Supplementary Appendix: Model Structure

Model Structure

AG-SIM is a deterministic, continuous-time, compartmental SD model implemented to run from the year 2020 to 2050. The model represents aggregated population stocks rather than individual-level trajectories. This approach allows efficient projection of long-term population-level outcomes while preserving core disease progression dynamics (Homer & Hirsch, 2006). The model consists of the following interconnected sectors: (a) Cleveland population dynamics, (b) risk factor sub-models, (c) Community Health Workers, and (d) Cardiovascular risk engine. The risk factor sub-models include the three risk factors in the Achieve-Greater interventions: hypertension, diabetes, and weight management. Feedback relationships connect these sectors, enabling representation of reinforcing processes such as untreated hypertension leading to higher CVD incidence and balancing processes such as treatment-induced SBP reduction, lowering CVD risk. The AG-SIM accounts for age, gender, and race.

Cleveland Population Sub-model

The Cleveland population sub-model (Figure 1) projects the demographic evolution of the Cleveland population and generates cohorts of individuals who age into adulthood (age 18 years) as inputs to the cardiometabolic risk-factor sub-models. Because the risk-factor modules represent adults aged 18 years and older, the population sub-model serves as the demographic foundation for downstream disease projections. The population is disaggregated by single-year age cohorts (0 to 100+ years), sex (female, male), and race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, and Other). This stratification preserves demographic heterogeneity and ensures alignment with age-, sex-, and race-specific disease risk parameters used in subsequent modules. Population size changes dynamically through births, deaths, and net migration. Births are determined by applying age-specific fertility rates to females of reproductive age (15–49 years). Deaths are calculated using age-specific mortality rates derived from life tables. Net migration is estimated through model calibration to historical population trends to ensure accurate replication of observed demographic trajectories.

Aging is represented using a cohort advancement structure. Newborns are assigned to the age 0 cohort. At the end of each annual cycle (cohort length = 1 year), the surviving population in each age cohort transitions to the next. This aging process continues sequentially through all age groups. Individuals in the terminal cohort (age 100 years and older) remain within that group, with reductions occurring only through mortality. This structure ensures demographic consistency over time and allows accurate projection of the adult population eligible for cardiometabolic risk modeling.

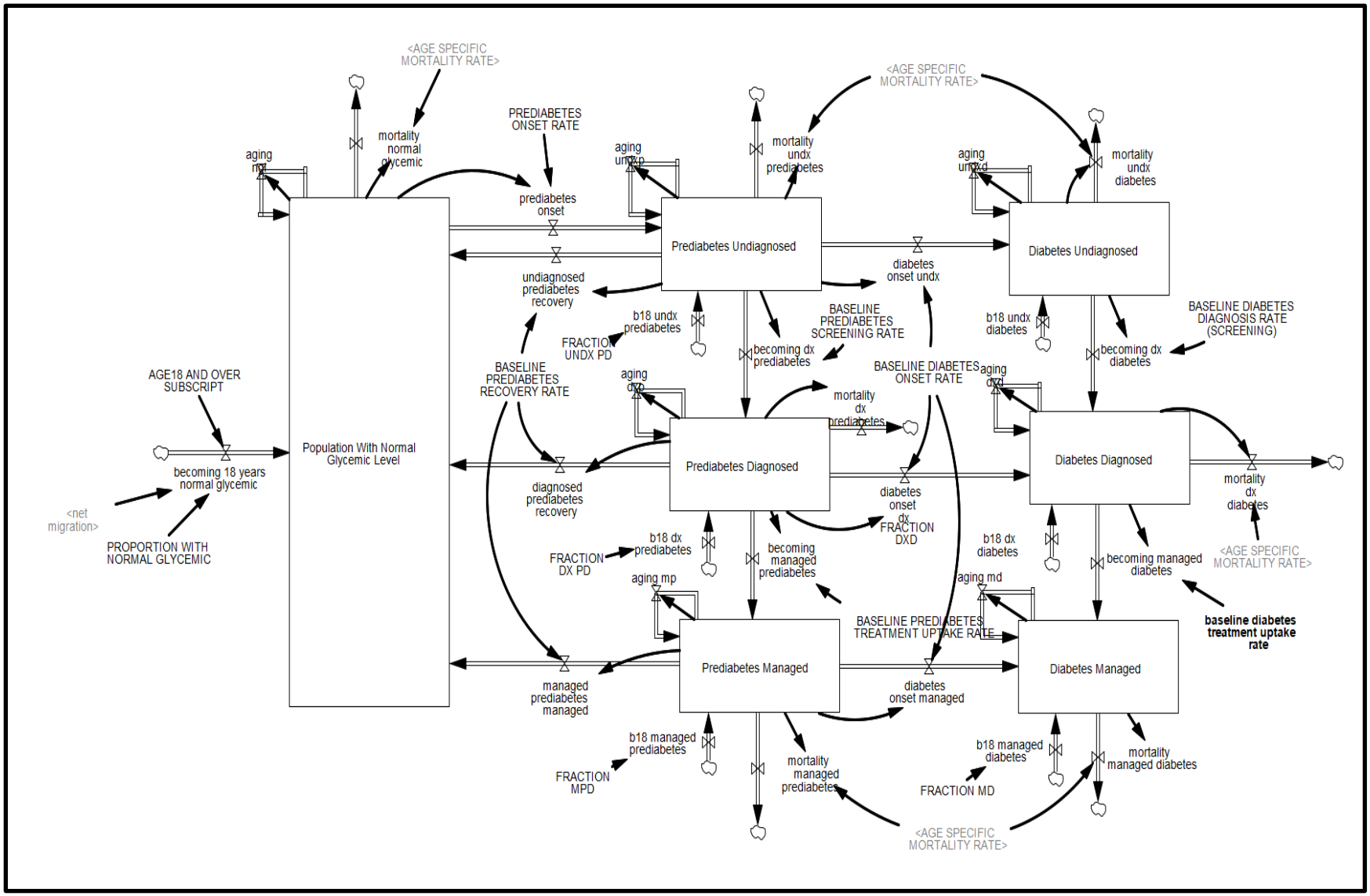
Diabetes Sub-model

The diabetes sub-model (Figure 2) simulates the progression and management of glycemic health states among adults aged 18 years and older. The adult population is stratified into three primary glycemic states: normal glycemia, prediabetes, and diabetes. To reflect heterogeneity in

screening, diagnosis, and treatment, each glycemic state is further disaggregated into diagnostic and management subgroups—undetected (undiagnosed), diagnosed, and managed. This layered structure enables the model to distinguish between biological disease progression and health system–mediated processes, such as screening and treatment, allowing for an explicit representation of how diagnosis and disease management modify downstream cardiovascular risk and mortality. Glycemic state classification follows current American Diabetes Association (ADA) criteria (ADA, 2025), with normal glycemia defined as HbA1c < 5.7%, prediabetes as HbA1c 5.7–6.4%, and diabetes as HbA1c ≥ 6.5%. Transitions between glycemic states are governed by age-, sex-, and race-specific rates of incidence, recovery, diagnosis, and treatment initiation. These parameters were derived from Cleveland-specific surveillance data (Healthy Northeast Ohio), epidemiologic studies, and empirical data from the Achieve-Greater Study. Mortality rates are adjusted to reflect the elevated risk associated with diabetes.

Population flows between glycemic states are modeled dynamically. The normal glycemic population increases as individuals age into adulthood (age 18), migrate into the city with normal glycemia, or revert from prediabetes through recovery. It decreases through progression to prediabetes or death. The undiagnosed prediabetes population grows as individuals develop impaired glycemia or enter adulthood, and declines through diagnosis, progression to diabetes, recovery, or death. Diagnosed prediabetes increases through screening and detection and may transition either to managed prediabetes with treatment initiation or to diabetes if glycemic control worsens. Managed prediabetes expands through treatment uptake among diagnosed individuals and contracts through recovery, treatment discontinuation, progression to diabetes, or death. Similarly, the undiagnosed diabetes population increases through incident diabetes cases and declines via diagnosis or death. Diagnosed diabetes increases through new detection or progression from prediabetes and may transition to managed diabetes upon treatment initiation. The managed diabetes population grows through sustained treatment engagement and decreases primarily through mortality or treatment dropout. Transitions across different health states were estimated by calibration. The model operates on an annual time scale from 2010 through 2050, consistent with long-term chronic disease projection frameworks.

