy from a healthy state, undetected EIN or detected EIN remain in the hysterectomy state until death.

Obesity is an important risk factor for uterine cancer.(7) Body mass index (BMI) is simulated as a categorical variable (< 25.0, 25.0-29.9, 30.0+) in the model by age, race, and birth cohort from population-based estimates from NHANES (https://wwwn.cdc.gov/nchs/nhanes/). The influence of obesity on the uterine cancer carcinogenic pathway is modeled through incorporation of fixed transition odds ratios for development of undetected EIN from a healthy state(13) and for the transition from undetected EIN to undetected endometrioid endometrial cancer.(24) Obesity in the model promotes the pathway to transition to detected cancer in patients with higher BMI values by increasing the transition probabilities of undetected states.

Both endometrioid and non-endometrioid cancers are classified based on the AJCC 7th edition stage I-IV. AJCC staging criteria are based on TNM (tumor, node, metastasis) criteria which have varied over time (Supplemental Table 3).(25) All cancers arise in an undetected state. While undetected, cancers may either persist in the given undetected stage,

progress to a higher undetected stage, or progress to a detected cancer of the corresponding stage if the tumor is identified clinically. Additionally, some individuals will die from other causes prior to the detection of the underlying uterine cancer. The model assumes that cancers cannot regress to a lower stage or to EIN.

Individuals with detected cancer may remain in the given stage-at-diagnosis, die from cancer, or die from other causes. Cancer-associated death rates are based on age at diagnosis, time elapsed since diagnosis, race, birth cohort, and histology-specific estimates from the SEER data as described below. The effects of cancer-directed therapy are captured in these survival estimates. Death from other causes is likewise based on age, race, and birth cohort-specific population estimates from the Centers for Disease Control and Prevention (CDC) WONDER database (https://wonder.cdc.gov). Individuals may transition to other-cause death from any state in the model.

Calibration and Validation

Model calibration was performed by varying unobservable parameters until the outputs from the model most closely matched available epidemiologic data.(26) Calibration of UTMO was performed in two phases, a multicohort phase and a cohort-specific phase. The motivation for this methodology was to incorporate as much of the available data as possible, while being able to forecast future trends. As some target data had limited sample sizes, high variance or insufficient information to stratify by birth cohort, this information was incorporated into the multicohort phase. The parameters from the multicohort phase were then used as starting values for the cohort-specific phase, where birth cohorts were grouped into 10-year intervals beginning with 1910-1920.(27, 28)

The primary calibration target was SEER-18 uterine cancer incidence stratified by age, birth cohort, race, histology and AJCC stage for patients 18-84 years of age. Our parameter set consisted of monthly transition probabilities for each connected edge in Figure 1, varying by age, birth cohort and race, with additional bounds imposed as outlined in Supplemental Table 4. We used simulated annealing (SA)(26), an optimization algorithm, to iteratively improve our model's performance as measured against a weighted combination of the targets outlined in Supplemental Table 5.

SA was repeated 3 times for the multicohort phase with 1,000,000 iterations each and step size, defined as the amount the parameters are adjusted by each iteration, of 0.001, 0.0001, and 0.00001 respectively. For the cohort-specific phase, SA with 1,000,000 iterations was run with a step-size of 0.00001. The motivation for this specification was to improve the stability of future projections by optimizing goodness-of-fit (GOF) with the smallest possible parameter changes by birth cohort. The number of iterations was determined by the available computational resources, while the step sizes were based on the order of magnitude of the background mortality inputs. After 1,000,000 iterations of simulated annealing, the resulting incidence curves from the model fit the SEER data well, indicating successful optimization.

Each phase of calibration utilized multiple targets linearly superposed as part of a composite objective function or GOF. The targets were broadly categorized into either incidence or

prevalence estimates. Each target was first weighted proportionally to the square root of the sample size of the source population. The weights were then scaled to the magnitude of the largest sample size, in this case SEER incidence data, as follows:

$$GOF_{Comp} = \sum_{target} \frac{\sqrt{N_{target}}}{\sqrt{N_{max}}} \cdot SSE(output, target).$$

Incidence targets were incorporated into the composite objective function by measuring the sum-squared-error (SSE) of the model-produced age-specific incidence compared to the target data stratified by race, histology, AJCC stage and birth cohort. SSE is defined as:

$$SSE(output, target) = \sum_{age} (output_{age} - target_{age})^2.$$

Prevalence targets were incorporated using the same weighting strategy but comparing the SSE of the mean annual age-specific prevalence of the model to the target data stratified by race, histology, and birth-cohort.

We visualized the calibration and validation process by comparing the cancer incidence and incidence-based mortality amongst women aged 40 and older estimated from UTMO to the estimation from SEER. Incidence-based mortality was calculated as the death rate among incident cases divided by the general population in SEER areas using the linked data between death certificate mortality and SEER incident cases.(29) We required 5 years of data on incident cases prior to each year of mortality (“burn-in” period). The “burn-in” period was determined by comparing the incidence-based mortality and death certificate mortality using methods recommended by NCI. To further examine the robustness of UTMO, we compared the 5-year cause-specific survival rates to the estimation from SEER 18. The distribution of AJCC stages was also compared between UTMO and SEER.

