MODELING THE EFFECTS OF TARGETED MASS MEDIA CAMPAIGNS AND TREATMENT ON ALCOHOL ABUSE IN KENYA

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DECLARATION

Declaration

This research is my work and has not been presented for a degree in any other University. No part of this thesis may be reproduced without the prior permission of the author and/or Kenyatta University.

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DEDICATION

I dedicate this work to my father in law Daniel Murungi for being the best dad to me and to my mother Margaret Muthuri for loving me always.

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ABBREVIATION AND ACRONYMS

WHO	World Health Organization
NACADA	National Authority for the Campaign against Alcohol and Drug Abuse
CASA	Court Appointed Special Advocates
DFE	Disease Free Equilibrium
AFE	Alcohol Free Equilibrium
EEP	Endemic Equilibrium Point

ABSTRACT

Alcoholism is a serious problem in Kenya today and many adults are addicted to alcohol. The harmful use of alcohol causes a large burden concerning diseases, social and economic problems to the society. Mass media campaigns against alcohol act as sources of information to halt alcohol abuse and its potentially harmful effects. In this research, we developed deterministic models for alcohol abuse driven by the light and heavy drinkers taking into consideration the influence of pre exposure to mass media campaigns. Two models were developed, one with perfect pre exposure campaigns where the campaigns were successful and the other with imperfect pre exposure campaigns where the campaigns were not successful. The two models were analyzed through the determination of the model's steady states and their respective stabilities analysis in terms of the alcohol abuse reproduction numbers R_0. The analysis shows that alcohol-free equilibrium (AFE) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. Numerical simulations were carried out and the sensitivity analysis of the model parameters was done to determine where the campaigns should be targeted for effective control of the abuse. The results from the simulations illustrated that increasing the rate of treatment reduces the number of alcohol addicts in the community. The results also showed that mass media campaign against alcohol consumption reduces alcohol abuse. From the study we conclude that if alcohol treatment is emphasized and mass media campaign regulated then alcohol addiction will be reduced from the community.

CHAPTER ONE: INTRODUCTION

1.1 Background

According to World Health Organization (2014), alcohol is a drug which is addictive and has been used by many people for centuries. 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 on the 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 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 bloodstream and the liver then clean alcohol from the blood through 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, according to (World, 2006).

In the past 1000 years, a grain that is fermented, juice from fruits and also honey are ingredients used to brew alcohol. Alcohol existed even in the past, for example, in China around 7000 B.C. India between 3000 and 2000 B.C had alcohol called *sura* made from rice, in 2700 B.C Babylonians worshipped wine goddess and in Greece, a drink called *mead* was used. In South America, Andes region corn, grapes or apples were used to make a variety of fermented beverages called *chichi*.

In the 16^{th} century, 'spirits' (the type of alcohol) was largely used as medicine. In Kenya, among all other abused substances, alcohol is leading. The most abused types of alcohol are the traditional brew because it is cheap and easily available. The next abused type is wine and spirits (NACADA, 2012).

When young adults are addicted to alcohol, the country has no future manpower to develop the nation. Researchers like (Eshiwani, 1993) and (Khamasi and Mutia, 2007) points out that alcohol and other abused substances may drive irresponsible behaviour among the youth. A study by (Pengpid and Peltzer, 2019) in Kenya concluded that 6.7% of Kenyan adults are harmful alcohol users and 12.8% are binge - drinkers. A report by (Kendagor et al., 2018) studied the prevalence of Heavy Episodic Drinking (HED) in Kenya. The results showed that the prevalence of HED was 12.6%. They also found out that the highest proportion of HED was in the 18-29 years age group at 35.5%.

A research by (Hornik et al., 2002), concluded that if alcohol is abused at an early age, this may continue even to adulthood which will affect the production of the affected in their professions and careers and even their families. Most alcoholic people started as light or occasional drinkers and later developed to full time or become dependent on alcohol. Many family and marriage problems arise from abusing alcohol.

Mass media is any means of transmission of information to reach as many people as possible. Mass media include broadcast media that uses videos and music from radio or television. Then there is also digital media that involves online and mobile mass communication from the internet. Examples of digital media include email, social media sites like Facebook, Instagram, Whatsapp, Twitter and Myspace. Outdoor media is also another form of mass media. These include AR-advertising, billboards in towns and on the roadsides, flying billboards mostly across major roads and placards. Another type of mass media is print media which

include physical objects like books, comics, magazine, newspaper or pamphlets.

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 (Grube, 2004). Many companies concentrate on alcohol advertising to increase sales. For example, in the 2000 year, the beverage industries spent 42 billion dollars of advertising alcohol in the United States of America. Whether young people are deliberately targeted by alcohol advertisers or not they are exposed to alcohol advertising on television, in print media, and on the radio. It is possible that advertising is important during initiation than during the addicted time. Analysis of data from 1970 to 1995 from 20 countries shows that banning alcohol adverts partially and completely might reduce alcohol abuse among the youth, (Saffer and Dave, 2002).

Alcohol marketing was found to increase the onset of binge drinking according to (De Bruijn, 2014). They studied the effects of alcohol marketing on adolescence in Kenya, Malawi, Nambia and Zambia. Kenya was the only country with legislation on alcohol consumption (Gazette, 2010), but did not seem to protect young people against large volumes of alcohol advertisement (De Bruijn, 2014). Studies show that 55.6% of the respondents are aware of alcohol adverts through television with 53.5% of them admitting that alcohol adverts attract them to drink (Anyange, 2014).

According to (Snyder et al., 2006), youth who saw more alcohol advertisements on average drank more (each additional advertisement seen increased the number of drinks consumed by one per cent). Youth in markets with greater alcohol advertising expenditures drank more (each additional dollar spent per capital raised the number of drinks consumed by 3 per cent). This shows that alcohol advertisement has an impact on alcohol consumption among the youth.

Internet users in Kenya by June 2016 were 21.6 million people which represent 53.3 per cent of people with internet access and 68.4 per cent data internet penetration. Kenya is ranked third at 9.4 per cent of the internet users in Africa according to the (Authority, 2014). By March 2019, the internet user in Kenya rose to 43.3 million with 83 % internet penetration,(Group, 2019). Social media sites are launched to cater to specific market needs and demands. The oldest social media site is My Space, which emphasis on music sharing, popular in the year 2000. Facebook was started in 2004, only available to Harvard University students but spread to other colleges and it was available to the public in 2006, (Moreno and Whitehill, 2014). Worldwide, 1.71 billion people are an active user of Facebook monthly which is about seventeen per cent increase. Facebook has approximately five million active users in Kenya and is growing.

Twitter emerged in 2006 and it uses short text messages with links to online and pictures with up to 140 characters called tweets. By 2012, Twitter had five million users worldwide who generate about three hundred forty tweets daily, according to (Huo and Wang, 2016) and (Dubey et al., 2016). These tweets may influence alcohol consumption if they target that. Instagram is used to share photos online. This site allows its user to upload and share photos with their friends who are following them in the same site. Linked In and YouTube are also social media sites but have less influence on alcohol consumption.

Social media is very important in people's lives because it affects the way they think and what they do. Social media is full of advertisements for alcohol and other drugs. Even restaurants and hotels use social media to promote alcohol and drinking special hours. It is in social media where people advertise parties, get-together and outings. According to (Vilage, 2017), research carried out by CASA Columbia at Columbia University in 2011, shows that American teens who use social media more at any time are more likely to smoke, drink alcohol and abuse drugs.

There is evidence that alcohol abuse spread like an infectious disease according to (Lenhart and Madden, 2007) and (Misra et al., 2011), hence can be formulated as a mathematical model. So far, few mathematical studies have been undertaken on the effects of mass media campaigns against alcohol abuse. Therefore, this study intends to develop and analyze mathematical models to look at alcohol abuse with the influence of treatment and mass media campaigns. Mathematical models help us to understand the extent of alcohol abuse with the effect of mass media and the impact of the intervention (rehabilitation). In Kenya, no mathematical model on the effect of treatment of alcohol and mass media campaign on alcohol abuse has been developed and the research is primarily designed to fill the gap.

1.2 Statement of the Problem

Alcohol abuse is one of the leading causes of diseases and deaths in youth and adults throughout the world. There are reports in Kenya of young people whose lives are destroyed because of alcohol, (NACADA, 2012). Young people mostly students are more likely to abuse alcohol because of peer pressure, media influence and lack of proper guidance, according to (**?**). The growth of mass media campaigns on alcohol consumption has a great influence on alcohol abuse hence there is a need to address the drinking problem.

According to (NACADA, 2010), alcohol abuse in central Kenya is a major problem. About two-thirds of the population consumes a high level of alcohol in the region. Consumption was estimated to be 18% and nearly 60% consumed alcohol before noon in central Kenya. They recommended enhanced enforcement of the Alcoholic Drinks Control Act, 2010 (Gazette, 2010) among other recommendations.

1.3 Justification of the Study

Thirty per cent of Kenyans aged between 15 and 65 years have ever drunk alcohol at some point in their life. 13.3 per cent of Kenyans are addicted to alcohol, which is about four million people according to (NACADA, 2012). When alcohol is taken in large quantities, it may cause death. Alcohol is estimated to cause 1.8 million fatalities every year worldwide. According to the latest survey by (Barometer, 2015), 89 per cent of people in Kenya use the internet for social media purposes with Facebook, Twitter, and Instagram leading. The same survey reviewed that 71 per cent of people in Kenya go online to post comments on social sites. There is zero per cent online use for 65 years and above, 64 per cent for below 25 years and below and 49 per cent for between 25 and 34 years. Studies conducted recently in the U.S.A shown that seventy-five per cent of teenagers who saw photos on social media of others drinking influenced them to do the same. A University of Nairobi student Selpher Cheloti did a study on Nairobi County secondary schools and showed that one out of the causes of alcohol and drug abuse was peer pressure at 84 per cent, (Cheloti et al., 2014). Thus alcohol abuse is a major concern in this country, hence the need to seek remedies.

1.4 General Objective

This research aims to develop a deterministic mathematical model describing the dynamics of alcohol abuse in the presence of mass media campaign and treatment of alcoholism (rehabilitation) in Kenya.

1.4.1 Specific Objectives

Specific objectives are:

i. Formulate model 1 on the impact of treatment on alcohol abuse incorporating mass media campaigns.

ii. Formulate model 2 on the effects of a targeted mass media campaign on alcohol abuse in Kenya.

iii. Establish the existence, stabilities of the equilibrium points and sensitivity analysis of each model.

iv. Determine the effects of mass media campaigns on alcohol abuse in Kenya

v. Determine the impact of the intervention (rehabilitation) in Kenya.

vi. Fit data from Kenya rehabilitation centres in the developed model using Matlab software to predict the impact of rehabilitation on alcoholism in Kenya.

1.5 Significance of the Study

The purpose of this research is to develop a deterministic model that illustrates the effects of treatment and mass media campaigns on alcoholism among the adult population in Kenya. Through this study, an understanding of the dynamics of alcohol abuse in the country is discussed. This study will thus help the health sector to know how the trends of alcohol abuse are, qualifying the potential effects of treating alcohol addicts, quantifying the potential impact, expanding the rehabilitation centres and emphasizing the data to be collected for future research. This study will also help the government regulate the media campaigns by promoting the campaign that shows the negative effects of alcohol and discourages the campaigns that encourage alcohol use.

