

A Compartmental Model of Obesity with Treatment Response and Relapse: Endemic Equilibrium Analysis and Long-Term Dynamics

Manoj Kumar Thapa

Department of Mathematics, Georgia Southwestern State University, Americus, GA, USA

Email: manoj.thapa@gsu.edu

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Abstract

In this paper, we examine the long-term dynamics of obesity under conditions of treatment response and relapse using a four-compartment dynamical model motivated by the growing use of GLP-1 receptor agonist therapy. The model is partially calibrated to Georgia Behavioral Risk Factor Surveillance System (BRFSS) data from 2011 to 2024 and reproduces the observed trends closely. We establish the existence of a unique endemic equilibrium, linking model parameters directly to long-run prevalence. Simulations show that increasing the effective treatment-transition rate on its own produces only modest reductions in prevalence. Sensitivity analysis shows that, under the baseline setting, equilibrium obesity prevalence is most strongly influenced by obesity progression and partial recovery. Under the enhanced treatment-response setting, the effective treatment-transition rate becomes the strongest negative driver, while obesity progression remains the strongest positive driver and the influence of relapse increases substantially. Improving access to GLP-1-based treatment may increase the effective treatment-transition rate, but access alone is not sufficient. Lasting reductions in prevalence also require managing relapse, so policy design needs to address adherence and long-term retention, not access alone.

Keywords

Obesity Modeling, Compartmental Systems, Endemic Equilibrium, Treatment Response, Relapse Dynamics, GLP-1 Therapy, Sensitivity Analysis

1. Introduction

Obesity has emerged as one of the most pressing chronic public-health concerns of the twenty-first century. A recent pooled analysis of more than 3,600 population-representative studies estimated that more than one billion children, adolescents, and adults worldwide were living with obesity in 2022, with adult rates more than doubling since 1990 [1]. Under continuation of recent trends, the prevalence of overweight and obesity is projected to increase through 2050 [2]. In the United States, adult obesity prevalence is approximately 40% [3], with substantial variation by state, age, race and ethnicity, and education level [4]. Clinical obesity is associated with major health consequences, including type 2 diabetes, cardiovascular disease, hypertension, and premature mortality [5], while overweight and obesity impose substantial economic costs worldwide [6].

Clinical understanding of obesity has changed substantially in recent years. Current consensus work defines clinical obesity as a chronic systemic illness and emphasizes long-term management [5]. This change has coincided with the introduction of highly effective pharmacological options. In phase 3 trials, semaglutide produced mean weight loss of approximately 15%, whereas tirzepatide produced reductions exceeding 20% [7] [8]. Phase 2 retatrutide results also showed substantial dose-dependent weight reduction [9]. Together, these developments define the current GLP-1 era of obesity pharmacotherapy.

However, maintaining population-level benefits is hindered by post-treatment weight regain. The STEP 4 trial, the STEP 1 extension, and SURMOUNT-4 documented substantial weight regain after treatment withdrawal or transition to placebo [10]-[12]. A 2026 systematic review and meta-analysis likewise found rapid weight regain and reversal of cardiometabolic benefits after stopping weight-management medication [13]. Real-world data also show incomplete one-year persistence and adherence to GLP-1 therapy for obesity [14]. The observed regain is consistent with persistent biological drivers after treatment withdrawal. Even highly effective treatment generally produces substantial but incomplete weight reduction [7] [8].

In light of these clinical findings, a natural mathematical question arises: How do treatment response and relapse interact to determine the long-run endemic burden, and how does that burden change as the effective treatment-transition rate increases? Previous studies have modeled obesity using system-dynamics and compartmental approaches and have examined population trends, intervention sensitivity, recovery, recurrence, and equilibrium behavior [15]-[18]. These studies provide an important foundation, but they do not explicitly represent the treatment-response pathway considered here, in which individuals leave the obese class after sufficient pharmacological response and subsequently either stabilize in the overweight class or relapse to obesity. In a chronic-condition context like obesity, the issue is not whether an obesity-free state can be reached, but whether interventions can alter the ongoing equilibrium point.

The present paper extends this literature. A four-compartment linear compartmental model is developed with explicit treatment response and relapse and partially calibrated to the Georgia Behavioral Risk Factor Surveillance System (BRFSS) data using a high-burden state-level case, and the unique endemic equilibrium is analytically obtained. Distinctive in this modeling is the treatment-induced recovery being channeled towards the overweight class, rather than towards the healthy class. This choice is motivated by trial evidence of substantial but often incomplete weight reduction [7] [8]. We then quantify how changes in the effective treatment-transition rate and relapse alter the relative influence of behavioral and treatment-response parameters on equilibrium obesity prevalence. Section 2 presents the model formulation; Section 3 develops the equilibrium and stability analysis; Section 4 describes parameter estimation; Section 5 reports the numerical results; Section 6 discusses the findings; and Section 7 concludes the paper.

2. Model Formulation

2.1. Population Structure and Assumptions

The normalized adult population is represented by four disjoint state variables, $H(t)$, $W(t)$, $O(t)$, and $T(t)$, corresponding to healthy weight, overweight, obesity, and a treatment-response state, respectively. The total population is normalized so that

$$H(t) + W(t) + O(t) + T(t) = 1, \quad t \geq 0. \quad (2.1)$$

The analysis concerns medium-term movement among weight states in a demographically closed adult population, and births, deaths, and migration are therefore omitted. The constant-population assumption is interpreted at the level of normalized population proportions rather than as a fixed cohort followed over several decades. Extended simulation horizons illustrate convergence toward equilibrium under constant transition rates; they are not demographic projections. The compartments are interpreted according to weight status and treatment response. Clinical frameworks recognize that BMI alone does not capture organ dysfunction and comorbidity [5]. For calibration, $O(t)$ represents the proportion of adults currently classified as obese in the BRFSS series, which defines obesity as BMI at least 30 kg/m² based on self-reported height and weight [4]. In particular, the treatment-response compartment $T(t)$ does not include every individual receiving anti-obesity medication. It represents individuals for whom treatment has produced sufficient weight reduction to move them out of the obese class, but who remain in active treatment or in the immediate post-treatment transition period before stabilization in the overweight class. This definition keeps $O(t)$ consistent with the observed BRFSS obesity-prevalence measure and avoids repeated reassignment across weight categories during treatment cycles that may produce approximately 15% to 21% body-weight loss [7] [8].

Individuals progress from healthy to overweight at rate α , reflecting gradual weight gain from lifestyle and environmental factors, and return from overweight to healthy at rate ρ , capturing sustained behavioral improvement. Progression from overweight to obesity occurs at rate β , while partial spontaneous recovery from obesity to overweight occurs at rate σ independent of formal treatment. Obese individuals enter the treatment-response state at the effective rate τ . This parameter combines treatment initiation, adherence, and sufficient early response to move an individual out of the obese class; therefore, it should not be interpreted as a direct prescription-uptake rate. Individuals leave the treatment-response compartment through one of two paths. Some move to the overweight class at rate γ , representing clinically meaningful but often incomplete weight reduction [7] [8]. Others return to the obese class at rate η , representing weight regain following discontinuation or loss of treatment benefit [10]-[13]. All transition rates are strictly positive constants.

2.2. Governing System of Equations

Under these assumptions, the population dynamics are governed by:

$$\begin{aligned}\frac{dH}{dt} &= -\alpha H + \rho W \\ \frac{dW}{dt} &= \alpha H - (\beta + \rho)W + \sigma O + \gamma T \\ \frac{dO}{dt} &= \beta W - (\tau + \sigma)O + \eta T \\ \frac{dT}{dt} &= \tau O - (\eta + \gamma)T\end{aligned}\tag{2.2}$$

This linear compartmental model conserves total population. The healthy compartment H receives inflow only from W via ρ . The overweight compartment W acts as the central hub. It receives inflow from H , from partial spontaneous recovery out of O via σ , and from individuals leaving the treatment-response state via γ . Movement from the treatment-response state to W rather than directly to H represents post-treatment stabilization following substantial but incomplete weight reduction, consistent with trial-reported mean losses [7] [8].