“Stress testing” of the model was performed for validation purposes using the CISNET-recommended maximum clinical incidence reduction (MCLIR) scenario analysis.(30) This consisted of creating 4 hypothetical screening and treatment interventions that could alter the incidence of uterine cancer (Supplemental Table 6). The purpose of each scenario is to explore the reduction in total uterine cancer incidence in an idealized setting as if a test with perfect sensitivity, specificity and treatment efficacy was introduced for patients of a given age. Patients exposed to the intervention would not be at risk for transition to uterine cancer (in our model moved to the hysterectomy state). Scenarios 1 and 2 are interventions applied to patients 45 years of age from the 1940-1950 birth cohort with undetected EIN for scenario 1 and for undetected endometrioid tumors and EIN for scenario 2. Scenarios 3 and 4 are interventions applied to patients 55 years of age from the same birth cohort with undetected EIN for scenario 3 and for undetected endometrioid tumors and undetected EIN for scenario 4 (Supplemental Table 6). MCLIR is a theoretic construct meant to test the robustness of the model through elimination of patients in an undetected state at one point in time.

Projection

To project cancer incidence and mortality in 2020-2050 using our model, parameter extrapolation was used in several different ways. BMI of future cohorts was estimated from NHANES data using the last observation carried forward (LOCF) method.(31) Similarly, survival hazard functions and hysterectomy rates for cohorts born after 2020 were extrapolated using LOCF. Consequently, obesity, survival and hysterectomy rates are assumed not to change from their most recent available data point to 2050 to minimize the level of uncertainty introduced in the projection.

To examine the implications of declining hysterectomy rate and increasing obesity rate on the projection, we also performed a series of scenario analyses and examined the projection after incorporating the forecasts of hysterectomy and obesity up to 2050.(32, 33) Hysterectomy trends are projected to decrease by 25.7% from 2020 to 2035. We imported data on the estimated incidence rate of hysterectomy procedures per 1000 women aged 18-64 years from 1935 to 2035 to WebPlotDigitizer 5.2 (Automeris LLC, California), estimated the data from 2020 to 2035 from the figure, and extended the projection to 2050 presuming the same trend continued (from 3.9 per 1000 by 2035). We applied the estimated hysterectomy data to our transition probabilities from healthy to benign hysterectomy for all age and race categories.(32) This constant decrease in transition probability was applied through 2050.

Obesity rates are rising, with the proportion of obese people in the United States increasing by about 6% every 5 years.(33) The proportion of people in the overweight category is remaining relatively constant whereas the proportions of obese people and healthy people are increasing and decreasing, respectively. In the projection, we assume an increase in the proportion of obese people of 1.17% per year. The people who join the obese category are taken from the healthy category. We compared future projections of endometrial cancer assuming obesity and hysterectomy rate were held constant, as well as if the hysterectomy rate declined and the rate of obesity increased.

The remaining calibrated parameters, including background mortality, were extrapolated to future cohorts using smooth cubic spline extrapolation(34) with a 1% deviation from linearity. The incidence results for each cohort were then combined and age-standardized using the SEER 2000 reference population, to get annual incidence for the projected years. (35, 36)

Data Availability

Data from the model are derived from publicly available sources as described above.

Results

The incidence and mortality for uterine cancer have gradually increased over time for both White and Black women (Figure 2, Table 2). Our model closely fit SEER incidence and mortality data available through 2018. For uterine cancer incidence, our model has a square root normalized SSE (NSEE), a measure of average variance from the target, of 0.064 in White women and 0.084 in Black women. For uterine cancer incidence-based mortality, our

model has an NSSE of 0.124 in White women and 0.333 in Black women. These results demonstrate excellent model validity.

The model performed well when additionally stratified by histology and age (Figure 3), while recognizing the additional noise present in single-age observations. The model-predicted median age of diagnosis across all cohorts for EM cancer was 65.3 and 65.7 for White and Black women, respectively (compared to actual median ages of 65.7 and 66.9). The predicted median age of diagnosis for non-EM cancer was 70.6 and 68.8 for White and Black women, respectively (compared to actual median ages of 70.7 and 69.7).

Predicted 5-year survival in 2013 (Table 2) was 92.6% (95% CI, 91.5%-93.6%) and 86.4% (95% CI, 84.7%-88.2%) for White and Black patients with endometrioid tumors, respectively, compared to an actual 5-year survival rate of 91.5% (95% CI, 90.8%-92.3%) and 85.6% (95% CI, 82.4%-88.3%). For non-endometrioid tumors in 2013, predicted 5-year survival was 45.9% (95% CI, 41.2%-50.7%) and 37.5% (95% CI, 34.5%-40.5%) for White and Black patients, respectively, compared to an actual survival of 50.7% (95% CI, 47.2%-54.1%) and 42.1% (95% CI, 36.7%-47.5%). In 2018, the stage distribution at diagnosis based on the UTMO was 70.5% stage I, 8.2% stage II, 12.1% stage III, and 9.2% stage IV, compared to the SEER incidence stage distribution of 74.5%, 4.5%, 11.9%, and 9.1%, respectively (Supplemental Table 7).

From 2020-2050, the incidence of uterine cancer is projected to continue increasing in both Black and White women. Based on current trends, the incidence of uterine cancer in White women will reach 74.2 cases per 100,000 by 2050 (compared to 57.7 cases per 100,000 in 2018) (Figure 2, Table 2). In Black women, the incidence rate will rise to 86.9 per 100,00 in 2050 (compared to 56.8 cases per 100,000 in 2018). Among White women, incidence-based mortality will increase from 6.1 per 100,000 in 2018 to 11.2 per 100,000 in 2050. The corresponding rise in mortality is greater in Black women, increasing from 14.1 per 100,000 in 2018 to 27.9 per 100,000 in 2050 (Supplemental Table 8).

