Modeling the Economic Impact of Muguka Addiction: A Deterministic Approach That Integrates Economic Burden Cost

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ABSTRACT

Muguka, a variant of miraa (khat), significantly influences the socio-economic dynamics of regions where it is widely cultivated and consumed. Despite its economic importance, addiction to Muguka has led to substantial health and economic challenges, yet limited research has quantified these impacts comprehensively. To address this gap, this study develops a deterministic compartmental model that integrates health costs to analyze the dynamics of Muguka addiction. The aim is to quantify and evaluate the economic and health consequences of Muguka addiction, providing insights for effective public health interventions. The methodology involves formulating a system of ordinary differential equations (ODEs) to capture the dynamics of addiction and health costs across defined compartments. Key aspects of the analysis include deriving the reproduction number, examining equilibrium points and their stability, and performing sensitivity analysis to explore how changes in critical parameters influence addiction prevalence and associated costs. Numerical simulations are employed to assess health cost dynamics and to design optimal, cost-effective control strategies. The findings reveal that Muguka addiction imposes significant economic burdens, including substantial income loss and escalating healthcare costs. The model provides policymakers and public health officials with a valuable tool for designing targeted interventions to mitigate the adverse effects of Muguka addiction.

Keywords: Muguka addiction; deterministic modeling; health costs; economic impact; sensitivity analysis

INTRODUCTION

Muguka, a variant of the khat plant (*Catha edulis*), is a stimulant commonly used in parts of East Africa, particularly in Kenya. Unlike traditional khat, Muguka consists of smaller, more potent leaves and is often favored for its stronger stimulating effects. The leaves of the Muguka plant are chewed to release cathinone, an amphetamine-like stimulant that produces feelings of euphoria, increased alertness, and hyperactivity. The cultivation and sale of Muguka provides substantial income for farmers and traders, contributing to the livelihoods of many households. In regions like Meru County in Kenya, Muguka cultivation has become a major economic activity, even outpacing other agricultural products in profitability [12]

However, these economic benefits are counterbalanced by several drawbacks. The focus on Muguka cultivation often leads to the neglect of other essential food crops, which can compromise local food security. Additionally, the income generated from Muguka trade is not always reinvested in sustainable activities. Instead, users may spend a significant portion of their earnings on the drug, perpetuating a cycle of poverty [10]. Furthermore, the financial gains are unequally distributed: middlemen and traders capture a larger share of the profits compared to the farmers who grow the plant. This disparity can lead to social tensions and exacerbate existing inequalities within communities [9]

The social implications of Muguka use are profound and multifaceted. Muguka is often consumed in group settings, fostering a sense of community among users. However, its use is also associated with negative social consequences. Prolonged use of Muguka can lead to social isolation, as users may prioritize the drug over familial and social responsibilities [7]. Muguka use has also been linked to increased crime rates and social unrest. The stimulant effects of Muguka can lead to aggressive behavior and impaired judgment, contributing to domestic violence and other criminal activities. Moreover, the presence of widespread Muguka use in communities can strain social services and law enforcement resources as they contend with the related social issues [11]

Muguka consumption poses significant health risks, both physical and mental. Physically, Muguka use can lead to dental problems, gastrointestinal disorders, and cardiovascular issues. Short-term effects include increased heart rate, elevated blood pressure, and hyperactivity, while long-term use can precipitate more severe problems such as cardiomyopathies, digestive tract ulcers, and tooth decay. The active ingredient in Muguka, cathinone, has stimulant properties like amphetamines, which can overwork the cardiovascular system and lead to complications like hypertension and heart disease [13]. Mentally, chronic Muguka use is associated with psychiatric disorders including anxiety, depression, and psychosis. Dependence on the drug can result in withdrawal symptoms and exacerbation of mental health issues. Studies have shown that Muguka users are at higher risk of developing mental health disorders compared to non-users [8]. Cognitive functions and emotional well-being are also impaired: users often experience memory deficits, reduced concentration, poor decision-making, mood swings, aggression, and social withdrawal. These cognitive and emotional impairments hinder personal development and strain social relationships [13]

The public health implications of widespread Muguka use are significant. Communities with high rates of Muguka abuse face increased healthcare burdens due to the drug's health impacts. Public health systems may struggle to address the needs of Muguka users, leading to strained resources and inadequate care. Moreover, the communal aspect of Muguka consumption (e.g., gathering in close quarters to chew) can facilitate the spread of infectious diseases, adding another layer of public health concern [12]. Healthcare systems thus face substantial challenges in addressing Muguka's health impacts. The legal status and cultural acceptance of Muguka complicate efforts to regulate its use and mitigate its risks. Policymakers must balance respect for cultural practices with the need to implement effective public health strategies. This balance requires comprehensive policies that include public education, accessible healthcare and rehabilitation services, and community engagement to reduce the adverse health effects of Muguka [10]. Although Muguka's physiological and psychological effects mirror those of other potent stimulants such as cocaine and methamphetamine, its cultural acceptability and legal status often downplay its dangers, complicating efforts to curb its use [5]. This underscores the need for targeted interventions to address the under-recognized risks of Muguka abuse in affected regions.

Recent advances in mathematical epidemiology have demonstrated the importance of compartmental models in understanding the transmission dynamics of addictive behaviors and in evaluating intervention strategies. Unlike traditional infectious disease models, substance-use models incorporate behavioural transitions, treatment adherence, relapse, and recovery processes that determine the persistence of addiction within a population. Mathematical models developed for substances such as alcohol, tobacco, opioids, and khat have provided valuable insights into the threshold conditions governing addiction persistence, the effectiveness of rehabilitation programmes, and the long-term consequences of substance abuse on public health systems [15].

From a health economics perspective, the burden of substance addiction extends beyond direct medical expenditures. Economic evaluations have increasingly recognized the importance of incorporating indirect costs, including productivity losses due to reduced labour participation, absenteeism, premature mortality, family instability, crime, and increased social welfare expenditures. Such comprehensive cost assessments provide policymakers with evidence required to allocate resources effectively between prevention programmes, treatment services, and community rehabilitation initiatives [16,17].

Although several mathematical models have examined the dynamics of substance dependence, there remains limited literature addressing Muguka-specific addiction, particularly in relation to the integration of epidemiological dynamics with economic burden analysis. Existing studies on khat and related stimulants have

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largely focused on physiological effects, social implications, and agricultural economics, with relatively little attention given to quantitative prediction of addiction progression and associated health-related expenditures [1,2,4]. This creates a significant knowledge gap, especially in regions of Eastern Kenya where Muguka cultivation, trade, and consumption constitute major socio-economic activities.