The obese compartment O is fed by progression from W at rate β and by relapse from T at rate η , and is depleted by the effective treatment transition τ and spontaneous partial recovery σ . The treatment-response compartment T is entered from obesity at the effective rate τ and is depleted by post-treatment stabilization γ and relapse η . Adding the four equations gives

$$\frac{d}{dt}(H + W + O + T) = 0,$$

which confirms that total population is conserved. **Figure 1** shows the compartmental flows.

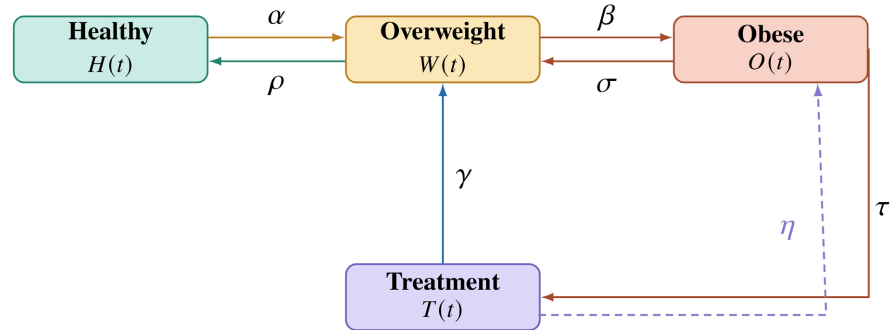


Figure 1. Compartmental flow diagram of the obesity model. The compartments $H(t)$, $W(t)$, $O(t)$, and $T(t)$ represent healthy-weight, overweight, obese, and treatment-response states, respectively. Arrows indicate progression, partial recovery, effective transition into treatment response, post-treatment stabilization, and relapse at the corresponding rates.

2.3. Parameter Summary

Table 1 summarizes all model parameters. Parameters β and σ are estimated in this study from Georgia BRFSS data [19] [20], while α , ρ , τ , γ , and η are fixed as baseline modeling assumptions. The cited studies motivate the corresponding transition pathways but do not provide direct estimates of the constant annual rates used here.

Table 1. Model parameters and values.

Param.	Description	Transition	Units	Type	Baseline	Range	Source/Basis
α	Lifestyle progression	$H \rightarrow W$	yr^{-1}	Fixed assumption	0.03	—	Pathway informed by [15]
β	Obesity progression	$W \rightarrow O$	yr^{-1}	Estimated	0.0607	0.01 - 0.15	Estimated in this study using Georgia BRFSS data [19] [20]
ρ	$W \rightarrow H$ transition rate	$W \rightarrow H$	yr^{-1}	Fixed assumption	0.02	—	Pathway informed by [15]
σ	Partial recovery	$O \rightarrow W$	yr^{-1}	Estimated	0.0355	0.00 - 0.06	Estimated in this study using Georgia BRFSS data [19] [20]
τ	Effective treatment transition	$O \rightarrow T$	yr^{-1}	Fixed assumption	0.03	—	Clinical treatment-response pathway motivated by [7] [8]
γ	Post-treatment stabilization	$T \rightarrow W$	yr^{-1}	Fixed assumption	0.05	—	Treatment-response pathway motivated by [7] [8]
η	Relapse to obesity	$T \rightarrow O$	yr^{-1}	Fixed assumption	0.40	—	Relapse pathway motivated by [10]-[13]

2.4. Long-Run Endemic Equilibrium

Let $E^* = (H^*, W^*, O^*, T^*)$ denote the positive steady state of system (2.2). Because obesity prevalence has remained persistently high in Georgia and nationally, the analysis focuses on how the transition parameters alter the long-run obese

proportion O^* , rather than on the existence of an obesity-free state. Section 3 derives this equilibrium and examines its stability properties.

3. Equilibrium and Global Stability Analysis

We now examine the steady states and long-term behavior of system (2.2). The analysis begins with the feasible region, proceeds to the explicit endemic equilibrium, and concludes with its global asymptotic stability.

3.1. Positivity and Invariant Region

Define the feasible simplex

$$\Omega = \{(H, W, O, T) \in \mathbb{R}_+^4 : H + W + O + T = 1\}.$$

When a compartment is zero, its differential equation contains only nonnegative inflow terms. Thus, a solution beginning in $\mathbb{R}_+^4$ cannot cross into the negative orthant.

Summing the equations yields

$$\frac{d}{dt}(H + W + O + T) = 0.$$

The normalization condition is therefore preserved along every solution. Hence, Ω is positively invariant, and trajectories beginning in Ω remain biologically admissible for all $t \geq 0$.

3.2. Existence and Uniqueness of the Endemic Equilibrium

Theorem 1: Suppose that all transition parameters are strictly positive. Then system (2.2) possesses exactly one equilibrium

$$E^* = (H^*, W^*, O^*, T^*) \in \Omega,$$

and each component of E^* is positive.

Proof. At equilibrium, the four derivatives in system (2.2) vanish. The treatment equation gives

$$T^* = \frac{\tau}{\eta + \gamma} O^* \quad (3.1)$$

From the obesity equation:

$$O^* = \frac{\beta(\eta + \gamma)}{D} W^*, \quad D = \sigma(\eta + \gamma) + \gamma\tau \quad (3.2)$$

From the healthy equation:

$$H^* = \frac{\rho}{\alpha} W^* \quad (3.3)$$

Substituting into the normalization condition gives

$$\Delta = (\alpha + \rho)D + \alpha\beta(\eta + \gamma + \tau).$$

and the closed-form equilibrium:

$$\begin{aligned} H^* &= \frac{\rho D}{\Delta}, & W^* &= \frac{\alpha D}{\Delta}, \\ O^* &= \frac{\alpha\beta(\eta + \gamma)}{\Delta}, & T^* &= \frac{\alpha\beta\tau}{\Delta}. \end{aligned} \quad (3.4)$$

Since all parameters are positive, $D > 0$ and $\Delta > 0$, ensuring positivity. The relations (3.1) - (3.3) express every component in terms of W^* , and the normalization condition fixes W^* uniquely. Therefore, no second equilibrium exists in Ω .

Formula (3.4) also displays the effect of the treatment-response loop on the steady state. Increasing the effective treatment-transition rate τ lowers O^* , whereas increasing the relapse rate η raises the equilibrium obese proportion.

3.3. Global Asymptotic Stability

Let $x(t) = (H, W, O, T)^\top$. System (2.2) may be written as

$$\dot{x} = Ax,$$

where

$$A = \begin{pmatrix} -\alpha & \rho & 0 & 0 \\ \alpha & -(\rho + \beta) & \sigma & \gamma \\ 0 & \beta & -(\sigma + \tau) & \eta \\ 0 & 0 & \tau & -(\eta + \gamma) \end{pmatrix}.$$

Theorem 2: The endemic equilibrium E^* is globally asymptotically stable in Ω .

Proof. The matrix A is Metzler and irreducible, and $\mathbf{1}^\top A = 0$. Hence, e^{At} is positive and column-stochastic for every $t > 0$. By Perron-Frobenius theory for irreducible compartmental systems [21], the eigenvalue 1 of e^{At} is simple, and all remaining eigenvalues have modulus strictly less than one.

