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MODELING THE EFFECTS OF TREATMENT ON ALCOHOL ABUSE IN KENYA INCORPORATING MASS MEDIA CAMPAIGN

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Abstract. The harmful use of alcohol causes a large burden with respect to diseases, social and economic problems to the society. Alcoholism is a serious problem in Kenya today. Many adults are addicted to alcohol. Mass media campaigns against alcohol acts as sources of information to halt alcohol abuse and its potential harmful effects. In this research we developed a deterministic models for alcohol abuse driven by the light and heavy drinkers taking into consideration the influence of pre -exposure to mass media campaigns. A model is developed with perfect pre-exposure campaigns. The model is analyzed through the determination of the model's steady states and it's respective stabilities analysis in terms of the alcohol abuse reproduction numbers R_0 . The analysis shown that alcohol free equilibrium (AFE) was locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. Numerical simulations were carried out o determine where the campaigns should be targeted for effective control of the abuse. The results from the simulations illustrated that increase in the rate of treatment reduces the number of alcohol addicts in the community. The model was validated using data from rehabilitation centers in Kenya. The implication of the results to policy makers is that alcohol treatment should be emphasized.

Keywords: alcohol abuse; reproduction number; alcohol free equilibrium; endemic equilibrium; local and global stabilities of equilibrium points; sensitivity analysis; numerical simulation.

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1. INTRODUCTION

According to WHO 2014, alcohol is a drug which is addictive and has been used by many people for centuries[1]. When alcohol is abused it causes harmful effects to the body and also causes social and economic problems in the society. Alcohol affects the body depending with amount of alcohol consumed, how often one drinks and sometimes the type of alcohol consumed. If taken lightly it acts as a stimulant and when consumed in large quantities, it can cause depression effects. The overdose causes insensitivity to pain, vomiting, seizures, unconsciousness and even death. Alcohol is absorbed into the body through the blood vessels which are in the walls of the stomach and small intestine. Immediately after drinking alcohol, it moves to the brain from the stomach where the the action of the nerve cells is slowed after the brain is affected. Alcohol absorbed in the stomach is approximately 20 per cent and the other remaining is absorbed through the small intestines. Alcohol moves to the liver through the blood stream and it cleans alcohol from the blood through a process called metabolism, whereby it is converted to substances that are not toxic. When alcohol is consumed in large amounts, the liver can not clean it all, thus the excess is left in the body. This excess alcohol circulates in the body and affects the body negatively. Thus the amount consumed directly affect the body of the consumer [3].

In Kenya, among all other abused substances, alcohol is leading. The most abused type of alcohol is the traditional brew because it is cheap and easily available. The next abused types are wine and spirit [2]. When young adults are addicted to alcohol, the country has no future manpower to develop the nation. Researchers like [4]and [5] point out that alcohol and other abused substances may drive the irresponsible behaviour among the youth [6].

Mass media is any means of transmission of information so as to reach as many people as possible. Young people mostly learn about alcohol from television, radio, film and popular music which acts as the main influence of the drinking problems according to [7]. According to [8], youth who saw more alcohol advertisements on average drank more (each additional advertisement seen increased the number of drinks consumed by one percent). Social media is very important in peoples lives because it affects the way they think and what they do. Social media is full of advertisement of alcohol and other drugs according to [9]. Research carried

out by CASA Columbia at Columbia University in 2011, shows that American teens who uses social media more at any time are more likely to smoke, drink alcohol and abuse drugs.

There is evidence that alcohol use spread like an infectious disease according to [10] and [11] hence can easily be formulated to a mathematical model. So far, few mathematical studies have been undertaken on the influence of mass media campaign on alcohol abuse. Therefore, this study intends to develop and analyse a mathematical model to look at the alcohol abuse with the impact of the intervention (rehabilitation). In Kenya, no mathematical model on effect of treatment of alcohol abuse has been developed and this paper is primarily designed to fill the gap.

Researchers like [12], [13], [14] and [15] have studied alcohol models including treatment or recovery. Other researchers [11], [16] and [17], studied the effect of mass media campaign on alcohol abuse. Hai-Feng Huo and Yan-Yan Wang (2016), developed a model which involved positive and negative role of Twitter on alcoholism. Our model include all forms of mass media not only twitter as done by [18].

2. MODEL ANALYSIS

2.1. Model Description. Our study uses seven compartments, six human population based compartments and one media compartment. The population based compartments are: S - Susceptible who have never used alcohol in their life, S_a - individuals exposed to media campaign and have never used alcohol, L - Light drinkers who drink two to three drinks one or two times a week, H - Heavy drinkers who are dependent on alcohol, T - individuals under treatment or in the rehabilitation centers and Q - individuals who have stopped drinking permanently. The media compartment M - is the density of media campaigns. Individuals are initiated to alcohol drinking due to contact with the light drinkers at a rate of λ where λ is given by $\beta(L+\eta_1H)$. The parameters and their descriptions are given in Table 2. The assumptions of the model include, exposure to media campaign before initiation to alcohol, which implies that only the susceptible are influenced by mass media campaigns, there is homogeneous mixing of the population in Kenya and individuals become alcoholic after contact with individual in the light drinking class and heavy drinking class. Alcohol becomes a problem when the individuals move to the heavy drinking class but there is no problem when they are in the light drinking class. For our

model, when individuals are exposed to mass media campaign they do get into drinking, they are exposed and get back to the susceptible class. Figure 1 represent flow of the model where the arrows represent the movement of individuals from one class to another.

The equations of the model are:

$$(1) \quad \left\{ \begin{aligned} \frac{dS}{dt} &= \Lambda + \omega S_a - \beta_m SM - (\lambda + \mu)S, \\ \frac{dS_a}{dt} &= \beta_m SM - (\mu + \omega)S_a, \\ \frac{dL}{dt} &= \lambda S - (\mu + \alpha_1 + \alpha_2)L, \\ \frac{dH}{dt} &= \alpha_1 L + \tau_2 T - (\sigma_1 + \mu + \sigma_2 + \delta)H, \\ \frac{dT}{dt} &= \sigma_1 H - (\mu + \tau_2 + \tau_1)T, \\ \frac{dQ}{dt} &= \tau_1 T + \sigma_2 H + \alpha_2 L - \mu Q, \\ \frac{dM}{dt} &= \theta_2 H - (\theta_1 + \rho)M. \end{aligned} \right.$$

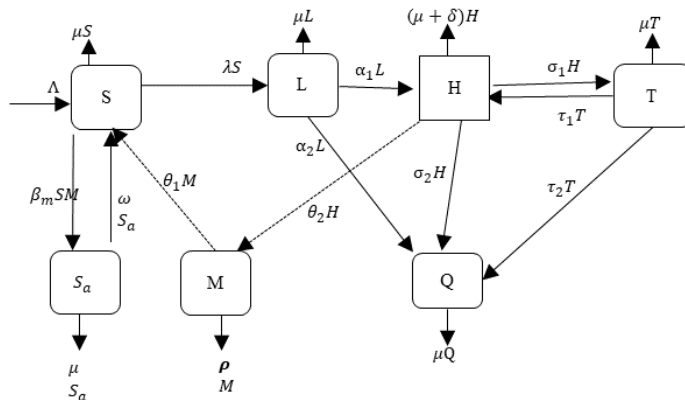


FIGURE 1. Flow chart of effective mass media campaign.