The present study addresses this gap by developing a deterministic compartmental model that integrates addiction dynamics with a health-economic framework. Unlike conventional addiction models that focus primarily on prevalence and control, the proposed approach quantifies the evolving economic consequences of addiction through a cost function associated with affected populations. This framework provides a quantitative basis for assessing the potential benefits of prevention strategies, treatment interventions, and recovery programmes aimed at reducing the long-term societal burden of Muguka addiction [3,18].

In summary, Muguka addiction is a serious socio-economic burden, exacerbating healthcare costs and reducing productivity in affected communities. Effective intervention requires an understanding of the interplay between addiction dynamics and economic factors. To this end, we develop a mathematical model that quantifies addiction prevalence, evaluates the impact of interventions, and predicts the economic burdens of Muguka use through an explicit health cost function.

Mathematical Formulation

This section introduces a deterministic compartmental model to study the economic impact of Muguka addiction by incorporating health costs into the population dynamics. The model tracks the progression of individuals through four stages: addiction and its control while quantifying the associated economic implications (such as healthcare costs). The population is divided into four compartments: Susceptible (S) individuals who have never used Muguka (but could potentially initiate use), Addicted (A) individuals who are actively using/abusing Muguka, Treatment (T) for individuals undergoing treatment or rehabilitation for Muguka addiction, and Recovered (R) individuals who have successfully overcome addiction (assumed to have temporarily or permanently quit Muguka). Key model parameters include those governing the rates of addiction initiation, treatment entry, recovery, relapse, and Muguka-related health complications, as well as per-capita cost coefficients for addicted and treated individuals (defined in the next section). Table 1 lists the state variables and their meanings, and Table 2 summarizes the model parameters and baseline values.

Table 1: State Variables

Variable	**Description**
$S(t)$	Population susceptible to initiating Muguka use
$A(t)$	Individuals actively addicted to Muguka
$T(t)$	Individuals receiving treatment or rehabilitation
$R(t)$	Individuals who have recovered from Muguka addiction

The model is structured as a system of ODEs representing the transitions between these compartments (Figure 1 illustrates the flow between compartments). We assume an open population with recruitment (e.g. immigration or maturation into adulthood) feeding into the susceptible class and natural mortality affecting all classes equally. The following were the key

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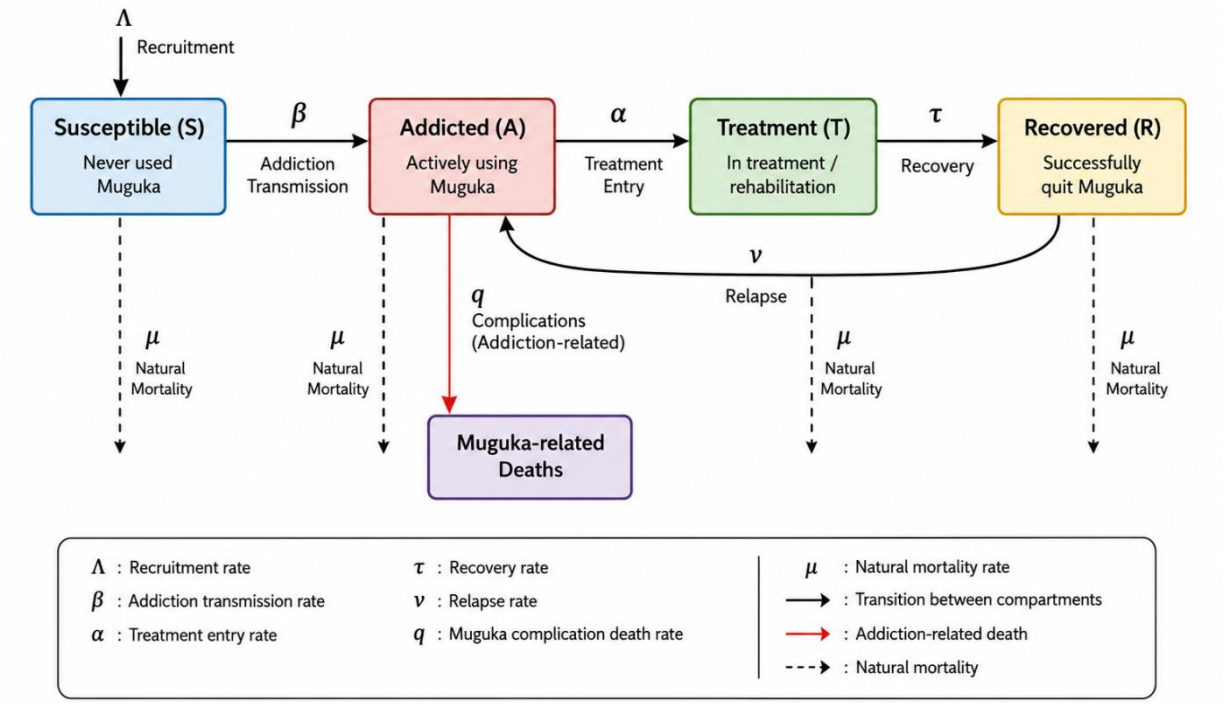

Figure 1: Schematic representation of the Muguka addiction model showing recruitment, addiction transmission, treatment entry, recovery, relapse, natural mortality, and addiction-related deaths.

Model Assumptions:

i. The total population $N(t)$ at time t is subdivided into four compartments based on Muguka use status: Susceptible $S(t)$, Addicted $A(t)$, in Treatment $T(t)$, and Recovered $R(t)$.

ii. New Muguka addictions occur through interactions between susceptible and addicted individuals (i.e., contact with addicted people encourages non-users to start using).

iii. Health-related costs accrue only from addicted and treatment populations (i.e., only active users and those in treatment incur direct health and social costs in the model).

iv. There is a constant inflow of new susceptible individuals (through birth or immigration) to the population. This "recruitment" replenishes the susceptible class.

v. Intervention programs (such as awareness campaigns or rehabilitation efforts) increase the rate at which addicts seek treatment and successfully recover (and thereby leave the addicted compartment).

Parameter Estimation and Data Sources

The reliability of any mathematical model depends significantly on the accuracy and justification of the parameters employed. In this study, model parameters were obtained from a combination of published literature on khat and substance-use disorders, demographic reports from Kenya, public health records, and reasonable assumptions where Muguka-specific data were unavailable. Since comprehensive epidemiological data on Muguka addiction remain limited, some parameters were estimated by adapting values from similar stimulant-use studies and calibrated to reflect the social and health conditions prevalent in Muguka-consuming regions of Eastern Kenya.