The corresponding normalized positive right eigenvector is E^* , since $AE^* = 0$ and $\mathbf{1}^\top E^* = 1$. Consequently,

$$e^{At} \rightarrow E^* \mathbf{1}^\top \text{ as } t \rightarrow \infty.$$

For any $x(0) \in \Omega$,

$$x(t) = e^{At} x(0) \rightarrow E^* \mathbf{1}^\top x(0) = E^*.$$

Therefore, E^* is globally asymptotically stable in Ω . In particular, it is locally asymptotically stable relative to Ω .

3.4. Interpretation

The results indicate that the system converges to the unique endemic equilibrium, regardless of the initial condition. Because $O^* > 0$, the limiting state retains a positive obese proportion. Policy interventions therefore act by shifting the equilibrium E^* , rather than eliminating obesity. The explicit expressions in (3.4) relate the parameters to long-run obesity prevalence O^* and are used for the sensitivity and scenario analyses in Section 5.

4. Parameter Estimation and Model Calibration

4.1. Data Sources

We use annual state-level adult obesity prevalence data from the Behavioral Risk Factor Surveillance System (BRFSS) for Georgia covering 2011-2024 [19] [20]. To maintain methodological consistency, we restrict attention to post-2011 estimates because BRFSS introduced cellular-telephone sampling and a new raking-based weighting method in 2011, creating a methodological break from earlier estimates [22]. The exact annual prevalence estimates and 95% confidence intervals used for calibration, together with the source-file identifier and extraction details, are provided in **Table S1**.

4.2. Parameter Estimation

Because $O(t)$ is the only directly observed model state, simultaneous estimation of all seven transition rates is not attempted. Under the compartment definitions in Section 2.1, $O(t)$ is the model quantity compared directly with the BRFSS obesity-prevalence series, while $T(t)$ is not directly observed in the available surveillance data. Because BRFSS provides repeated cross-sectional prevalence estimates rather than longitudinal observations of the same individuals [19] [20], the fitted parameters are interpreted as population-level effective transition rates rather than individual-level transition hazards estimated from longitudinal follow-up. We therefore estimate only the key progression and partial-recovery parameters

$$\theta = (\beta, \sigma),$$

while fixing the remaining parameters at the baseline values $\alpha = 0.03$, $\rho = 0.02$, $\tau = 0.03$, $\gamma = 0.05$, and $\eta = 0.40$. These fixed values define the baseline modeling scenario and are not treated as direct empirical estimates of annual transition hazards. The cited clinical and modeling studies motivate the associated pathways, while the influence of the selected values is examined through sensitivity and scenario analyses. The absence of a directly observed treatment-response series in the BRFSS data further supports this restricted estimation approach. Practical identifiability of the fitted pair is assessed using the output-sensitivity matrix and profile likelihood [23].

Parameters are estimated by minimizing the sum of squared errors:

$$\text{SSE}(\theta) = \sum_{i=1}^n \left[O_{\text{model}}(t_i; \theta) - O_{\text{data}}(t_i) \right]^2. \quad (4.1)$$

The calibration interval is defined by $t_i = \text{year}_i - 2011$, $i = 1, \dots, 14$. The initial obesity proportion is set equal to the first observed BRFSS value, $O(0) = 0.28$. Because corresponding overweight and treatment-response observations are unavailable, we set $W(0) = 0.35$, $T(0) = 0.01$, and the normalization condition then gives $H(0) = 0.36$.

The values assigned to $W(0)$ and $T(0)$ are baseline assumptions rather than directly observed Georgia estimates.

Parameters β and σ are estimated by minimizing the unweighted sum of squared errors in (4.1) over $0.01 \leq \beta \leq 0.15$, $0 \leq \sigma \leq 0.06$. System (2.2) is solved using MATLAB's ode45 solver [24], and minimization is performed with the Nelder-Mead algorithm implemented in fminsearch. The numerical solution is evaluated at the annual observation times using shape-preserving piecewise cubic interpolation (pchip).

4.3. Goodness of Fit and Calibrated Parameters

Under the fixed baseline assumptions and the estimated values of β and σ , the partially calibrated model closely matches the observed trajectory of obesity in Georgia:

$SSE = 6.61 \times 10^{-4}$, $RMSE = 0.0069$, $R^2 = 0.923$. The best estimates of the parameters are $\beta = 0.0607 \text{ yr}^{-1}$ and $\sigma = 0.0355 \text{ yr}^{-1}$.

These values minimize the objective function under the imposed baseline assumptions and parameter bounds. The practical-identifiability analysis in Section 4.4 shows that the estimates are strongly correlated, and the individual value of σ is weakly constrained by the obesity-prevalence series. They should therefore be interpreted as conditional best-fit values rather than precise independent estimates of the two transition rates.

The endemic equilibrium under the parameter set is $O^* = 0.4687$ (46.87%), indicating a substantially higher long-run obesity burden than the current level. Under the fixed baseline rates and initial conditions, the model gives the conditional point projection

$$O(2030) \approx 0.381 \text{ (38.10\%)},$$

reflecting gradual convergence toward the endemic equilibrium over multiple decades. Because uncertainty in the fixed baseline rates is not propagated, this value should not be interpreted as a probabilistic forecast. The model explains 92.3% of the variation.

Refitting over alternative assumed values of $W(0)$ and $T(0)$ gave conditional 2030 projections of 37.66% - 38.63%, compared with 38.10% at baseline (Table S2). The estimated transition rates and equilibrium prevalence were more sensitive to these assumptions, with O^* ranging from 43.39% to 56.54%.

A supplementary weighted least-squares fit using variances approximated from the published confidence intervals gave a conditional 2030 projection of 38.05%, compared with 38.10% under unweighted calibration; the fitted trajectories differed by less than 0.02 percentage points over 2011-2024 (Table S3).

4.4. Practical Identifiability and Parameter Uncertainty

Because β and σ are estimated from a single observed state, their practical identifiability was examined before interpreting the fitted values individually. The output-sensitivity matrix evaluated at the least-squares estimate has numerical rank two, indicating that the fitted parameters are locally distinguishable under the fixed baseline rates and initial conditions. However, the corresponding local

covariance approximation gives

$$\text{Corr}(\hat{\beta}, \hat{\sigma}) = 0.9985,$$

showing strong positive correlation between the two estimates.

Profile-likelihood analysis was also used to assess parameter uncertainty [23].

The approximate 95% profile interval for β is

$$0.0252 \leq \beta \leq 0.0874 \text{ yr}^{-1}.$$

In contrast, the profile for σ does not cross the 95% threshold within the prescribed fitting range $0 \leq \sigma \leq 0.06 \text{ yr}^{-1}$, indicating weak practical identifiability. The fitted values should therefore be interpreted as a strongly correlated parameter pair rather than as independently precise empirical transition rates.

5. Results

The parameter modifications used in the numerical experiments serve different illustrative purposes. **Figure 2** uses the combined setting $(2\tau, 0.85\eta)$ to compare baseline and altered treatment-response dynamics over a twenty-year horizon. **Figure 6** uses the enhanced setting $(5\tau, 0.5\eta)$ to examine how the sensitivity structure changes when the treatment-response pathway becomes more influential. **Figure 7** compares the baseline setting with moderate $(1.3\tau, 0.75\eta)$ and stronger $(1.6\tau, 0.50\eta)$ combined scenarios. These parameter settings are illustrative and should not be interpreted as empirically estimated intervention levels.