Model calibration was performed to ensure that the simulated baseline prevalence of prediabetes and diabetes from 2010 to 2025 reproduced observed epidemiologic trends in Cleveland. Calibration involved iteratively adjusting incidence, diagnosis, and management parameters within empirically plausible ranges until model outputs aligned with surveillance data. Sensitivity analyses were conducted to evaluate the robustness of projections to uncertainty in key parameters, including incidence rates, diagnosis probabilities, treatment initiation rates, and diabetes-related excess mortality. This structure allows the model to capture both biological progression and healthcare system effects, enabling estimation of how improvements in screening, diagnosis, and management—such as those driven by Community Health Worker interventions—translate into long-term reductions in diabetes prevalence and cardiovascular risk.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prediabetes onset
aging undxp
mortality undx prediabetes
aging undxd
mortality undx diabetes
Prediabetes Undiagnosed
Diabetes Undiagnosed
diabetes onset undx
undiagnosed prediabetes recovery
b18 undx prediabetes
FRACTION UNDX PD
BASELINE PREDIABETES SCREENING RATE
becoming dx prediabetes
b18 undx diabetes
becoming dx diabetes
BASELINE DIABETES DIAGNOSIS RATE (SCREENING)
AGE18 AND OVER SUBSCRIPT
BASELINE PREDIABETES RECOVERY RATE
BASELINE DIABETES ONSET RATE
aging dxp
mortality dx prediabetes
aging dxd
Population With Normal Glycemic Level
Prediabetes Diagnosed
Diabetes Diagnosed
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diagnosed prediabetes recovery
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mortality dx diabetes
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FRACTION DX PD
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b18 dx diabetes
becoming managed diabetes
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aging md
baseline diabetes treatment uptake rate
BASELINE PREDIABETES TREATMENT UPTAKE RATE
Prediabetes Managed
Diabetes Managed
managed prediabetes managed
diabetes onset managed
b18 managed prediabetes
FRACTION MPD
mortality managed prediabetes
b18 managed diabetes
FRACTION MD
mortality managed diabetes
<AGE SPECIFIC MORTALITY RATE>

Figure xx:

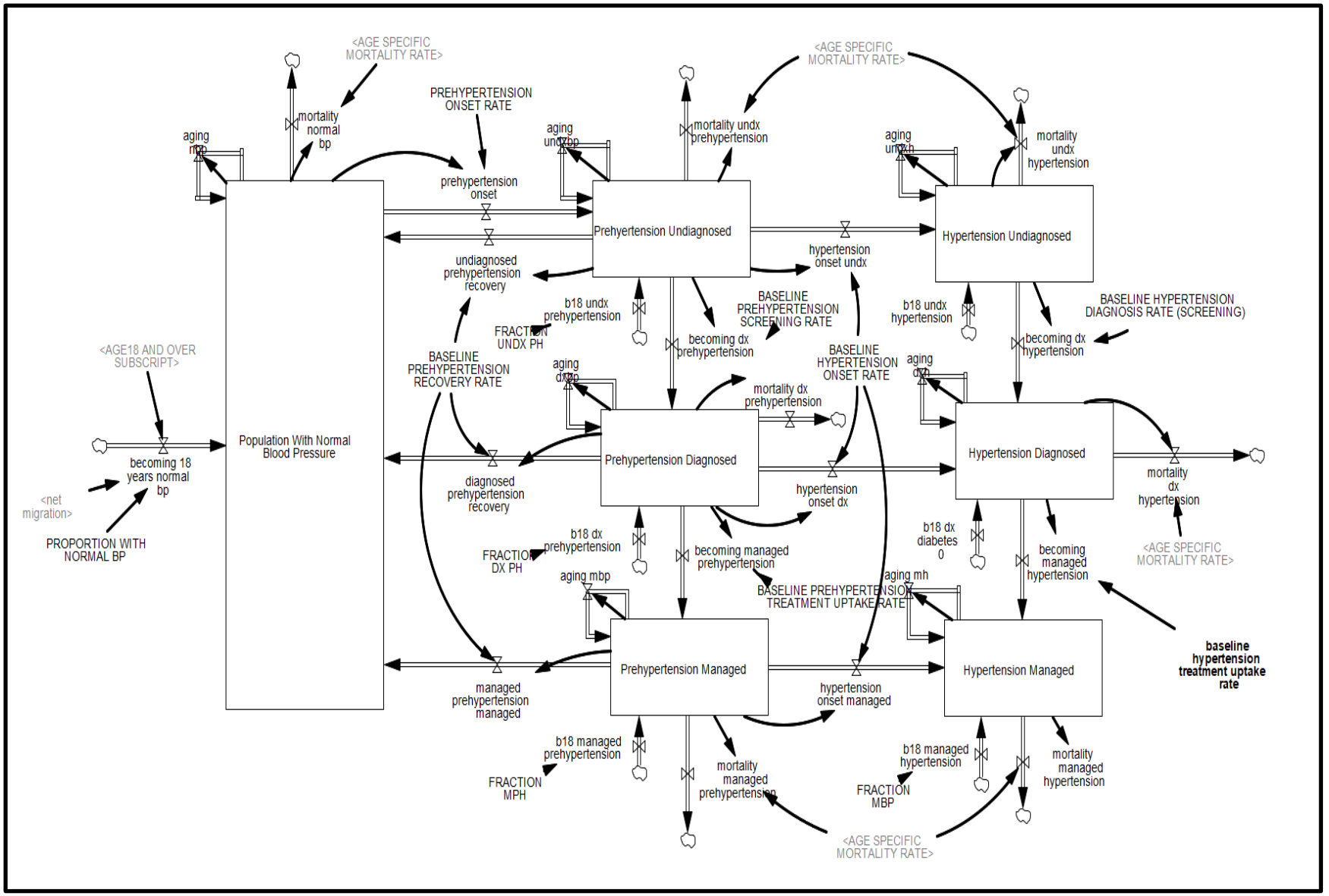
Hypertension Sub-Model

The hypertension sub-model (Figure X) simulates the progression, detection, and management of blood pressure states among adults aged 18 years and older. The adult population is categorized into three clinically meaningful blood pressure states: normal blood pressure, prehypertension (elevated blood pressure), and hypertension. Normal blood pressure is defined as systolic blood pressure (SBP) < 120 mmHg and diastolic blood pressure (DBP) < 80 mmHg. Prehypertension represents early vascular risk, defined as SBP 120–139 mmHg or DBP 80–89 mmHg. Hypertension is defined as SBP ≥ 140 mmHg or DBP ≥ 90 mmHg. These categories allow the model to represent early risk accumulation and the potential for intervention before sustained hypertension develops. Within each blood pressure category, individuals are further distinguished by awareness and management status. Specifically, people may be unaware of their elevated blood pressure, be diagnosed but untreated, or have it actively managed. This distinction is essential because cardiovascular risk differs substantially between controlled and uncontrolled hypertension, and because screening and treatment processes represent modifiable policy levers.

Blood pressure progression occurs gradually over time. Individuals may transition from normal blood pressure to prehypertension, and from prehypertension to hypertension, at rates calibrated to Cleveland-specific epidemiologic data. Regression to lower-risk states is also permitted, reflecting lifestyle modification or treatment effects. These bidirectional transitions allow the model to represent both worsening and improvement in vascular risk. Screening influences movement from undiagnosed to diagnosed states. Diagnosis enables entry into the treatment pathway, where pharmacologic and behavioral interventions reduce mean SBP. Treatment effectiveness is represented as a reduction in average SBP among managed individuals, which, in turn, lowers the probability of cardiovascular events through the integrated risk engine. Treatment discontinuation or loss of adherence returns individuals to an uncontrolled state, increasing their risk profile. Mortality risk is state-dependent. Individuals with uncontrolled hypertension experience elevated cardiovascular mortality relative to those with normal blood pressure or well-controlled hypertension. Excess mortality risk parameters were informed by large prospective cohort studies and incorporated into the cardiovascular event calculation module. The hypertension sub-model operates on an annual time step and is dynamically linked to the BMI and diabetes sub-models. Weight gain increases the probability of progression to prehypertension and hypertension, while weight reduction reduces progression rates. Similarly, the coexistence of diabetes modifies cardiovascular risk multiplicatively within the risk engine.

Model calibration ensured that simulated baseline hypertension prevalence, awareness, treatment, and control rates aligned with Cleveland surveillance data for the period 2010–2025. Parameter adjustment was performed within empirically plausible ranges to reproduce observed epidemiologic trends. Sensitivity analyses were conducted to evaluate the stability of long-term projections under variation in incidence rates, diagnosis probabilities, adherence levels, treatment efficacy, and excess mortality associated with uncontrolled hypertension. By explicitly representing early vascular risk (prehypertension), detection processes, and sustained management, the hypertension sub-model enables assessment of how improvements in