The natural recruitment and mortality rates were estimated using demographic statistics obtained from the Kenya National Bureau of Statistics (KNBS) and national population reports. The addiction transmission coefficient (β) was estimated based on behavioural interaction patterns between susceptible individuals and active Muguka users, following approaches used in previous addiction transmission models [15]. The treatment-seeking rate (α) recovery rate (τ), and relapse rate (ν) were informed by studies on substance

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rehabilitation outcomes and addiction recovery dynamics [16,18]. The Muguka-related complication rate (q) was approximated from documented health complications associated with chronic khat consumption, including cardiovascular and psychiatric disorders [1,5,8].

The cost parameters associated with addiction and treatment were derived from available health economic studies on substance abuse, incorporating direct healthcare expenditures and estimated productivity losses due to reduced labour participation and impaired social functioning [16,17]. In the absence of detailed Muguka-specific economic records, these cost estimates serve as representative indicators of the relative economic burden of addiction.

Table 2: Model Parameters, Description, Baseline Values, and Data Sources

Parameter	Description	Baseline Value	Unit	Source/Justification
Λ	Recruitment rate into susceptible population	KNBS estimate	individuals/year	Kenyan demographic statistics
β	Muguka addiction transmission rate	Calibrated	person^{-1} year^{-1}	Estimated from addiction contact models [15]
μ	Natural mortality rate	KNBS estimate	year^{-1}	National demographic reports
α	Rate of entering treatment	0.05	year^{-1}	Substance rehabilitation studies [15]
τ	Recovery rate from treatment	0.30	year^{-1}	Addiction treatment literature [16,18]
ν	Relapse rate after recovery	0.45	year^{-1}	Drug dependence and relapse studies [18]
q	Muguka-related health complication rate	0.02	year^{-1}	Khat-related medical studies [1,5,8]
Ca	Cost per addicted individual	Estimated	KES/person/year	Health economic assessment [16,17]
Ct	Cost per treated individual	Estimated	KES/person/year	Rehabilitation and treatment expenditure studies [16,17]

Some parameter values were obtained from published demographic and health studies, while parameters lacking Muguka-specific empirical estimates were calibrated using available regional data and evidence from comparable substance-use models.

Model Calibration and Validation

To improve the predictive reliability of the proposed model, a calibration procedure was undertaken using available information on Muguka consumption patterns, addiction prevalence, and reported health-related effects from affected regions of Eastern Kenya, particularly Meru and surrounding counties where Muguka cultivation and consumption are widespread [11,14]. Unknown parameters, particularly the addiction transmission coefficient (β), were adjusted within biologically realistic ranges so that the model-generated addiction trajectory reproduced the observed trends reported in available public health and socio-economic studies.

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The model was validated by comparing simulated addiction prevalence and health burden trends with available observational evidence from regional studies and public health reports. The comparison showed that the model reproduced the expected pattern of increasing addiction burden in the absence of effective interventions and a decline in prevalence when treatment and preventive measures were strengthened. Although the scarcity of detailed longitudinal Muguka-specific data limited the use of advanced statistical validation techniques, the qualitative agreement between simulation outcomes and observed regional trends supports the applicability of the model as a decision-support framework.

Future studies should employ comprehensive epidemiological surveys and healthcare expenditure databases to perform rigorous parameter estimation using statistical optimization methods such as least squares fitting, maximum likelihood estimation, and Bayesian inference. Such approaches would provide more precise estimates of uncertainty and improve the predictive capacity of the model.

For mathematical clarity, all state variables and parameters used in the model are explicitly defined before the formulation of the governing equations. Let $S(t)$, $A(t)$, $T(t)$, and $R(t)$ denote the populations of susceptible individuals, active Muguka users, individuals undergoing treatment, and recovered individuals at time (t), respectively. The total population at time (t) is given by

$$N(t) = S(t) + A(t) + T(t) + R(t)$$

The model assumes that the population changes due to recruitment, natural mortality, addiction-related transitions, treatment processes, recovery, and relapse. The dynamics are therefore represented by the following system of ordinary differential equations.

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \beta S A - \mu S, \\ \frac{dA}{dt} &= \beta S A + \nu R - (\alpha + q + \mu)A, \\ \frac{dT}{dt} &= \alpha A - (\tau + \mu) T, \\ \frac{dR}{dt} &= \tau T - (\nu + \mu) R, \end{aligned} \tag{1}$$

where:

- Λ represents the recruitment rate into the susceptible population.
- β represents the effective Muguka addiction transmission rate.
- μ denotes the natural mortality rate.
- α represents the rate at which addicted individuals enter treatment.
- τ denotes the successful recovery rate from treatment.
- ν represents the relapse rate of recovered individuals.
- q denotes the Muguka-related health complication or addiction-induced removal rate.

Model Analysis

Positivity And Boundedness of Solutions

Given non-negative initial conditions $S(0)$, $A(0)$, $T(0)$, $R(0)$, the system's trajectories remain non-negative for all future times, which is crucial since the variables represent population counts. This can be verified by standard arguments showing that each equation prevents the corresponding state from becoming negative if it starts non-negative (for instance, dS/dt at $S = 0$ is $\Lambda > 0$, pushing S positive). The population $N(t)$ is

bounded above by a carrying capacity determined by the balance of inflow Λ and outflow μ. Specifically, in the absence of addiction ($A = T = R = 0$), the susceptible population tends to the equilibrium $S = \Lambda/\mu$. With addiction present, total population may decline due to the additional removals qA, but solutions remain bounded for all $t \geq 0$. These properties ensure the model dynamics are epidemiologically and demographically well-posed (solutions stay in the feasible region $S, A, T, R \geq 0$ and $N < \infty$ for all time).

Equilibrium Points

To determine the equilibrium points of the system, we set all time derivatives to zero ($dS/dt = dA/dt = dT/dt = dR/dt = 0$). Solving the resulting algebraic equations yields the equilibrium populations S^*, A^*, T^*, R^*. Two important equilibrium scenarios are of interest:

Muguka-Free Equilibrium (MFE): This equilibrium corresponds to the absence of active addiction in the population. Setting $A^* = T^* = R^* = 0$, the equations simplify and yield a nonzero susceptible population at steady state. From the first equation, $0 = \Lambda - \mu S^*$ (since $A^* = 0$), giving $S^* = \Lambda/\mu$. The other equations are satisfied by $A^* = T^* = R^* = 0$. Thus, the Muguka-free equilibrium is

$$(S^*, A^*, T^*, R^*) = \left(\frac{\Lambda}{\mu},\ 0,\ 0,\ 0\right).$$

This scenario represents a stable society with a constant population Λ/μ in which no one is using Muguka.