When adding the projected hysterectomy and obesity trends, the projected incidence and mortality in both White and Black women is significantly higher. Based on these projected trends, the incidence of uterine cancer in White women will reach 92.9 cases per 100,000 by 2050 (compared to 74.2 without obesity and hysterectomy projections) (Figure 1, Supplemental Figure 1, Supplemental Table 9). In Black women, the incidence rate will reach 102.0 (compared to 86.9 without projections). Among White women, incidence-base mortality will rise to 12.9 per 100,000 (compared to 11.2 without projections). The corresponding incidence-based mortality in 2050 with obesity and hysterectomy projections is projected to be 31.4 per 100,000 (compared to 27.9 without projections).

When stratified by histology, the incidence of endometrioid tumors is projected to increase considerably in both Black and White women (Figure 3, Table 2). In contrast, while there is a slight increase in the rate of non-endometrioid tumors in White women, the incidence rate of non-endometrioid tumors in Black women increases more substantially from 22.5 per 100,000 in 2018 to 36.3 per 100,000 in 2050. The stage distribution is projected to change slightly over time, with the percentage of cases diagnosed at stage I declining from 74.5%

in 2018 to 68.9% in 2050 (Supplemental table 7). Additionally, all stage-combined 5-year survival for endometrioid and non-endometrioid tumors is projected to remain relatively stable (Table 2). These survival estimates are based on current treatment paradigms and are unable to account for future therapeutic advances.

Two sojourn times were estimated from model outputs by race and histology: 1) pre-invasive sojourn time, which reflects the time from first pre-invasive lesion (undetected EIN state) to detected cancer, and 2) malignancy sojourn time, which reflects the time from first malignant cell (undetected cancer) to clinically detected cancer (Table 3). The sojourn time was shorter for non-endometrioid compared to endometrioid cancers. . For example, the malignancy sojourn time for endometrioid uterine cancer was 22.4 months in Black women compared to 21.9 months in White women. The corresponding sojourn times for non-endometrioid tumors were 8.2 and 8.7 months, respectively.

Results from stress testing of the model using the MCLIR methodology are shown in Figure 4. Scenarios 1 and 2 were applied for women at age 45 and eliminated further risk for patients with undetected EIN alone (scenario 1) or undetected EIN and endometrioid tumors (scenario 2). For both White and Black women there was a decline in cancer incidence starting at age 45 and lasting up to 7 years and 8 years, respectively. Women 55 years of age for scenario 3 (removal of undetected EIN alone) and scenario 4 (removal of undetected EIN and endometrioid tumors) saw a decline in cancer incidence lasting up to 15 and 16 years for White and Black women respectively. The magnitude of decline and the absolute number of cases eliminated was larger when the intervention was applied at 55 compared to 45 years of age. This indicates that potential screening and intervention options are more effective when targeting women aged 55 than those aged 45.

Discussion

Using population-based data, we developed and calibrated a natural history model of uterine cancer that accurately reflects incidence and mortality of the disease. Based on available data, this model suggests that the incidence and mortality of uterine cancer will increase substantially over the next three decades. Black women will experience a disproportionate increase in uterine cancer incidence and mortality compared to their White counterparts.

In recent decades, the incidence of uterine cancer has risen at a concerning rate. Between 2010 and 2020, the number of uterine cancer cases diagnosed in the U.S. increased from 43,470 cases to 65,620, a more than 50% increase over the course of a decade.(37, 38) Our model predicts that these trends will continue for the foreseeable future. From 2018 to 2050, the incidence of uterine cancer is predicted to increase by 51.4% in Black women and 28.6% in White women. By 2050 we project that the incidence of uterine cancer will be 86.9 per 100,000 in Black women and 74.2 per 100,000 in White women.

Our model also projects that racial disparities in uterine cancer will become more pronounced. By 2050, the incidence-based mortality rate from uterine cancer is expected to be nearly three times greater in Black compared to White women. Prior work has suggested that racial disparities for uterine cancer are driven by numerous factors.(39) Black

women more often have delays in diagnosis, face barriers in accessing care, present with more advanced stage disease and more aggressive tumor histologic subtypes and receive suboptimal care.(39–41) While the rate of endometrioid tumors will increase among both Black and White women, our modeling data predict that Black women will experience a more marked rise in non-endometrioid uterine cancers. Non-endometrioid tumors are aggressive tumor subtypes that account for a disproportionate number of deaths from uterine cancer.(11) The disparate rise in the rate of non-endometrioid cancers in Black compared to White women is a driving factor in the increasing mortality rate from uterine cancer that we noted among Black women.

Our natural history model provides a powerful tool to explore how past, present, and future changes in epidemiologic trends and other risk factors influence the incidence of uterine cancer.(6) Both age and obesity are important risk factors for uterine cancer and are incorporated into the model. Reproductive risk factors including age at menarche and menopause, parity, and exposure to exogenous hormones have all changed over time and influence the prevalence of uterine cancer.(42) Hysterectomy rates have fluctuated substantially over time, particularly recently, and will have an important influence on the risk profile of uterine cancer.(43) Importantly, there is substantial racial variation in hysterectomy rates that could exacerbate disparities in the incidence and mortality of uterine cancer.

The stress testing performed using MCLIR methodology provides important insights into potential screening strategies for uterine cancer.(30) At present, screening for uterine cancer is not recommended. Prior modeling studies examining transvaginal ultrasound and endometrial biopsy, the most common diagnostic tests for uterine cancer, have suggested that these modalities are not cost-effective screening tests for asymptomatic women.(44, 45) Simulation of an effective screening test at age 55 in our model resulted in a significant reduction in uterine cancer cases. Importantly, given the natural history of uterine cancer, this reduction in cases persisted for more than ten years. A number of new tests for the early diagnosis of uterine cancer leveraging genomic changes in serum and exfoliated uterine cells have demonstrated promising preliminary results.(46) Analyses of the potential role of these tests for screening and early detection of uterine cancer using our natural history model are ongoing.