screening coverage, treatment adherence, and blood pressure control—such as those achieved through Community Health Worker–led interventions—translate into reductions in long-term cardiovascular events and mortality at the population level.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mortality undx prehypertension
Prehyertension Undiagnosed
undiagnosed prehypertension recovery
b18 undx prehypertension
FRACTION UNDX PH
becoming dx prehypertension
BASELINE PREHYPERTENSION SCREENING RATE
BASELINE PREHYPERTENSION RECOVERY RATE
aging dxbp
mortality dx prehypertension
Prehypertension Diagnosed
diagnosed prehypertension recovery
b18 dx prehypertension
FRACTION DX PH
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Prehypertension Managed
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b18 managed prehypertension
FRACTION MPH
mortality managed prehypertension
<AGE SPECIFIC MORTALITY RATE>
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mortality undx hypertension
Hypertension Undiagnosed
hypertension onset undx
b18 undx hypertension
BASELINE HYPERTENSION DIAGNOSIS RATE (SCREENING)
becoming dx hypertension
BASELINE HYPERTENSION ONSET RATE
aging dxh
Hypertension Diagnosed
hypertension onset dx
b18 dx diabetes 0
becoming managed hypertension
mortality dx hypertension
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BASELINE PREHYPERTENSION TREATMENT UPTAKE RATE
Hypertension Managed
baseline hypertension treatment uptake rate
hypertension onset managed
b18 managed hypertension
FRACTION MBP
mortality managed hypertension
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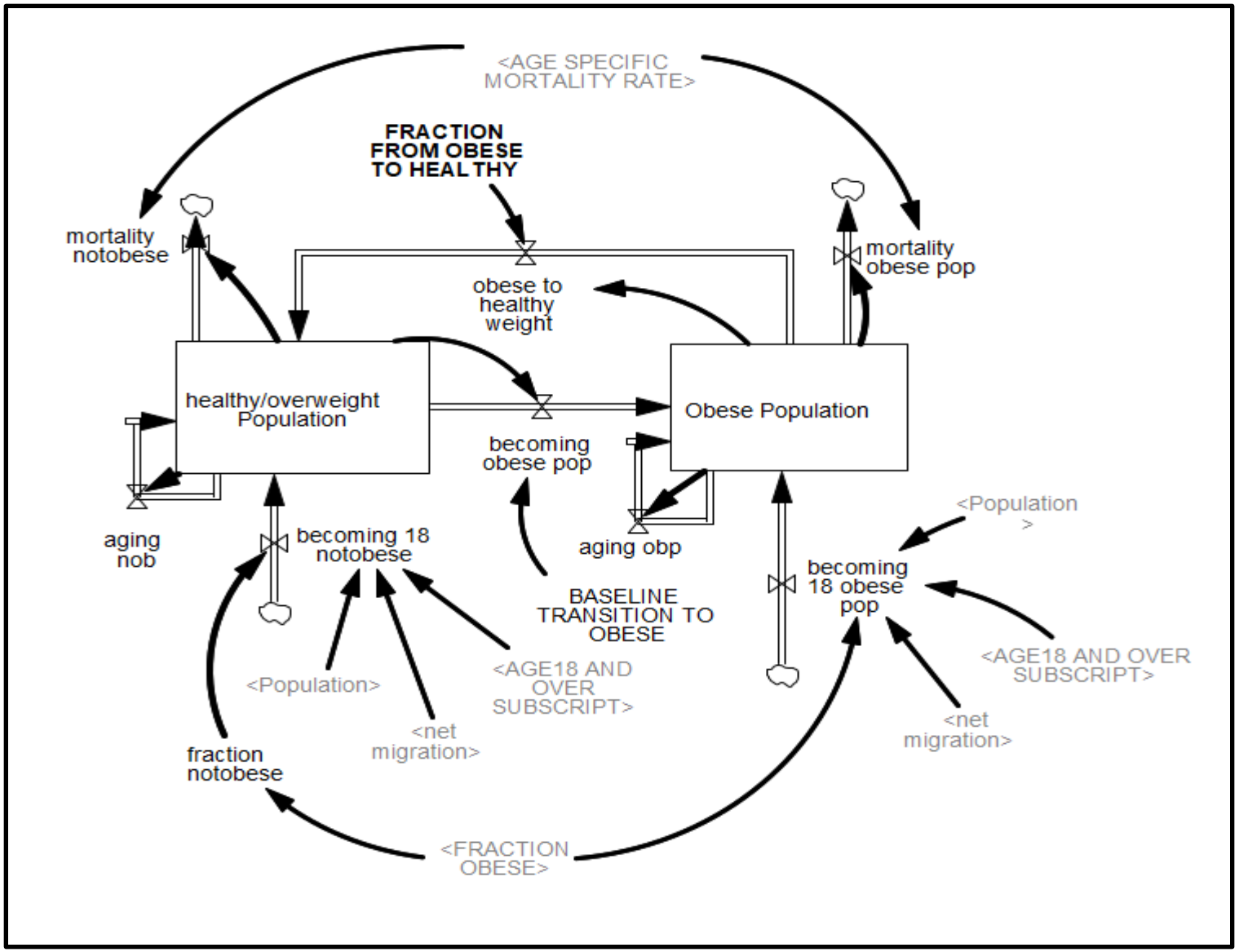
Figure xx:

Body Mass Index (BMI) Sub-Model

The Body Mass Index (BMI) sub-model represents the distribution and evolution of weight status among adults aged 18 years and older and serves as a key upstream determinant within the broader cardiometabolic system. Rather than modeling granular BMI categories, the structure focuses on two clinically meaningful population states: healthy/overweight and obese. This binary classification captures the threshold at which excess adiposity substantially amplifies cardiometabolic risk while maintaining parsimony in long-term population projections.

Individuals are stratified by age (18 years and older), sex (female, male), and race/ethnicity (non-Hispanic White, non-Hispanic Black, Hispanic, and Other). This disaggregation enables the model to capture demographic heterogeneity in obesity prevalence, weight-gain trajectories, and associated health risks. At any given time, adults are in one of two BMI states. Transitions occur bidirectionally: individuals may move from healthy/overweight to obese through net weight gain, or from obese to healthy/overweight through sustained weight reduction. These transitions represent the combined effects of aging, environmental exposure, behavioral patterns, socioeconomic context, and intervention effects. Progression to obesity is influenced by secular weight trends and structural factors such as food access and economic stability, which are modeled in linked sub-systems. Movement from obesity to healthy/overweight reflects lifestyle modification, clinical weight management, and CHW-supported interventions.

Both BMI states are subject to mortality, with obesity-associated excess mortality incorporated into the cardiovascular risk engine rather than applied directly within the BMI module. This ensures that the downstream impact of obesity operates through increased cardiometabolic risk rather than isolated mortality adjustments. Population inflows occur as individuals age into adulthood (age 18), at which point they enter the BMI distribution consistent with observed baseline prevalence in Cleveland. Net migration effects are incorporated through the broader population sub-model. The BMI sub-model operates dynamically on an annual time step from 2025 to 2050. Weight transitions are estimated using calibration to reproduce historical trends in Cleveland obesity prevalence prior to the intervention period. Importantly, the BMI sub-model interacts bidirectionally with both the diabetes and hypertension sub-models. Obesity increases the probability of progression to prediabetes and diabetes and accelerates progression to prehypertension and hypertension. Conversely, reductions in obesity prevalence lower incident rates of diabetes and hypertension over time. Through these linkages, changes in the BMI distribution indirectly influence long-term projections of cardiovascular events. Calibration procedures ensured that simulated baseline obesity prevalence and secular trends aligned with Cleveland surveillance data from 2010 to 2025. Sensitivity analyses were conducted to assess the robustness of long-term projections to variations in weight-gain rates, weight-loss effectiveness, and intervention coverage.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FRACTION FROM OBESE TO HEALTHY
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mortality obese pop
obese to healthy weight
healthy/overweight Population
Obese Population
becoming obese pop
aging nob
becoming 18 notobese
aging obp
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BASELINE TRANSITION TO OBESE
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<AGE18 AND OVER SUBSCRIPT>
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fraction notobese
<FRACTION OBESE>

Figure xx:

CVD Risk Engine

The CVD sub-model (Figure 4) projects CVD events, CVD deaths, and the post-CVD population aged 18 years and older in Cleveland. The Prevent Risk Engine for CVD served as a foundation for estimating the probability of CVD events based on risk factors. To estimate the probability of CD events, the population was divided into eight risk groups, allowing estimation of different CVD event probabilities for each group. The risk groups are (a) normal—individuals with no known risk factors; (b) hypertension only (SBP); (C) diabetes only; (d) BMI only; (e) hypertension and Diabetes; (f) hypertension and BMI; (g) diabetes and BMI; (H) hypertension and Diabetes and BMI. The equations for the probability of CVD events given the risk group are:

$$P(CVD_{normal}) = {}^{1}/_{(1+EXP(-(\beta_0)))} \cdots\cdots 1$$

$$P(CVD_{hypertension}) = {}^{1}/_{(1+EXP(-(\beta_0+\beta_1 hypertension_i)))} \cdots\cdots 2$$

$$P(CVD_{diabetes}) = {}^{1}/_{(1+EXP(-(\beta_0+\beta_2 diabetes)))} \cdots\cdots 3$$

$$P(CVD_{BMI}) = {}^{1}/_{(1+EXP(-(\beta_0+\beta_3 BMI_i)))} \cdots\cdots 4$$

$$P(CVD_{hypertension\&diabetes}) = {}^{1}/_{(1+EXP(-(\beta_0+\beta_1 hypertension+\beta_2 diabetes)))} \cdots\cdots 5$$

$$P(CVD_{hypertension\&BMI}) = {}^{1}/_{(1+EXP(-(\beta_0+\beta_1 hypertension+\beta_3 BMI)))} \cdots\cdots 6$$

$$P(CVD_{diabetes\&BMI}) = {}^{1}/_{(1+EXP(-(\beta_0+\beta_2 diabetes+\beta_3 BMI)))} \cdots\cdots 7$$

$$P(CVD_{hypertension\&diabetes\&BMI}) = {}^{1}/_{(1+EXP(-(\beta_0+\beta_1 hypertension+\beta_2 diabetes+\beta_3 BMI)))} \cdots\cdots 8$$

Where β is the coefficient; β_0 is the intercept; β_1 is the coefficient for hypertension; β_2 is the coefficient for diabetes; and β_3 is the coefficient for BMI. The coefficients ($\beta_1, \beta_2, \beta_3$)were derived from the Prevent Risk Engine as cited (), while the coefficient β_0 was derived through calibration. The distribution of CVD patients by risk groups was generated from electronic medical records of 600,000 patient visits at University Hospital in Cleveland (see Table 1). This table allows us to distribute the CVD events data into CVD events by risk groups to calibrate the model to obtain β_0 for all risk groups.