CHAPTER TWO: LITERATURE REVIEW

2.1 Literature Review

A study by (Sánchez et al., 2007), studied drinking alcohol as an acquired state because of high contacts or intense interaction with the drinking classes. The purpose of their study was to identify mechanisms that influence and affect the population to start drinking or be initiated to the drinking classes. They divided the population into three classes which include susceptible, problem drinkers and temporarily recovered individuals. They concluded that a high relapse rate will be present when treatment has short-lived positive effects and that drinking behaviour is a result of the movement of susceptible to the drinker and recovered to the drinker. Results also showed that the sudden growth of the number of drinkers was possibly caused by a large number of individuals joining the problem drinking class and that it was more effective to control the average time the susceptible spends in the drinking environment.

Research by (Manthey et al., 2008), considered the college population which was divided into three groups depending on their drinking patterns. These groups were non-drinkers (N), social drinkers (S) and problem drinkers (P). They modelled drinking as a disease that spread depending on their social interactions. They assumed that problem drinkers could recruit both non-drinkers and social drinkers. They used systems of differential equations to develop a SIS model. Their analysis revealed that recruitment plays an important role in the pattern of campus drinking. According to their research, the reproduction number does not fully determine the dynamics of college drinking. They emphasized that campus drinking may be reduced by minimizing the recruitment of the non-drinkers by the problem drinkers. This could be attained by reducing the social interaction of students to heavy drinking environment which facilitates rapid progression from the non-drinking to the problem drinking class.

Bhunu (2012), studied the dynamics of alcoholism in a community taking into account that some people voluntarily quit alcohol consumption and some as a result of being on treatment. The model subdivided the human population into four classes which include S- those who do not consume alcohol and never consumed it, D- alcohol consumer but not become alcohol dependent A- alcohol consumers and independent on alcohol and R- those recovered with or without treatment. Analysis of the reproduction number showed conditions (which included who is encouraged to quit alcohol, either S, D or R) under which supporting the encouragement of moderate drinkers to quit alcohol consumption leads to a decrease in alcoholism better than alcoholics only quitting and vice versa. They used Center Manifold Theory to analyze the equilibrium point which shown that the equilibrium point is locally asymptotically stable when the reproduction number is less than unity. Numerical simulations were performed to illustrate various scenarios. The results from numerical simulation show that, in the long term, encouraging and supporting more moderate drinkers to quit alcoholic consumption will achieve a better result than supporting and encouraging alcoholics only to quit.

A research by (Walters et al., 2013), developed a three-stage model and represented the effect of social influence on drinking habits. Their main interest was the total recovery of individuals in the treatment class. They divided the population into three groups namely; susceptible (S) who do not consume alcohol, individuals with alcohol problem (A) and individuals in treatment (R). They assumed alcoholism develop due to social interaction with the individuals with an alcohol problem. Individuals in the treatment class may relapse back to A class or stay in the treatment class until they are completely recovered and go back to the susceptible class S. They used the stability analysis to calculate the reproduction number R_0, which when it exceeded one, alcohol persists in the population and when it is less than one the alcohol problem will die out with time. They concluded that β (rate of susceptible developing alcohol problem), which was based on contact with the drinking class, had a great influence on R_0. A decrease in β causes R_0 to decrease

and increasing β will increase R_0, hence to reduce alcohol problem in the population it is important to prevent susceptible from entering the alcohol problem class.

Sharma and Samanta (2013), developed a mathematical model for alcohol abuse and used four compartments which include S- susceptible who are moderate and occasional drinkers, D- heavy drinkers, T- drinkers in treatment and R- temporarily recovered drinkers. They found that the basic reproduction number of the model system is

$$R_0 = \frac{\beta_1}{\mu + \delta_1 + \phi}.$$

Where β_1 is the transmission coefficient from S class to the D class, μ is the natural death rate, δ_1 is death due to alcohol abuse and ϕ is the rate of getting treatment. Sensitivity analysis of R_0 identified β_1 as and as the parameter to be targeted for the reduction of R_0. Sensitivity analysis showed that it is more effective to control how the drinking spreads than to increase the number in the treatment class. They used numerical simulation to verify disease-free equilibrium E_0 is stable when $R_0 < 1$ and when $R_0 > 1$ endemic equilibrium E^* becomes stable and disease-free equilibrium become unstable then forward bifurcation occurs. The backward bifurcation occurs when the other endemic equilibrium E_1 is unstable.

Researchers (Bani et al., 2013), modelled the environmental factors concerning alcohol drinking patterns in college. Their model considered the social factors that mostly influence alcohol drinking patterns and the strategies that would reduce the social interaction of serious drinkers patterns so that the population of heavy drinkers can be reduced. They divided the college population into light drinkers, moderate drinkers, and heavy drinkers. They captured two drinking environments defined as low and high-risk environments. The moderate drinkers were divided into two classes based on the 2 distinct drinking environments. They concluded that the success of intervention programs to reduce the heavy drinking in college depended on the availability of resources and the people's ability to identify where

and how much to intervene. Analyzing the model mathematically suggested that heavy drinking can be reduced if the drinking reproduction number which depends on social and environmental factors is brought below one. Uncertainty and sensitivity analysis was carried out on the metric representing time to eradicate the high-risk drinkers in the presence of treatment which provided a direct measure of the success of the intervention programs. This measure showed that time to eradication of high-risk drinkers decreases with an increase in the level of intervention meant to reduce the rate of recruitment of new alcohol drinkers.

A research by (Ma et al., 2015), analyzed the impact of awareness programs a time delays on alcohol consumption behaviour. They divided the human population into three classes; $S(t)$- individuals who drink moderately or do not drink, $A(t)$- heavy drinkers and $X(t)$- population aware of the risk of drinking and do not drink alcohol. They also included the media class $M(t)$ which represented the cumulative density of the awareness programs that are driven by the media. They concluded that awareness programs reduce the population in the heavy drinking class. The time delays showed that for a lower value of time delay parameter τ, the system was stable but when the parameter had a higher value then the system could lose its stability. They analyzed the conditions for the existence of transcritical and Hopf bifurcation using τ as the bifurcation parameter. and the study suggested that the equilibrium of the system was locally asymptotically stable for $\tau \in [0, \tau_0]$ and unstable for $\tau > \tau_0$.

Huo and Wang (2016), constructed a model of binge drinking with the media coverage influence and did not consider recruitment and death. They divided the population N into n groups (n is the maximum degree) depending on the degree of the nodes and divided these groups into three classes depending on how they take or use alcohol. They developed a binge drinking model considering the media using mean-field theory. After analysis, they showed that the equilibrium points E_0 and E^* (representing Alcohol-Free Equilibrium and Unique Alcohol Equilibrium), are

all globally asymptotically stable. Alcohol abuse will disappear if the reproduction number is less than one and persist if the reproduction is greater than one. Network heterogeneity made drinking behaviour to spread faster hence they concluded that mixing freely makes drinking behaviour spread easily and media coverage is the best way to reduce alcohol problems though it does not change how it spreads.

Studies by (Dubey et al., 2016), presented a SIR model and studied the impact of 2 important parameters: awareness programs and treatment on the spread of an infectious disease. They divided the population into, S - Susceptible population, I -Infective population and R - Recovery population. Then they assumed that a part of the S class forms another class called susceptible awareness population, S_a. This class develops due to awareness programs by social/electronic media density M at any time t. They concluded that there exist only two equilibrium points: DFE (total elimination of the infection) $I = 0$ and EEP (disease will persist). The analysis showed that DFE was locally asymptotically stable when the reproduction number was less than one and endemic equilibrium existed when the reproduction number was greater than one and was globally asymptotically stable. The numerical simulation revealed that the infected population decrease as they increased the media awareness rate, β. They also showed that if the media dissemination was not available in the population then infection increased which was further reduced by treatment.

Huo and Zhang (2016), developed a model that involved positive and negative role of Twitter on alcoholism. They divided the total population into 4 compartments: Those who drink moderately, light drinkers, heavy drinkers, and quitters. These people may use their Twitter accounts at any time and comment about their drinking. They carried out the sensitivity analysis of the basic reproduction number R_0 and heavy drinkers. The parameters that are related to Twitters appear in basic reproduction numbers. They analyzed R_0, considering when the positive tweets (information discouraging alcoholism) are less than the negative ones (information

encouraging alcoholism) or when the positive tweets are more than the negative tweets. They concluded that if the number of positive tweets is increased or if the number of negative tweets is decreased would help reduce alcohol abuse. Also controlling the number of tweets by the moderate drinkers would reduce alcohol consumption. Positive and negative tweets played an important role in the model, although positive tweets played a more vital role than negative tweets.

Our study is developed to improve the study of (Huo and Zhang, 2016) They studied alcoholism with the influence of Twitter. They did not consider other mass media campaigns and treatment of alcohol abuse. There is no mathematical model of the influence of mass media on alcohol abuse in Kenya. This study intends to fill that gap by developing and analyzing a mathematical model of treatment of alcohol abuse incorporating mass media campaign. This study will help us to understand the extent of alcohol abuse with the effect of mass media and the impact of the intervention (rehabilitation).

CHAPTER THREE: FORMULATION AND ANALYSIS OF THE MODELS

3.1 Model 1

Our research involves a mathematical modelling approach where alcohol abuse is modelled as a socially contagious disease, hence we can use theories of disease modelling to formulate models of alcohol abuse. Model 1 is a perfect pre-exposure campaign model where the campaign against alcohol abuse is effective.

3.1.1 Model Formulation

In this section, we consider model assumptions, model description, model flow chart and model equations.

3.1.2 Models Assumptions

The assumptions of model 1 are:

i. There is exposure to media campaign against alcohol consumption before initiation to alcohol, which implies that only the susceptible are influenced by mass media campaigns.

ii. There is homogeneous mixing of the population in Kenya and individuals become alcoholic after contact with an individual in the light drinking class and heavy drinking class.

iii. 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.

iv. When individuals are exposed to the media campaign, they do not get into drinking habits but go back to susceptible class.

v. The growth of awareness programs is assumed to be proportional to the number of heavy drinkers.

vi. When the individuals relapse, they move to the heavy drinking class.

3.1.3 Model Description

We will have six human compartments and one media compartment where we consider the adult population only. These classes 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 centres and Q -individuals who have stopped drinking permanently. The media compartment M - is the density of media campaign against alcohol consumption. The progression from one class to another will be formulated into seven nonlinear ordinary differential equations as shown in Figure 3.1. We will use data for simulation from rehabilitation centres in Kenya.

Individuals are recruited into the model at a rate of Λ. Individuals are initiated to alcohol drinking due to contact with the light drinkers at a rate of λ where λ is given by $\beta(L+\eta_1 H)$ and β is the effective contact rate of light and heavy drinkers with the non-drinking classes and η_1 is the rate of the heavy drinkers contacting the susceptible which is always less than the rate of contact of the light drinkers with the susceptible. Hence η_1 is always less than one. The rate of dissemination of media awareness to the susceptible is β_m. The human population die due to natural causes at a rate μ and they die due to alcohol-related causes at a rate δ. The rate of increase of alcohol intake from a few drinks a week to dependence to alcohol is α_1 and light drinkers quit drinking at a rate of α_2. The rate at which heavy drinkers seek treatment or go to rehabilitation centres for treatment is σ_1 and the rate at which heavy drinkers quit drinking without treatment is σ_2. The effective treatment rate of the heavy drinking class is τ_2, which partly due to media awareness programs. The rate of relapse after rehabilitation back to heavy drinking is τ_1. The rate of the media awareness campaign on the light drinkers

is represented by θ_1 and θ_2 represents the rate of media awareness programs of the heavy drinking classes which will increase the rate of joining the treatment class. The depletion (depreciation) of the media campaigns due to ineffectiveness of the programs or other factors is represented by ρ. The rate of effective media campaigns against alcohol is denoted by ω. Table 3.1 gives the variables and Table 3.2 gives the summary of the parameters and their description used in our research.