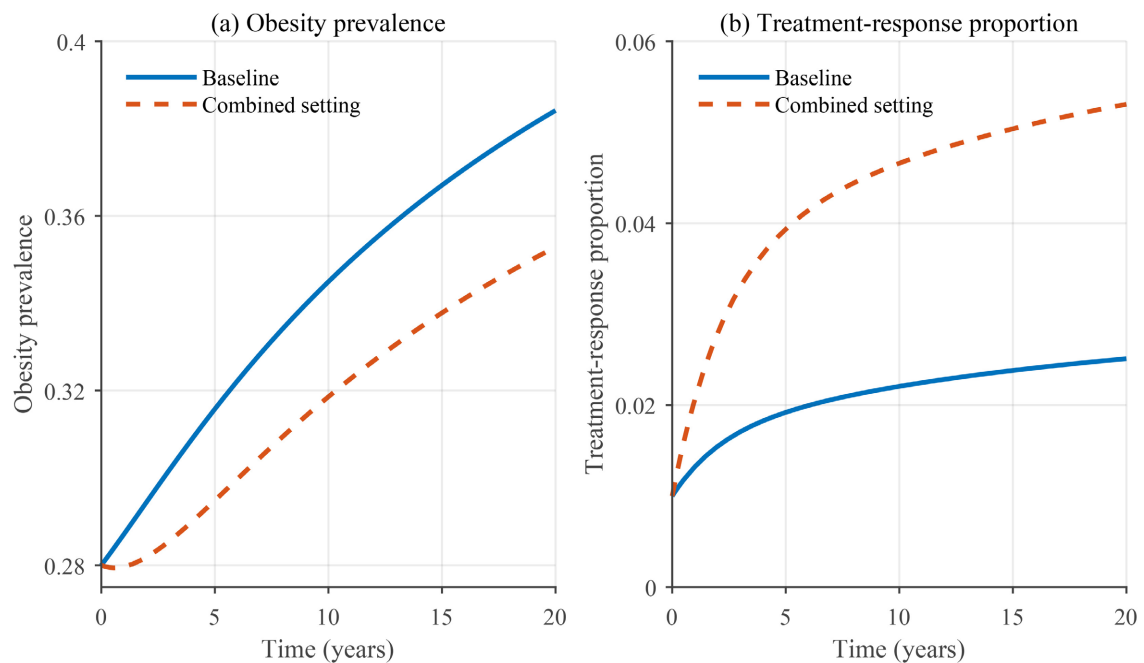


Figure 2. Baseline and combined treatment-response dynamics. Panel (a) compares obesity prevalence under the baseline and intervention settings, while panel (b) compares the corresponding treatment-response proportions. In the combined setting, the effective treatment-transition rate τ is doubled and the relapse rate η is reduced by 15%.

5.1. Baseline Dynamics

Under the conditional best-fit parameter set, the system exhibits gradual and persistent growth in obesity over a twenty-year horizon, as shown in **Figure 2**. This behavior is consistent with the observed Georgia BRFSS trend [19] [20].

The overweight compartment both feeds progression into obesity and receives individuals returning from treatment. Consequently, it does not simply serve as a reservoir of the endemic burden, but as an amplifier of it. The treatment-response compartment remains comparatively small under the baseline effective treatment-transition rate. All trajectories converge toward the endemic equilibrium, consistent with the global stability result established in Section 3.

5.2. Model Fit and Conditional Projection

The partially calibrated model closely matches the observed Georgia BRFSS obesity prevalence over the 2011-2024 calibration interval (**Figure 3**). This in-sample agreement is conditional on the fixed baseline assumptions and does not by itself establish predictive validity outside the calibration period.

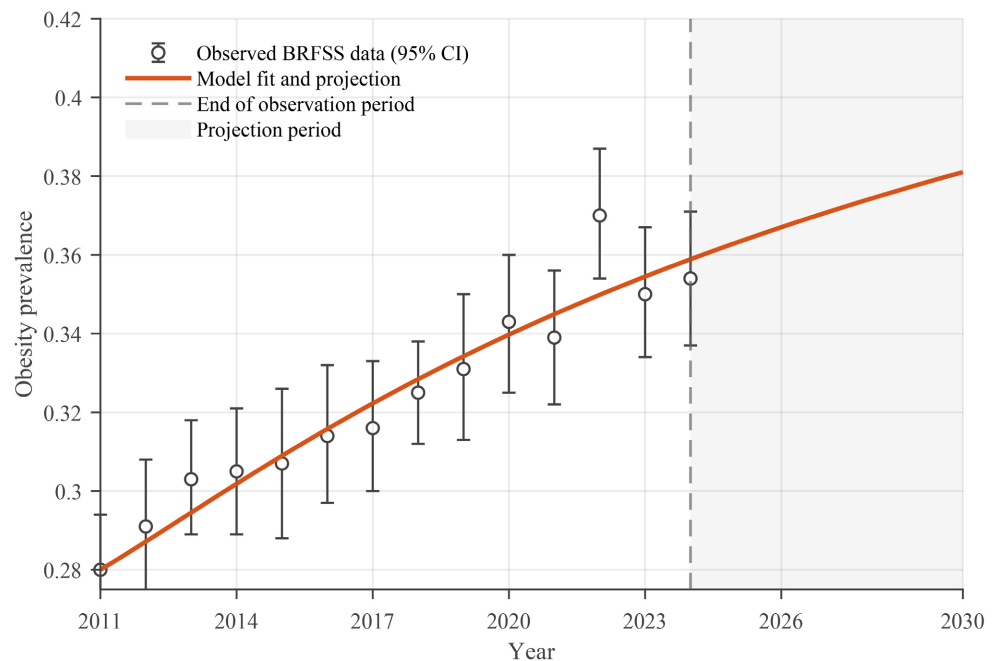


Figure 3. Model fit to Georgia BRFSS obesity data (2011-2024) and conditional projection to 2030. The calibrated trajectory reproduces the observed prevalence pattern over the calibration interval; the shading marks the conditional projection period.

With the baseline parameters fixed, the conditional point projection is 38.1% by 2030. The trajectory remains below the theoretical endemic level $O^* = 46.87\%$, which is approached over several decades, beyond the near-term projection horizon. Uncertainty in the fixed baseline rates is not propagated, so the reported 2030 value remains a conditional model output rather than a predictive forecast.

5.3. Effect of the Treatment-Response Rate

Increasing the effective treatment-transition rate shifts the obesity trajectory downward relative to baseline, although the effect remains modest under current relapse conditions, as shown in **Figure 4**. The deviations from baseline increase gradually over time, consistent with the sensitivity index $\Upsilon_{\tau} = -0.0742$.