Evidence from many studies (citation) suggests that CVD events can be significantly reduced if patients with hypertension, diabetes, and high BMI are treated to target. To account for the effect of hypertension and diabetes treatment, as well as weight loss on CVD events, the probability of CVD events is adjusted by the proportion of the population in each risk group receiving treatment

and the effect of treatment on CVD event probability (i.e., the relative probability of CVD event due to treatment). An example of an adjusted probability for individuals with diabetes only is:

$$AP(CVD_{diabetes}) = \left(P(CVD_{diabetes}) * \left(1 - \left({}^{fd_{tot}}/_{fd_0} \right) \right) \right) + \left(P(CVD_{diabetes}) * \left({}^{fd_{tot}}/_{fd_0} \right) * (1 - RR_d) \right) \quad \text{..... ..... 9}$$

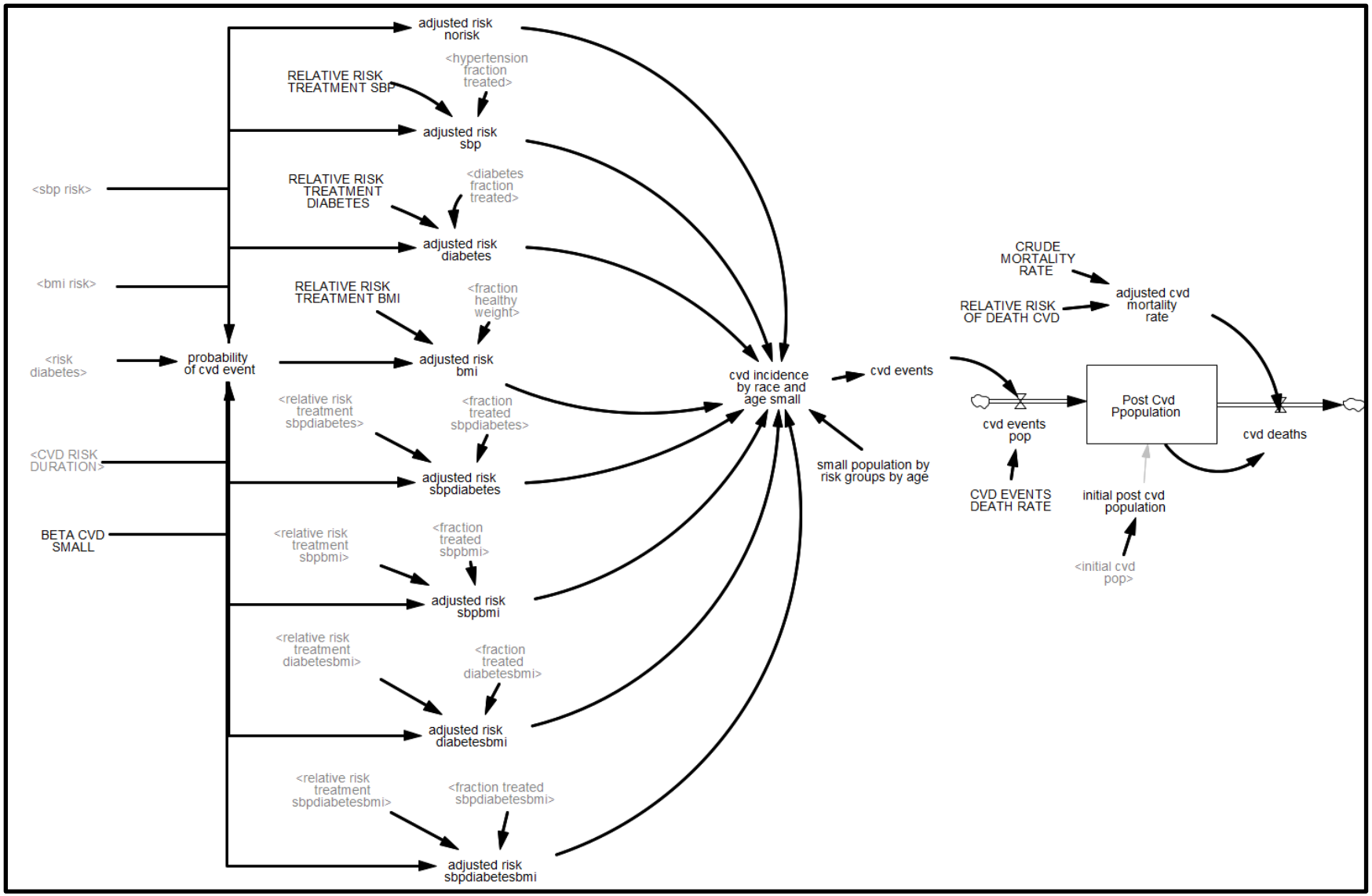


Figure xx: Model Parameters

Parameter	Base-case value	Uncertainty range	Distribution	Source	Notes
Fertility rate			Uniform	Census Bureau	
Age-specific mortality			Uniform	Census Bureau	
Net migration	0.004		Uniform	Census Bureau	
Prediabetes onset rate	0.092		Uniform	Calibration	
Prediabetes recovery rate	0.005		Uniform	Calibration	
Prediabetes screening rate	0.001		Uniform	Calibration	
Prediabetes treatment uptake rate	0.0001		Uniform	Calibration	
Diabetes onset rate	0.0076		Uniform	Calibration	
Diabetes screening rate	0.001		Uniform	Calibration	
Diabetes treatment uptake rate	0.005		Uniform	Calibration	
Prehypertension onset rate	0.1		Uniform	Calibration	
Prehypertension recovery rate	0.018		Uniform	Calibration	
Prehypertension screening rate	0.021		Uniform	Calibration	
Prehypertension treatment uptake rate	0.0066		Uniform	Calibration	
Hypertension onset rate	0.029		Uniform	Calibration	
Hypertension screening rate	0.025		Uniform	Calibration	
Hypertension treatment uptake rate	0.001		Uniform	Calibration	
SBP reduction	13		Uniform	AchieveGreater	
BMI reduction	3.7		Uniform	AchieveGreater	
Transition to obesity	0.001		Uniform	Calibration	
SBP risk coefficient			Uniform	Prevent Risk Equation	
BMI risk coefficient			Uniform	Prevent Risk Equation	
Diabetes risk coefficient	2.39		Uniform	Prevent Risk Equation	
CVD risk duration	10		Uniform	Prevent Risk Equation	

Model Validation and Sensitivity Analysis

The structure and behavioral validation test [29] was used to validate the model. In the structure test, the model was presented to clinicians with expert knowledge of CVD to verify its structure and assumptions about causal relationships. Therefore, the model is grounded in current knowledge and available evidence on CVD risk factors and interventions to prevent CVD events. The behavior test simulates the prevalence of key diabetes, hypertension, smoking, and CVD variables, compared with available data (see supporting documents for behavior validation graphs). The results suggest that the simulated model's behavior compares favorably with data, indicating that the model performs credibly for the visual fit test.

For the sensitivity analysis, a two-way sensitivity analysis was performed on the base case and the policy experiments to evaluate the likely impact of changes in the most important parameters on CVD outcomes. The most important parameter included in the sensitivity analysis was the onset rate for diabetes, hypertension, and obesity. These rates were varied by ±50 %, and the model was run 1000 times. The estimated average and the minimum and maximum values at 95% confidence level for each run were used to show the credible interval.

Intervention Experiment

We modelled four scenarios to assess the population impact of scaling community health worker (CHW)–led cardiometabolic interventions.

Baseline (status quo): Current trends in screening, diagnosis, treatment, and management of hypertension, diabetes, and obesity were assumed to continue without additional CHW scale-up. Diagnosis rates, treatment uptake, adherence, and weight trajectories remained unchanged. This scenario provided the counterfactual against which interventions were evaluated.

Hypertension-focused intervention: CHW activities were expanded to improve blood pressure screening, care linkage, medication adherence, and home monitoring. The model increased annual hypertension diagnosis, treatment initiation, and sustained adherence, resulting in reductions in mean systolic blood pressure consistent with observed Achieve-Greater program effects. No changes were applied to diabetes or weight trajectories.

Hypertension plus diabetes intervention: This scenario extended hypertension management to include improved detection of diabetes and better glycemic control. Screening, diagnosis, and sustained treatment of prediabetes and diabetes were increased, with HbA1c reductions informed by Achieve-Greater data, modifying both vascular and metabolic risk.

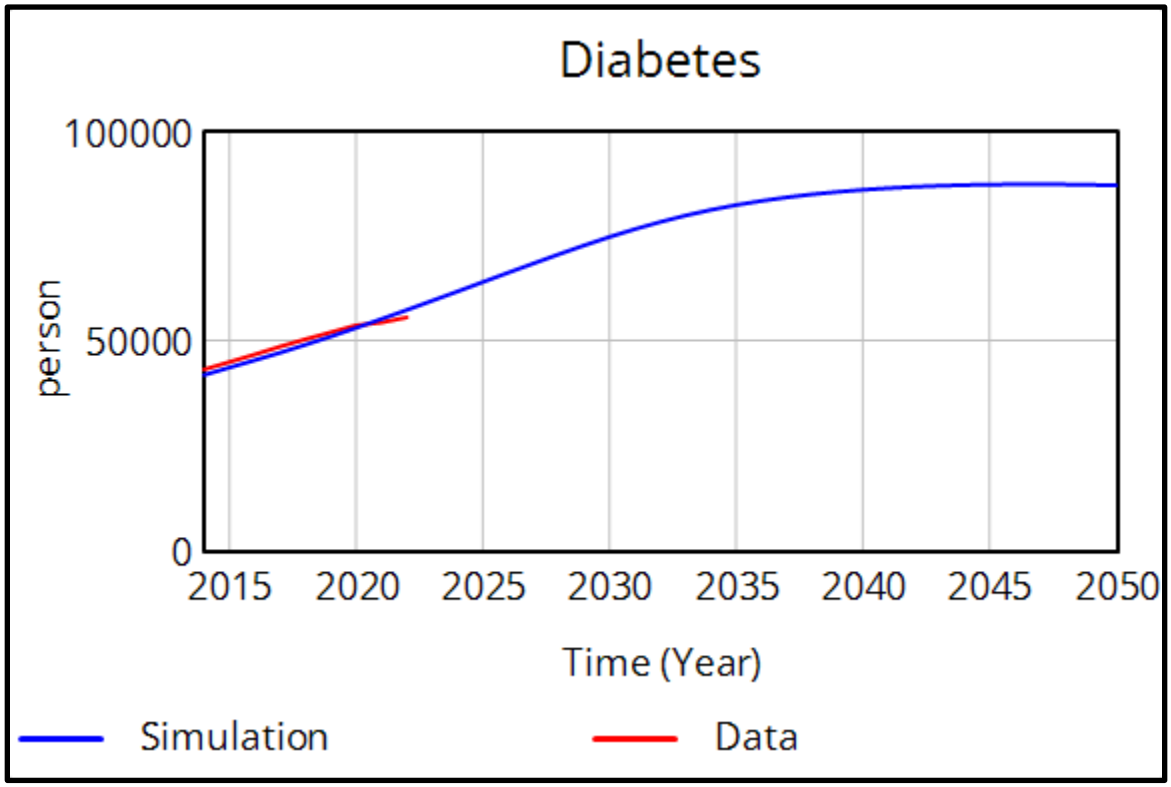
Comprehensive cardiometabolic intervention: The final scenario integrated hypertension and diabetes management with sustained weight support. Reduced progression to obesity and increased regression to healthier weight states lowered the incidence of hypertension and diabetes, enabling assessment of synergistic multi-risk-factor prevention.