2.2. Invariant Region. To investigate the boundedness of the model or the region where the solutions of the model are feasible, we first add all the human compartments to get the total population N . Where $N = S + S_a + L + H + T + Q$. Taking the time derivatives of our total

population along the solution path gives:

$$(2) \quad \frac{dN}{dt} = \Lambda - \mu N - \delta H.$$

In the absence of mortality rate due to alcohol, equation (2) reduces to

$$(3) \quad \frac{dN}{dt} \leq \Lambda - \mu N.$$

After integrating the differential equation (3) with respect to time and as t tends to infinity, the limit of $N(t)$ becomes;

$$(4) \quad \lim_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}.$$

Thus $0 < N(t) \leq \frac{\Lambda}{\mu}$. We conclude that the feasible solutions set of the system equation enters and remains in the region Ω for all future time, where the region Ω is given by:

$$(5) \quad \Omega = \left\{ (S, S_a, L, H, T, Q) \in \mathbb{R}_+^6 \mid 0 < N(t) \leq \frac{\Lambda}{\mu} \right\}.$$

Therefore the model is well posed and we can study the dynamics of the model in Ω .

2.3. Positivity of the Model. The positivity of the model is calculated by first assuming that the initial condition are: $S(0) > 0, S_a(0) > 0, L(0) > 0, H(0) > 0, T(0) > 0, Q(0) > 0, M(0) > 0$. Using comparison theory and letting $k_1 = \alpha_1 + \alpha_2 + \mu, k_2 = \sigma_1 + \sigma_2 + \mu + \delta, k_3 = \tau_1 + \tau_2 + \mu$, we solve equation (1) to get;

$$(6) \quad \frac{dM}{dt} = \theta_2 M - (\theta_1 + \rho)M \geq -(\theta_1 + \rho)M, \frac{dM}{dt} \geq -(\theta_1 + \rho)M.$$

After integration with respect to time t and substituting $t=0$ we get

$$(7) \quad M(t) \geq M(0)e^{-(\theta_1 + \rho)t} > 0.$$

We do the same to all other equations to get

$$(8) \quad \begin{cases} \frac{dQ}{dt} \geq -\mu Q, Q(t) \geq Q(0)e^{-\mu t} > 0 \\ \frac{dT}{dt} \geq -k_3 T, T(t) \geq T(0)e^{-k_3 t} > 0 \\ \frac{dH}{dt} \geq -k_2 H, H(t) \geq H(0)e^{-k_2 t} > 0 \\ \frac{dL}{dt} \geq -k_1 L, L(t) \geq L(0)e^{-k_1 t} > 0 \\ \frac{dS_a}{dt} \geq -(\mu + \omega) S_a, S_a(t) \geq S_a(0)e^{-(\mu + \omega)t} > 0 \\ \frac{dS}{dt} \geq -(\lambda_1 + \mu) S, S(t) \geq S(0)e^{\int -(\lambda_1 + \mu) dt} > 0 \end{cases}$$

Equation (7) and (8) show that S, S_a, L, H, T, Q and M are always positive for all time t .

2.4. Alcohol Free Equilibrium(AFE) and alcohol abuse Reproduction Number. The AFE of the system (1) is obtained by setting all alcohol users classes and the class exposed to media campaigns to zero. This means $L = H = T = Q = 0$ and

$$(9) \quad \begin{cases} \Lambda + \omega S_a - \beta_m SM - \mu S = 0 \\ \beta_m SM - \omega S_a - \mu S_a = 0 \end{cases}$$

Adding equations (9) we get

$$\Lambda - \mu(S + S_a) = 0$$

We assume at AFE, $S = S_a$, hence

$$(10) \quad S = \frac{\Lambda}{2\mu}, S_a = \frac{\Lambda}{2\mu}$$

The AFE of the model is given by:

$$(11) \quad E^0 = \left\{ S^0, S_a^0, L^0, H^0, T^0, Q^0 \right\} = \left\{ \frac{\Lambda}{2\mu}, \frac{\Lambda}{2\mu}, 0, 0, 0, 0 \right\}$$

The point E^0 is the point at which there is no alcohol in the population. The total population at this point E^0 is equal to the susceptible population plus susceptible individuals exposed to media campaigns.

We use the next generation matrix method as used by [19], to determine the alcohol abuse reproduction number R_0 , of the model. Using the notation F to represent the new infection and V to represent transfer of infection in our model we get:

$$F = \begin{pmatrix} \frac{\Lambda\beta}{2\mu} & \frac{\Lambda\beta\eta_1}{2\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} k_1 & 0 & 0 \\ -\alpha_1 & k_2 & -\tau_2 \\ 0 & -\sigma_1 & k_3 \end{pmatrix}$$

Where $k_1 = \alpha_1 + \alpha_2 + \mu$, $k_2 = \sigma_1 + \sigma_2 + \mu + \delta$, $k_3 = \tau_1 + \tau_2 + \mu$. The reproduction of the model is the largest eigenvalue of the matrix FV^{-1} , where V^{-1} is the inverse of V and Matrix FV^{-1} is given by

$$F.V^{-1} = \begin{pmatrix} \frac{\Lambda\beta(k_3(k_2 + \alpha_1\eta_1) - \sigma_1\tau_2)}{2\mu k_1(k_2k_3 - \sigma_1\tau_2)} & \frac{\Lambda\beta\eta_1}{2\mu k_2k_3 - \mu\sigma_1\tau_2} & \frac{\Lambda\beta\eta_1\tau_2}{2\mu k_2k_3 - \mu\sigma_1\tau_2} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

The reproduction number is given by :

$$(12) \quad R_0 = \frac{\beta\Lambda(k_3(k_2 + \eta_1\alpha_1) - \sigma_1\tau_2)}{2\mu k_1(k_2k_3 - \sigma_1\tau_2)}$$

Our model has two steady states, the alcohol free equilibrium and endemic equilibrium points. We study each at a time.