Muguka Endemic Equilibrium (MEE): This is a situation where Muguka addiction persists in the population at steady state, so all compartments are potentially nonzero. Solving the full equilibrium equations for S^*, A^*, T^*, R^* in terms of the model parameters is algebraically involved. However, the endemic equilibrium can be expressed in closed form by a sequence of substitutions. From the S-equation at equilibrium: $0 = \Lambda - \beta S^* A^* - \mu S^*$. At the endemic equilibrium, typically $N^* = \Lambda/\mu$ (meaning the inflow balances total outflow in the long term), so one can solve for S^* in terms of A^*. Similarly, the equilibrium conditions for A, T, R can be manipulated. In general, the endemic equilibrium will exist only if the system's parameters satisfy a threshold condition (related to the reproduction number exceeding 1, discussed below). For brevity, we do not reproduce the full expressions for A^*, T^*, R^* here, but they can be obtained by algebraic elimination. These equilibrium values depend on a complex interplay of parameters $\beta, \alpha, \tau, \nu, q$, etc. When it exists, the endemic equilibrium is denoted

$$(S^*, A^*, T^*, R^*) = \left(\tilde{S},\ \tilde{A},\ \tilde{T},\ \tilde{R}\right),$$

with all components $\tilde{S}, \tilde{A}, \tilde{T}, \tilde{R} > 0$. This equilibrium forms the basis for analyzing the long-term burden of Muguka addiction in the population.

Local Stability Analysis

We assess the local stability of the equilibrium points by examining the Jacobian matrix of the system. The Jacobian J is a 4×4 matrix of partial derivatives $J_{ij} = \partial f_i / \partial x_j$ (where f_i is the right-hand side of the ith ODE and $x_j \in \{S, A, T, R\}$). Evaluating J at an equilibrium and analyzing its eigenvalues reveals whether that equilibrium is stable (eigenvalues with negative real parts) or unstable (any eigenvalue with a positive real part).

At the Muguka-Free Equilibrium $(\Lambda/\mu, 0,0,0)$, the Jacobian takes a block triangular form due to the absence of addicts and patients. The eigenvalues in this case are found to be $-\mu$ (with multiplicity three for the S, T, R directions) and $\beta\, S^* - (\alpha + q + \mu)$ for the A direction (since the linearized growth of A when rare is driven by $\beta S^* - (\alpha + q + \mu)$). Here $S^* = \Lambda/\mu$ is the susceptible population at MFE. The sign of the eigenvalue associated with the addicted class determines the stability of the MFE. If $\beta S^* < \alpha + q + \mu$, then all eigenvalues are negative and the MFE is locally asymptotically stable (addiction cannot invade). Conversely, if $\beta S^* > \alpha + q + \mu$, the eigenvalue for the addicted class becomes positive, making the MFE unstable and

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indicating that an invasion of addiction can occur from rarity. This condition leads naturally to the definition of a threshold parameter for the system.

Basic Reproduction Number R_0

Using the next-generation matrix approach, the basic reproduction number is obtained as

$$R_0 = \frac{\beta S^0}{\alpha+q+\mu} \quad (2)$$

where $S^0 = \frac{\Lambda}{\mu}$ is the susceptible population at the Muguka-free equilibrium. The parameter R_0 represents the expected number of secondary addictions generated by one addicted individual introduced into a fully susceptible population.

The threshold condition $R_0 < 1$ indicates that addiction cannot sustain itself and the Muguka-free equilibrium remains locally asymptotically stable. Conversely, $R_0 > 1$ implies persistent addiction and the existence of a Muguka-endemic equilibrium.

Stability of the Endemic Equilibrium

When $R_0 > 1$, the model admits the endemic equilibrium $(\tilde{S}, \tilde{A}, \tilde{T}, \tilde{R})$. The local stability of this Muguka-endemic equilibrium can be analyzed by evaluating the Jacobian at that point. Computation is more involved because all compartments have nonzero values. However, by applying the Ruth-Hurwitz criteria or numerical eigenvalue computation, one can check that the endemic equilibrium is locally asymptotically stable whenever it exists (provided model parameters remain in a biologically realistic range). In simpler terms, if $R_0 > 1$, the system will tend toward the endemic state of persistent Muguka use, and small perturbations around that state will die out, keeping the system near the endemic equilibrium [4].

Total Economic Burden Analysis

An important contribution of this model is the incorporation of a health cost function that quantifies the direct healthcare expenditure of Muguka addiction over time [5,6]. We define the total health-related cost at time t as:

$$C(t) = c_1 A(t) + c_2 T(t) \quad (3)$$

where:

- c_1 is the average economic cost associated with one addicted individual.
- c_2 is the average treatment and rehabilitation cost per individual under treatment.

Although direct healthcare expenditure represents an important component of the economic burden of Muguka addiction, the total societal cost extends beyond medical treatment. Chronic Muguka use can lead to reduced productivity due to absenteeism, decreased work performance, unemployment, family economic instability, increased criminal activities, and additional expenditure on social support and law enforcement. Therefore, to provide a more comprehensive assessment of the economic consequences of Muguka addiction, the total economic burden is extended to include indirect and social costs. This approach is consistent with contemporary health economic evaluations of substance abuse [16,17,18].

The total economic burden of Muguka addiction is therefore represented as

$$E(t) = c_1 A(t) + c_2 T(t) + c_P A(t) + c_S R(t) \quad (4)$$

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where:

- $E(t)$ is the total economic burden at time t;
- $c_1 A(t)$ represents direct healthcare costs associated with active Muguka addiction;
- $c_2 T(t)$ represents treatment and rehabilitation costs;
- $c_P A(t)$ represents productivity losses arising from reduced work capacity, absenteeism, and unemployment among addicted individuals;
- $c_S R(t)$ represents broader social response costs including community support programmes, family assistance, crime prevention, and law enforcement interventions.

Differentiating Equation (4) with respect to time (t) gives

$$\frac{dE}{dt} = (c_1 + c_P)\frac{dA}{dt} + c_T\frac{dT}{dt} + c_T\frac{dT}{dt} + c_S\frac{dR}{dt} \quad (5)$$

Substituting the governing model equations results in

$$\frac{dE}{dt} = (c_1 + c_P)(\beta SA + \nu R - (\alpha + q + \mu)A + c_2(\alpha A - (\tau + \mu)T + c_S(\tau T - (\nu + \mu)R \quad (6)$$

Sensitivity Analysis

The extended economic model highlights that the societal burden of Muguka addiction is multidimensional. In addition to healthcare expenditures, the indirect losses associated with reduced labour productivity and social consequences may constitute a substantial proportion of the total economic burden. Consequently, interventions that prevent addiction initiation, increase treatment effectiveness, and reduce relapse rates have economic benefits beyond reducing medical costs, as they preserve productivity and reduce pressure on social support systems.