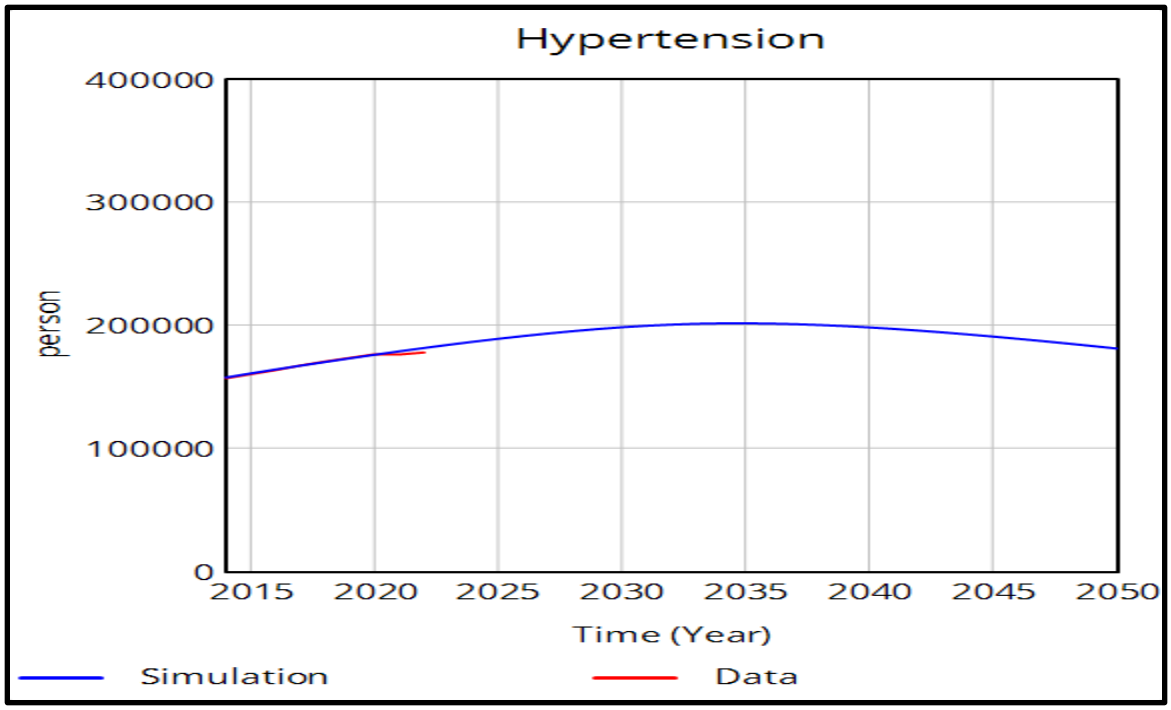
Supplementary Appendix: Model Calibration and Behavioral Validation

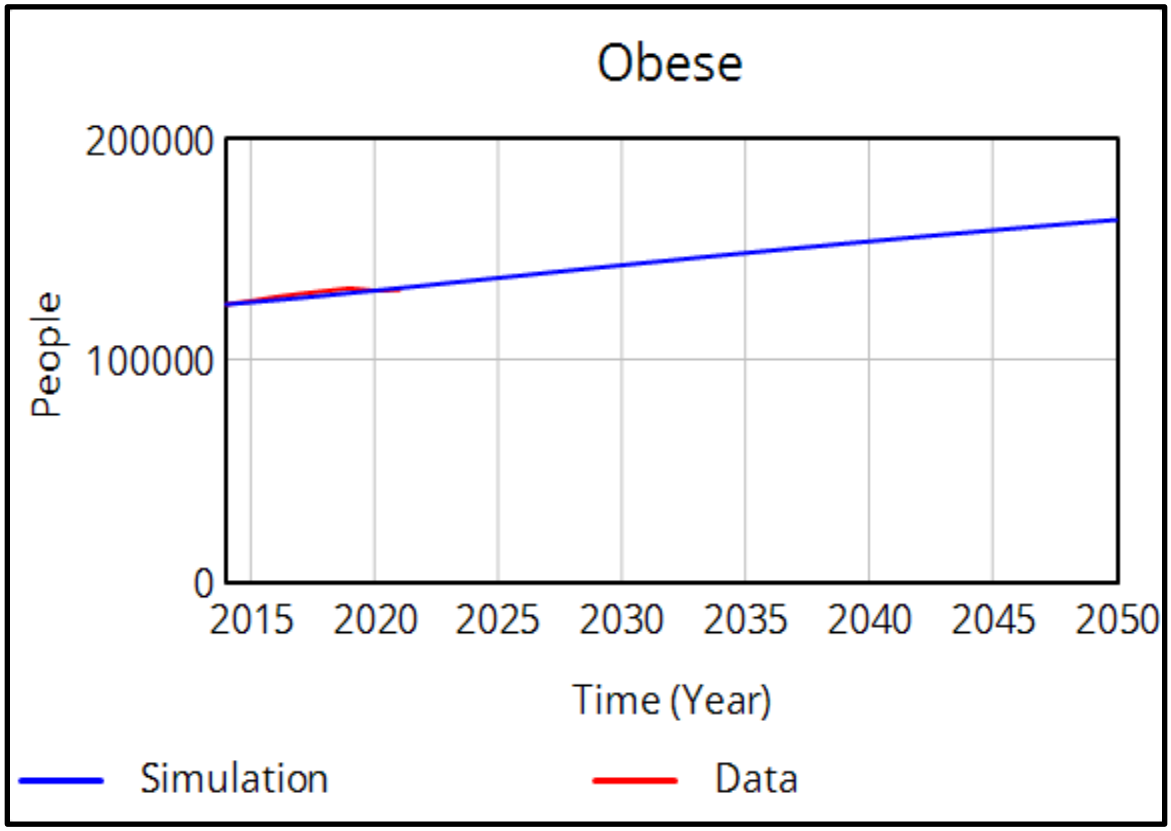
To assess the internal validity and empirical credibility of the Achieve-Greater Simulation Model (AG-SIM), simulated trajectories were compared with observed data for key cardiometabolic and cardiovascular outcomes in Cleveland from 2015 to 2024 (Supplementary Figures S1–S5). Outcomes examined included the prevalence of diabetes, hypertension, and obesity; annual CVD events; and CVD prevalence.

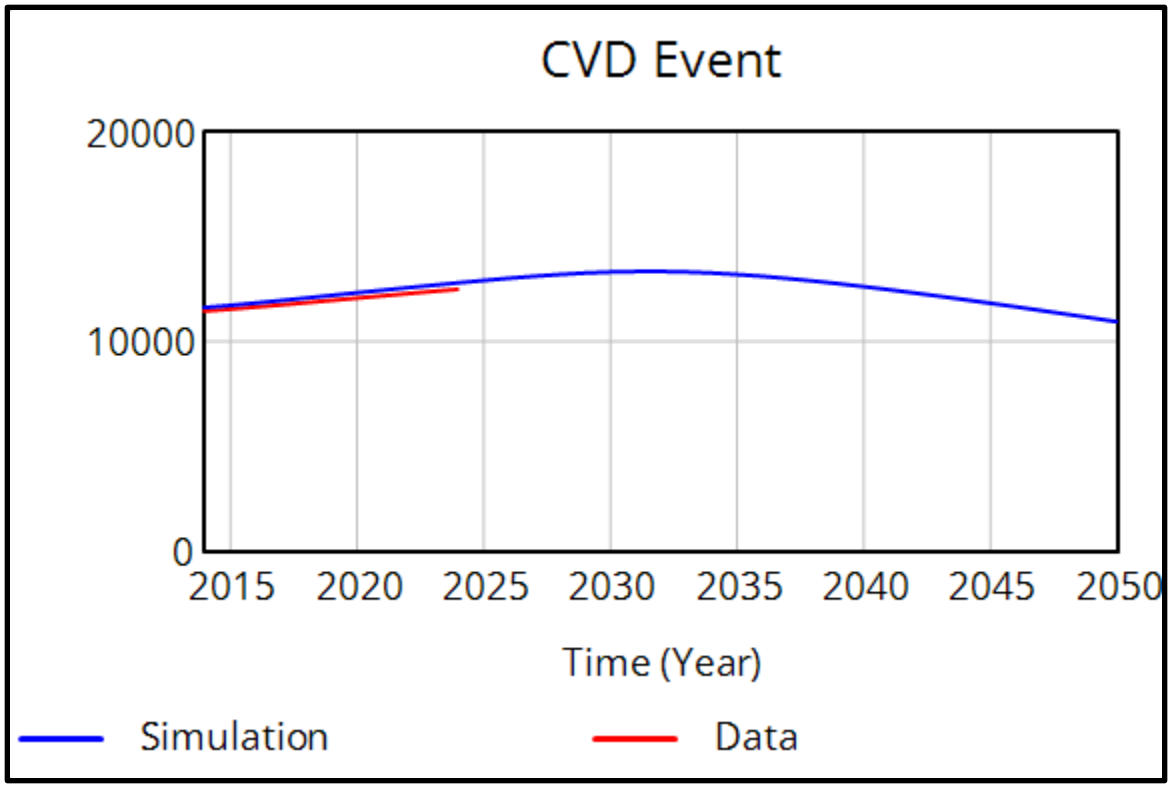
Across all outcomes, simulated trends closely reproduced the direction, magnitude, and curvature of observed epidemiological data. For diabetes, the model captured the steady rise in prevalence through the calibration period, with simulated values overlapping empirical data within plausible bounds. Hypertension prevalence was similarly reproduced, including the gradual increase followed by stabilization observed in recent years. Obesity trends demonstrated strong concordance, with simulated trajectories aligning with observed secular patterns upward.