Table 3.1: Definition of Variables

Variables	Definitions
S	Susceptible who have never drunk alcohol
S_a	Susceptible who are pre-exposed to effects of alcohol abuse
L	Light drinkers who drink about five drinks, two or three times a week
H	Heavy drinkers who drink daily and dependent on alcohol
T	Individuals on rehabilitation and not exposed to alcohol
Q	Individuals who completely quit alcohol and leave the model
M	Awareness programs are driven by mass media

Table 3.2: Parameters and their Description

Parameter	Description
Λ	Recruitment rate of drinkers into population
μ	Natural death rate
λ	Contact rate of susceptible with the light drinkers
β	Effective contact rate
β_m	Rate of dissemination of media awareness of the susceptible
α_1	Transfer rate of light drinkers to heavy drinkers
α_2	Rate of quitting alcohol of the light drinkers
σ_1	Treatment rate of the heavy drinkers (rate of joining rehabilitation)
σ_2	Rate of heavy drinkers quitting the drink
τ_2	Rate of effective treatment
τ_1	Rate of relapse back to heavy drinking from rehabilitation
δ	Death rate due to alcohol abuse
θ_1	Rate of awareness programs being implemented on the light drinkers
θ_2	Rate of awareness programs being implemented on the heavy drinkers
ρ	Rate of depletion of media programs
ω	Rate of effective (successful) media campaign
η_1	Modification Parameter

3.1.3.1 Model Flow Charts and Equations of Model 1

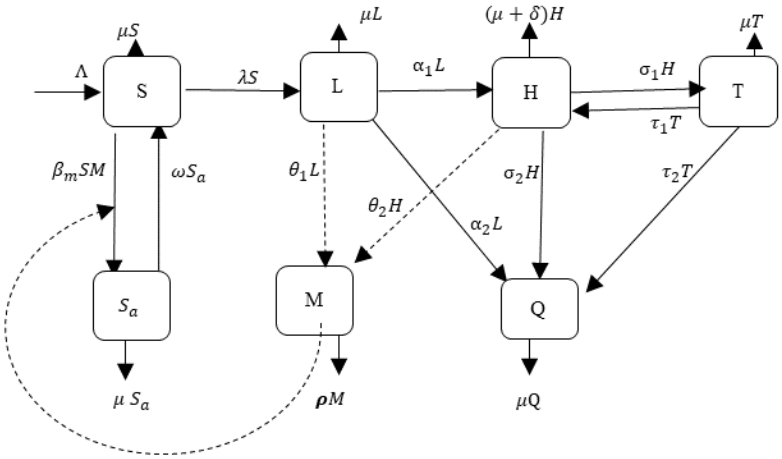

Figure 3.1: Flow chart of effective mass media campaign

From Figure 3.1, we get the following differential equations.

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda + \omega S_a - \beta_m SM - (\lambda + \mu)S, \\
\frac{dS_a}{dt} &= \beta_m SM - (\mu + \omega)S_a, \\
\frac{dL}{dt} &= \lambda S - (\mu + \alpha_1 + \alpha_2)L, \\
\frac{dH}{dt} &= \alpha_1 L + \tau_1 T - (\sigma_1 + \mu + \sigma_2 + \delta)H, \\
\frac{dT}{dt} &= \sigma_1 H - (\mu + \tau_2 + \tau_1)T, \\
\frac{dQ}{dt} &= \tau_2 T + \sigma_2 H + \alpha_2 L - \mu Q, \\
\frac{dM}{dt} &= \theta_1 L + \theta_2 H - \rho M.
\end{aligned}\right\} \tag{3.1}$$

We let $k_1 = \mu+\alpha_1+\alpha_2$, $k_2=\mu+\sigma_1+\sigma_2+\delta$, $k_3=\mu+\tau_1+\tau_2$ and

$$\lambda = \beta(L + \eta_1 H) \tag{3.2}$$

3.1.4 Model Analysis

In this section, we analyze model 1 where the analysis include; invariant region, the positivity of the model, reproduction number of the alcohol abuse, alcohol-free equilibrium(AFE), local and global stability of AFE, endemic equilibrium point and the bifurcation of the model.

3.1.5 Invariant Region and Positivity

To investigate 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:

$$\left.\begin{aligned} \frac{dN}{dt} = \frac{dS}{dt} + \frac{dS_a}{dt} + \frac{dL}{dt} + \frac{dH}{dt} + \frac{dT}{dt} + \frac{dQ}{dt}, \\ \frac{dN}{dt} = \Lambda - \mu N - \delta H. \end{aligned}\right\} \tag{3.3}$$

Equation (3.3) can also be written as:

$$\frac{dN}{dt} \leq \Lambda - \mu N. \tag{3.4}$$

Integrating the linear differential equation (3.4) with respect to time, using the integrating factor e^{ut} we obtain,

$$Ne^{\mu t} \leq \frac{\Lambda}{\mu} e^{\mu t} + e^{c}. \tag{3.5}$$

This is simplified to,

$$N \leq \frac{\Lambda}{\mu} + \frac{C}{e^{\mu t}}, N \leq \frac{\Lambda}{\mu} + Ce^{-\mu t}, \tag{3.6}$$

where $C = e^c$ is a constant of integration. As t tends to infinity, the limit of $N(t)$ becomes

$$\lim_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}. \tag{3.7}$$

From equation (3.7), it is clear that $N(t)$ is bounded and

$$0 < N(t) \leq \frac{\Lambda}{\mu}. \tag{3.8}$$

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:

$$\Omega = \left\{ \Big(S, S_a, L, H, T, Q\Big) \in \mathbb{R}^6_+ \mid 0 < N(t) \leq \frac{\Lambda}{\mu} \right\}. \tag{3.9}$$

Therefore from equation (3.9), the model is well posed and we can study the dynamics of the model in Ω.

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 get from the last equation of (3.1);

$$\frac{dM}{dt} = \theta_1 L + \theta_2 H - \rho M, \qquad \frac{dM}{dt} \geq -\rho M. \tag{3.10}$$

Separating the variables we obtain,

$$\frac{dM}{M} \geq -\rho \; dt. \tag{3.11}$$

Integrating this equation (3.11) with respect to time t we obtain,

$$ln \; M(t) \geq -\rho \, t + C, \tag{3.12}$$

where C is a constant of integration. Introducing exponential both sides we obtain;

$$M(t) \geq e^{-\rho+C}. \tag{3.13}$$

Substituting $t = 0$ and solving for C we get,

$$M(0) \geq e^{0}.e^{C}, \qquad \text{hence} \qquad e^{C} = M(0). \tag{3.14}$$

Thus our solution now is

$$M(t) \geq M(0)e^{-\rho t} > 0. \tag{3.15}$$

Equation (3.15) is positive for all time t. We do the same to the equation of Q,

$$\frac{dQ}{dt} = \tau_2 T + \sigma_2 H + \alpha_2 L - \mu Q, \;\; \frac{dQ}{dt} \geq -\mu Q. \tag{3.16}$$

Separating the variables and integrating equation (3.16), introducing exponential and solving for t=0, we obtain,

$$Q(t) \geq Q(0)e^{-\mu t} > 0. \tag{3.17}$$

Equation (3.17) is positive for all time t. The same applies to the differential equation involving treatment (T),

$$\frac{dT}{dt} = \sigma_1 H - k_3 T \; \frac{dT}{dt} \geq -k_3 T. \tag{3.18}$$

Integrating equation (3.18) and solving as done in equation (3.10) we obtain;

$$Q(t) \geq Q(0)e^{-k_3 t} > 0. \tag{3.19}$$

Equation (3.19) is also positive for all time t. This will apply to all other differential

equation involving H, L, S_a and S. This is given in the equation (3.20) below.

$$\left.\begin{aligned} \frac{dH}{dt} &\geq -k_2 H,\ H(t) \geq H(0)e^{-k_2 t} > 0 \\ \frac{dL}{dt} &\geq -k_1 L,\ L(t) \geq L(0)e^{-k_1 t} > 0 \\ \frac{dS_a}{dt} &\geq -(\mu+\omega)S_a,\ S_a(t) \geq S_a(0)e^{-(\mu+\omega)t} > 0 \\ \frac{dS}{dt} &\geq -(\lambda_1+\mu)S,\ S(t) \geq S(0)e^{\int -(\lambda_1+\mu)dt} > 0 \end{aligned}\right\} \tag{3.20}$$

Equation (3.20) show that S, S_a, L and H are always positive for all time t.

3.1.6 Alcohol Free Equilibrium(AFE) and Alcohol Reproduction Number

The AFE of the system (3.1) is obtained by setting all alcohol drinking classes and media class to zero. This means $L = H = T = Q = M = 0$ which implies that $S_a = 0$ hence;

$$S^0 = \frac{\Lambda}{\mu}.$$

The AFE of the model is therefore given by:

$$E^0 = \left\{S^0, S_a^0, L^0, H^0, T^0, Q^0, M^0\right\} = \left\{\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0\right\}. \tag{3.21}$$

The **reproduction number** is the number of average secondary infections produced by an infectious individual in a susceptible population. In the case of alcohol abuse model, R_0 is the average number of secondary cases generated by one alcohol user over the alcoholic period. We use the next generation matrix method to determine the **alcohol abuse reproduction number** R_0, as used by (Castillo-Chavez et al., 2002). Using the notation F to represent the new infection and V to represent the transfer of infection in our model noting that $S^0 = \frac{\Lambda}{\mu}$ we obtain:

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

$$V = \begin{pmatrix} k_1 & 0 & 0 \\ -\alpha_1 & k_2 & -\tau_1 \\ 0 & -\sigma_1 & k_3 \end{pmatrix}$$

The reproduction number of the model is the largest eigenvalue of the matrix FV^{-1}, where V^{-1} is the inverse of V. The matrix FV^{-1} is given by;

$$F.V^{-1} = \begin{pmatrix} \frac{\Lambda\beta(k_3(k_2+\alpha_1\eta_1)-\sigma_1\tau_1)}{\mu k_1(k_2k_3-\sigma_1\tau_1)} & \frac{\Lambda\beta\eta_1}{\mu k_2k_3-\mu\sigma_1\tau_1} & \frac{\Lambda\beta\eta_1\tau_1}{\mu k_2k_3-\mu\sigma_1\tau_1} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

The reproduction numberR_0 is therefore given by:

$$R_0 = \frac{\beta\Lambda(k_3(k_2+\eta_1\alpha_1)-\sigma_1\tau_1)}{\mu k_1(k_2k_3-\sigma_1\tau_1)}. \tag{3.22}$$