2.5. Local Stability of AFE.

Theorem 1. *The AFE point (E^0) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. To prove the theorem we obtain the Jacobian matrix of the system (1) at the AFE E^0 .

$$J_{E^0} = \begin{pmatrix} -\mu & \omega & \frac{-\Lambda\beta}{2\mu} & \frac{-\Lambda\beta\eta_1}{2\mu} & 0 \\ 0 & -(\mu + \omega) & 0 & 0 & 0 \\ 0 & 0 & \frac{\Lambda\beta}{2\mu} - k_1 & \frac{\Lambda\beta\eta_1}{2\mu} & 0 \\ 0 & 0 & \alpha_1 & -k_2 & \tau_2 \\ 0 & 0 & 0 & \sigma_1 & -k_3 \end{pmatrix}$$

The characteristic polynomial of the matrix J_{E^0} is

$$(13) \quad (-\lambda_1 - \mu)(-\lambda_1 - (\mu + \omega))(\lambda_1^3 + P_1\lambda_1^2 + P_2\lambda_1 + P_3) = 0$$

where

$$P_1 = k_1 + k_2 + k_3 - \frac{\beta\Lambda}{2\mu}$$

$$P_2 = k_1k_2 + k_1k_3 + k_2k_3 + k_2\beta\frac{\Lambda}{2\mu} + k_3\beta\frac{\Lambda}{2\mu} + \alpha_1\beta\eta_1\frac{\Lambda}{2\mu} - \sigma_1\tau_2$$

$$P_3 = k_1k_2k_3 - k_2k_3\beta\frac{\Lambda}{2\mu} - k_3\alpha_1\eta_1\beta\frac{\Lambda}{2\mu} - k_1\tau_2\sigma_1 + \sigma_1\beta_1\tau_2\frac{\Lambda}{2\mu}$$

Simplifying P_3 and writing it in terms of R_0 we get

$$P_3 = k_1(k_2k_3 - \sigma_1\tau_2) + \frac{\Lambda\beta}{2\mu}(-k_3(k_2 + \alpha_1\eta_1) + \sigma_1\tau_2)$$

$$P_3 = k_1(k_2k_3 - \sigma_1\tau_2) - \frac{\Lambda\beta k_1}{2\mu k_1(k_2k_3 - \sigma_1\tau_2)}(k_3(k_2 + \alpha_1\eta_1) - \sigma_1\tau_2)(k_2k_3 - \sigma_1\tau_2)$$

$$P_3 = k_1(k_2k_3 - \sigma_1\tau_2)\left(1 - \frac{\Lambda\beta}{2\mu k_1(k_2k_3 - \sigma_1\tau_2)}(k_3(k_2 + \alpha_1\eta_1) - \sigma_1\tau_2)\right)$$

We know that

$$R_0 = \frac{\Lambda\beta}{2\mu k_1(k_2k_3 - \sigma_1\tau_2)}(k_3(k_2 + \alpha_1\eta_1) - \sigma_1\tau_2),$$

$$\text{So that } P_3 = k_1(k_2k_3 - \sigma_1\tau_2)(1 - R_0)$$

From equation (13) we see that

$$-\lambda_1 - \mu = 0, -\lambda_1 - (\mu + \omega) = 0, (\lambda_1^3 + P_1\lambda_1^2 + P_2\lambda_1 + P_3) = 0, \text{ so that } \lambda_{11} = -\mu < 0 \text{ and } \lambda_{12} = -(\mu + \omega) < 0$$

For the expression

$$(14) \quad \lambda_1^3 + P_1\lambda_1^2 + P_2\lambda_1 + P_3 = 0$$

We apply Routh - Hurwitz Criteria where (14) should have negative real root if and only if $P_1 > 0, P_2 > 0, P_3 > 0$ and $P_1P_2 > P_3$. In our case P_1 is positive because it is the sum of positive variables. $P_2 > 0$ because only $\sigma_1\tau_2$ is negative and $k_2 = \sigma_1 + \sigma_2 + \delta + \mu, k_3 = \tau_1 + \tau_2 + \mu$. The product of $P_1P_2 > 0$ since both are positive and if we choose $R_0 < 1$, then $P_1P_2 > P_3$. For P_3 to be positive, $1 - R_0$ must be positive since $k_2k_3 > \sigma_1\tau_2$ as explained above. This means that $R_0 < 1$ hence AFE point E^0 is locally asymptotically stable.

2.6. Global Stability of AFE. Global stability of AFE of the model is investigated using the theorem by [19].

Theorem 2. *The fixed point $\widetilde{U}_0 = (X^*, 0) = (\frac{\Lambda}{2\mu}, \frac{\Lambda}{2\mu}, 0, 0, 0, 0)$ is globally asymptotically stable, if $R_0 < 1$ is locally asymptotically stable and assumption (H1) and (H2) are satisfied.*

Proof. The equation (1) is written as $\frac{dX}{dt} = F(X, Z), \frac{dZ}{dt} = G(X, Z)$, where $X = (S)$ represents the alcohol free classes and $Z = (L, H, T)$ represents the alcohol drinking classes. $G(X, 0) = 0, \widetilde{U}_0 = (X^*, 0) = (\frac{\Lambda}{2\mu}, \frac{\Lambda}{2\mu}, 0, 0, 0, 0)$ denotes the AFE point of the model. The conditions (H1) and (H2) are

(H1) for $\frac{dX}{dt} = F(X, 0), X^0$ is globally asymptotically stable.

(H2), $G(X, Z) = AZ - \widetilde{G}(X, Z), \widetilde{G}(X, Z) \geq 0$ for $(X, Z) \in R_+^6$ where $A = D_Z G(X, 0)$ is an M-matrix (the off diagonal element of A are nonnegative) and R_+^6 is the region where the model makes biological sense.