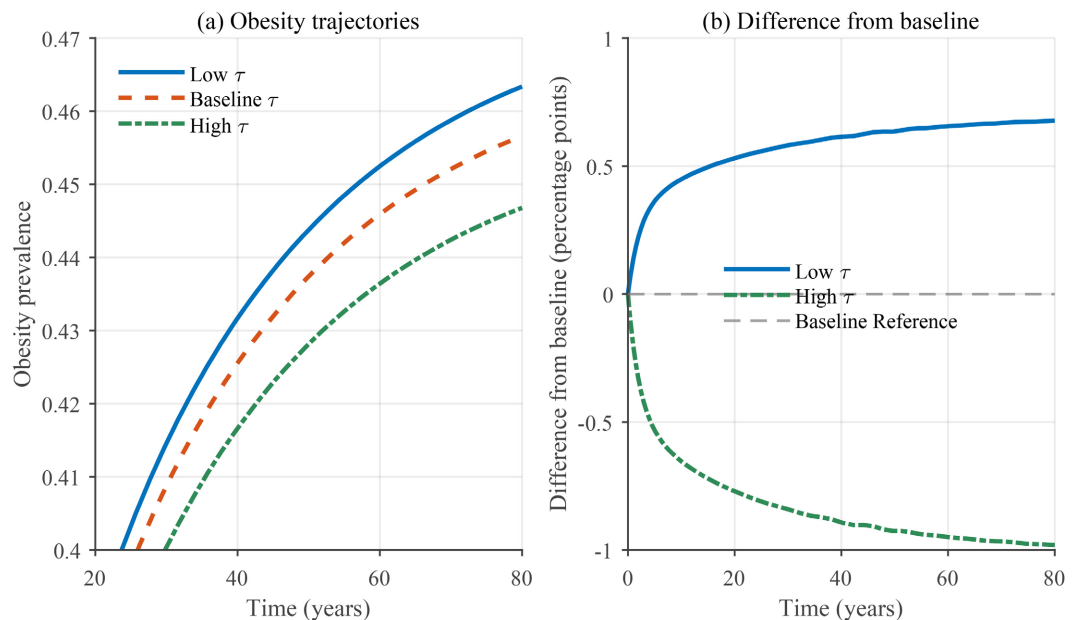


Figure 4. Effect of the effective treatment-transition rate τ on obesity dynamics. Panel (a) shows a zoomed long-term view of obesity prevalence for low, baseline, and high values of τ ; panel (b) gives the corresponding differences from baseline in percentage points. The low, baseline, and high settings are $\tau = 0.024$, 0.030 , and 0.039 yr^{-1} , respectively. All other parameters and initial conditions retain their baseline values.

The normalized sensitivity index for the effective treatment-transition rate is $\Upsilon_{\tau} = -0.0742$ under the baseline parameter setting. A 10% local increase in τ therefore corresponds to an approximate 0.74% decrease in equilibrium obesity prevalence. Direct evaluation with a 10% perturbation also gives a reduction of approximately 0.74%. The $T \rightarrow W$ pathway explains why this effect remains limited. In the model, individuals leaving the treatment-response state enter the overweight rather than the healthy compartment, so renewed progression into obesity remains possible. Clinically, substantial weight regain after treatment withdrawal has been documented [10]-[13].

5.4. Effect of Relapse Rate

The relapse rate η affects long-term outcomes. Over a forty-year time frame, the trajectories for different values of η separate progressively, each tending toward its own endemic level (**Figure 5**).

Higher values of η raise both transient and long-run obesity prevalence, whereas lower values produce sustained reductions. The increasing separation of the trajectories shows that relapse control becomes especially important when the

effective treatment-transition rate is increased. These findings align with clinical evidence that substantial weight regain is common after GLP-1 treatment withdrawal [10]-[13].

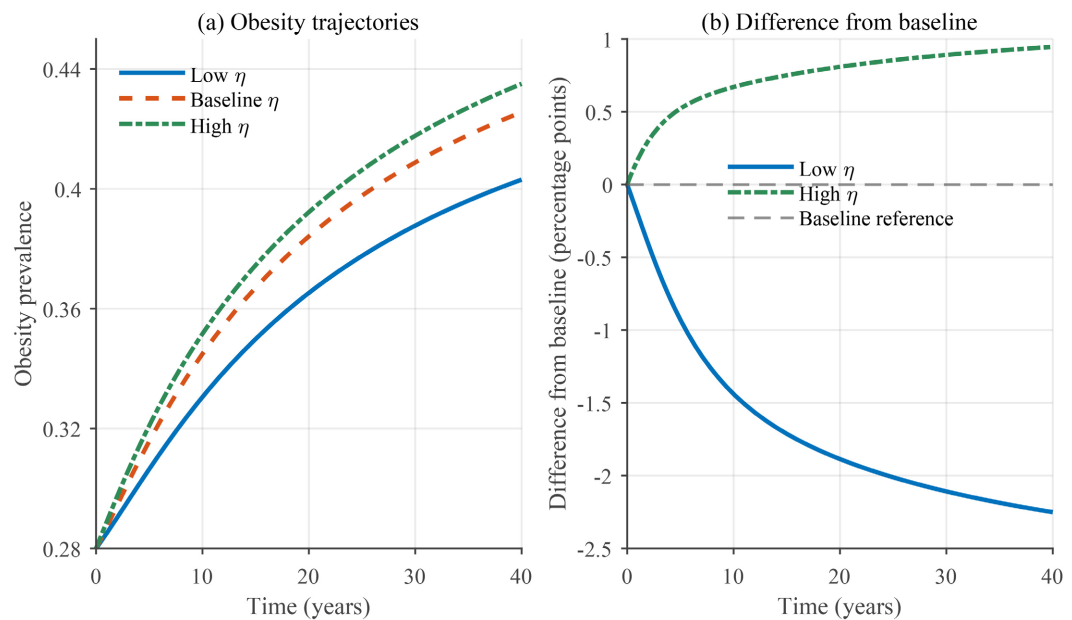


Figure 5. Effect of relapse rate η on long-run obesity dynamics. Panel (a) shows the obesity trajectories; panel (b) shows deviation from baseline in percentage points. Higher relapse shifts the trajectory toward a larger endemic obese proportion, whereas lower relapse produces a lower limiting level. The low, baseline, and high settings are $\eta = 0.20$, 0.40 , and 0.60 yr^{-1} , respectively. All other parameters and initial conditions are held at their baseline values.

5.5. Sensitivity Analysis

The sensitivity structure shows a clear variation in the relative influence of the model parameters on endemic obesity (Figure 6). The normalized sensitivity index of the endemic equilibrium O^* with respect to a parameter p is defined by

$$\Upsilon_p = \frac{\partial O^*}{\partial p} \frac{p}{O^*}.$$

The indices are evaluated at the conditional best-fit parameter set. Because β and σ are strongly correlated in the calibration, their individual sensitivity values describe local model behavior and should not be interpreted as independently estimated population effects.

Under the baseline treatment-response setting, the obesity-progression parameter β has the largest positive influence, while the partial-recovery parameter σ has the largest negative influence, with $\Upsilon_\beta = 0.4999$ and $\Upsilon_\sigma = -0.4570$. The progression and recovery parameters α and ρ have equal and opposite sensitivities, $\Upsilon_\alpha = 0.2000$ and $\Upsilon_\rho = -0.2000$. The treatment-response parameters have comparatively smaller effects under the baseline setting:

$$\Upsilon_\tau = -0.0742, \Upsilon_\eta = 0.0659, \Upsilon_\gamma = -0.0347.$$

Under the enhanced treatment-response setting,

$$\tau_{enh} = 5\tau, \eta_{enh} = 0.5\eta,$$

the relative influence of the treatment-response pathway increases substantially. The effective treatment-transition rate τ becomes the strongest negative influence, with $\Upsilon_{\tau} = -0.4189$, while the sensitivity to relapse increases to $\Upsilon_{\eta} = 0.3351$. Nevertheless, the obesity-progression parameter remains the strongest positive influence, with $\Upsilon_{\beta} = 0.5292$. The remaining sensitivity indices under the enhanced setting are

$$\Upsilon_{\sigma} = -0.2868, \Upsilon_{\alpha} = 0.2117, \Upsilon_{\rho} = -0.2117, \Upsilon_{\gamma} = -0.1586.$$