The reproduction number can also be written as,

$$R_0 = \frac{\beta\Lambda(k_3\eta_1\alpha_1+k_3k_2-\sigma_1\tau_1)}{\mu k_1(k_2k_3-\sigma_1\tau_1)}$$

Factoring out k_2k_3 in the numerator of the equation of R_0, we obtain;

$$R_0 = \frac{\beta\Lambda(k_3\eta_1\alpha_1+k_3k_2(1-\frac{\sigma_1\tau_1}{k_2k_3}))}{\mu k_1(k_2k_3-\sigma_1\tau_1)}$$

Considering the fraction $\frac{\sigma_1\tau_1}{k_2k_3}$, where $k_2 = \mu+\sigma_1+\sigma_2+\delta$ and $k_3 = \mu+\tau_1+\tau_2$, we observe that this is a fraction less one. Further the fraction,

$$\frac{\sigma_1}{\mu+\sigma_1+\sigma_2+\delta}, \tag{3.23}$$

represent the probability that an individual getting treatment and the fraction,

$$\frac{\tau_1}{\mu+\tau_1+\tau_2}, \tag{3.24}$$

represent the probability that an individuals from treatment class relapsing back to the heavy drinking class. Hence the product,

$$\frac{\sigma_1\tau_1}{(\mu+\sigma_1+\sigma_2+\delta)(\mu+\tau_1+\tau_2)}, \tag{3.25}$$

represent the probability that an individual will be in the treatment class. We can also let

$$\frac{\sigma_1\tau_1}{(\mu+\sigma_1+\sigma_2+\delta)(\mu+\tau_1+\tau_2)} = \phi,$$

hence R_0 now becomes,

$$R_0 = \frac{\Lambda}{\mu k_1}\Big(\frac{\beta(\eta_1\alpha_1 k_3 + k_2k_3(1-\phi))}{k_2k_3(1-\phi)}\Big) = \frac{\Lambda}{\mu k_1}\Big(\frac{\beta(\eta_1\alpha_1 + k_2(1-\phi))}{k_2(1-\phi)}\Big). \tag{3.26}$$

From the equation (3.26), we conclude that,

$$R_0 = \frac{\Lambda}{\mu}\Big(\frac{\beta}{k_1} + \frac{\beta\eta_1\alpha_1}{k_1k_2(1-\phi)}\Big), \tag{3.27}$$

where

$$R_L = \frac{\Lambda\beta}{\mu k_1}$$

represents the reproduction number of light drinking class L and

$$R_H = \frac{\Lambda\beta\alpha_1\eta_1}{\mu k_1k_2(1-\phi)},$$

represents the reproduction number of the heavy drinking class H.

3.1.7 Local and Global 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 (3.1) at the AFE E^0.

$$J_{E^0} = \begin{pmatrix} -\mu & \omega & \frac{-\Lambda\beta}{\mu} & \frac{-\Lambda\beta\eta_1}{\mu} & 0 & 0 & -\frac{\beta_m\Lambda}{\mu} \\ 0 & -(\mu+\omega) & 0 & 0 & 0 & 0 & \frac{\beta_m\Lambda}{\mu} \\ 0 & 0 & \frac{\Lambda\beta}{\mu} - k_1 & \frac{\Lambda\beta\eta_1}{\mu} & 0 & 0 & 0 \\ 0 & 0 & \alpha_1 & -k_2 & \tau_1 & 0 & 0 \\ 0 & 0 & 0 & \sigma_1 & -k_3 & 0 & 0 \\ 0 & 0 & 0 & \alpha_2 & \sigma_2 & -\mu & 0 \\ 0 & 0 & \theta_1 & \theta_2 & 0 & 0 & -\rho \end{pmatrix}$$

From the Jacobian matrix, it can be seen that the first three eigenvalues are; $-\mu, -(\omega+\mu)$ and $-\rho$. The other eigenvalues are evaluated from the reduced matrix below.

$$\begin{pmatrix} \frac{\beta\Lambda}{\mu} - k_1 & \frac{\Lambda\beta\eta_1}{\mu} & 0 \\ \alpha_1 & -k_2 & \tau_1 \\ 0 & \sigma_1 & -k_3 \end{pmatrix}$$

The characteristic polynomial of the matrix J_{E^0} given by;

$$\lambda_1^3 + P_1\lambda_1^2 + P_2\lambda_1 + P_3 = 0. \tag{3.28}$$

where,

$$\begin{aligned}
P_1 =& -\frac{\beta\Lambda}{\mu} + k_1 + k_2 + k_3,\\
P_2 =& -\frac{\alpha_1\beta\eta_1\Lambda}{\mu} - \frac{\beta k_2\Lambda}{\mu} - \frac{\beta k_3\Lambda}{\mu} + k_1k_2 + k_3k_2 + k_1k_3 - \sigma_1\tau_1,\\
P_3 =& \frac{\beta\Lambda\sigma_1\tau_1}{\mu} - \frac{\alpha_1\beta\eta_1k_3\Lambda}{\mu} - \frac{\beta k_2k_3\Lambda}{\mu} - k_1\sigma_1\tau_1 + k_1k_2k_3.
\end{aligned}$$

Simplifying P_3 and writing it in terms of R_0 we get:

$$\begin{aligned}
P_3 =& k_1(k_2k_3 - \sigma_1\tau_1) + \frac{\Lambda\beta}{\mu}(-k_3(k_2 + \alpha_1\eta_1) + \sigma_1\tau_1),\\
P_3 =& k_1(k_2k_3 - \sigma_1\tau_1) - \frac{\Lambda\beta k_1}{\mu k_1(k_2k_3 - \sigma_1\tau_1)}(k_3(k_2 + \alpha_1\eta_1) - \sigma_1\tau_1)(k_2k_3 - \sigma_1\tau_1),\\
P_3 =& k_1(k_2k_3 - \sigma_1\tau_1)\Big(1 - \frac{\Lambda\beta}{\mu k_1(k_2k_3 - \sigma_1\tau_1)}(k_3(k_2 + \alpha_1\eta_1) - \sigma_1\tau_1)\Big).
\end{aligned}$$

We know that from equation (3.23) that,

$$R_0 = \frac{\Lambda\beta}{\mu k_1(k_2k_3 - \sigma_1\tau_1)}(k_3(k_2 + \alpha_1\eta_1) - \sigma_1\tau_1)\Big),$$

therefore

$$P_3 = k_1(1 - \phi)(1 - R_0).$$

We apply Routh - Hurwitz Criteria (Routh, 1877), where equation (3.28) 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 *and* P_2 are positive. 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. This means that AFE point E^0 is locally asymptotically stable whenever $R_0 < 1$.

□

Global stability of AFE of system (3.1) is investigated using the theorem by (Castillo-Chavez and Song, 2004).

Theorem 2. *The fixed point* $\widetilde{U_0} = (X^*, 0) = (\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0)$ *is globally asymptotically stable, if* $R_0 < 1$ *is locally asymptotically stable and assumption* $(H1)$ *and* $(H2)$ *are satisfied where 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 non-negative) and* R^6_+ *is the region where the model makes biological sense. The equation* (3.1) *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.* $G(X, 0) = 0, \widetilde{U_0} = (X^*, 0) = (\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0)$ *denotes the AFE point of the model.*

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

$$A = \begin{pmatrix} \beta - k_1 & \beta\eta_1 & 0 \\ \alpha_1 & -k_2 & \tau_1 \\ 0 & \sigma_1 & -k_3 \end{pmatrix},$$

$$AZ = \begin{pmatrix} (\beta - k_1)L + \beta\eta_1 H \\ \alpha_1 L - k_2 H + \tau_1 T \\ \sigma_1 H - k_3 T \end{pmatrix},$$

$$G(X, Z) = \begin{pmatrix} \lambda S - k_1 L \\ \alpha_1 L - k_2 H + \tau_1 T \\ \sigma_1 H - k_3 T \end{pmatrix},$$

$$\widetilde{G}(X, Z) = AZ - G(X, Z)$$

.

$$\widetilde{G}(X,Z) = \begin{pmatrix} \beta(L+\eta_1 H)(1-\frac{S}{N}) \\ 0 \\ 0 \end{pmatrix}.$$

Since $S \leq N, \frac{S}{N} \leq 1$, it is clear that $\widetilde{G}(X,Z) \geq 0$. It is also clear that $X^* = (\frac{\Lambda}{\mu}, 0, 0)$ is g.a.s equilibrium of $\frac{dX}{dt} = F(X,0)$. Hence the system (3.1) is globally asymptotically stable. □

3.1.8 Endemic Equilibrium Point (EEP)

To find conditions for the existence of an equilibrium for which alcohol abuse is endemic in the population, the system (3.1) is solved in terms of force of infection at steady state λ^* , where $\lambda^* = \beta(L^* + \eta_1 H^*)$. Setting the right hand side of equation (3.1) to zero and noting that $\lambda = \lambda^*$ at equilibrium gives:

$$\left.\begin{aligned}
S^* =& \xi_5 \\
S_a^* =& \frac{(\Lambda-\mu\xi_5)\left((\mu+\omega)\left(-\beta\left(\eta_1+\xi_2\right)+\beta\eta+\xi_1\beta_m\right)+\xi_3\left(\beta(\mu+\omega)-\mu\beta_m\right)\right)}{\beta\omega\left(\eta_1+\xi_2\right)(\mu+\omega)+\mu\xi_3\omega\beta_m} \\
L^* =& \frac{(\mu+\omega)(\Lambda-\mu\xi_5)}{\xi_5\left(\beta\left(\eta_1+\xi_2\right)(\mu+\omega)+\mu\xi_3\beta_m\right)}\xi_2 \\
H^* =& \frac{(\mu+\omega)(\Lambda-\mu\xi_5)}{\xi_5\left(\beta\left(\eta_1+\xi_2\right)(\mu+\omega)+\mu\xi_3\beta_m\right)} \\
T^* =& \xi_1\frac{(\mu+\omega)(\Lambda-\mu\xi_5)}{\xi_5\left(\beta\left(\eta_1+\xi_2\right)(\mu+\omega)+\mu\xi_3\beta_m\right)} \\
Q =& \xi_4\frac{(\mu+\omega)(\Lambda-\mu\xi_5)}{\xi_5\left(\beta\left(\eta_1+\xi_2\right)(\mu+\omega)+\mu\xi_3\beta_m\right)} \\
M^* =& \xi_3\frac{(\mu+\omega)(\Lambda-\mu\xi_5)}{\xi_5\left(\beta\left(\eta_1+\xi_2\right)(\mu+\omega)+\mu\xi_3\beta_m\right)}
\end{aligned}\right\} \tag{3.29}$$

where $\xi_1 = \frac{\sigma_1}{k_3}$, $\xi_2 = \frac{k_2-\xi_1\tau_1}{\alpha_1}$, $\xi_3 = \frac{\theta_1\xi_2+\theta_2}{\rho}, \xi_4 = \frac{\alpha_2\xi_2+\xi_1\tau_2+\sigma_2}{\mu}, \xi_5 = \frac{k_1\xi_2}{\beta(\eta_1+\xi_2)}$.
Writing equation of (3.23) in terms of β we obtain;

$$\beta = \frac{k_1\mu R_0\left(1-\phi\right)}{\Lambda\left(\alpha_1\eta_1k_3+k_2k_3-\sigma_1\tau_1\right)}. \tag{3.30}$$