To understand which factors, influence health cost the most, we performed a sensitivity analysis on key parameters[13]. In each case, we vary one parameter and observe the effect on the cost function $C(t)$ (either on its peak value or long-term level), while holding other parameters at baseline values (see Table 2). The findings can be summarized as follows:

Cost per Addict vs. per Treatment (c_1 and c_2): Increasing the cost coefficient for addiction c_1 leads to a directly proportional rise in total cost $C(t)$ at any given prevalence of addiction. A larger c_1 means each addicted individual imposes a higher economic burden, which raises both the peak and cumulative cost of an growth in addiction prevalence [13]. Similarly, increasing c_2 (cost per person in treatment) raises the portion of cost due to treatment programs. Higher c_2 would reflect more expensive or intensive treatment efforts, increasing total costs especially if a large fraction of addicts enter treatment. In practice, a higher c_1 emphasizes the need for preventive measures (since addiction is very costly), whereas a higher c_2 highlights the economic impact of investing in recovery programs.

*Treatment-Seeking Rate (α)***:** This is the rate at which addicts voluntarily enter treatment. A higher α means addicts are moving to treatment faster. In the short term, raising α can increase T(t) and thus $C(t)$ (because more individuals are incurring treatment costs) [14]. However, because treatment removes individuals from the addicted pool, a sufficiently large α will eventually lead to a lower number of active addicts $A(t)$, which can reduce long-term costs. Our model suggests there may be an initial cost surge when scaling up treatment programs, followed by a reduction in the overall addiction prevalence and a subsequent decline in total costs as the epidemic of addiction is brought under control.

Addiction Transmission Rate (β): This "contact rate" determines how quickly susceptible individuals become addicted through interactions with addicts. As expected, a higher β dramatically increases the spread of

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addiction [12]. When β is large, the model predicts a rapid rise in $A(t)$,leading to a higher and earlier peak in the health cost C(t). Faster addiction spread means that more individuals succumb before interventions can take effect, significantly raising the total burden. This result underlines the importance of preventive programs (education, awareness, restrictions on Muguka availability) in reducing effective contact rates and thereby mitigating the growth of addiction cases.

Recovery Rate (τ): This is the rate at which individuals in treatment successfully recover (i.e., leave the T compartment for R). Increasing τ means treatment is more effective or faster, so individuals spend less time in treatment and more quickly become recovered [12]. A higher τ tends to reduce the number of active addicts $A(t)$ (because treatment success removes people from the addictive cycle) and reduces T(t) (since people do not stay in treatment as long). Both effects act to lower the health cost $C(t)$ over time. Thus, improving the efficacy of treatment programs (higher recovery rate) can substantially decrease the long-term economic burden of Muguka addiction by curbing the population of addicts and the duration of costly interventions.

In addition to the above, we note that other parameters like the relapse rate ν and the complication removal rate q can influence the dynamics and cost, even though they were not explicitly varied in the four cases above. For instance, a higher relapse rate \nu means recovered individuals are more likely to fall back into addiction, which can sustain higher A(t) and costs for longer. Conversely, a higher Muguka-related death rate q will tend to remove addicts faster (reducing $A(t)$, but it also means more loss of life and productivity; its net effect on C(t) depends on how one accounts for the cost of fatalities (our $C(t)$captures ongoing costs, not the implicit cost of lost life years). Generally, reducing ν(through better aftercare to prevent relapse) will lower long-term addiction prevalence and costs, while reducing q (through better healthcare for addicts) will keep addicts alive longer, potentially increasing $C(t)$ unless coupled with effective treatment to move them into recovery. A full multi-parameter sensitivity analysis could quantify these effects, but qualitatively our model suggests that the most influential parameters on total cost are those directly affecting the number of active addicts (such as β, τ, ν and the unit cost parameters c_1, c_2.

To solidify these insights, we conducted numerical simulations of the model, illustrating how $C(t)$ changes over time under variations in specific parameters. We used baseline parameter values estimated for the Muguka context (see Table 2)

The numerical simulations were performed using the fourth-order Runge-Kutta method implemented through MATLAB's ODE45 solver over a period of 100 years. The simulations investigate the sensitivity of the Total Economic Burden $E(t)$, which incorporates direct addiction costs, treatment expenditures, productivity losses, and social welfare costs. For each experiment, a single parameter was varied while all remaining parameters were maintained at their baseline values.

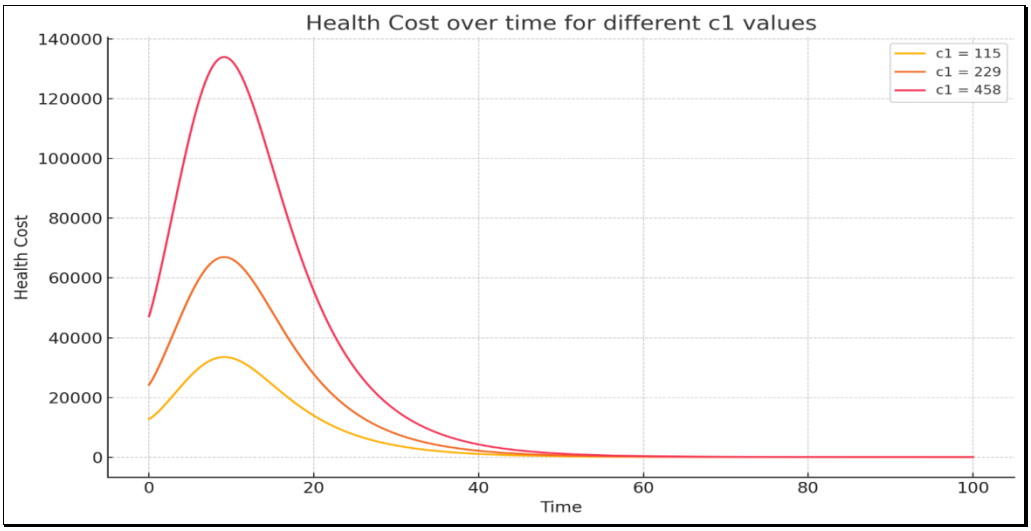

Figure 2: Total economic burden E(t) for different values of the direct addiction cost coefficient c_1

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The numerical simulations were performed using the fourth-order Runge-Kutta method implemented in MATLAB over a simulation period of 100 times units. Baseline parameter values were obtained from literature estimates and model calibration using available Muguka-related demographic and health information. The figure illustrates the sensitivity of the total economic burden $E(t)$ to variations in the direct addiction cost coefficient c_1 while all other parameters remain fixed at their baseline values.

An increase in c_1 results in a proportional increase in the total economic burden throughout the simulation period because each addicted individual contributes more directly to the overall cost. Conversely, reducing c_1 lowers the magnitude of the economic burden without altering the underlying addiction dynamics. The timing and shape of the addiction trajectory remain unchanged because c_1 affects only the economic evaluation and does not influence transitions between epidemiological compartments.