In this case, $F(X, 0) = (\Lambda - 2\mu S), X = (S, S_a, Q)$ and $Z = (L, H, T)$.

$$A = \begin{pmatrix} \beta - k_1 & \beta\eta_1 & 0 \\ \alpha_1 & -k_2 & \tau_2 \\ 0 & \sigma_1 & -k_3 \end{pmatrix}$$

$$\begin{aligned}
 AZ &= \begin{pmatrix} (\beta - k_1)L + \beta\eta_1H \\ \alpha_1L - k_2H + \tau T \\ \sigma_1H - k_3T \end{pmatrix} \\
 G(X, Z) &= \begin{pmatrix} \lambda S - k_1L \\ \alpha_1L - k_2H + \tau_2T \\ \sigma_1H - k_3T \end{pmatrix} \\
 \tilde{G}(X, Z) &= AZ - G(X, Z) \\
 \tilde{G}(X, Z) &= \begin{pmatrix} \beta(L + \eta_1H)(1 - \frac{S}{N}) \\ 0 \\ 0 \end{pmatrix}
 \end{aligned}$$

Since $S \leq N$, $\frac{S}{N} \leq 1$, then $\tilde{G}(X, Z) \geq 0$. This implies that the second condition of (H2) is satisfied, hence $X^* = (\frac{\Lambda}{2\mu}, \frac{\Lambda}{2\mu}, 0)$ is globally asymptotically stable equilibrium of $\frac{dX}{dt} = F(X, 0)$ when $R_0 < 1$

2.7. Endemic Equilibrium Point (EEP). We find conditions for the existence of an equilibrium for which our model is endemic in the population. We solve equation (1) in terms of force of infection at steady state λ^* , where $\lambda^* = \beta(L^* + \eta_1H^*)$. Endemic equilibrium occurs when the alcohol persists in the community. Setting the right hand side of the model to zero and noting that $\lambda = \lambda^*$ at equilibrium gives

$$(15) \quad \begin{cases} S^* = \frac{\Lambda^*(\mu + \omega)}{(\lambda^* + \mu)(\mu + \omega) + M\mu\beta_m} \\ S_a^* = \frac{M\Lambda\beta_m}{(\lambda^* + \mu)(\mu + \omega) + M\mu\beta_m} \\ L^* = \frac{\lambda^*\Lambda(\mu + \omega)}{(\lambda^* + \mu)(\mu + \omega) + M\mu\beta_mk_1} \\ H^* = \frac{\lambda^*(\Lambda(\mu + \omega))k_3\alpha_1}{((\lambda^* + \mu)(\mu + \omega) + M\mu\beta_m)k_1(k_2k_3 - \sigma_1\tau_2)} \\ T^* = \frac{\lambda^*(\Lambda(\mu + \omega)\alpha_1\sigma_1)}{((\lambda^* + \mu)(\mu + \omega) + M\mu\beta_m)k_1(k_2k_3 - \sigma_1\tau_2)} \end{cases}$$

Substituting the above expression in the force of infection and solving we obtain two values of λ^*

$\lambda^* = 0$, which correspond to alcohol free equilibrium and

$$(16) \quad \lambda^* = \frac{-\mu(\mu + \omega + M\beta_m)}{\mu + \omega} + \frac{\beta\Lambda}{k_1} \left(1 + \frac{k_3\alpha_1\eta_1}{k_2k_3 - \alpha_1\tau_2}\right)$$

Which corresponds to endemic equilibrium point. We write equation (12) in terms of β to obtain

$$(17) \quad \beta = \frac{2\mu k_1 R_0 (k_2 k_3 - \sigma_1 \tau_2)}{\Lambda (k_2 k_3 + k_3 \alpha_1 \eta_1 - \sigma_1 \tau_2)}$$

Substituting β into equation of λ^* we get

$$(18) \quad \lambda^* = \mu \left(2R_0 - 1 + \frac{(\Lambda\beta_m M)}{(\mu + \omega)}\right)$$

Substituting the value of λ^* into equation (16) we obtain:

$$(19) \quad \begin{cases} S^* = \frac{\Lambda}{2\mu R_0} \\ S_a^* = \frac{M\beta_m \Lambda}{2R_0\mu(\mu + \omega)} \\ L^* = \frac{\Lambda(-\beta_m M + (\mu + \omega)(2R_0 - 1))}{2k_1 R_0 (\mu + \omega)} \\ H^* = \frac{k_3 \alpha_1 \Lambda (-M\beta_m + (\mu + \omega)(2R_0 - 1))}{k_1 2R_0 (\mu + \omega) (k_2 k_3 - \sigma_1 \tau_2)} \\ T^* = \frac{\alpha_1 \sigma_1 \Lambda (-M\beta_m + (\mu + \omega)(2R_0 - 1))}{2k_1 R_0 (\mu + \omega) (k_2 k_3 - \sigma_1 \tau_2)} \end{cases}$$

Endemic equilibrium exist and is positive if $R_0 > 1$.

2.8. Bifurcation of the Model. We use Centre Manifold Theory in [19] to investigate the nature of the bifurcation of the model. We let $S = x_1, S_a = x_2, L = x_3, H = x_4, T = x_5, Q = x_6$.

Then $N = x_1 + x_2 + x_3 + x_4 + x_5 + x_6$.

The model can be written as

$\frac{dN}{dt} = F(x)$ with $F = (f_1, f_2, f_3, f_4, f_5, f_6)$ and

$$(20) \quad \begin{cases} \frac{dx_1}{dt} = f_1 = \Lambda + \omega x_2 - \beta_m x_1 M - \beta(x_3 + \eta_1 x_4)x_1 - \mu x_1 \\ \frac{dx_2}{dt} = f_2 = \beta_m x_1 M - \mu x_2 - \omega x - 2 \\ \frac{dx_3}{dt} = f_3 = \beta(x_3 + \eta_1 x_4)x_1 - k_1 x_4 \\ \frac{dx_4}{dt} = f_4 = \alpha_1 x_3 + \tau_2 x_4 - k_2 x_4 \\ \frac{dx_5}{dt} = f_5 = \sigma_1 x_4 - k_3 x_5 \\ \frac{dx_6}{dt} = f_6 = \tau_1 x_5 + \sigma_2 x_4 + \alpha_2 x_3 - \mu x_6 \end{cases}$$

Where $k_1 = \alpha_1 + \alpha_2 + \mu, k_2 = \sigma_1 + \sigma_2 + \delta + \mu$ and $k_3 = \tau_1 + \tau_2 + \mu$. By choosing $\beta = \beta^*$ as the bifurcation parameter and investigating the case when $R_0 = 1$ gives

$$(21) \quad \beta = \frac{2\mu k_1(k_2 k_3 - \sigma_1 \tau_2)}{\Lambda(k_2 k_3 + k_2 \alpha_1 \eta_1 - \sigma_1 \tau_2)}$$

It can be shown that the Jacobian of system (1) at $\beta = \beta^*$ has a zero eigenvalue which is simple. To investigate the stability of the model we use the following model of Castillo -Chevez and Song (2004).