For cardiovascular outcomes, simulated annual CVD events and prevalent CVD closely tracked surveillance estimates over the calibration period. The model reproduced both the magnitude of annual events and the gradual rise in CVD prevalence associated with improved survival. Minor slope deviations in the early years were within acceptable calibration tolerances and did not materially affect long-term projections. Collectively, these comparisons demonstrate that the model adequately reproduces historical cardiometabolic and cardiovascular trends before the implementation of policy experiments. This behavioral validation supports the model's structural integrity and provides confidence that projected intervention effects emerge from realistic underlying population dynamics rather than artefacts of model specification.




Hypertension
400000
300000
200000
100000
0
person
2015
2020
2025
2030
2035
2040
2045
2050
Time (Year)
Simulation
Data


Obese
200000
100000
0
People
2015
2020
2025
2030
2035
2040
2045
2050
Time (Year)
Simulation
Data



Supplementary Figure X: Uncertainty Analysis of Projected CVD Outcomes

Sensitivity Analysis of Model Projections

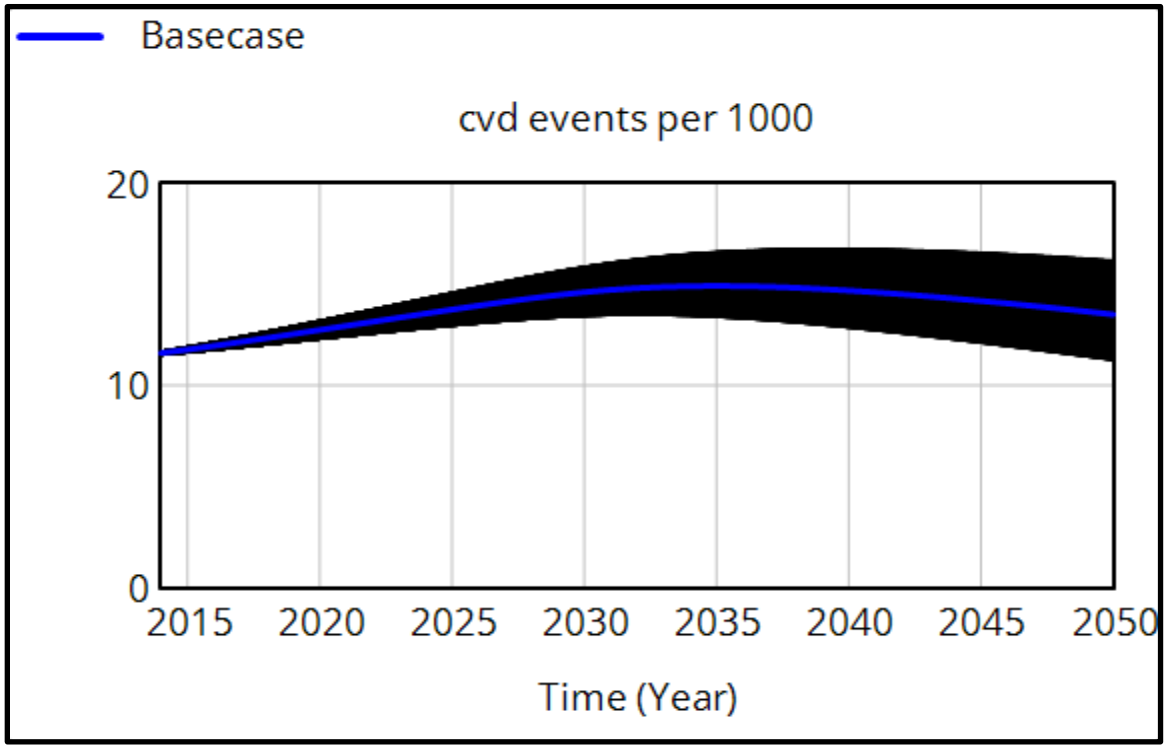
We conducted probabilistic sensitivity analyses to assess the robustness of model projections to uncertainty in key parameters. Parameter values governing cardiometabolic risk transitions, intervention effectiveness, and disease progression were varied simultaneously within ±50% of their nominal values. For each scenario, 1000 simulation runs were performed, and the resulting outcome distributions were summarized as 95% uncertainty interval around the mean, as shown in the table below.

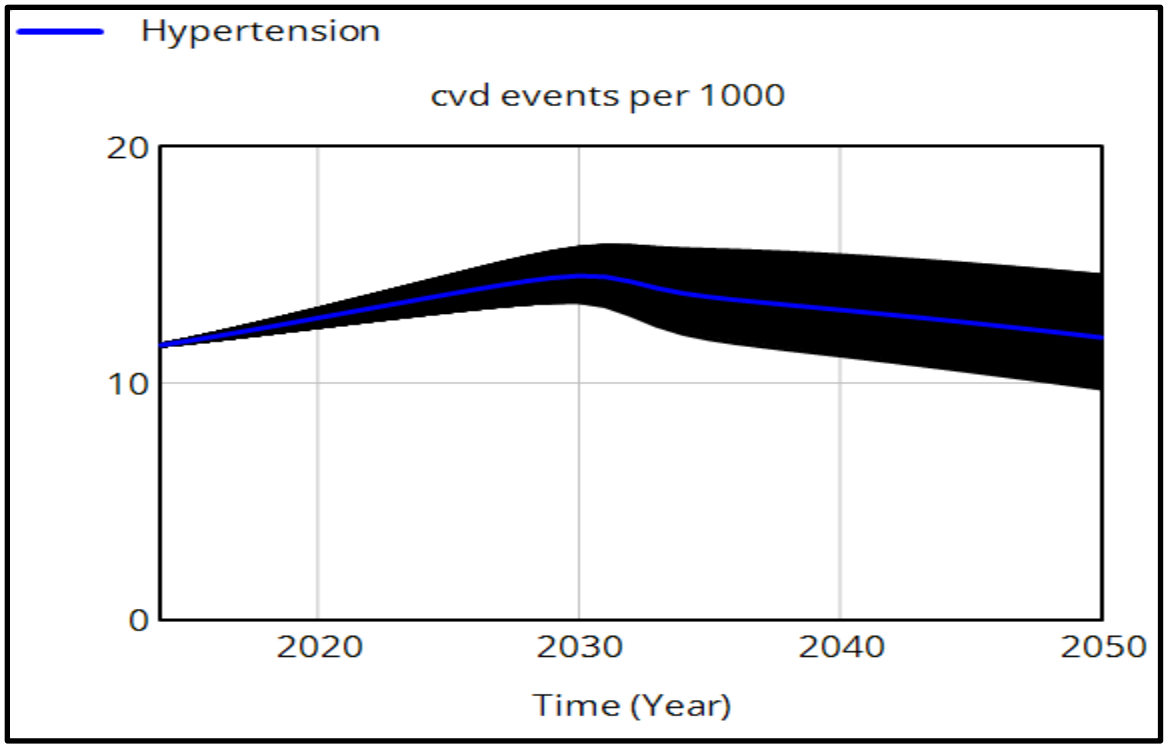
Across all scenarios, the model produced consistent qualitative behavior. Under baseline conditions, projected cardiovascular disease (CVD) incidence increased gradually through approximately 2030–2035, then stabilized and declined slightly toward 2050. CVD mortality followed a similar but more gradual upward trajectory over the simulation horizon. The sensitivity envelopes surrounding the base case trajectory indicate moderate uncertainty but preserve the overall direction and timing of these trends.

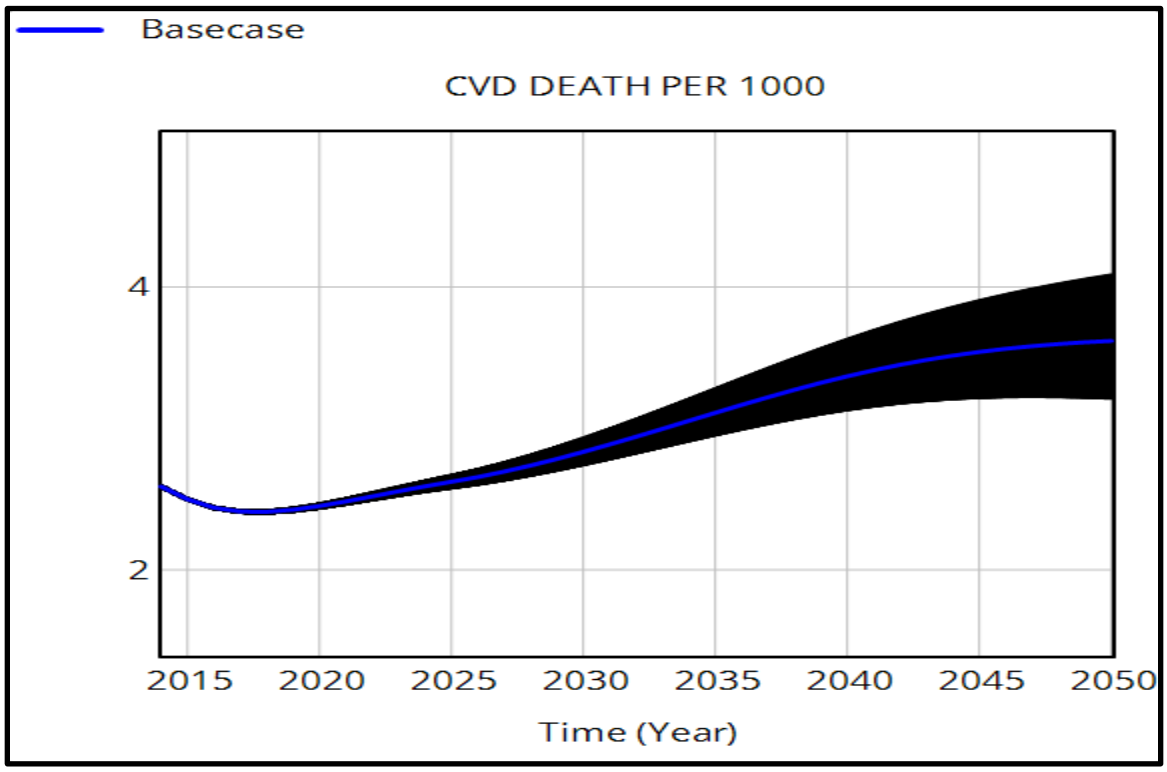
Intervention scenarios demonstrated similar robustness. Hypertension-focused interventions consistently reduce projected CVD events and mortality relative to baseline across the full range of parameter values. Adding diabetes management produced additional reductions in both outcomes, while the comprehensive cardiometabolic intervention (hypertension, diabetes, and obesity management) yielded the largest reductions in projected CVD incidence and mortality.