We recognize a number of important limitations. First, as with any microsimulation model, our model is based on population-level estimates of numerous parameters including risk factors, protective factors, cancer incidence, and mortality. We performed comprehensive literature searches to identify the best available sources of data. Further, we utilized model development methodology common to the CISNET modeling consortium which has produced natural history models for many solid tumors that have been used to inform policy and practice decisions.(47–49) Second, it is difficult to forecast future rates of hysterectomy and obesity. We provide modeling data based on current estimates of the hysterectomy rate, obesity, and survival for patients with endometrial cancer as well as the best available projections of future trends. Third, we did not model all potential risk factors. Work is ongoing to develop more precise estimates of other risk factors for uterine cancer including reproductive factors such as parity, age at menopause and age at menarche.

While these factors are not specifically modeled as parameter estimates, the trends in the incidence of uterine cancer naturally account for changing trends over time. Fourth, although non-endometrioid tumors account for a disproportionate number of deaths from uterine cancer, the smaller annual number of cases leads to less precision in model estimations. Further, non-endometrioid cancers were modeled using the same parameters as endometrioid cancers, despite potential differences in their risk factor associations, and that EIN is a recognized precursor for endometrioid but not for all non-endometrioid cancers. Given that the UT-MO model estimates closely align with the SEER estimates, we hypothesize that the UT-MO model projections are reliable for both endometrioid and non-endometrioid subtypes. Fifth, we performed extensive literature reviews to inform model inputs. We recognize some sources are dated, however for each parameter and calibration target we prioritized the highest quality and most recent data. Lastly, the current iteration of UTMO only includes Black and White women and does not incorporate uterine sarcomas. The present model represents our base-case analysis, and further model development will include other racial and ethnic groups and patients with sarcomas.

In summary, we have developed and calibrated a natural history model of uterine cancer that accurately reflects the incidence and mortality of the disease. This model suggests that the number of new cases of uterine cancer will rise dramatically over the next three decades. These population-level trends support the urgent need to develop and implement novel primary and secondary prevention strategies for uterine cancer.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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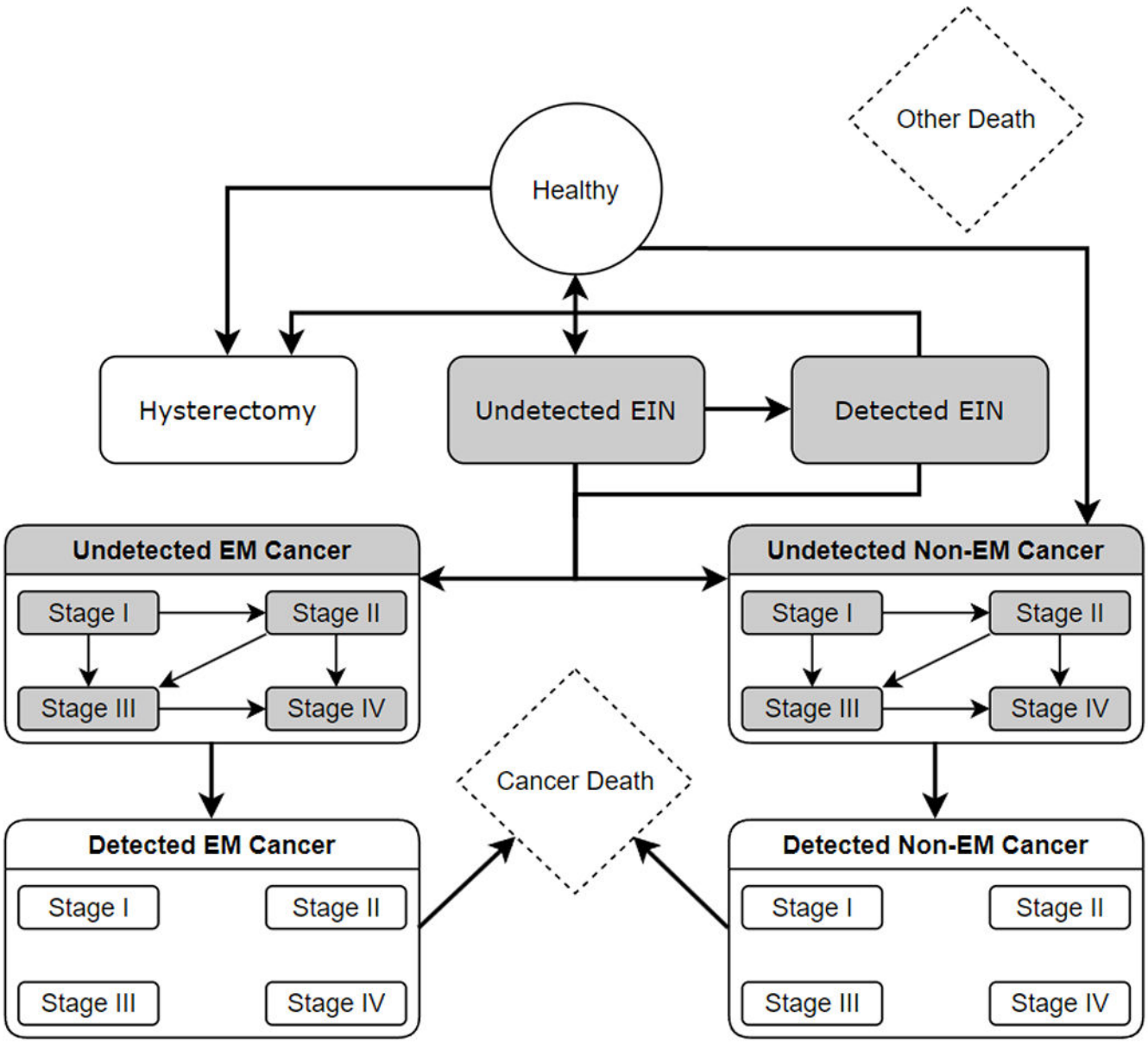

Figure 1.
Columbia University uterine cancer microsimulation model (UT-MO). Incidence and mortality projections for uterine cancer.