Theorem 3. Consider the following general systems of ordinary differential equation with parameter β

$$\frac{dx}{dt} = f(x, \beta), R^n \times R \rightarrow R \text{ and } f \in C^2(R^n \times R),$$

Where 0 is the equilibrium point of the system (that is, $f(x, \beta) \equiv 0$ for all β) and assume

A1: $A = D_x f(0, 0, 0, 0) = (\frac{df_i}{dx_j}(0, 0, 0, 0))$ is the linearization matrix of the system (1) around the equilibrium 0 with β evaluated at 0. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts: **A2:** Matrix a has a non negative right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue. let f_k be the k^{th} component of f and

$$a = \sum_{k,i,j=1}^n v_k w_i w_j \frac{d^2 f_k}{dx_i dx_j}(0, 0, 0, 0)$$

$$b = \sum_{k,i=1}^n (0, 0, 0, 0) v_k w_i \frac{d^2 f_k}{dx_i d\beta}(0, 0, 0, 0)$$

The local dynamics of the system around 0 is totally determined by the signs of a and b.

- i. $a > 0, b > 0$. When $\beta < 0$ with $|\beta| \ll 1, 0$ is locally asymptotically stable and there exist a positive unstable equilibrium: when $0 < \beta \ll 1.0$ is unstable and there exists a negative, locally asymptotically stable equilibrium:

- ii. $a < 0, b < 0$. When $\beta < 0$ with $\phi \ll 1$, 0 is unstable, when $0 < \beta \ll 1$, 0 is locally asymptotically stable equilibrium, and there exist a positive unstable equilibrium.
- iii. $a > 0, b < 0$. When $\beta < 0$ with $|\beta| \ll 1$, 0 is unstable, and there exist a locally asymptotically stable negative equilibrium: when $0 < \beta \ll 1$, 0 is stable, and a positive unstable equilibrium appears.
- iv. $a < 0, b > 0$. When β changes from negative to positive, 0 changes its stability from stable to unstable. Corresponding a negative unstable equilibrium becomes positive and locally asymptotically stable.

2.8.1. *Eigenvectors of $J_{(E^0)} = J_\beta$.* The Jacobian of the model at β denoted by J_β has a right eigenvector denoted by $w = (w_1, w_2, w_3, w_4, w_5, w_6)^T$ given by:

$$(22) \quad \begin{pmatrix} -\mu & \omega & -g_1 & -g_2 & 0 & 0 \\ 0 & -g_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & g_1 - k_1 & g_2 & 0 & 0 \\ 0 & 0 & \alpha_1 & -k_2 & \tau_2 & 0 \\ 0 & 0 & 0 & \sigma_1 & -k_3 & 0 \\ 0 & 0 & \alpha_2 & \sigma_2 & \tau_1 & -\mu \end{pmatrix} \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Where $g_1 = \frac{\beta\Lambda}{\mu}$, $g_2 = \frac{\beta\eta - 1\Lambda}{\mu}$, $g_3 = (\mu + \omega)$ Equation (22) can also be written as:

$$(23) \quad \begin{cases} -\mu w_1 + \omega w_2 - g_1 w_3 - g_2 w_4 = 0 \\ -g_3 w_2 = 0 \\ (g_1 - k_1) w_3 + g_2 w_4 = 0 \\ \alpha_1 w_3 - k_2 w_4 + \tau_2 w_5 = 0 \\ \sigma_1 w_4 - k_3 w_5 = 0 \\ \alpha_2 w_3 + \sigma_2 w_4 + \tau_1 w_5 - \mu w_6 = 0 \end{cases}$$

After solving equation (23), we get:

$$(24) \quad \begin{cases} w_1 = -\frac{(g_1 w_3 + g_2 w_4)}{\mu} < 0 \\ w_2 = 0 \\ w_3 = \frac{g_2 w_4}{g_1 - k_1} > 0 \\ w_4 = \frac{k_3 w_5}{\sigma_1} > 0 \\ w_5 = \frac{w_1 w_4}{k_3} < 0 \\ w_6 = \frac{\alpha_2 g_2 w_4}{(g_1 - k_1)\mu} + \frac{\sigma_3 k_3 w_5}{\sigma_1 \mu} + \frac{\tau_1 w_1 w_4}{k_3 \mu} < 0 \end{cases}$$

The Jacobian matrix has a left eigenvector denoted v given by $v = (v_1, v_2, v_3, v_4, v_5, v_6)^T$

$$(25) \quad \begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 \\ \omega & -g_3 & 0 & 0 & 0 & 0 \\ -g_1 & 0 & g_1 - k_1 & \alpha_1 & 0 & \alpha_2 \\ -g_2 & 0 & \alpha_1 & -k_2 & \sigma_1 & \sigma_2 \\ 0 & 0 & 0 & \tau_2 & -k_3 & \tau_1 \\ 0 & 0 & 0 & 0 & 0 & -\mu \end{pmatrix} \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \\ v_6 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Solving equation (25), we obtain:

$$(26) \quad \begin{cases} v_1 = v_2 = 0 \\ v_3 = \frac{\alpha_1 v_4}{g_1 - k_1} > 0 \\ v_4 = \frac{k_3 v_5}{\tau_2} > 0 \\ v_5 = \frac{\tau_2 v_4}{k_3} > 0 \\ v_6 = 0 \end{cases}$$

We find the sign of \mathbf{a} and \mathbf{b} as follows:

$v_k w_i w_j \frac{d^2 f_k}{dx_i dx_j}$ for $k = 3; i, j = 1, 3, 4$, gives;

$$(27) \quad \begin{cases} v_3 w_1 w_1 \frac{d^2 f_3}{dx_1 dx_1} = 0 \\ v_3 w_1 w_3 \frac{d^2 f_3}{dx_1 dx_3} = \beta v_3 w_1 w_3 < 0 \\ v_3 w_3 w_1 \frac{d^2 f_3}{dx_3 dx_1} = v_3 w_3 w_1 \beta < 0 \\ v_3 w_1 w_4 \frac{d^2 f_3}{dx_1 dx_4} = v_3 w_1 w_4 \beta \eta_1 < 0 \\ v_3 w_4 w_1 \frac{d^2 f_3}{dx_4 dx_1} = v_3 w_4 w_1 \beta \eta_1 < 0 \end{cases}$$

The sum of equation (26) and (27) is the value of \mathbf{a} given by:

$$(28) \quad \mathbf{a} = 2v_3 w_1 \beta (w_2 + w_4 \eta_1) < 0$$

$\mathbf{a} < 0$ because $w_1 < 0$ and $v_3, w_2, w_4 > 0$.