Solving for $\lambda = \beta(L + \eta_1 H)$, we obtain;

$$\lambda = \frac{k_1\mu\left(R_0 - 1\right)R_0(\mu+\omega)\left(1-\phi\right)}{\alpha_1 B k_3 \Lambda \xi_3 + k_1 R_0(\mu+\omega)\left(1-\phi\right)}.$$

Substituting the value of β into equation (3.29) we obtain:

$$\left.\begin{aligned}
S^* =& \frac{\Lambda}{\mu R_0} \\
S_a^* =& \frac{\alpha_1\beta_m k_3 \Lambda^2 \xi_3\left(R_0 - 1\right)}{\mu R_0\left(\alpha_1\beta_m k_3\Lambda\xi_3 + k_1 R_0(\mu+\omega)\left(1-\phi\right)\right)} \\
L^* =& \frac{\Lambda\left(R_0 - 1\right)(\mu+\omega)\left(1-\phi\right)}{\alpha_1\beta_m k_3\Lambda\xi_3 + k_1 R_0(\mu+\omega)\left(1-\phi\right)} \\
H^* =& \frac{\alpha_1 k_3\Lambda\left(R_0 - 1\right)(\mu+\omega)}{\alpha_1\beta_m k_3\Lambda\xi_3 + k_1 R_0(\mu+\omega)\left(1-\phi\right)} \\
T^* =& \frac{\alpha_1\Lambda\left(R_0 - 1\right)\sigma_1(\mu+\omega)}{\alpha_1\beta_m k_3\Lambda\xi_3 + k_1 R_0(\mu+\omega)\left(1-\phi\right)} \\
Q^* =& \frac{\alpha_1 k_3\Lambda\xi_4\left(R_0 - 1\right)(\mu+\omega)}{\alpha_1\beta_m k_3\Lambda\xi_3 + k_1 R_0(\mu+\omega)\left(1-\phi\right)} \\
M^* =& \frac{\alpha_1 k_3\Lambda\xi_3\left(R_0 - 1\right)(\mu+\omega)}{\alpha_1\beta_m k_3\Lambda\xi_3 + k_1 R_0(\mu+\omega)\left(1-\phi\right)}.
\end{aligned}\right\} \tag{3.31}$$

From equation (3.31) we conclude that;

Theorem 3. *Endemic equilibrium exist and is positive if* $R_0 > 1$.

3.1.9 Bifurcation of the Model

We use Center Manifold Theory to investigate the nature of the bifurcation. We let $S = x_1, S_a = x_2, L = x_3, H = x_4, T = x_5, Q = x_6, M = x_7$.

Equation (3.1) can be written as,

$\frac{dN}{dt} = F(x)$ with $F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)$ and,

$$\left.\begin{aligned}
\frac{dx_1}{dt} &= f_1 = \Lambda + \omega x_2 - \beta_m x_1 x_7 - \beta(x_3 + \eta_1 x_4)x_1 - \mu x_1 \\
\frac{dx_2}{dt} &= f_2 = \beta_m x_1 x_7 - \mu x_2 - \omega x_2 \\
\frac{dx_3}{dt} &= f_3 = \beta(x_3 + \eta_1 x_4)x_1 - k_1 x_4 \\
\frac{dx_4}{dt} &= f_4 = \alpha_1 x_3 + \tau_1 x_4 - k_2 x_4 \\
\frac{dx_5}{dt} &= f_5 = \sigma_1 x_4 - k_3 x_5 \\
\frac{dx_6}{dt} &= f_6 = \tau_2 x_5 + \sigma_2 x_4 + \alpha_2 x_3 - \mu x_6 \\
\frac{dx_7}{dt} &= f_7 = \theta_1 x_3 + \theta_2 x_4 - \rho x_7
\end{aligned}\right\}. \tag{3.32}$$

By choosing $\beta = \beta^*$ as the bifurcation parameter and investigating the case when $R_0 = 1$ gives;

$$\beta^* = \frac{\mu k_1(1-\phi)}{\Lambda(k_2 k_3 + k_2\alpha_1\eta_1 - \sigma_1\tau_1}. \tag{3.33}$$

It can be shown that the Jacobian of equation (3.1) at $\beta = \beta^*$ has a zero eigenvalue which is simple. To investigate the stability of equation (3.1), we use the following model of (Castillo-Chavez and Song, 2004), Appendix A.

Eigenvectors of $J_{(E^0)} = J^*_\beta$

The Jacobian of the model at $\beta = \beta^*$ denoted by J_{β^*} has a right eigenvector denoted by $w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T$ given by:

$$\begin{pmatrix} -\mu & \omega & -g_1 & -g_2 & 0 & 0 & 0 \\ 0 & -g_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & g_1 - k_1 & g_2 & 0 & 0 & 0 \\ 0 & 0 & \alpha_1 & -k_2 & \tau_1 & 0 & 0 \\ 0 & 0 & 0 & \sigma_1 & -k_3 & 0 & 0 \\ 0 & 0 & \alpha_1 & \sigma_2 & \tau_2 & -\mu & 0 \\ 0 & 0 & \theta_1 & \theta_2 & 0 & 0 & -\rho \end{pmatrix} \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \\ w_7 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \tag{3.34}$$

where $g_1 = \frac{\beta^* \Lambda}{\mu}, g_2 = \frac{\beta^* \eta - 1\Lambda}{\mu}, g_3 = -(\mu + \omega)$. Equation (3.34) can also be written as:

$$\left.\begin{aligned} -\mu w_1 + \omega w_2 - g_1 w_3 - g_2 w_4 = 0 \\ -g_3 w_2 = 0 \\ (g_1 - k_1) w_3 + g_2 w_4 = 0 \\ \alpha_1 w_3 - k_2 w_4 + \tau_2 w_5 = 0 \\ \sigma_1 w_4 - k_3 w_5 = 0 \\ \alpha_1 x_3 + \sigma_2 x_4 + \tau_2 x_5 - \mu x_6 = 0 \\ \theta_1 x_3 + \theta_2 x_4 - \rho x_7 = 0 \end{aligned}\right\}. \tag{3.35}$$

After solving equation (3.35), we get:

$$\left.\begin{aligned} w_1 = -\frac{(g_1 w_3 + g_2 w_4)}{\mu} < 0 \\ w_2 = 0 \\ w_3 = \frac{g_2 w_4}{g_1 - k_1} > 0 \\ w_4 = \frac{k_3 w_5}{\sigma_1} > 0 \\ w_5 = \frac{\omega_1 w_4}{k_3} > 0 \\ w_6 = \frac{\alpha_1 x_3 + \sigma_2 x_4 + \tau_2 x_5}{\mu} > 0 \\ w_7 = \frac{\theta_1 x_3 + \theta_2 x_4}{\rho} > 0 \end{aligned}\right\}. \tag{3.36}$$

The Jacobian matrix has a left eigenvector denoted v given by:

$$\begin{pmatrix} -\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ \omega & -g_3 & 0 & 0 & 0 & 0 & 0 \\ -g_1 & 0 & g_1 - k_1 & \alpha_1 & 0 & \alpha_1 & \theta_1 \\ -g_2 & 0 & \alpha_1 & -k_2 & \sigma_1 & \sigma_2 & \theta_2 \\ 0 & 0 & 0 & \tau_2 & -k_3 & \tau_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\rho \end{pmatrix} \begin{pmatrix} v_1 \\ v_2 \\ v_3 \\ v_4 \\ v_5 \\ v_6 \\ v_7 \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}. \tag{3.37}$$

Solving equation (3.37), we obtain:

$$\left.\begin{aligned} v_1 =& v_2 = 0 \\ v_3 =& \frac{\alpha_1 v_4}{g_1 - k_1} > 0 \\ v_4 =& \frac{k_3 v_5}{\tau_2} > 0 \\ v_5 =& \frac{\tau_2 v_4}{k_3} > 0 \\ v_6 =& 0 \\ v_7 =& 0 \end{aligned}\right\}. \tag{3.38}$$

We find the sign of **a** and **b** as follows using the expression,

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

$$\left.\begin{aligned} v_3 w_1 w_1 \frac{d^2 f_3}{dx_1 dx_1} =& 0 \\ v_3 w_1 w_3 \frac{d^2 f_3}{dx_1 dx_3} =& \beta^* v_3 w_1 w_3 < 0 \\ v_3 w_3 w_1 \frac{d^2 f_3}{dx_3 dx_1} =& v_3 w_3 w_1 \beta^* < 0 \\ v_3 w_1 w_4 \frac{d^2 f_3}{dx_1 dx_4} =& v_3 w_1 w_4 \beta^* \eta_1 < 0 \\ v_3 w_4 w_1 \frac{d^2 f_3}{dx_4 dx_1} =& v_3 w_4 w_1 \beta^* \eta_1 < 0 \end{aligned}\right\}. \tag{3.39}$$

The sum of equations in (3.39) is the value of **a** given by:

$$\mathbf{a} = 2 v_3 w_1 \beta^* (w_3 + w_4 \eta_1) < 0 \tag{3.40}$$

$\mathbf{a} < 0$ since $w_1 < 0$ and $v_3, w_3, w_4 > 0$.

To find the value of **b** as per theorem 3, we let $k = 3, i = 3, 4$. When $k = 1, 2, 4, 5$ and $i = 1, 2, 5$, the second derivative of x_i and $x_j$will be zero.

$$v_3 w_3 \frac{df_3}{dx_3 d_{\beta^*}} = \frac{\Lambda}{\mu} v_3 w_4 \frac{df_3}{dx_4 b_{\beta^*}} = \frac{\eta_1 \Lambda}{\mu} \tag{3.41}$$

From equation (3.41) the expression of **b** is;

$$\mathbf{b} = v_3 \frac{\Lambda}{\mu}(w_3 + w_4\eta_1) > 0. \tag{3.42}$$

Hence from theorem 8 item iv, β^* changes from negative to positive, 0 changes its stability from stable to unstable. This means a negative unstable equilibrium becomes positive and locally asymptotically stable.

3.1.10 Sensitivity Analysis of the Model

The Partial Rank Correlation coefficient (PRCC) is one of the efficient methods used for testing the sensitivity analysis of the parameters. We use Latin Hypercube Sampling (LHS) to sample the parameters as it densely stratifies the input parameters. PRCC measures the strength between the outputs and inputs of the model correlation through sampling done by LHS method (Bidah et al., 2020; Pennington, 2015).

Our model has sixteen parameters and we desire to determine the most significant parameters i.e the parameters that impact the model output significantly. We performed 1000 runs in our simulations. The parameter with large PRCC values ($0.5 >$ or $-0.5 <$) are deemed the most influential in the model. The closer the PRCC value is to +1 or -1 the more strongly the parameter influences the outcome measure. A negative sign indicates that the parameter is inversely proportional to the outcome measure (Gomero, 2012).