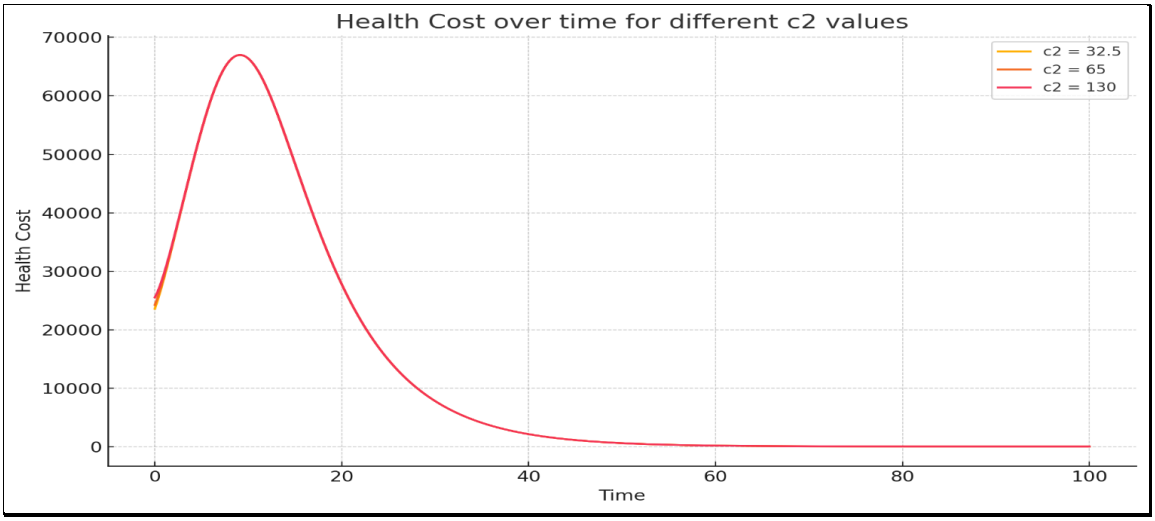

Figure 3: Total economic burden $E(t)$ for different values of the treatment cost coefficient c_2

The numerical simulations were performed using the fourth-order Runge-Kutta method implemented in MATLAB over a simulation period of 100 times units. Baseline parameter values were obtained from literature estimates and calibrated model parameters. The figure shows the effect of varying the treatment cost coefficient c_2 on the total economic burden $E(t)$.

The results indicate that changes in c_2 produce relatively small variations in the total economic burden compared with changes in c_1. This behaviour suggests that the major proportion of the economic burden is attributable to active addiction rather than treatment expenditure under the baseline parameter settings. The results further indicate that treatment costs become increasingly important only when treatment uptake and rehabilitation participation increase substantially.

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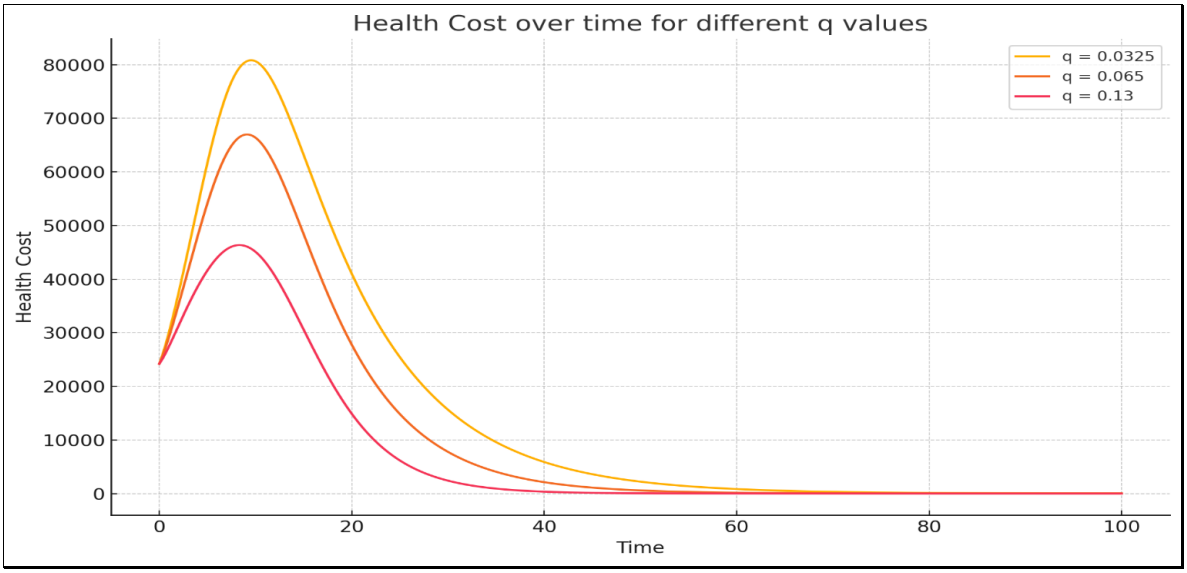

Figure 4: Total economic burden E(t) for different values of the Muguka-related complication rate q

The figure illustrates the effect of varying the Muguka-related complication rate q while maintaining all other parameters at baseline values. A lower value of q prolongs the duration of addiction within the population, resulting in higher cumulative economic losses and a delayed peak in E(t). Higher values of q reduce the duration of addiction but represent increased addiction-related morbidity and mortality. The results highlight the public health consequences associated with severe addiction complications.