To find the value of \mathbf{b} as per theorem 3, we let $k = 3, i = 3, 4$. When $k = 1, 2, 4, 5, 6$ and $i = 1, 2, 5, 6$, the second derivative will be zero.

$$(29) \quad \begin{cases} v_3 w_3 \frac{df_3}{dx_3 d\beta^*} = \frac{\Lambda}{\mu} \\ v_3 w_4 \frac{df_3}{dx_4 d\beta^*} = \frac{\eta_1 \Lambda}{\mu} \end{cases}$$

From equation (29) the expression of \mathbf{b} is;

$$(30) \quad \mathbf{b} = v_3 \frac{\Lambda}{\mu} (w_3 + w_4 \eta_1) > 0.$$

Hence from theorem 3 item iv, β changes from negative to positive, 0 changes its stability from stable to unstable. Correspondingly a negative unstable equilibrium becomes positive and locally asymptotically stable.

2.9. Sensitivity Analysis of the Model. We carry out the sensitivity analysis of some parameters to identify which parameters have great impact on the reproduction number R_0 . We use partial derivatives with respect to the parameters. Considering the parameters $\sigma_1, \sigma_2, \alpha_2, \tau_1$ and τ_2 which represent the rate of treatment of alcohol, rate of quitting alcohol for the heavy

drinkers, rate of light drinkers quitting alcohol, rate of treated class quitting alcohol and rate of relapse respectively.

$$(31) \quad \begin{cases} \frac{\partial R_0}{\partial \sigma_1} = -\frac{\Lambda \beta \alpha_1 \eta_1 (\sigma_1 (\mu + \tau_1) k_3)}{2 \mu k_1 (\sigma_1 (\mu + \tau_1) + (\delta + \mu + \sigma_2) k_3)} < 0 \\ \frac{\partial R_0}{\partial \sigma_2} = -\frac{\Lambda \beta \alpha_1 \eta_1 k_3}{2 \mu k_1 (\sigma_1 (\mu + \tau_1) + (\delta + \mu + \sigma_2) k_3)^2} < 0 \\ \frac{\partial R_0}{\partial \alpha_2} = -\frac{\Lambda \beta (-\sigma_1 \tau_2 + (\delta + \mu + \alpha_1 \eta_1 + \delta_1 + \sigma_1 + \sigma_2) k_3)}{2 \mu k_1^2 (\sigma_1 (\mu + \tau_1) + (\delta + \mu + \sigma_2) k_3)} < 0 \\ \frac{\partial R_0}{\partial \tau_1} = -\frac{\Lambda \alpha_1 \beta \eta_1 \tau_2 \sigma_1}{2 \mu k_1 (\sigma_1 (\mu + \tau_1) + (\delta + \mu + \sigma_2) k_3)^2} < 0 \\ \frac{\partial R_0}{\partial \tau_2} = \frac{\Lambda \alpha_1 \beta \eta_1 \sigma_1 (\mu + \tau_1)}{2 \mu k_1 (\sigma_1 (\mu + \tau_1) + (\delta + \mu + \sigma_2) k_3)^2} > 0 \end{cases}$$

The parameters with negative partial derivatives of the reproduction number (with respect to $\sigma_1, \sigma_2, \alpha_2$ and τ_1) means that if their values are increase, may greatly decrease alcohol abuse in the community, while other parameters are not changed. These parameters reduce the alcohol abuse reproduction number which implies that alcohol abuse is reduced in the community. The positive partial derivatives of the reproduction number (with respect to τ_2), show that increase in the value of τ_2 (relapse), increases the reproduction number hence increases the risk of alcohol addicts in the community.

Table 1 gives the sensitivity analysis of the model. We used $\beta_1 = 0.00000002, \Lambda = 1674000, \mu = 0.025, \alpha_1 = 0.031, \alpha_2 = 0.7, \sigma_1 = 0.004, \sigma_2 = 0.05, \tau_1 = 0.1, \tau_2 = 0.002, \delta = 0.2, \eta_1 = 0.001$

TABLE 1. Sensitivity Analysis Index

Symbol	Description	Sensitivity Index
σ_1	Treatment rate of heavy drinkers	-0.0157006
σ_2	Rate of heavy drinkers quitting	-0.0195371
α_1	Transfer rate of light drinkers to heavy drinkers	-1.17171
α_2	Rate of light drinkers quitting alcohol	-1.16853
τ_1	Rate of effective treatment	-0.00147973
τ_2	Rate of relapse back to heavy drinkers	0.0000155532

2.10. Numerical Simulation. To study the dynamics of system (1), we used MATLAB (ode45) software. Table 2, list the parameters used in simulation together with their source. The initial

population of S, S_a, L, H and Q are from [21]. We collected secondary data from rehabilitation centres in Kenya to identify the initial population of the treatment class. To estimate the initial conditions of the steady states, we used the idea that, Kenyan population is estimated to be approximately 52 million according to [21]. This is equivalent to $N = S + S_a + L + H + T + Q$. Alcohol prevalence is estimated to be 30 percent and 13.3 percent of this is estimated to be addicted to alcohol [2]. This translates to 15.6 million people in the classes $L + H + T + Q$ and about 2.028 million people addicted to alcohol. So the initial conditions used are: $S(0) = 7280000$, $S_a(0) = 29100000$, $L(0) = 11570000$, $H(0) = 2028000$, $T(0) = 20000$, $Q(0) = 2000000$.