Table 3.3: Sensitivity Analysis Index of Model 1

Serial No	Parameter	600 run
1	Λ	0.0228
2	μ	-0.4057
3	β	0.9310
4	β_m	-0.5145
5	α_1	0.0351
6	α_2	-0.2562
7	σ_1	0.9239
8	σ_2	-0.1038
9	τ_1	-0.6327
10	τ_2	-0.6713
11	δ	-0.8454
12	θ_1	-0.7394
13	θ_2	-0.0321
14	ρ	0.5720
15	ω	-0.0135
16	η_1	-0.0507

The parameters on the left of Figure 3.2 which have sensitivity index greater than 0.5 are $\beta_m, \tau_1, \tau_2, \delta$ and θ_1. Increasing any of these parameters decrease the reproduction number R_0 hence decrease alcohol consumption in the community. The parameters on the right of Figure 3.2 have positive sensitivity analysis index, which means that increase their values increase the reproduction number hence increase the alcohol in the community. These parameters that sensitivity analysis index less than -0.5 are β, σ_1 and ρ.

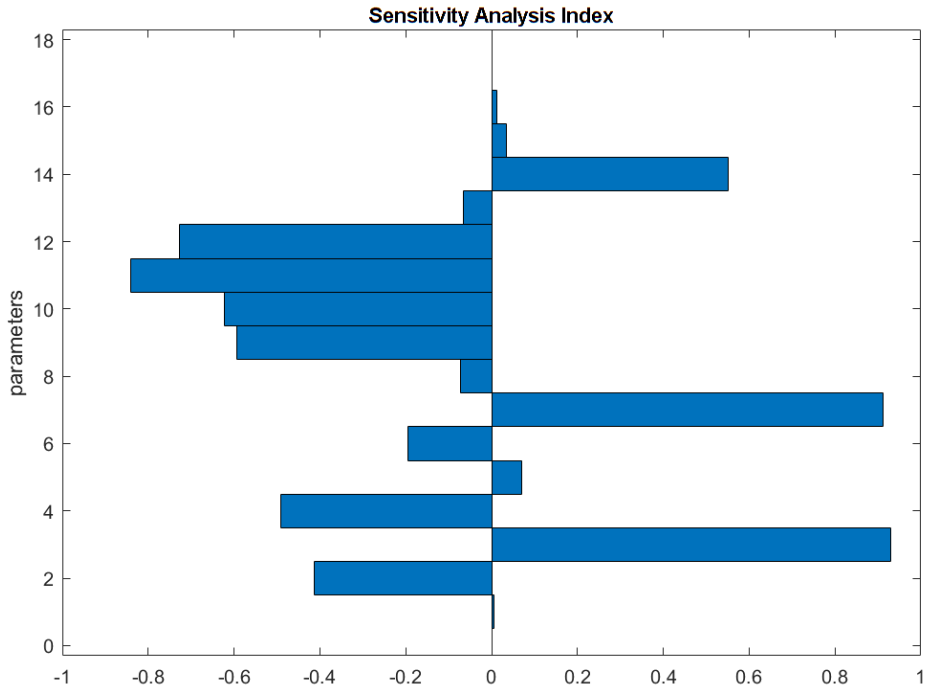

Figure 3.2: Tornado Plots for model 1

3.2 Model 2

In this section, we consider model 2 which has imperfect or ineffective mass media campaign. When individuals are exposed to mass media campaign against alcohol abuse, some still get into alcohol drinking habits and while others get to susceptible class. The population that get to drinking class after exposure to mass media campaign against alcohol abuse shows that the campaigns were not effective. Another fraction of the population effects the campaigns and do not join the drinking class but gets back to the susceptible class.

3.2.1 Model Formulation

This section involves model assumptions, model descriptions, model flow chart and model equations.

3.2.2 Model Description

Our study uses seven compartments, six human population-based compartments and one media compartment. The population-based compartments are S - Sus-

ceptible 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 centres and Q -individuals who have stopped drinking permanently. The media compartment M - is the density of the media campaign. The progression from one class to another will be formulated into seven nonlinear ordinary differential equations as shown in Figure 3.2.

Individuals are recruited into the model at a rate of Λ. Individuals are initiated to alcohol drinking due to contact with the light drinkers at a rate of λ where λ is given by $\beta(L+\eta_1 H)$ and β is the effective contact rate of light and heavy drinkers with the non drinking classes and η_1 is the rate of the heavy drinkers contacting the susceptible which is always less than the rate of contact of the light drinkers with the susceptible. Hence η_1 is always less than one. The rate of dissemination of media awareness to the susceptible is β_m. The human population die due to natural causes at a rate μ and they die due to alcohol related causes at a rate δ. The rate of increase of alcohol intake from a few drinks a week to dependence to alcohol is α_1 and light drinkers quit drinking at a rate of α_2. The rate at which heavy drinkers seek treatment or go to rehabilitation centers for treatment is σ_1 and the rate at which heavy drinkers quit drinking without treatment is σ_2, where $\sigma_2 << \sigma_1$ because very few addicts quit drinking without treatment. The effective treatment rate of the heavy drinking class is τ_1, which partly due to media awareness programs. The rate of relapse after rehabilitation back to heavy drinking is τ_2. The rate of the media awareness campaign on the light drinkers is represented by θ_1 and θ_2 represents the rate of media awareness programs of the heavy drinking classes which will increase the rate of joining the treatment class. ρ represents the depletion(depreciation) of the media campaigns due to ineffectiveness of the programs or other factors. The rate of effective media campaigns against alcohol is denoted by ω. The efficacy(effectiveness) of the media campaign is measured by ϵ. Table 3.4 gives the variables and Table 3.5 gives the summary of the parameters

and their description used in our research.

Table 3.4: Definitions of Variables

Variables	Definitions
S	Susceptible who have never drank alcohol
S_a	Susceptible who are pre - exposed to effects of alcohol abuse
L	Light drinkers who drink about five drinks, two or three times a week
H	Heavy drinkers who drink daily and dependent on alcohol
T	Individuals on rehabilitation and not exposed to alcohol
Q	Individuals who completely quit alcohol and leave the model
M	Awareness programs driven by mass media

Table 3.5: Parameters and their Description

Parameter	Description
Λ	Recruitment rate of drinkers into population
μ	Natural death rate
λ	Contact rate of susceptible with the light drinkers
β_2	Effective contact rate
β_m	Rate of dissemination of media awareness of the susceptible
α_1	Transfer rate of light drinkers to heavy drinkers
α_2	Rate of quitting alcohol of the light drinkers
σ_1	Treatment rate of the heavy drinkers (rate of joining rehabilitation)
σ_2	Rate of heavy drinkers quitting the drink
τ_1	Rate of effective treatment
τ_2	Rate of relapse back to heavy drinking from rehabilitation
δ	Death rate due to alcohol abuse
θ_1	Rate of awareness programs being implemented on the light drinkers
θ_2	Rate of awareness programs being implemented on the heavy drinkers
ρ	Rate of depletion of media programs
ω	Rate of effective (successful) media campaign
η_1	Modification Parameter
ϵ	Efficacy(effectiveness) of media Campaign

3.2.3 Model Assumptions

These are the assumptions of model 2.

- There is exposure to media campaign before initiation to alcohol, which implies that only the susceptible are influenced to drink alcohol 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.
- When individuals are exposed to media campaign, they may become alcoholic or not, where this rate is determined by ϵ and $0 \leq \epsilon \leq 1$. When $\epsilon = 1$, we have model 1 and when $\epsilon = 0$ the campaign is ineffective.
- Those in rehabilitation and quitters do not cause initiation and media campaign depletes the rate of initiation.
- When the individuals relapse, they move to the heavy drinking class.

3.2.3.1 Model Flow Charts and Equations of Model 2

From Figure 3.3 we formulate linear differential equations below.

$$\left.\begin{aligned}
\frac{dS}{dt} &= \Lambda + \omega S_a - \beta_m SM - (\lambda + \mu)S \\
\frac{dS_a}{dt} &= \beta_m SM - (\omega + \mu)S_a - \lambda(1-\epsilon)S_a \\
\frac{dL}{dt} &= \lambda S + \lambda(1-\epsilon)S_a - (\mu + \alpha_1 + \alpha_2)L \\
\frac{dH}{dt} &= \alpha_1 L + \tau_2 T - (\mu + \sigma_1 + \sigma_2 + \delta)H \\
\frac{dT}{dt} &= \sigma_1 H - (\mu + \tau_1 + \tau_2)T \\
\frac{dQ}{dt} &= \alpha_2 L + \sigma_2 H + \tau_1 T - \mu Q \\
\frac{dM}{dt} &= \theta_1 L + \theta_2 H - \rho M
\end{aligned}\right\} \tag{4.1}$$

Where $\lambda = \beta(L + \eta_1 H)$. To make the mathematical analysis easier and convenient, we introduce the following: $\Phi_1 = \omega + \mu, \quad \Phi_2 = 1 - \epsilon, \quad \Phi_3 = \mu + \alpha_1 + \alpha_2, \quad \Phi_4 = \mu + \sigma_1 + \sigma_2 + \delta, \quad \Phi_5 = \mu + \tau_1 + \tau_2, \quad \Phi_6 = \rho$

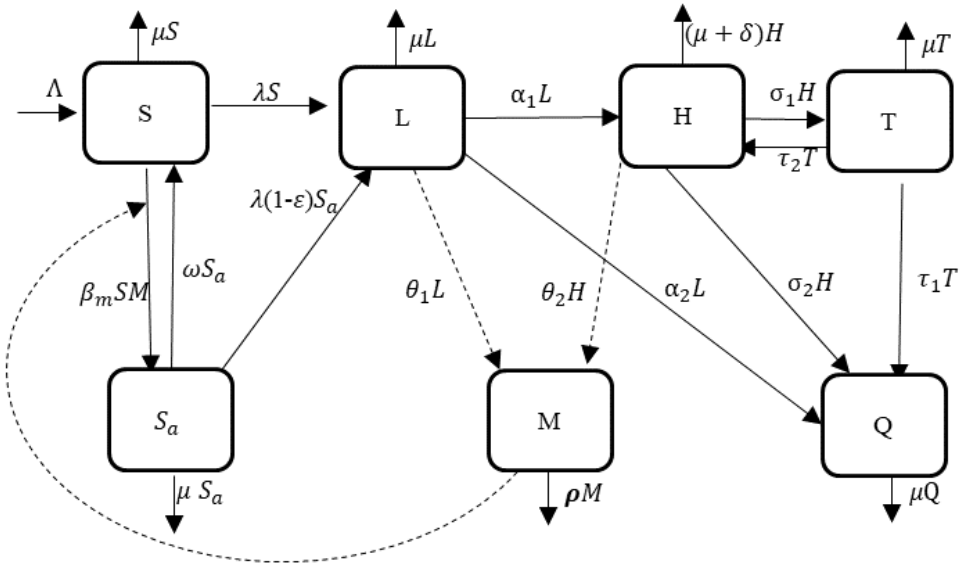

Figure 3.2: Flow chart of ineffective mass media campaign

3.2.4 Model Analysis

In this section we consider model analysis of model 2 which include: invariant region, positivity of the model, alcohol abuse reproduction number, alcohol free equilibrium(AFE),local and global stability of AFE, endemic equilibrium point, the bifurcation of the model and sensitivity analysis of the model.

3.2.5 Invariant Region and Positivity

We use the following Lemma to show the region where our model epidemiologically meaningful and if it is well posed.