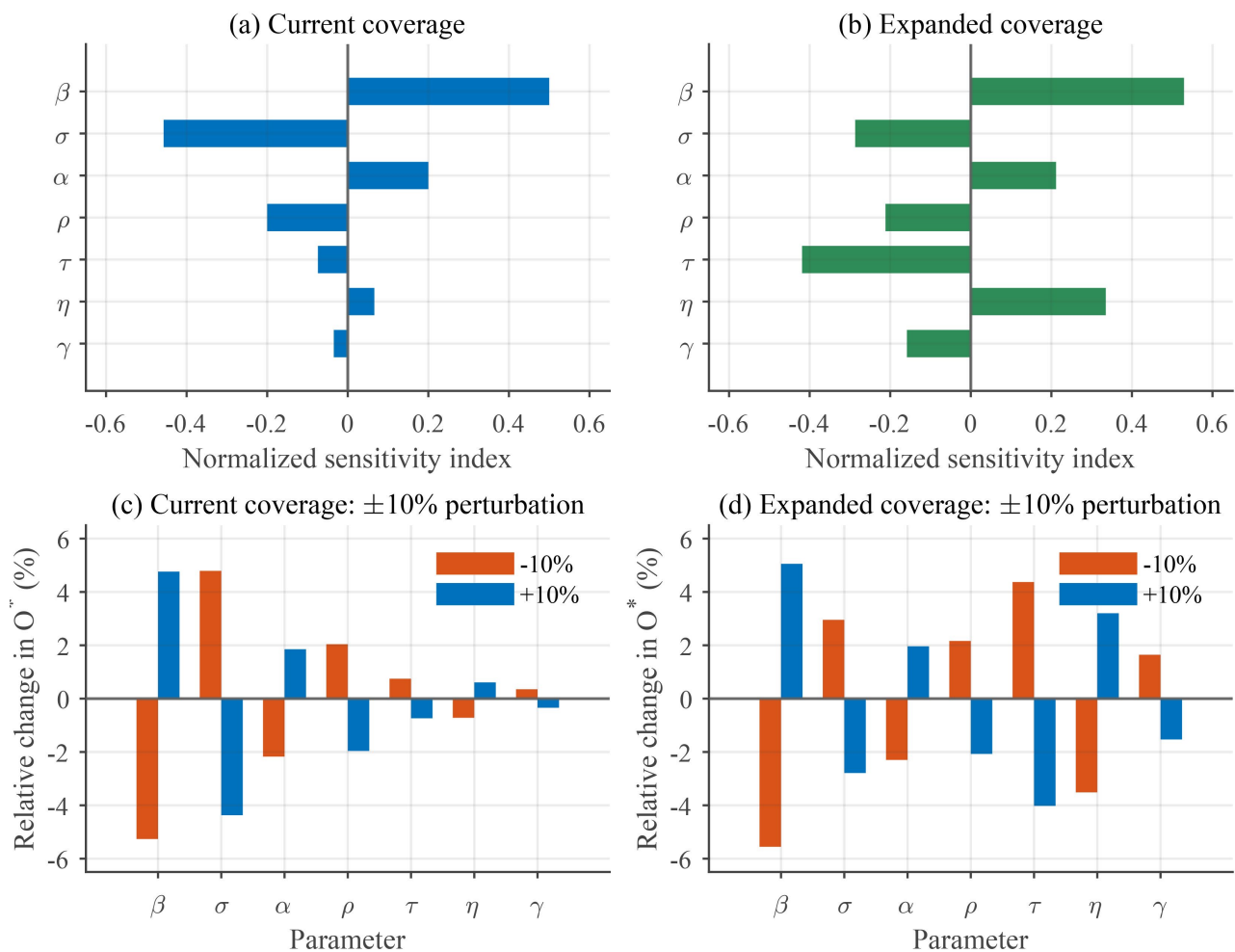


Figure 6. Sensitivity analysis of equilibrium obesity prevalence under the baseline and enhanced treatment-response settings. Panels (a)-(b) show the exact normalized sensitivity indices, while panels (c)-(d) show the relative changes in O^* under $\pm 10\%$ parameter perturbations. Under the enhanced setting, the effective treatment-transition rate becomes the strongest negative driver and the influence of relapse increases substantially, while obesity progression remains the strongest positive driver.

The finite perturbation results are consistent with these local indices. Under the baseline setting, a 10% increase in τ lowers O^* by approximately 0.74%, whereas a 10% increase in η raises O^* by approximately 0.61%. Under the en-

hanced setting, the corresponding changes are approximately -4.02% for τ and $+3.20\%$ for η .

Thus, increasing the effective treatment-transition rate changes the sensitivity structure by increasing the influence of both treatment response and relapse. It does not eliminate the importance of behavioral progression, since β remains the largest positive driver in both settings.

Supplementary refits over the alternative fixed-rate ranges listed in **Table S4** preserved the ordering of the reference, moderate, and stronger combined intervention scenarios in 2030 and at equilibrium, but not all parameter rankings (**Table S5**). The numerical equilibrium level was more sensitive to the fixed-rate assumptions than the conditional 2030 projection.

5.6. Combined Intervention Scenarios

Three intervention settings are considered: the baseline parameter set, a moderate combined intervention $(1.3\tau, 0.75\eta)$, and a stronger combined intervention $(1.6\tau, 0.50\eta)$, as shown in **Figure 7**.

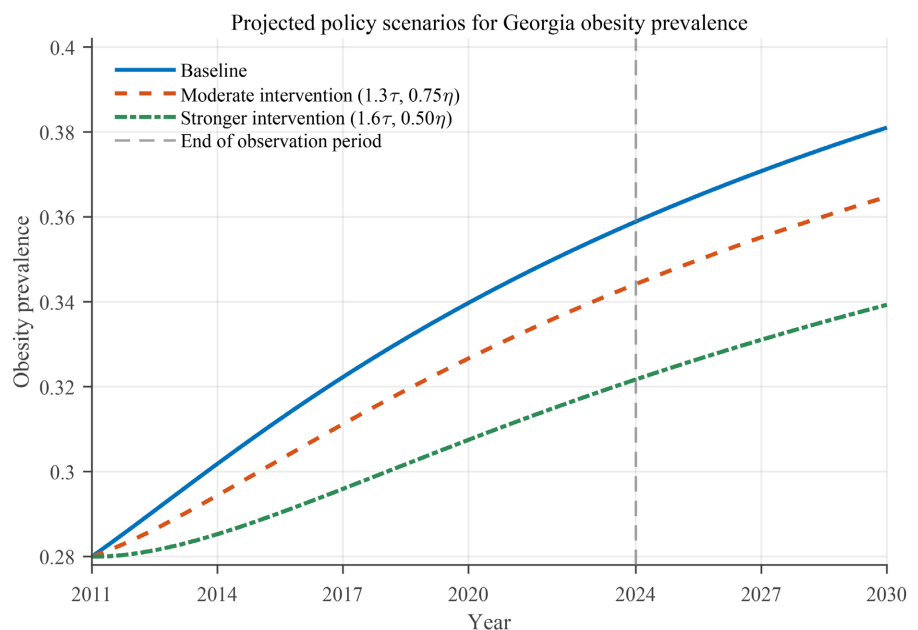


Figure 7. Illustrative scenarios combining the effective treatment-transition rate and relapse. Baseline, moderate, and strong intervention settings are shown. The combined parameter changes produce larger reductions in obesity prevalence than the baseline setting.

The stronger combined setting produces a larger reduction than the moderate combined setting. Even with these improvements, none of the scenarios reverses the upward trend during the projection horizon. Instead, the trajectories remain in transient convergence toward new, lower endemic equilibria.

These results have policy implications. This pattern is broadly consistent with approaches that pair medication access with broader support. The CMS BALANCE model, for example, combines expanded access to GLP-1 treatment with

lifestyle support [25]. Increasing the effective treatment-transition rate without reducing relapse limits the long-run improvement in equilibrium obesity prevalence. Because τ combines treatment initiation, adherence, and sufficient early response, the scenarios do not represent changes in prescription access alone.