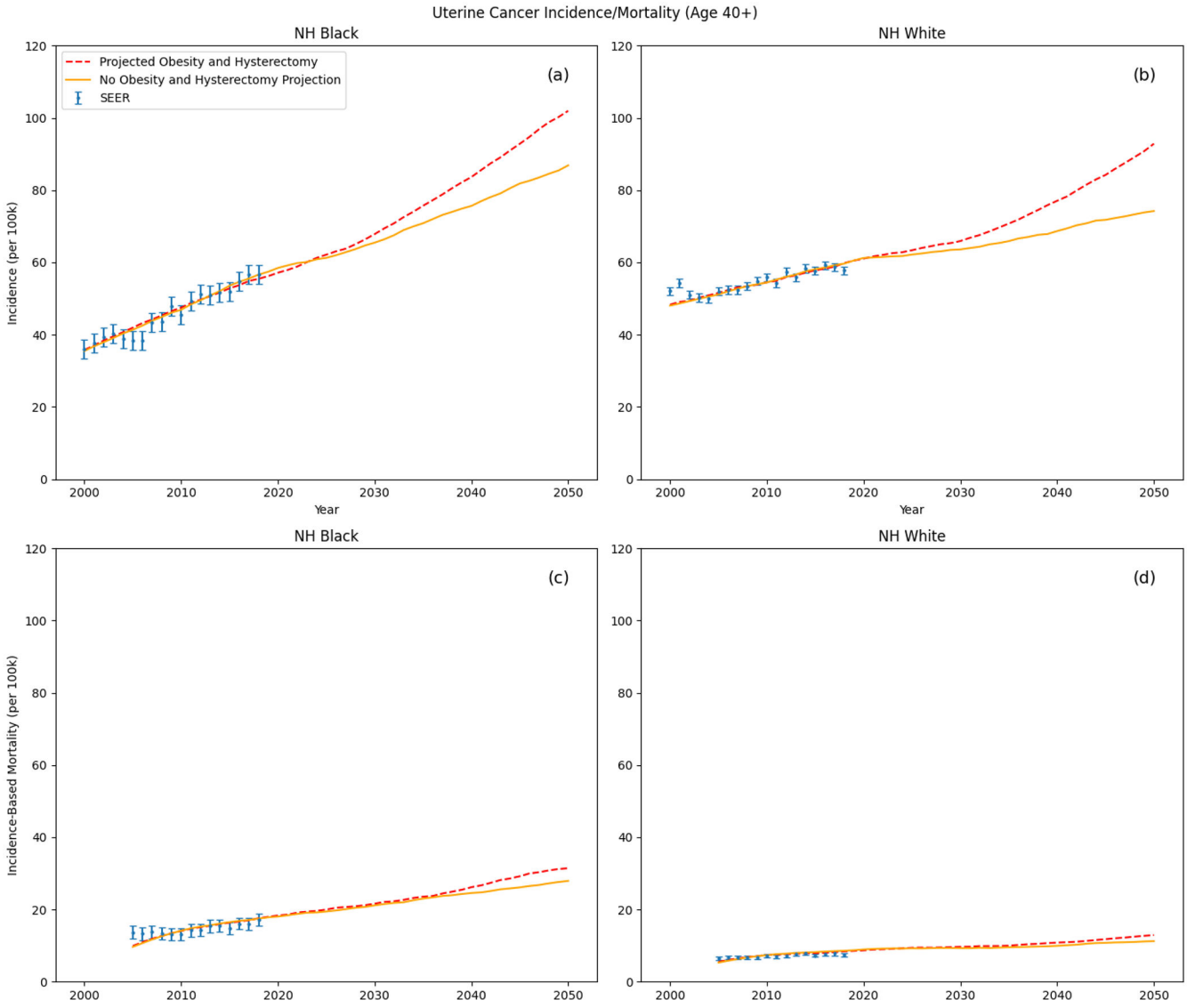

Figure 2.

Projected age adjusted uterine cancer incidence and incidence-based mortality among women aged 40+ stratified by race and ethnicity to 2050. A. Incidence in Black women. B. Incidence in White women. C. Incidence-Based Mortality in Black women. D. Incidence-Based Mortality in White women. Orange solid line: using LOCF method for future hysterectomy and obesity trend; red dotted line: incorporating projected hysterectomy and obesity trend.