Lemma 1. *The feasible region* Ω_1 *is defined by the set*

$$\Omega_1 = \left\{ (S, S_a, L, H, T, Q) \in R_+^6 : 0 \leq N(t) \leq \frac{\Lambda}{\mu} \right\}.$$

with initial data $S > 0, S_a, L > 0, H > 0, T > 0, Q > 0, M > 0$*, which is positively invariant for all* $t \geq 0$*.*

Proof. The sum of the human compartments give the total population N, where $N = S + S_a + L + H + T + Q$. Taking the sum of the derivatives we obtain

$$\frac{dN}{dt} = \Lambda - \mu N - \delta H. \tag{4.2}$$

Since $\delta H \geq 0$ for all $t \geq 0$ Equation (4.2) can be re-written as

$$\frac{dN}{dt} \leq \Lambda - \mu N. \tag{4.3}$$

Integrating equation (4.3) using integrating factor $e^{\mu t}$, we obtain

$$N(t) = \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}. \tag{4.4}$$

As $t \to \infty$, we get $\limsup_{t \to \infty} N(t) \leq \frac{\Lambda}{\mu}$. This, means that the region Ω_1 is attracting all the solutions in $\mathbb{R}^6_+$, which gives the feasible solution set of the model system (4.1) as;

$$\Omega_1 = \left\{ (S, S_a, L, H, T, Q) \in R^6_+ : 0 \leq N(t) \leq \frac{\Lambda}{\mu} \right\}. \tag{4.5}$$

If $N(0) \leq \frac{\Lambda}{\mu}$, then $N(t) \leq \frac{\Lambda}{\mu}$, as t tends to ∞, and if $N(0) > \frac{\Lambda}{\mu}$, then it implies that either the solution enters Ω_1 in finite time or $N(t)$ approaches $\frac{\Lambda}{\mu}$ asymptotically. Therefore the region Ω_1 is mathematically well-posed and epidemiologically meaningful. In addition, the usual existence, uniqueness and continuation result hold for the model system (4.1). □

To show the **Positivity of the model** we use the following theorem.

Theorem 4. *If all parameters of the model system 4.1 are positive and the initial conditions satisfy,*

$$\{(S(0), S_a(0), L(0), H(0), T(0), Q(0), M(0)) \geq 0\} \in \Omega_1,$$

then the solutions set $\{S(t), S_a(t), L(t), H(t), T(t), Q(t), M(t)\}$ *of the model system* (4.1) *is non-negative for all* $t \geq 0$.

Proof. From the first equation of the model system (4.1), we have

$$\frac{dS}{dt} = \Lambda + \omega S_a - \beta_m SM - (\lambda + \mu)S. \tag{4.6}$$

$\omega S_a \geq 0$ for all $t \geq 0$ equation (4.6) can be re-written as

$$\frac{dS}{dt} \leq \Lambda - \zeta S - \mu S, \tag{4.7}$$

where $\zeta = \lambda + \beta_m M$. The expression in 4.7 can be written as

$$\frac{d}{dt}\left(S \exp\left\{\int_0^t \zeta(u)du + \mu t\right\}\right) \leq \Lambda \exp\left\{\int_0^t \zeta(u)du + \mu t\right\}, \tag{4.8}$$

Integrating both sides of (4.8) from 0 to t, we obtain

$$S(\hat{t}) \exp\left\{\int_0^t \zeta(u)du + \mu t\right\} - S(0) \leq \int_0^t \Lambda \exp\left\{\int_0^x \zeta(x)dx + \mu y\right\} dy, \tag{4.9}$$

then multiplying both sides of (4.9) by $\exp\left\{-\int_0^{t^*} \zeta(u)du - \mu_v t\right\}$, we have

$$\begin{aligned} S(\hat{t}) \leq S(0) \exp\left\{-\int_0^t \zeta(u)du - \mu t\right\} + \exp\left\{-\int_0^t \zeta_v(u)du - \mu\hat{t}\right\} \\ \times \int_0^{\hat{t}} \Lambda \exp\left\{\int_0^x \zeta(x)dx + \mu y\right\} dy > 0. \end{aligned} \tag{4.10}$$

Since, the right-hand side of the expression (4.10) is always positive, the solution $S(t)$ will always remain positive for all $t > 0$. Using the same argument, it can be shown that the quantities S_a, L, H, T, Q and M are positive for all $t > 0$. □

3.2.6 Alcohol Free Equilibrium (AFE) and Alcohol Reproduction Number

The AFE of the system (4.1) given by $E_a^0 = \{S^0, S_a^0, L^0, H^0, T^0, Q^0, M^0\}$ is obtained by equating all the alcohol classes to zero and solving the equations. We assume there is no alcohol in the community. This means that $L = H = T = Q =$

$M = 0$ and $S_a = 0$, hence;

$$S^0 = \frac{\Lambda}{\mu}.$$

The AFE is given by

$$E_a^0 = \{S^0, S_a^0, L^0, H^0, T^0, Q^0, M^0\} = \{\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0\} \tag{4.11}$$

We use the next generation method as in (Castillo-Chavez et al., 2002), to find the **alcohol abuse reproduction number** R_{0a}. The reproduction number R_{0a} is worked from the largest eigenvalue of the matrix $F.V^{-1}$, where, F represent the new infection and V represent transfer of infection. In our model, equations of F are given by, $f_1 = \beta_2(L + \eta_1 H)S^0, f_2 = 0, f_3 = 0$, where $S^0 = \frac{\Lambda}{\mu}$ at AFE point. Since there no new infections with an ineffective media campaign, the reproduction number of model 2 is similar to that of model 1. Finding the partial derivatives of F with respect to L, H, T we obtain

$$F = \begin{pmatrix} \beta_2\frac{\Lambda}{\mu} & \beta_2\frac{\eta_1\Lambda}{\mu} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \Phi_6 & 0 & 0 \\ -\alpha_1 & \Phi_3 & -\tau_2 \\ 0 & -\sigma_1 & \Phi_4 \end{pmatrix}.$$

Therefore the alcohol abuse reproduction number is;

$$R_{0a} = \frac{\beta\Lambda(\Phi_3(1-\Psi_1) + \alpha_1\eta_1)}{\Phi_3\Phi_6\mu(1-\Psi_1)}, \text{ where } \Psi_1 = \frac{\delta_1\tau_2}{\Phi_3\Phi_4}. \tag{4.12}$$

From (4.12), Ψ_1 refers to the proportion of individuals who move from H to T and

back. On the other hand $(1 - \Psi_1)$ are the individuals who do not cycle between the two compartments.

3.2.7 Local and Global Stability of AFE

Theorem 5. *The AFE point* (E_a^0) *is locally asymptotically stable if* $R_{0a} < 1$ *and unstable if* $R_{0a} > 1$.

Proof. To prove the theorem, we obtain the Jacobian matrix of the system 4.1 at E_a^0. We let $\Phi_1 = \omega + \mu$, $\Phi_2 = 1 - \epsilon$, $\Phi_3 = \mu + \sigma_1 + \sigma_2 + \delta$, $\Phi_4 = \mu + \alpha_1 + \alpha_2$, $\Phi_5 = \rho$, $\Phi_6 = \mu + \tau_1 + \tau_2.$, hence our system 4.1reduces to:

$$\left.\begin{aligned} \frac{dS}{dt} &= \Lambda + \omega S_a - \beta_m SM - (\lambda + \mu)S \\ \frac{dS_a}{dt} &= \beta_m SM - \phi_1 S_a - \lambda(1 - \epsilon)S_a \\ \frac{dL}{dt} &= \lambda S + \lambda\phi_2 S_a - \phi_4 L \\ \frac{dH}{dt} &= \alpha_1 L + \tau_2 T - \phi_3 H \\ \frac{dT}{dt} &= \sigma_1 H - \phi_6 T \\ \frac{dQ}{dt} &= \alpha_2 L + \sigma_2 H + \tau_1 T - \mu Q \\ \frac{dM}{dt} &= \theta_1 L + \theta_2 H - \phi_5)M \end{aligned}\right\} \tag{4.13}$$

The Jacobian of equation 4.1 is given by;

$$J_{E_a^0} = \begin{pmatrix} -\mu & \omega & \frac{-\Lambda\beta}{\mu} & \frac{-\Lambda\beta\eta_1}{\mu} & 0 & 0 & \frac{-\beta_m\Lambda}{\mu} \\ 0 & -\Phi_1 & 0 & 0 & 0 & 0 & \frac{\beta_m\Lambda}{\mu} \\ 0 & 0 & \frac{\Lambda\beta}{\mu} - \Phi_6 & \frac{\Lambda\beta\eta_1}{\mu} & 0 & 0 & 0 \\ 0 & 0 & \alpha_1 & -\Phi_3 & \tau_2 & 0 & 0 \\ 0 & 0 & 0 & \sigma_1 & -\Phi_4 & 0 & 0 \\ 0 & 0 & 0 & \alpha_2 & \sigma_2 & -\mu & 0 \\ 0 & 0 & \theta_1 & \theta_2 & 0 & 0 & -\phi_5 \end{pmatrix} \tag{4.14}$$

From equation 4.14 it can easily be seen that $-\mu$, $-\Phi_1$ and ϕ_5 are the first three eigenvalues which have negative real parts. The remaining eigenvalues are obtained from the following reduced matrix in equation 4.15.

$$J_{E_a^0} = \begin{pmatrix} \frac{\Lambda\beta}{\mu} - \Phi_6 & \frac{\Lambda\beta\eta_1}{\mu} & 0 \\ \alpha_1 & -\Phi_3 & \tau_2 \\ 0 & \sigma_1 & -\Phi_4 \end{pmatrix} \tag{4.15}$$

The characteristic polynomial of (4.15) is given by

$$y(\nu) = \nu^3 + a_1\nu^2 + a_2\nu + a_3, \tag{4.16}$$

where

$$
\begin{aligned}
a_1 &= -\frac{\beta\Lambda}{\mu} + \phi_3 + \phi_4 + \phi_6 \\
a_2 &= -\frac{\alpha_1\beta\Lambda\eta_1}{\mu} - \frac{\beta\Lambda\phi_3}{\mu} - \frac{\beta\Lambda\phi_4}{\mu} - \sigma_1\tau_2 + \phi_3\phi_4 + \phi_3\phi_6 + \phi_4\phi_6, \\
a_3 &= -\frac{\alpha_1\beta\Lambda\eta_1\phi_4}{\mu} + \frac{\beta\Lambda\sigma_1\tau_2}{\mu} - \frac{\beta\Lambda\phi_3\phi_4}{\mu} - \sigma_1\tau_2\phi_6 + \phi_3\phi_4\phi_6.
\end{aligned}
$$

We can write a_3 in terms of R_{0a} as

$$
a_3 = (1 - \psi_1)\,\phi_3\phi_4\phi_6\,(1 - R_{0a})\,.
$$

We then use Routh-Hurwitz criterion (Routh, 1877), to establish the necessary and sufficient conditions for all the roots of $y(\nu)$ to have negative real parts. The Routh-Hurwitz criterion of stability of the AFE is given by

$$
\begin{cases} H_1 > 0 \\ H_2 > 0 \\ H_3 > 0 \end{cases} \Longleftrightarrow \begin{cases} H_1 > 0 \\ H_2 > 0\,, \\ H_3 > 0 \end{cases}
$$

where

$$
H_1 = a_1, \quad H_2 = \begin{vmatrix} a_1 & 1 \\ a_3 & a_2 \end{vmatrix}, \quad H_3 = \begin{vmatrix} a_1 & 1 & 0 \\ a_3 & a_2 & a_1 \\ 0 & 0 & a_3 \end{vmatrix}.
$$