TABLE 2. Parameters values and their Description

Parameter	Description	Value	Source
Λ	Recruitment rate	1674000	[21]
μ	Natural death rate	0.025	Assumed
β	Alcohol contact rate	0.00000002	[11]
β_m	Rate of dissemination of media awareness	0.00005	Assumed
α_1	Transfer rate of light drinkers	0.031	Assumed
α_2	Rate of quitting alcohol of the light drinkers	0.7	Assumed
σ_1	rate of joining rehabilitation	variable	Assumed
σ_2	Rate of heavy drinkers quitting the drink	0.05	Assumed
τ_1	Rate of effective treatment	0.1	Assumed
τ_2	Rate of relapse	0.2	Assumed
δ	Death rate due to alcohol abuse	0.2	Assumed
θ_1	Rate of awareness programs on S	0.0005	[11]
θ_2	Rate of awareness programs on H	0.0001	Assumed
ρ	Rate of depletion of media programs	0.06	[11]
ω	Rate of effective media campaign	0.0002	Assumed
η_1	Modification parameter	0.01	Assumed

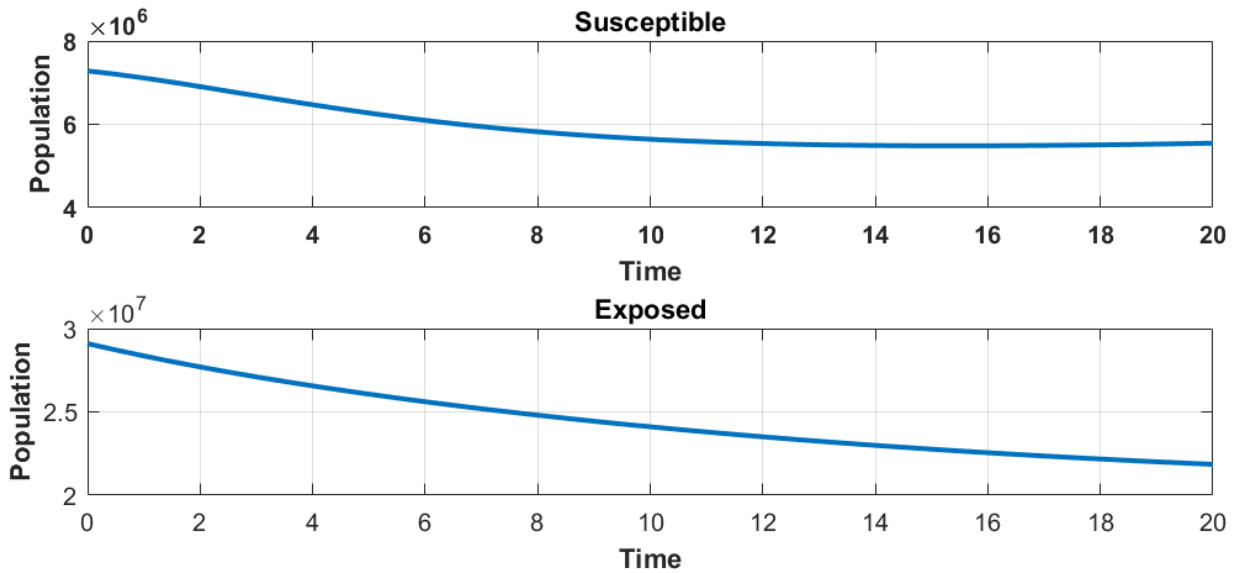


FIGURE 2. Non alcoholic classes for 20 years.

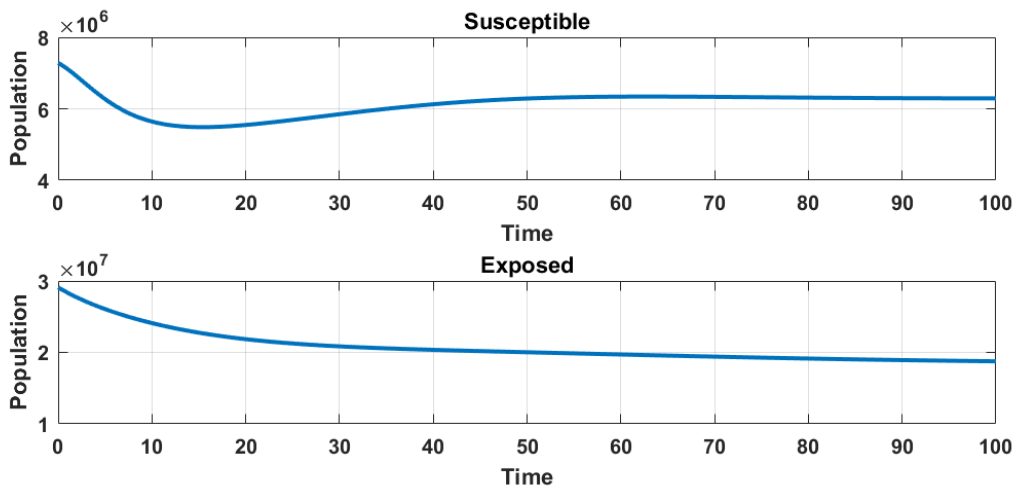


FIGURE 3. Non alcoholic classes for 100 years.

Figure 2 represents the rate of change in the classes that have never taken alcohol with respect to time. These are the susceptible class and the susceptible and exposed to media class. For the first twenty years the population in two classes decrease with time but thereafter they get to an equilibrium point as shown in Figure 3.

Figure 4 represent classes that have ever used alcohol in their lifetime These include the treated and the quitters who are not using alcohol. The number of light drinkers increases

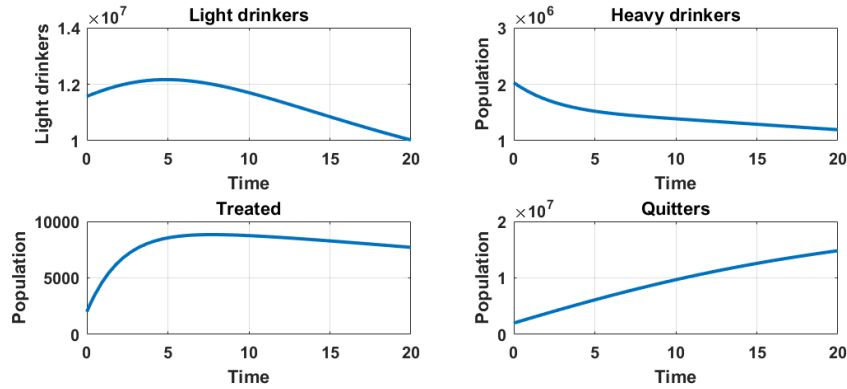


FIGURE 4. Alcohol Classes

but later decrease as they either get to treatment or quit alcohol. The heavy drinking class decrease due to the individuals being encouraged to go for treatment and others die due alcohol related complications. The people under treatment increase with time and eventually gets to an equilibrium point after some time.