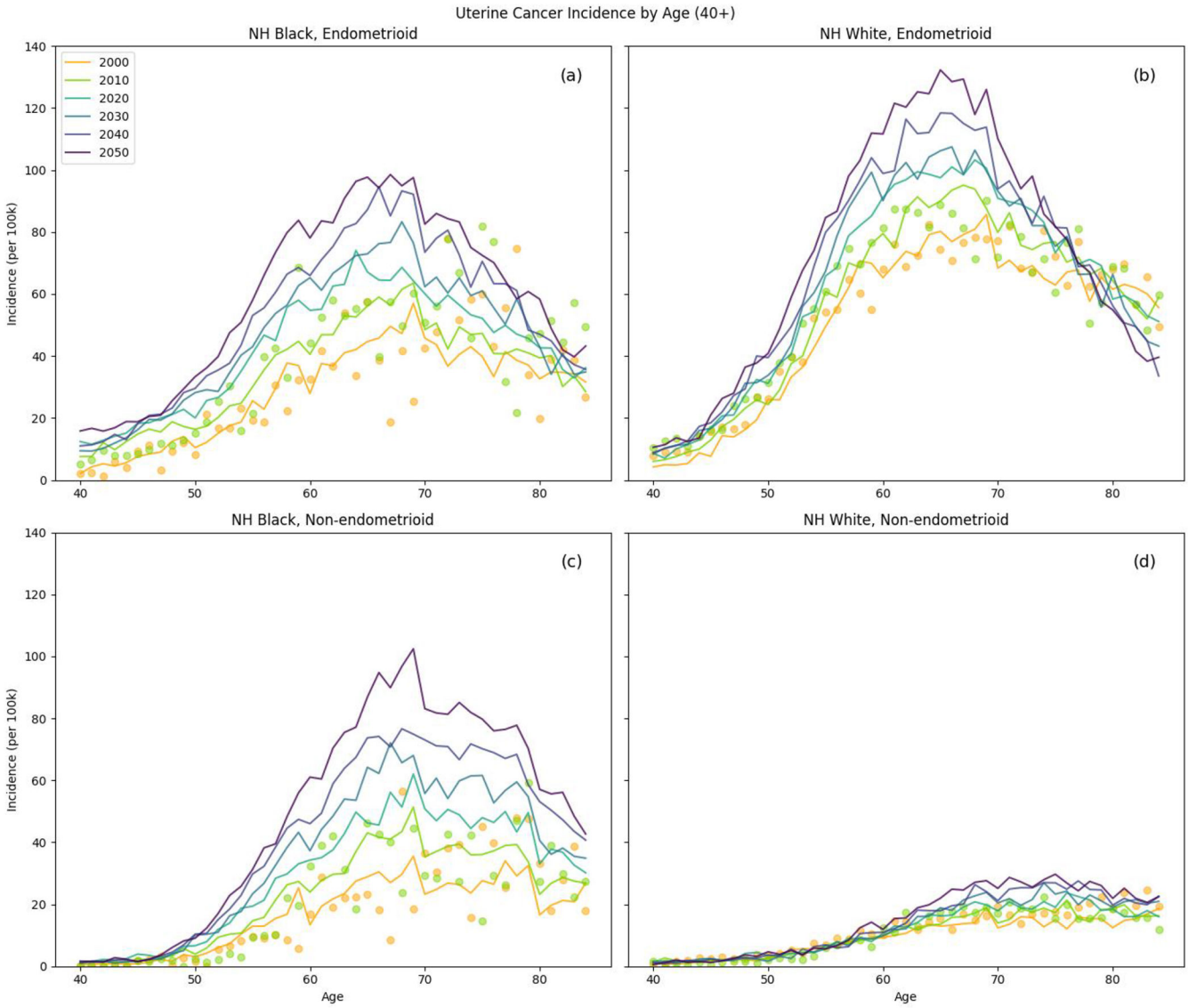

Figure 3.

Histology-specific age adjusted uterine cancer incidence stratified by race and ethnicity and birth cohort among women aged 40+. A. Endometrioid cancer in Black women. B. Endometrioid cancer in White women. C. Non-endometrioid cancer in Black women. D. Non-endometrioid cancer in White women.