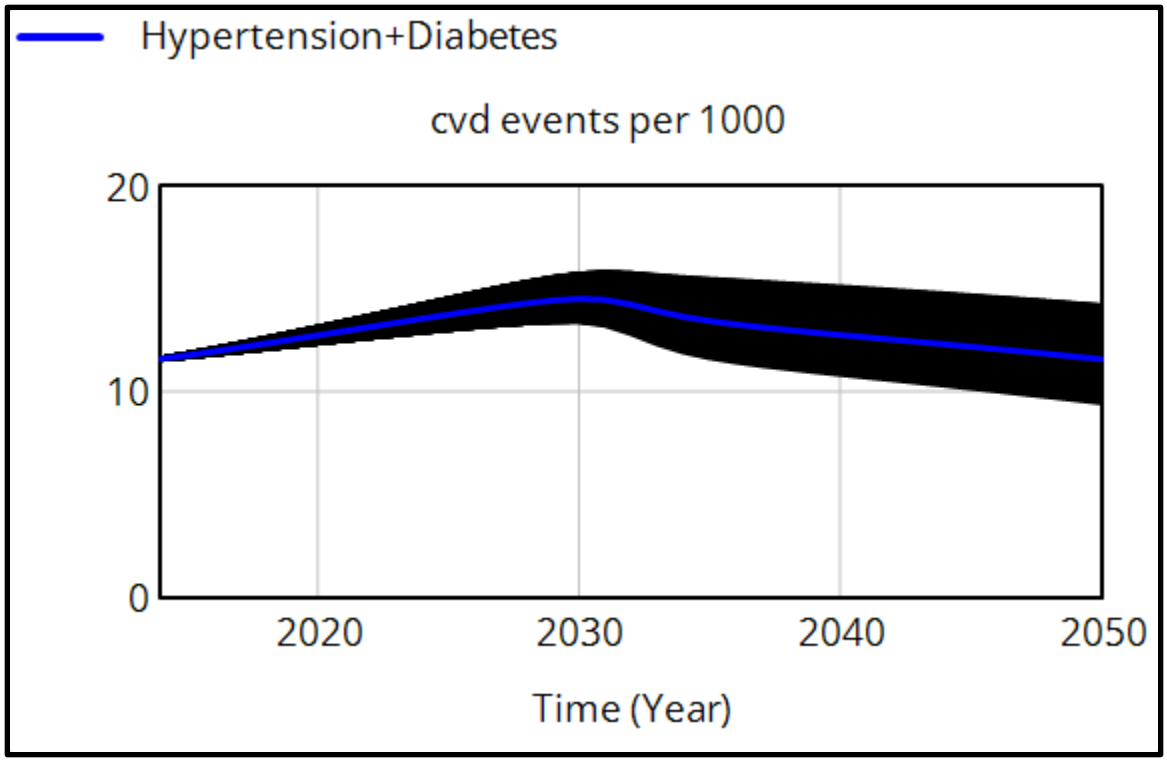
Although the magnitude of effects varied across simulations, the relative ordering of intervention strategies remained stable across the uncertainty ranges.

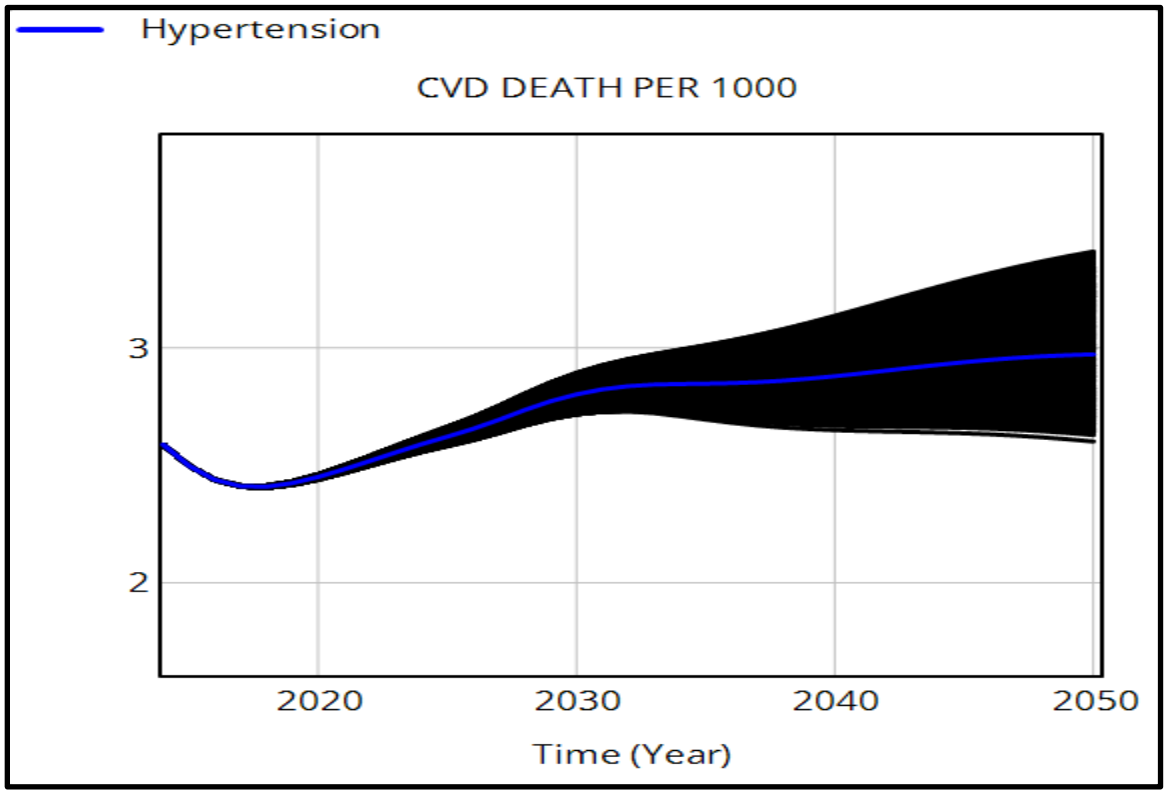
The widening uncertainty intervals over time reflect the cumulative effects of parameter uncertainty and feedback dynamics within the model. Nonetheless, the persistence of the intervention effect across simulations suggests that the projected reductions in CVD burden are not driven by a narrow set of parameter assumptions. Overall, the sensitivity analyses indicate that the model's principal findings—namely the substantial potential impact of scaling community health worker–supported cardiometabolic interventions—are robust to plausible uncertainty in key epidemiological and intervention parameters.