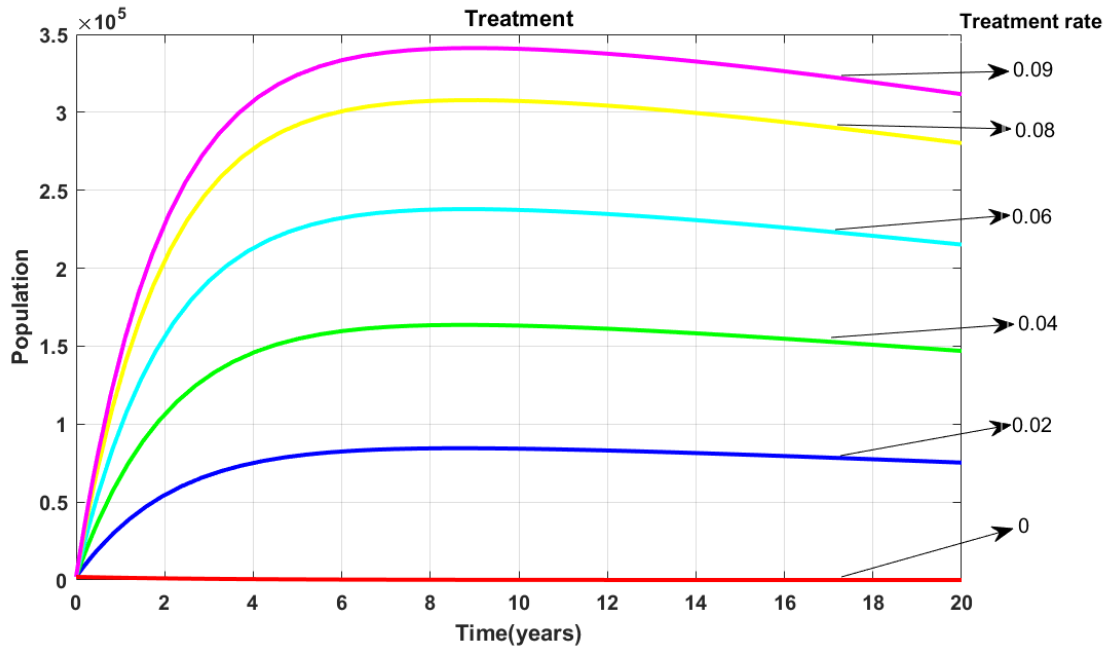


FIGURE 5. Alcohol treatment varied from $\sigma_1 = 0$ to $\sigma_1 = 0.09$.

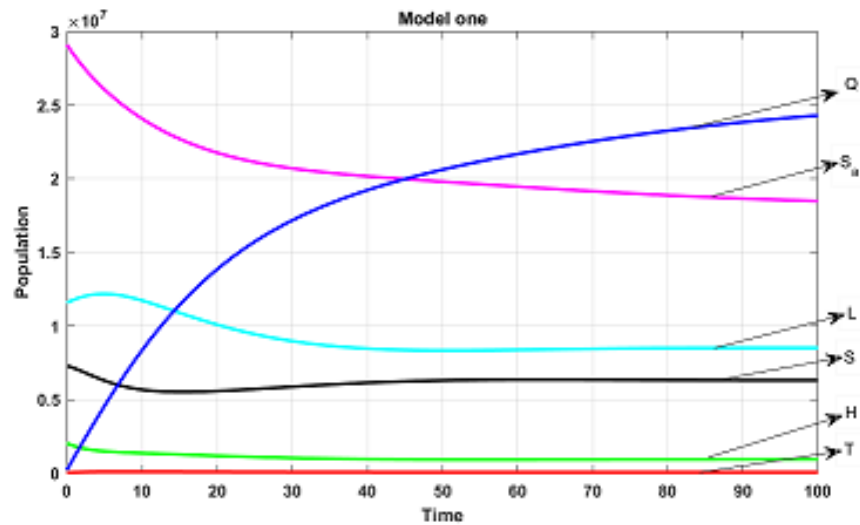


FIGURE 6. All population classes

In Figure 5, we varied the rate of treatment from 0 to 0.09. The red plot shows a horizontal line which implies there is no change of the treatment with respect to time. When the rate of treatment is increased, the number in the treatment class increase up to an equilibrium point. Figure 6 represent all population classes in our model, where the quitters increase with time due to increase in treatment and quitting from other classes. The other classes have slight increase except the exposed susceptible which decrease slightly with time.

2.11. Discussion and Conclusion. We developed a deterministic model of alcohol abuse incorporating treatment and mass media campaign against alcohol abuse. Figure 1 shows a flow chart of an effective media campaign model where those exposed to mass media do not drink alcohol. In section 2, we analyzed the model by obtaining the invariant point, positivity of the model solutions, alcohol abuse reproduction number, equilibrium points and their stabilities. The bifurcation of the model was analyzed using theorems in [19]. Sensitivity analysis was analyzed to identify the parameters that has great impact on alcohol abuse reproduction number R_0 . Numerical simulation shows that higher treatment rate reduces those in alcohol addicted class. We estimated data (accurate data not available) from the data collected from the rehabilitation centers in Kenya.

These are some of the challenges encountered in our data collection from the rehabilitation

centers. Most of the rehabilitation centers in Kenya are not run by the government but are privately owned while others are run by Non Government Organizations (NGO). They do not have centralized data and it is very difficult to ascertain how many rehabilitation centers are there in the country and the number of patients admitted in these institutions. We were able to visit a few and we estimated the data of individuals under treatment. The number of patients admitted in these institution is very low compared to the addicted individuals in the community (heavy drinking class). The cost of these facilities is also high and they do not allow patients to use insurance schemes like NHIF, which is enjoyed by majority in Kenya. Many of the patients are not admitted voluntarily but they are taken there by the relatives or guardians.

The government of Kenya need to increase the number of people in rehabilitation centers, by treating alcohol abuse like other contiguous diseases. This can be achieved by owning, supporting and running the rehabilitation centers. This will make the centers cheaper, accessible and affordable. The government should sensitized the citizens on the need of treatment of alcohol addiction.

From sensitivity analysis and numerical analysis we conclude that increase in the rate of treatment(rate of joining rehabilitation), reduces alcohol addicts in the community. Also mass media campaign should emphasize on risks of alcohol abuse to reduce individuals joining heavy drinking class.

Conflict of Interests

The authors declare that there is no conflict of interests.

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