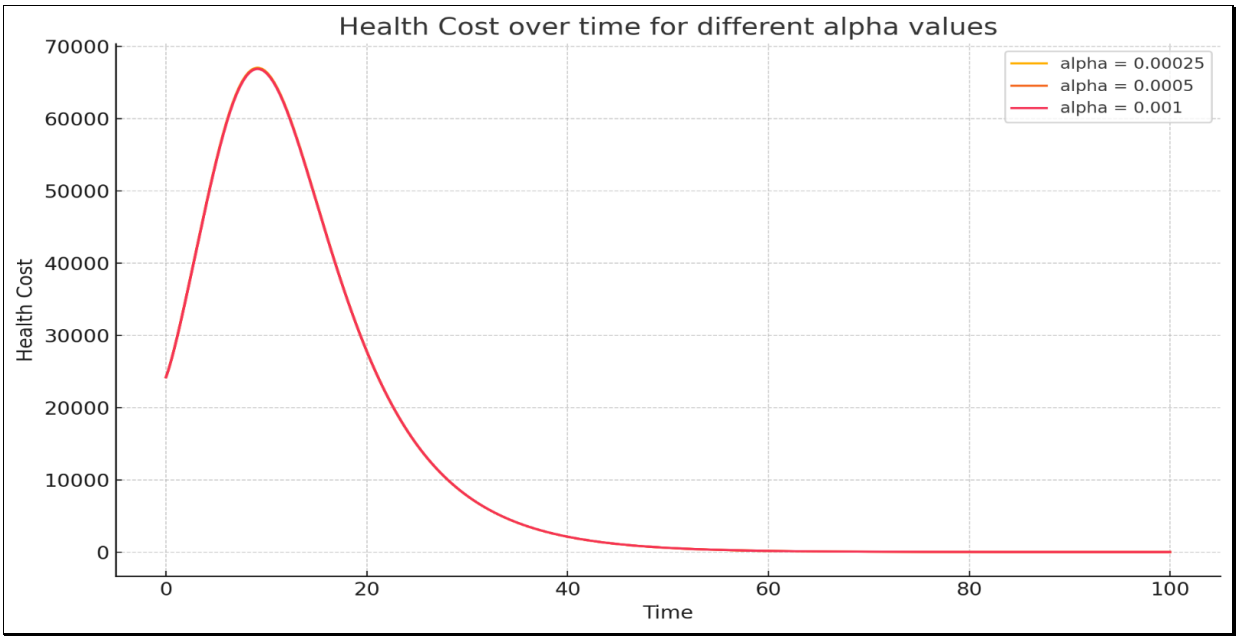

Figure 5: Total economic burden E(t) for different values of the treatment-seeking rate α

The figure examines the sensitivity of E(t) to changes in the treatment-seeking rate α. Under the baseline scenario, variations in α produce only modest changes in the economic burden because treatment participation remains relatively low. The findings suggest that increasing treatment uptake alone may not substantially reduce addiction prevalence unless accompanied by improved treatment effectiveness and reduced relapse rates.

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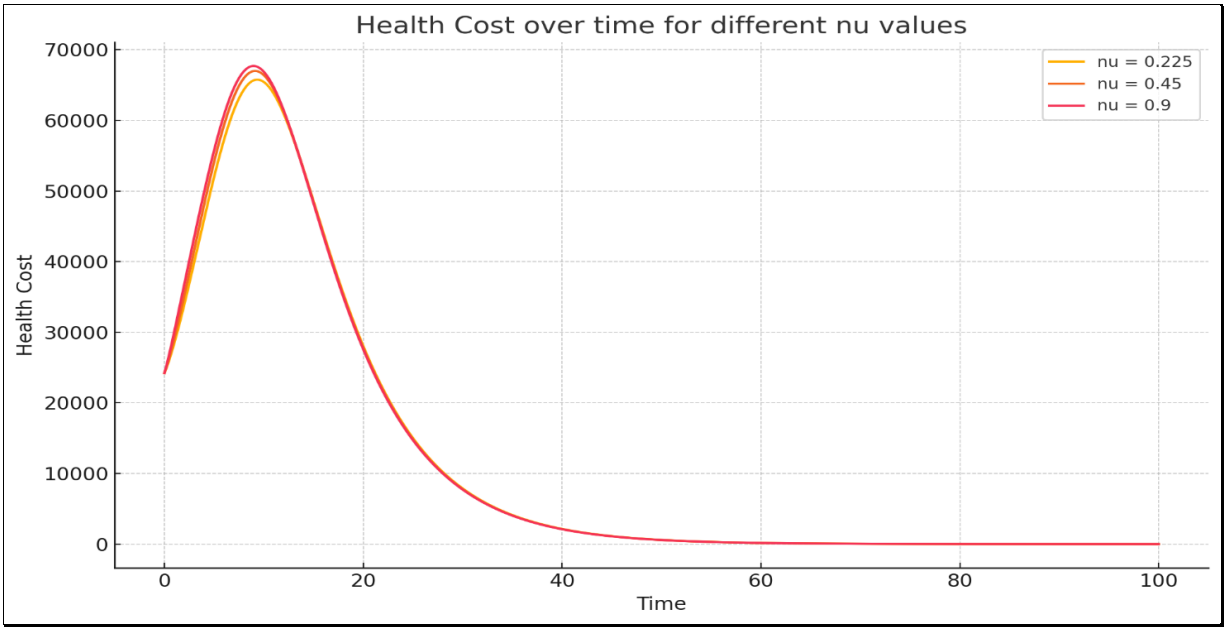

Figure 6: Total economic burden E(t) for different values of the relapse rate ν.

The figure illustrates the effect of relapse on the long-term economic burden of Muguka addiction. Higher relapse rates increase the number of individuals returning to active addiction, thereby sustaining higher levels of economic loss over time. Lower relapse rates facilitate long-term recovery and reduce the cumulative societal burden. These results emphasize the importance of post-treatment support programmes and long-term rehabilitation strategies in minimizing recurring addiction-related costs.

These simulation results align with the earlier sensitivity analysis and reinforce key insights: preventing the spread of addiction (lowering β) and improving recovery (increasing τ or equivalently reducing ν are crucial for reducing the magnitude and duration of the Muguka addiction burden. Direct costs (c_1, c_2) scale the economic impact but do not change the underlying epidemic trajectory, while the rate of seeking treatment (α) has limited effect unless it is made substantially large or paired with effective treatment (high τ).

DISCUSSION OF RESULTS

The inclusion of indirect economic losses provides a broader understanding of the true societal consequences of Muguka addiction. Previous health economic studies have shown that productivity losses and social consequences often exceed direct treatment expenditures in substance-use disorders [16,17]. Therefore, public policies targeting Muguka abuse should not only focus on reducing healthcare costs but also consider strategies aimed at improving employment opportunities, social reintegration, family support systems, and community-based prevention programmes.

The present study developed a deterministic compartmental model to investigate the dynamics of Muguka addiction and its associated economic burden. The integration of addiction progression, treatment interventions, recovery mechanisms, and economic costs provides a quantitative framework for understanding how different factors influence the long-term societal impact of Muguka use. The model results demonstrate that parameters associated with addiction initiation, treatment success, relapse, and health-related costs play significant roles in determining both the prevalence of addiction and the resulting economic burden.

Model Assumptions And Their Implications

The model is based on several assumptions that facilitate mathematical analysis but may simplify real-world Muguka addiction patterns. First, the model assumes homogeneous mixing within the population, meaning that every susceptible individual is assumed to have an equal probability of interaction with an addicted individual. In practice, Muguka consumption is influenced by age, peer groups, cultural practices, socioeconomic status,

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and geographical location. Therefore, addiction risk may vary considerably among different demographic groups.

Second, the model assumes that transition parameters, such as addiction initiation, treatment-seeking, recovery, and relapse rates, remain constant over time. In reality, these rates may change due to public awareness campaigns, government regulations, economic conditions, accessibility of rehabilitation services, and changes in social attitudes towards Muguka use. Introducing time-dependent parameters would provide a more realistic representation of the changing dynamics of addiction.