6. Discussion

6.1. Treatment Response and Relapse as Joint Determinants

The equilibrium expression shows how the effective treatment-transition rate τ and relapse rate η jointly determine long-run obesity prevalence. From (3.4), the equilibrium obese proportion may be written as

$$O^* = \frac{\alpha\beta(\eta + \gamma)}{(\alpha + \rho)[\sigma(\eta + \gamma) + \gamma\tau] + \alpha\beta(\eta + \gamma + \tau)}.$$

The two treatment-response-loop parameters act in opposite directions: increasing τ reduces O^* , whereas increasing η raises it. Their effects are interdependent: low relapse amplifies the benefit of increasing the effective treatment-transition rate, whereas high relapse substantially limits its long-term impact.

This interaction becomes more pronounced as the effective treatment-transition rate increases. The treatment-response pathway has less influence when τ is small, but as the treatment-response compartment expands, the system becomes more sensitive to both τ and relapse. In that setting, long-run prevalence is influenced by the balance between entry into the treatment-response state and loss of treatment benefit.

The $T \rightarrow W$ pathway explains why successful treatment response does not remove the risk of recurrence. Individuals leaving the treatment-response state enter the overweight compartment, where progression through β remains possible. Repeated movement through the obese, treatment-response, and overweight states can therefore sustain a positive obesity level when relapse is frequent. This interpretation is consistent with evidence from the STEP and SURMOUNT discontinuation trials and the subsequent meta-analysis, which documented substantial weight regain after treatment withdrawal [10]–[13]. Here, effective treatment response and retention should not be viewed in isolation, but as two policy levers.

6.2. Behavioral Progression and Increasing Treatment-Response Influence

Under the baseline treatment-response setting, equilibrium obesity prevalence is influenced most strongly by the obesity-progression parameter β and the partial-recovery parameter σ . Movement from healthy weight to overweight and from overweight to obesity therefore remains important when the treatment-response loop is small. Within the model, prevention strategies that reduce α or β remain relevant because they act directly on the progression pathways.

As the effective treatment-transition rate increases, the treatment-response

loop becomes considerably more influential. Under the enhanced setting, τ becomes the strongest negative driver, with $\Upsilon_{\tau} = -0.4189$, while the relapse sensitivity increases to $\Upsilon_{\eta} = +0.3351$. However, obesity progression remains the strongest positive driver, $\Upsilon_{\beta} = +0.5292$.

The enhanced treatment-response setting therefore changes the relative sensitivity structure without making behavioral progression unimportant. Prevention, effective treatment response, and relapse reduction should be regarded as complementary mechanisms rather than substitutes.

6.3. Policy Implications

Georgia is used as a state-level calibration case because the 2024 adult obesity prevalence in the analyzed BRFSS series was 35.4% [19] [20]. The fitted parameter values and conditional numerical projections are specific to Georgia; the equilibrium formulas and stability results are not. After recalibration, the framework can be applied to other populations represented by the same four-compartment transition structure.

In the simulations, increasing the effective treatment-transition rate can reduce both transient and equilibrium obesity prevalence. The size of the reduction depends on the durability of treatment benefit. A large return flow from T to O offsets the gain produced by increasing τ , so improvement in the treatment-response pathway may deliver less long-term benefit when relapse remains high. Because τ aggregates treatment initiation, adherence, and sufficient early response, the present model does not isolate the effect of access alone. Instead, it represents the combined rate at which individuals move from obesity into the treatment-response state.

This interpretation is consistent with policy approaches that combine medication access with supportive care. The CMS BALANCE model, for example, pairs expanded access to GLP-1 treatment with lifestyle support [25]. The present model combines treatment initiation, adherence, and sufficient early response within the effective transition rate τ rather than estimating their separate effects. In Section 5.6, the stronger combined setting gives a larger reduction than the moderate setting. Nevertheless, even the stronger intervention $(1.6\tau, 0.50\eta)$ does not reverse the upward trajectory during the projection horizon because the system converges slowly toward its new endemic equilibrium.

6.4. Limitations and Future Research

The conclusions are subject to the following modeling limitations.

First, the homogeneous formulation does not distinguish age, sex, race and ethnicity, education, or insurance-related treatment access. Population surveillance and real-world treatment studies document variation across these dimensions [4] [26]. Including demographic structure would allow a more detailed analysis of disparities in long-run outcomes.

Second, births, deaths, and migration are not modeled explicitly, and the tran-

sition parameters are assumed constant over time. Consequently, the extended 40- and 80-year trajectories should be interpreted as mathematical illustrations of convergence in normalized population proportions rather than as literal projections of a fixed adult cohort. The constant-rate assumption also cannot represent changes in treatment uptake and persistence or future therapeutic developments [9] [14]. Extensions incorporating demographic turnover and time-varying parameters would improve the realism. Accordingly, the 2030 value is a conditional point projection and does not include uncertainty in the fixed transition rates.

Third, practical-identifiability analysis shows that the estimates of β and σ are strongly correlated. Although the output-sensitivity matrix has full numerical rank, the estimated-parameter correlation is approximately 0.9985, and the profile likelihood for σ does not close within the prescribed fitting range. Consequently, the individual parameter values and long-run outcomes should be interpreted conditionally on the fixed baseline assumptions. Additional observations of the overweight or treatment-response states would be needed to separate the progression and partial-recovery rates more precisely. In addition, the unweighted least-squares objective assigns equal weight to annual estimates despite differences in their reported sampling precision. A supplementary weighted-calibration check using confidence-interval-derived variance estimates produced only small changes in the fitted trajectory and conditional 2030 projection (Table S3).

Fourth, the effective treatment-transition rate τ is represented as an exogenous parameter that combines treatment initiation, adherence, and sufficient early response. Insurance coverage, medication cost, supply, adherence behavior, and health-system capacity are not modeled separately. Real-world studies show incomplete treatment persistence and associations between insurance-plan structure and semaglutide initiation [14] [26]. Incorporating these mechanisms explicitly would support a more detailed policy analysis.

Finally, the stability analysis relies on the linear structure of the system. Extensions involving nonlinear relapse, adherence behavior, or treatment-capacity constraints could be analyzed using methods from monotone dynamical systems [27] [28].

Future work could include demographic stratification, uncertainty analysis, cost-effectiveness evaluation of combined treatment and adherence strategies, and incorporation of health-economic models. Despite these restrictions, the explicit expression for E^* provides a tractable and transparent model of how treatment response and relapse contribute to the prevalence of obesity in the long run.

7. Conclusion

This study examines the long-run behavior of obesity under treatment response and relapse using a compartmental dynamical framework. Under the conditional best-fit scenario, the observed trend is consistent with gradual convergence toward a higher endemic level. Increasing the effective treatment-transition rate

shifts trajectories downward, although high relapse limits the effect. As the treatment-response pathway expands, equilibrium obesity prevalence becomes substantially more sensitive to the effective treatment-transition rate and relapse. Nevertheless, under the reference and enhanced parameter settings used in the main analysis, obesity progression remains the strongest positive driver, so behavioral prevention, effective treatment response, and relapse reduction remain complementary components of long-term obesity control. Improving access to GLP-1-based therapies may contribute to a larger effective transition rate, but access alone is not sufficient because the rate also reflects adherence and sufficient early response. Combining improved treatment response with reduced relapse produces larger sustained reductions in obesity prevalence.

Data Availability Statement

All data used in this study are publicly available. Obesity prevalence data were obtained from the Behavioral Risk Factor Surveillance System (BRFSS) maintained by the Centers for Disease Control and Prevention (CDC). The exact Georgia series used for calibration is provided in the Supplementary Material.

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Conflicts of Interest

The author declares no conflict of interest.

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Appendix. Supplementary Material

A Compartmental Model of Obesity with Treatment Response and Relapse: Endemic Equilibrium Analysis and Long-Term Dynamics.

S1. Calibration Data

Table S1 reports the annual Georgia adult obesity prevalence estimates and published 95% confidence limits used for calibration. Values were verified against the Georgia rows in the CDC annual tables [1].