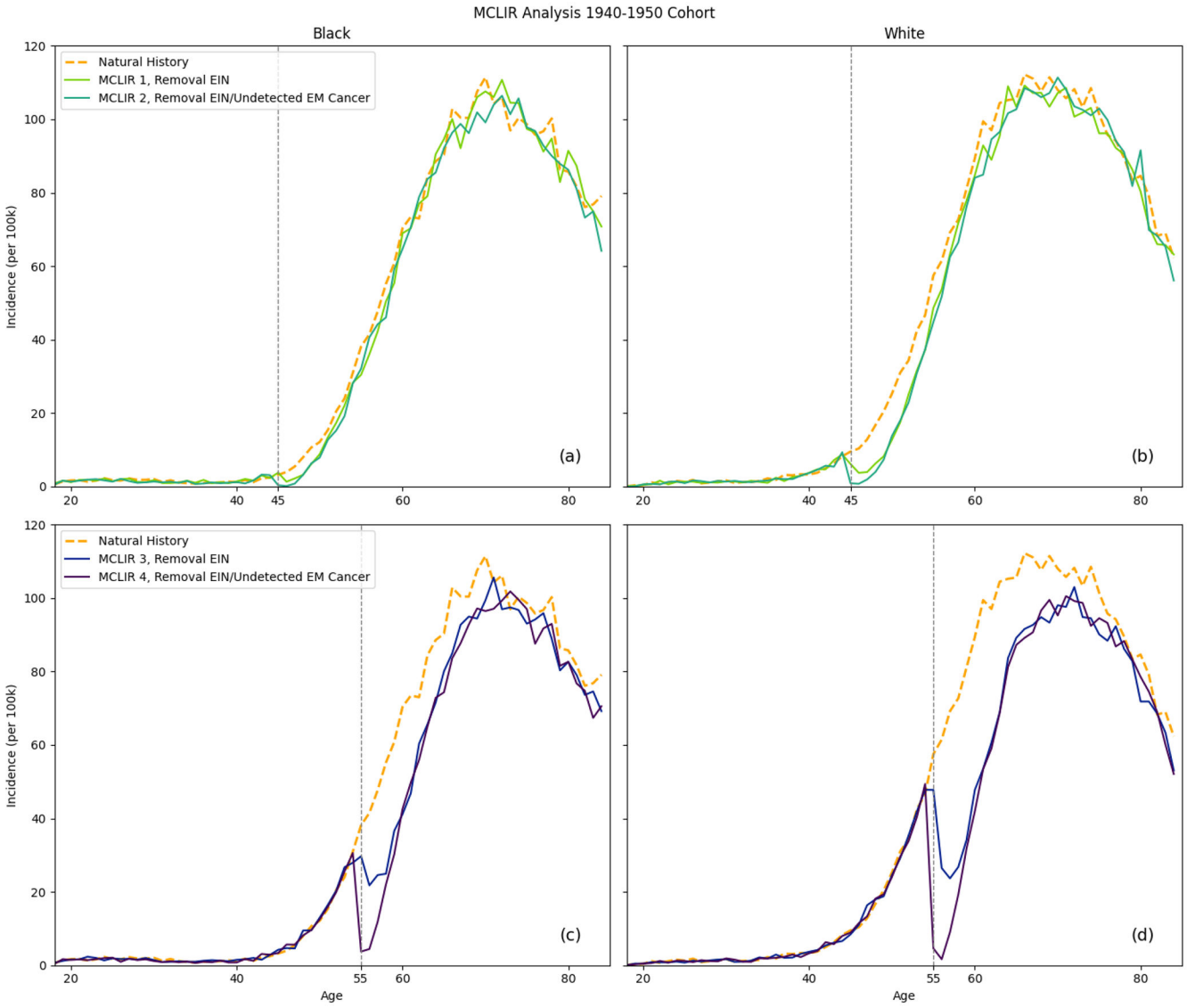

Figure 4.

Sensitivity analysis using maximum clinical incidence reduction (MCLIR) scenario analysis in the birth cohort of 1940-1950. A. Removal of undetected endometrial intraepithelial neoplasia (scenario 1) or undetected endometrioid cancer (scenario 2) in 45-year-old Black patients.

B. Removal of undetected endometrial intraepithelial neoplasia (scenario 1) or undetected endometrioid cancer (scenario 2) in 45-year-old White patients.

C. Removal of undetected endometrial intraepithelial neoplasia (scenario 3) or undetected endometrioid cancer (scenario 4) in 55-year-old Black patients.

D. Removal of undetected endometrial intraepithelial neoplasia (scenario 3) or undetected endometrioid cancer (scenario 4) in 55-year-old White patients.

Table 1.

Input parameters and calibration targets for the natural history model.

	Implementation	Data Source	Example
Model inputs			
Obesity prevalence	Simulated BMI category by age and birth cohort	NHANES (2000-2020) (www.cdc.gov/nchs/nhanes/index.html)	1960-1970 birth cohort, NH White female, age 50, 40.5% BMI ≥ 30 kg/m^2
Impact of obesity	Fixed transition risk ratio	Zhao et al (2021)(24),Epplein et al (2008)(13)	Women with BMI ≥ 25 kg/m^2 OR 2.7 for developing EMC as compared to BMI < 25
Obesity Projections *	Projected Increase in obesity proportion	GBD 2024(33)	From 2020-2025, the proportion of obese people will go up by 6%
Hysterectomy rates	Monthly, race- & age-specific probability of hysterectomy	NHANES (2000-2020) (www.cdc.gov/nchs/nhanes/index.html)	NH Black women Aged 45-49, P(Hysterectomy) = 0.002
Hysterectomy Projections *	Projected decrease in hysterectomy rate	Simms et al (2020)(32)	From 2020-2035, the hysterectomy rate will decrease by 25.7%
Hysterectomy mortality	Competing hazard	Wingo et al (1985)(23)	Hysterectomy mortality excluding pregnancy and cancer = 6.0 per 10k
All-cause mortality	Monthly, race, cohort & age-specific probability of death (excluding uterine cancer)	CDC Wonder (1968-2016)(https://wonder.cdc.gov/)	1950-1960 birth cohort, NH White women aged 55-59, P(Death) = 0.0008
Cancer-specific survival	10-year follow up survival hazards by race/ethnicity, histology, stage, age and cohort	SEER (2000-2018)(https://seer.cancer.gov/)	NH White women aged 40-44, EM stage IV, 1st year monthly P(cancer death) = 0.0078
Cancer sojourn time	Undetected state progression upper and lower bounds	Broder et al (2021)(10)	Stage III to stage IV progression median time 1.5 years
Calibration targets			
EIN incidence	Age-specific incidence rates	Reed et al (2009)(20)	Women aged 60-65, 28.9 per 100,000
EIN progression	Undetected state progression upper and lower bounds	Lacey et al (2008)(14)	3-year risk of progression for patient diagnosed with EIN of 8.2%
EIN prevalence	Age-specific prevalence rates	Korhonen et al (1997)(15)	Women aged 45-55, subclinical prevalence 0-2 per 3000
Endometrial cancer prevalence	Age-specific prevalence rates	Horwitz et al (1981)(27),Göl et al (2001)(28)	Rate of EM cancer detected at autopsy 22-31 per 10,000
Cancer incidence	Race/ethnicity-, histology-, stage-, age- and cohort-specific incidence rates	SEER (2000-2018)(https://seer.cancer.gov/)	1940-1950 birth cohort, NH Black women aged 70, EM stage I, 38.8 per 100,000

EIN: endometrial intraepithelial neoplasia. EM: endometrioid. Non-EM: non-endometrioid.

* Only present in projected obesity and hysterectomy trend analysis

Table 2.

Observed and projected incidence and mortality of uterine cancer overall and stratified by histology.