Third, the model does not explicitly incorporate age structure, gender differences, or social network effects. Previous studies have shown that substance-use behaviour is strongly influenced by demographic characteristics and social interactions. An age-structured or network-based modelling approach may therefore provide more detailed insights into vulnerable populations and targeted intervention strategies.

Limitations Related to Data Availability and Parameter Estimation

A major limitation of the study is the scarcity of comprehensive longitudinal data specifically related to Muguka addiction in Kenya. As a result, some model parameters were obtained from studies involving khat and other comparable substance-use disorders, while others were calibrated using available regional information. Although this approach is commonly employed in emerging public health modelling studies, it introduces uncertainty in the quantitative predictions of the model.

The model validation process was primarily based on qualitative agreement between simulated trends and available observations from Muguka-affected regions. The absence of extensive epidemiological records and detailed healthcare expenditure databases limited the application of advanced statistical validation methods. Future studies should utilize large-scale surveys, hospital records, rehabilitation centre data, and national health databases to enable more accurate parameter estimation and rigorous model validation.

Implications For Public Health Policy

Despite these limitations, the model provides useful policy insights. The results suggest that preventive interventions aimed at reducing the initiation of Muguka use can substantially lower future addiction prevalence and associated economic losses. Improving access to effective treatment programmes and reducing relapse rates are also essential for achieving sustainable reductions in the societal burden of Muguka addiction.

The expanded economic analysis further demonstrates that the burden of Muguka addiction extends beyond healthcare expenditure to include productivity losses, family economic stress, reduced workforce participation, and broader social costs. Therefore, policy responses should adopt an integrated approach involving public health authorities, educational institutions, community organizations, rehabilitation providers, and policymakers.

Future Research Directions

Future research should extend the current deterministic framework by incorporating stochastic effects, age-structured populations, and spatial heterogeneity to account for regional differences in Muguka cultivation and consumption patterns. The integration of optimal control theory may also help determine cost-effective combinations of prevention, treatment, and rehabilitation strategies that minimize both addiction prevalence and economic burden.

Additionally, future studies should focus on collecting detailed Muguka-specific epidemiological and economic data from affected regions in Kenya to improve model calibration, uncertainty analysis, and predictive accuracy.

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CONCLUSIONS

In this study, we developed a mathematical model for Muguka addiction that integrates epidemiological dynamics with health cost analysis. The model captures the transitions of individuals through susceptibility, addiction, treatment, and recovery, while explicitly accounting for the economic costs associated with addiction and intervention efforts. Our analysis yielded several important findings. First, the model's basic reproduction number R_0 provides a threshold criterion for the persistence of Muguka use: if $R_0 < 1$, the addiction will eventually die out, whereas if $R_0 > 1$, Muguka addiction can invade the population and reach an endemic equilibrium. For plausible parameter estimates in Kenya, R_0 is well above 1, indicating that Muguka use has the potential to remain endemic without effective interventions. Second, the Muguka-free equilibrium was shown to be locally stable when $R_0 < 1$, and conversely, when $R_0 > 1$, a unique endemic equilibrium exists and is stable, meaning the system will gravitate toward a persistent level of addiction in the long run. These theoretical results highlight the need for reducing transmission potential (e.g., through awareness and prevention) to push R_0 below unity if eradication is desired.

From a socio-economic perspective, the incorporation of the total economic burden function $E(t) = c_1 A(t) + c_2 T(t) + c_P A(t) + c_S R(t)$ allowed us to estimate the economic burden of Muguka addiction over time. The model simulations indicate that, even with moderate parameter values, the costs can escalate rapidly as addiction spreads, reflecting lost productivity and increased healthcare and enforcement expenses. The cost tends to peak when the number of active addicts is highest and then declines as either interventions take effect or a significant portion of the population succumbs to addiction and related removals. For example, using notional cost values (per addict and per patient) appropriate to the Kenyan context, our simulations showed a potential cost peak on the order of tens of thousands of cost units (which could correspond to US dollars or Kenyan shillings in the appropriate scale) within months of an uncontrolled spread. Such costs underscore the urgency for public health action.

Based on our model findings, we can offer several policy insights and optimal control considerations for mitigating the impact of Muguka addiction:

Prevention Programs: Curbing the initiation of Muguka use is crucial. Public awareness campaigns, restrictions on Muguka access (especially for youth), and community-based interventions should aim to reduce the effective contact rate β. Lowering β directly reduces R_0 and prevents new addictions, providing the most cost-effective long-term control of the problem. Our model suggests that prevention has a multiplicative benefit by avoiding both the health harms and the economic costs associated with each averted case of addiction.

Enhanced Treatment and Aftercare: Strengthening the treatment infrastructure (increasing α) and improving treatment efficacy (increasing τ and reducing relapse ν are vital. This includes expanding access to rehabilitation centers, improving the quality of counseling and medical management for addiction, and providing support systems for those in recovery to prevent relapses. Efficient treatment drives down the number of active addicts (cutting $c_1 A$ costs) and ensures that recovering individuals do not revert to costly addiction cycles. While our model showed that simply increasing treatment uptake α without high success can have limited immediate effects, combining it with high success rates and low relapse yields substantial reductions in long-term costs.

Resource Allocation: Policymakers need to balance investments between preventive and treatment measures. In our cost formulation, c_1 and c_2 represent spending on prevention vs. treatment per individual. A balanced approach is necessary for cost-effectiveness [14]. For instance, if c_2 (treatment cost) is very high relative to c_1, it may indicate that resources are heavily skewed towards treating existing addicts, whereas increasing funding for prevention could lower the number of new addicts and reduce future costs. On the other hand, under-investment in treatment (c_2 too low) could lead to persistently high addiction prevalence and societal costs. An optimal control strategy would involve determining the mix of prevention and treatment investment that minimizes the total cost $C(t)$ over a multi-year horizon.

In conclusion, the deterministic model presented here provides a framework for understanding and quantifying the complex dynamics of Muguka addiction and its economic impact. The integration of health cost analysis into the addiction model is a novel step that reveals how different intervention strategies might "pay off" in economic terms. While the model is a simplification of reality, it offers valuable qualitative guidance: aggressive prevention to reduce initiation, combined with effective treatment to shorten addiction duration, is likely to yield the greatest benefit both in health and economic outcomes. Future work could extend this model by incorporating additional complexities such as stochastic effects, age structure, or more detailed economic feedback. Nonetheless, even in its current form, the model is a useful tool for policymakers. It highlights the importance of curbing the Muguka menace not just for the health and social well-being of the community, but also to avert significant economic losses associated with addiction.

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