Table S1. Georgia adult obesity prevalence, BRFSS, 2011-2024.

Year	Prevalence (%)	Lower 95% limit (%)	Upper 95% limit (%)	Source PDF page
2011	28.0	26.6	29.4	19
2012	29.1	27.4	30.8	20
2013	30.3	28.9	31.8	21
2014	30.5	28.9	32.1	22
2015	30.7	28.8	32.6	23
2016	31.4	29.7	33.2	24
2017	31.6	30.0	33.3	25
2018	32.5	31.2	33.8	26
2019	33.1	31.3	35.0	27
2020	34.3	32.5	36.0	28
2021	33.9	32.2	35.6	29
2022	37.0	35.4	38.7	30
2023	35.0	33.4	36.7	31
2024	35.4	33.7	37.1	32

Source pages are counted from the first page of the PDF.

The source file is 2011-2024-overall-obesity-prevalence-map-508.pdf, pages 19 - 32 [1]. All 14 years were retained without imputation or smoothing. Percentages and confidence limits were divided by 100 for computation.

Numerical Methods

Supplementary refits were performed in MATLAB R2021a using the same model equations, data, parameter bounds, and baseline assumptions as the main analysis. The linear system was evaluated with its matrix-exponential solution, $y(t) = e^{At} y_0$, and β and σ were estimated with fminsearch using multiple starting values and explicit enforcement of the parameter bounds,

$$0.01 \leq \beta \leq 0.15, \quad 0 \leq \sigma \leq 0.06 \text{ (yr}^{-1}\text{)}.$$

The original ode45/pchip/fminsearch baseline was reproduced separately; the

two implementations agreed to the reported precision. The 14 annual observations were fitted by unweighted least squares, except in S3. Time was measured from 2011; 2030 corresponds to year 19.

S2. Robustness to Initial Conditions

The model was refitted at all nine combinations of $W(0) \in \{0.30, 0.35, 0.40\}$ and $T(0) \in \{0, 0.01, 0.02\}$, with $O(0) = 0.28$ and $H(0) = 1 - O(0) - W(0) - T(0)$. The five fixed rates remained at their manuscript baseline values. These are assumed sensitivity ranges, not empirical intervals for Georgia.

Table S2. Refitted rates, equilibrium prevalence, and conditional 2030 projection.

$W(0)$	$T(0)$	$H(0)$	$\hat{\beta}$ (yr ⁻¹)	$\hat{\sigma}$ (yr ⁻¹)	O^* (%)	$O(2030)$ (%)
0.30	0.00	0.42	0.10088	0.06000†	47.33	38.26
0.30	0.01	0.41	0.08349	0.04870	47.50	38.16
0.30	0.02	0.40	0.04888	0.02099	52.74	38.61
0.35	0.00	0.37	0.08570	0.05692	44.67	37.75
0.35	0.01	0.36	0.06069	0.03552	46.87	38.10
0.35	0.02	0.35	0.03547	0.01296	54.58	38.62
0.40	0.00	0.32	0.06741	0.04675	43.39	37.66
0.40	0.01	0.31	0.04776	0.02808	46.23	38.07
0.40	0.02	0.30	0.02784	0.00840	56.54	38.63

†The estimate $\hat{\sigma} = 0.06000$ reaches the imposed upper bound.

The bold row is the baseline initial state. The estimate reaches the imposed upper bound $\sigma = 0.06$. Equilibrium values change because β and σ are refitted, not because a fixed-parameter equilibrium depends on the initial state.

The conditional 2030 projection ranges from 37.66% to 38.63%, whereas equilibrium prevalence ranges from 43.39% to 56.54%. These are sensitivity ranges, not confidence intervals.

S3. Weighted Calibration

A weighted fit [2] was compared with the unweighted baseline. The data provide rounded 95% confidence limits, not separate sampling variances. For limits L_i and

U_i expressed as proportions, variances were approximated by $\hat{v}_i = \left[\frac{U_i - L_i}{3.92} \right]^2$

under a symmetric normal approximation. The weighted objective was

$$\text{WSSE}(\theta) = \sum_{i=1}^{14} \frac{[O_{\text{model}}(t_i; \theta) - O_{\text{data}}(t_i)]^2}{\hat{v}_i}, \theta = (\beta, \sigma)^T$$

The initial state $y_0 = (0.36, 0.35, 0.28, 0.01)^T$, five fixed rates, parameter bounds, and observations were unchanged. These are precision weights for annual

estimates, not respondent survey weights.

Table S3. Unweighted and approximate inverse-variance-weighted calibration.

Calibration	$\hat{\beta}$ (yr ⁻¹)	$\hat{\sigma}$ (yr ⁻¹)	O^* (%)	$O(2030)$ (%)
Unweighted (baseline)	0.06069	0.03552	46.87	38.10
Weighted (CI-derived)	0.06208	0.03691	46.57	38.05

The largest trajectory difference over 2011-2024 was 0.018 percentage points. The 2030 projection changed from 38.10% to 38.05%, and the equilibrium from 46.87% to 46.57%. The unweighted fit remains the main analysis.

S4. Robustness to Fixed Transition Rates

Each of the five fixed rates took its lower, baseline, and upper value in **Table S4**, giving $3^5 = 243$ combinations in each grid. These ranges are assumed departures from baseline. At every combination, β and σ were refitted using the baseline initial state, unweighted objective, and original bounds; all boundary fits were retained.

Table S4. Fixed-rate robustness. Parameter ranges (yr⁻¹).

Rate	Baseline	Moderate grid $\pm 20\%$	Broader stress test $\pm 50\%$
α	0.030	0.024 - 0.036	0.015 - 0.045
ρ	0.020	0.016 - 0.024	0.010 - 0.030
τ	0.030	0.024 - 0.036	0.015 - 0.045
γ	0.050	0.040 - 0.060	0.025 - 0.075
η	0.400	0.320 - 0.480	0.200 - 0.600

S5. Sensitivity Rankings and Intervention Outcomes

Exact normalized sensitivity indices were evaluated at each refitted reference setting and its enhanced setting $(5\tau, 0.5\eta)$, holding other parameters fixed for each derivative. Rankings compare all seven rates. The moderate $(1.3\tau, 0.75\eta)$ and stronger $(1.6\tau, 0.5\eta)$ combined scenarios were applied from 2011 without further refitting. Obesity prevalence was compared at 2030 and at equilibrium.

Table S5. Sensitivity rankings and intervention outcomes.

Result	Moderate grid $\pm 20\%$	Broader grid $\pm 50\%$
Fixed-rate settings evaluated	243	243
β largest positive index: reference	243/243	241/243
σ most negative index: reference	243/243	222/243
β largest positive index: enhanced	243/243	231/243
τ most negative index: enhanced	232/243	195/243

Continued

Magnitudes of both τ and η indices increase under enhancement	243/243	243/243
Stronger < moderate < reference: $O(2030)$	243/243	243/243
Stronger < moderate < reference: O^*	243/243	243/243
Reference $O(2030)$ range (%)	37.92 - 38.37	37.62 - 39.37
Reference O^* range (%)	44.79 - 52.47	43.14 - 90.31
Refits at the upper bound for σ	9	54
Refits at the lower bound for σ	0	5

Counts refer to evaluated settings, not probabilities. Reported ranges are sensitivity ranges, not confidence or prediction intervals.

The intervention ordering was preserved in all tested settings, but the exact sensitivity rankings were not. Equilibrium prevalence was more sensitive to the fixed-rate assumptions than the 2030 projection. The upper stress-test equilibrium of 90.31% is a conditional model output, not an estimate of Georgia prevalence.

Supplementary References

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