	Calibration						*Projection*					
Outcome	**2000**		**2010**		**2018**		**2030**		**2040**		**2050**	
	Black	**White**	**Black**	**White**	**Black**	**White**	**Black**	**White**	**Black**	**White**	**Black**	**White**
Incidence (40+)												
Overall												
SEER	36.0 (33.4, 38.6)	52.1 (51.0, 53.2)	45.5 (42.9, 48.1)	55.9 (54.8, 57.0)	56.8 (54.2, 59.4)	57.7 (56.6, 58.9)	-	-	-	-	-	-
UT-MO	35.5 (32.1, 38.9)	48.0 (46.3, 49.7)	46.9 (43.5, 50.3)	54.4 (52.7, 56.2)	56.6 (53.2, 60.0)	59.7 (57.9, 61.4)	65.5 (62.1, 68.9)	63.6 (61.9, 65.3)	75.6 (72.2, 79.0	68.7 (67.0, 70.4)	86.9 (83.5, 90.3)	74.2 (72.5, 75.9)
Endometrioid												
SEER	23.5 (22.1, 24.9)	43.6 (42.7, 44.6)	30.7 (29.3, 32.2)	48.2 (47.3, 49.2)	34.2 (32.8, 35.7)	49.2 (48.3, 50.2)	-	-	-	-	-	-
UT-MO	23.0 (21.2, 24.8)	41.0 (39.5, 42.5)	29.6 (27.9, 31.4)	46.7 (45.2, 48.2)	35.2 (33.4, 37.0)	51.2 (9.8, 52.7)	39.3 (37.5, 41.1)	54.5 (53,0, 56.0)	44.7 (42.9, 46.5)	58.8 (57.3, 60.3)	50.5 (48.7, 52.3)	63.4 (61.9, 64.9)
Non-endometrioid												
SEER	12.5 (11.2, 13.8)	8.5 (8.3, 8.7)	14.8 (13.5, 16.0)	7.7 (7.4, 7.9)	22.5 (21.2, 23.8)	8.5 (8.3, 8.8)	-	-	-	-	-	-
UT-MO	12.5 (10.9, 14.1)	7.0 (6.8, 7.3)	17.2 (15.6, 18.8)	7.8 (7.5, 8.0)	21.4 (19.8, 23.0)	8.4 (8.2, 8.7)	26.2 (24.6, 27.8)	9.1 (8.8, 9.3)	30.9 (29.3, 32.5)	9.9 (9.6, 10.1)	36.3 (34.7, 37.9)	10.8 (10.6, 11.1)
5-Year Survival (%)	2013 *											
Overall												
SEER	66.5 (62.1, 70.5)	86.6 (85.7, 87.5)	68.5 (65.3, 71.6)	86.6 (85.8, 87.4)	69.1 (66.1, 71.8)	89.0 (85.1, 86.8)	-	-	-	-	-	-
UT-MO	71.4 (69.5, 73.3)	86.3 (85.0, 87.5	67.2 (65.4, 69.0)	85.7 (84.4, 87.0)	67.1 (65.3, 68.9)	85.7 (84.4, 87.0)	66.7 (65.2, 68.3)	85.1 (83.9, 86.3)	64.3 (62.8, 65.7)	85.1 (83.9, 86.2)	68.2 (67.0, 69.5)	83.8 (82.6, 84.9)
Endometrioid												
SEER	81.8 (75.4, 86.2)	91.5 (90.5, 92.5)	84.5 (80.9, 87.5)	92.5 (91.7, 93.2)	86.5 (83.3, 89.1)	92.2 (91.4, 92.9)	-	-	-	-	-	-

	Calibration						*Projection*					
Outcome	**2000**		**2010**		**2018**		**2030**		**2040**		**2050**	
	Black	**White**	**Black**	**White**	**Black**	**White**	**Black**	**White**	**Black**	**White**	**Black**	**White**
UT-MO	88.8 (87.2, 90.5)	92.3 (91.2, 93.3)	86.5 (84.8, 88.2)	92.4 (91.3, 93.4)	86.4 (84.7,88.2)	92.6 (91.5, 93.6)	86.9 (85.4, 88.3)	91.8 (90.8, 92.8)	86.4 (85.0, 87.8)	91.3 (90.3, 92.3)	88.0 (86.8, 89.1)	91.5 (90.5, 92.5)
Non-endometrioid												
SEER	38.4 (30.0, 46.7)	50.0 (45.6, 54.3)	39.0 (32.4, 45.5)	50.0 (46.0, 53.8)	42.9 (37.3, 48.4)	52.4 (48.4, 55.9)	-	-	-	-	-	-
UT-MO	44.5 (41.2, 47.8)	50.5 (45.6, 55.4)	40.2 (37.3, 43.2)	52.8 (48.3, 57.3)	37.5 (34.5, 40.5)	45.9 (41.2, 50.7)	41.7 (39.3, 44.0)	48.4 (44.1, 52.7)	40.5 (38.3, 42.6)	51.6 (47.6, 55.6)	43.2 (41.2, 45.1)	45.5 (41.7, 49.3)

SEER: Surveillance, Epidemiology, and End Results database. UT-MO: Columbia University uterine cancer model.

*2013 was the last year with 5-year survival data from SEER 18.

Table 3.

Sojourn times stratified by race and histology.

Sojourn time (months)					
		Black		*White*	
Sojourn Time	***Histology***	***Mean***	***SD***	***Mean***	***SD***
Pre-invasive	Endometrioid	127.6	105.1	97.8	72.9
	Non-endometrioid	27.1	54.6	52.3	73.0
Malignancy	Endometrioid	22.4	19.7	21.9	19.8
	Non-endometrioid	8.2	6.6	8.7	6.6

SD: Standard deviation