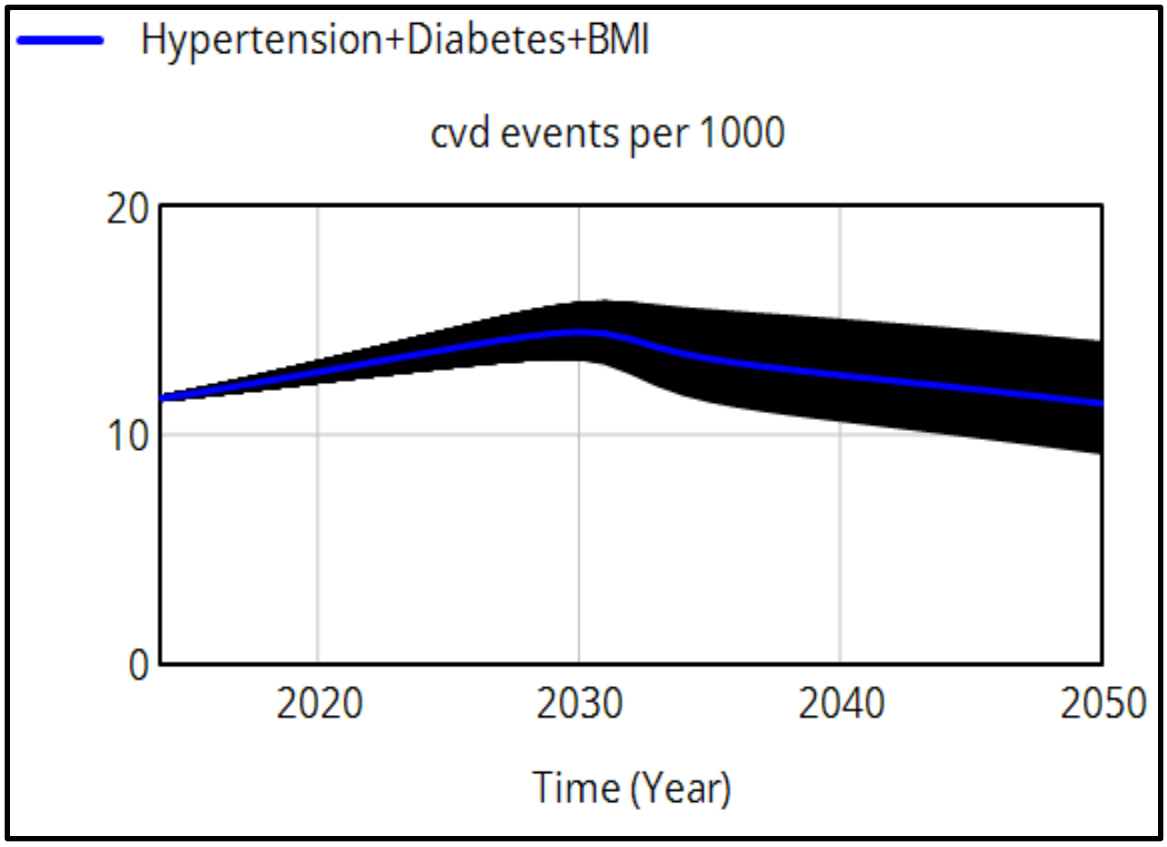


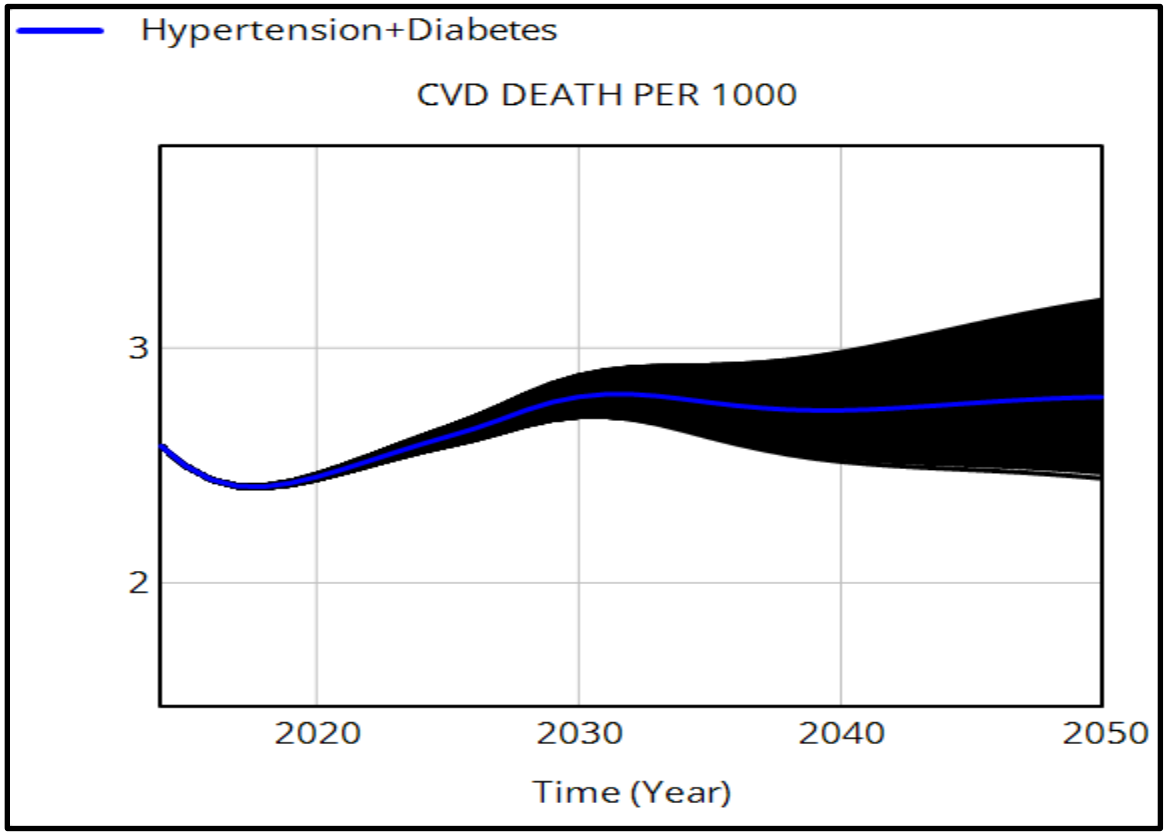

Hypertension
cvd events per 1000
20
10
0
2020
2030
2040
2050
Time (Year)

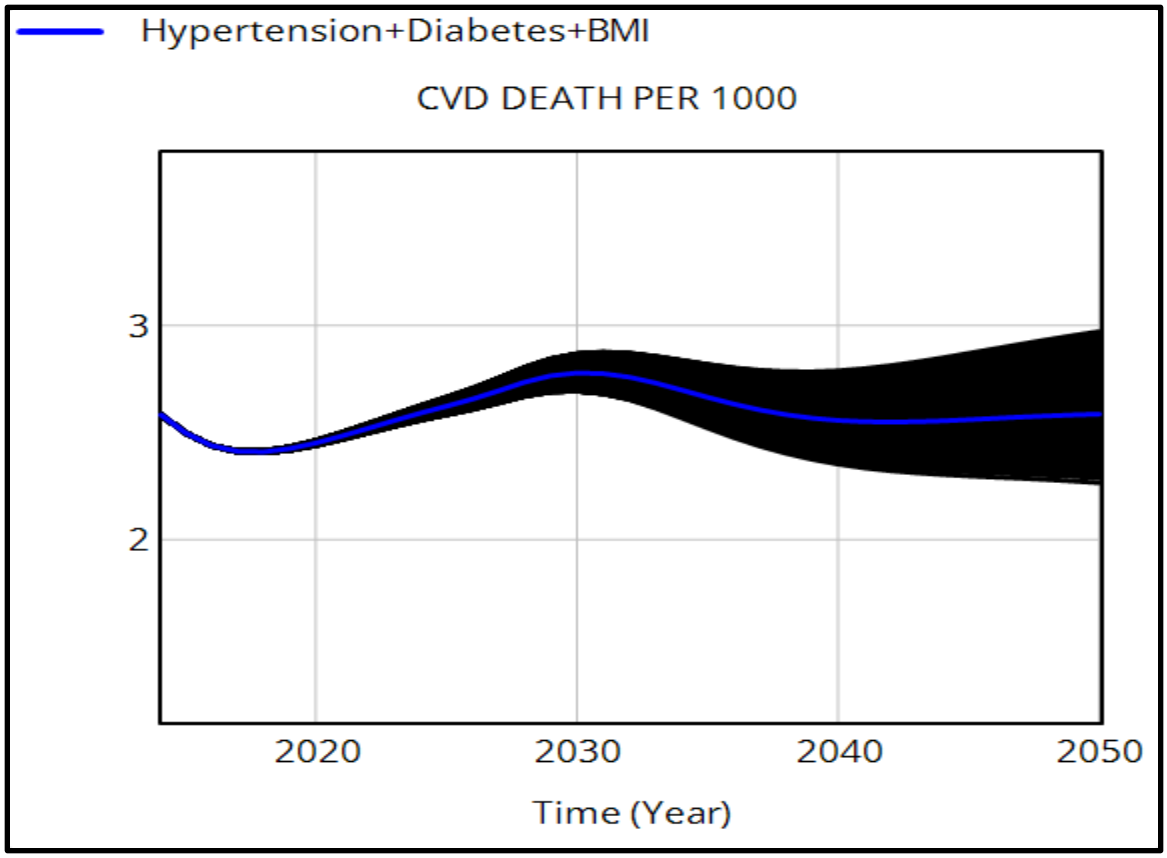

Basecase
CVD DEATH PER 1000
4
2
2015
2020
2025
2030
2035
2040
2045
2050
Time (Year)


Hypertension+Diabetes
cvd events per 1000
20
10
0
2020
2030
2040
2050
Time (Year)


Hypertension
CVD DEATH PER 1000
3
2
2020
2030
2040
2050
Time (Year)


Hypertension+Diabetes+BMI
cvd events per 1000
20
10
0
2020
2030
2040
2050
Time (Year)


Hypertension+Diabetes
CVD DEATH PER 1000
3
2
2020
2030
2040
2050
Time (Year)



Outcome	2020	2035	2050
CVD Events per 1000			
BAU	12.75 (12.41—13.11)	14.92 (13.57—16.45)	13.53 (11.47—15.97)
Hypertension	12.75 (12.41—13.11)	13.66 (12.24—15.29)	11.97 (10.10—14.16)
Hypertension Diabetes	12.75 (12.41—13.11)	13.48 (12.05—15.11)	11.63 (9.80—13.78)
Hypertension Diabetes Obesity	12.75 (12.41—13.11)	13.36 (11.93—14.99)	11.42 (9.61—13.54)
CVD Mortality per 1000			
BAU	2.45 (2.44—2.46)	3.11 (2.97—3.27)	3.62 (3.24—4.06)
Hypertension	2.45 (2.44—2.46)	2.85 (2.72—2.99)	2.98 (2.66—3.34)
Hypertension Diabetes	2.45 (2.44—2.46)	2.77 (2.64—2.91)	2.80 (2.50—3.14)
Hypertension Diabetes Obesity	2.45 (2.44—2.46)	2.66 (2.54—2.80)	2.59 (2.32—2.90)

Table 1: CVD Outcomes under alternative interventions with 95% uncertainty interval.