We then have

$$
\begin{aligned}
H_1 &= a_1 = -\frac{\beta\Lambda}{\mu} + \phi_3 + \phi_4 + \phi_6, \\
H_2 &= a_1 a_2 - a_3 = \left(-\frac{\beta\Lambda}{\mu} + \phi_3 + \phi_4 + \phi_6\right) \\
&\quad \left(-\frac{\alpha_1\beta\Lambda\eta_1}{\mu} - \frac{\beta\Lambda\phi_3}{\mu} - \frac{\beta\Lambda\phi_4}{\mu} - \sigma_1\tau_2 + \phi_3\phi_4 + \phi_3\phi_6 + \phi_4\phi_6\right) \\
&\quad - (1 - R_0)(1 - \psi_1)\phi_3\phi_4\phi_6 \\
H_3 &= a_1 a_2 a_3 - a_3^2 = (1 - R_0)\phi_3\phi_4\phi_6\left(-\frac{\beta\Lambda}{\mu} + \phi_3 + \phi_4 + \phi_6\right) \\
&\quad (1 - \psi_1)\left(-\frac{\alpha_1\beta\Lambda\eta_1}{\mu} - \frac{\beta\Lambda\phi_3}{\mu} - \frac{\beta\Lambda\phi_4}{\mu} - \sigma_1\tau_2 + \phi_3\phi_4 + \phi_3\phi_6 + \phi_4\phi_6\right) \\
&\quad - (1 - R_0)^2 (1 - \psi_1)^2 \phi_3^2\phi_4^2\phi_6^2.
\end{aligned}
$$

The above result shows that we have $H_1 > 0$, $H_2 > 0$ and $H_3 > 0$ if and only if $R_{0a} < 1$. Therefore we conclude that the AFE is locally asymptotically stable whenever $R_{0a} < 1$. □

We use the theorem by (Castillo-Chavez and Song, 2004) as used by (Tilahun et al., 2018), to investigate the **global stability** of AFE of system (4.1).

Theorem 6. *The fixed point $\widetilde{U_0} = (X^*, 0) = (\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0)$ is globally asymptotically stable equilibrium of the model 2, if $R_{0a} < 1$ (locally asymptotically stable) and assumption $(H1)$ and $(H2)$ are satisfied where $(H1)$ for $\frac{dX}{dt} = F(X, 0), X^0$ is globally asymptotically stable.*

$(H2), G(X, Z) = AZ - \widetilde{G}(X, Z), G(X, Z) \geq 0$ 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.

The equation 4.1 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. $G(X, 0) = 0, \widetilde{U_0} = (X^*, 0) = (\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0)$ denotes the AFE point of the model.

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

$$A = \begin{pmatrix} \beta_2 - k_1 & \beta_2\eta_1 & 0 \\ \alpha_1 & -k_2 & \tau_2 \\ 0 & \sigma_1 & -k_3 \end{pmatrix}$$

$$AZ = \begin{pmatrix} (\beta_2 - k_1)L + (\beta_2\eta_1)H \\ \alpha_1 L - k_2 H + \tau_2 T \\ \sigma_1 H - k_3 T \end{pmatrix}$$

$$G(X,Z) = \begin{pmatrix} \lambda S - k_1 L \\ \alpha_1 L - k_2 H + \tau_2 T \\ \sigma_1 H - k_3 T \end{pmatrix}$$

$$\widetilde{G}(X,Z) = AZ - G(X,Z)$$

$$\widetilde{G}(X,Z) = \begin{pmatrix} \beta_2(L + \eta_1 H)(1 - S^0) \\ 0 \\ 0 \end{pmatrix}$$

The matrix for $\widetilde{G}(X,Z)$ reduces to:

$$\widetilde{G}(X,Z) = \begin{pmatrix} \beta_2(L+\eta_1 H)(1-\frac{S}{N}) \\ 0 \\ 0 \end{pmatrix}$$

Since $S \leq N$, $\frac{S}{N} \leq 1$, it is clear that $\widetilde{G}(X,Z) \geq 0$. It is also clear that $X^* = (\frac{\Lambda}{\mu}, 0, 0)$ is globally asymptotically stable equilibrium of $\frac{dX}{dt} = F(X,0)$. Hence the model is globally asymptotically stable. □

3.2.8 Endemic Equilibrium Point (EEP)

Endemic equilibrium of our model occurs when alcohol persists in the community. This equilibrium is denoted by $E_a^* = \{S^*, S_a^*, L^*, H^*, T^*, Q^*, M^*\}$. To obtain E_a^*, we equate the RHS of system 4.1 to zero. After solving the system in terms of L^* we obtain;

$$\left.\begin{aligned} S^* &= \frac{\omega S_a^* + \Lambda}{\beta\eta_1 H^* + \mu + \beta L^* + \beta_m M^*}, \quad H^* = \frac{\alpha_1 L^* + \tau_2 T^*}{\Phi_3}, \\ T^* &= \frac{\alpha_1 L^* \sigma_1}{\Phi_3\Phi_4(1-\Psi_1)}, \\ S_a^* &= \frac{\Lambda\beta_m M^*}{() \left(\beta\Phi_2\lambda^* + \Phi_1\right) + M^*\beta_m \left(\beta\Phi_2\lambda^* + \Phi_1 - \omega\right)}, \\ Q^* &= \frac{L^* \left(\alpha_1 \left(\sigma_1\tau_1 + \sigma_2\Phi_4\right) + \Phi_3\Phi_4\alpha_2 \left(1-\Psi_1\right)\right)}{\Phi_3\Phi_4\mu \left(1-\Psi_1\right)}, \\ M^* &= \frac{L^*(\Phi_4(\theta_1\Phi_3(1-\Psi_1) + \alpha_1\theta_2))}{\Phi_3\Phi_4\Phi_5 \left(1-\Psi_1\right)}. \end{aligned}\right\} \tag{4.17}$$

Substituting the solutions of the state variables in (4.17) into the third equation of (4.1) we get;

$$A_2 L^{*2} + A_1 L^* + A_0 = 0, \tag{4.18}$$

where,

$$\left.\begin{aligned} A_2 &= \beta\Phi_2\Phi_6(\Phi_4(\Phi_3(1-\Psi_1)+\alpha_1\eta_1))(\beta\Phi_5(\Phi_4(\Phi_3(1-\Psi_1)+\alpha_1\eta_1)) \\ &\quad + \beta_m(\Phi_4(\theta_1\Phi_3(1-\Psi_1)+\alpha_1\theta_2))), \\ A_1 &= -[\beta_m(\Phi_1-\omega)(\Phi_4(\alpha_1\theta_2+\Phi_3\theta_1(1-\Psi_1)))+ \\ &\quad \beta\Phi_5(\mu\Phi_2+\Phi_1)(\Phi_4(\alpha_1\eta_1+\Phi_3(1-\Psi_1))] \\ &\quad + \mu\Phi_2R_{0a}(\beta\Phi_5(\Phi_4(\alpha_1\eta_1+\Phi_3(1-\Psi_1))+\beta_m(\Phi_4(\alpha_1\theta_2+\theta_1\Phi_3(1-\Psi_1))))), \\ A_0 &= \mu\Phi_1\Phi_5\Phi_6\Phi_3^2\Phi_4^2\,(1-\Psi_1)^{\,2}(1-R_{0a}). \end{aligned}\right\}$$

Note that A_2 is positive, and that A_1 may be rearranged as:

$$A_1 = \left[\frac{\mu\Phi_2R_{0a}(\beta\Phi_5(\Phi_4(\alpha_1\eta_1+\Phi_3\phi_7)+\beta_m(\Phi_4(\alpha_1\theta_2+\theta_1\Phi_3\phi_7)))}{\beta_m(\Phi_1-\omega)(\Phi_4(\alpha_1\theta_2+\Phi_3\theta_1\phi_7))+\beta\Phi_5(\mu\Phi_2+\Phi_1)(\Phi_4(\alpha_1\eta_1+\Phi_3\phi_7)} - 1\right],$$

where $\phi_7 = 1-\psi_1$ It follows that:

(i) There is a unique endemic equilibrium if $A_0 < 0$ (i.e. if $R_{0a} > 1$);

(ii) There is a unique endemic equilibrium if $A_1 < 0$; and $A_0 = 0$; or $A_1^2 - 4A_2A_0 = 0$;

(iii) There are two endemic equilibria if $A_0 > 0$; $A_1 < 0$ and $A_1^2 - 4A_2A_0 > 0$;

(iv) There are no endemic equilibria otherwise.

We need to note that equation (4.1) has a backward bifurcation at $R_{0a} = 1$ if and only if $A_0 < 0$ and $A_1^2 - 4A_0A_2 > 0$, (Omondi et al., 2017). This means that the conditions that $R_{0a} < 1$ is no longer applicable for a situation where alcohol is eradicated in the community. For us to achieve the this goal, we have to bring R_{0a} below the critical values R_c. To obtain R_c, we equate the discriminate of the equation (4.18) to zero and make R_{0a} the subject. Thus R_c is given by;

$$R_c = \left[1 - \frac{A_1^2}{4A_2\mu\Phi_1\Phi_3^2\Phi_4^2\Phi_5\Phi_6\,(1-\Psi_1)^{\,2}}\right].$$

We note that the hypothesis $A_0 > 0$ is equivalent to $R_{0a} < 1$, and the hypothesis

$A_1 < 0$ is equivalent to $R_{0a} > R_c$. The results of this section may be summarized in the following theorem.

Theorem 7. *If $R_{0a} < 1$, then E_0 is an equilibrium of system (4.1) and it is locally asymptotically stable. Furthermore, there exists an endemic equilibrium if conditions in item (ii) are satisfied, or two endemic equilibria if conditions in item (iii) are satisfied. If $R_{0a} > 1$, then E_0 is unstable and there exists a unique endemic equilibrium.*

3.2.9 Bifurcation of the Model

Here, we prove that the occurrence of multiple alcohol persistent equilibria for $R_{0a} < 1$ comes from a backward bifurcation. We investigate the nature of the bifurcation by using the method introduced in (Castillo-Chavez and Song, 2004), which is based on the centre manifold theory (Lee and Milgram, 1983; Omondi et al., 2018b,a). In the centre manifold theorem, there are two important quantities: the coefficients, say a and b of the normal form representing the dynamics of the system on the central manifold. These coefficients give information on the direction of the transcritical bifurcation. In particular, if $a < 0$ and $b > 0$, then the bifurcation is forward; if $a > 0$ and $b > 0$, then the bifurcation is backward. Using this approach, the following result is may be obtained.

Theorem 8. *The system (4.1) has a backward bifurcation at $\mathcal{R}_{0a} = 1$ if and only if $A_1 < 0$ and $A_1^2 - 4A_2A_0 > 0$.*

Proof. Considering $R_{a0} = 1$, let β^* be given by

$$\beta^* = \frac{\Phi_3\Phi_4\mu(1-\Psi_1)}{\Lambda(\Phi_4(1-\Psi_1)+\alpha_1\eta_1)}.$$

The Jacobian matrix of the system (4.1) evaluated at AFE is as given in (4.14). The linearised system (4.14) with $\beta = \beta^*$ admits a simple zero eigenvalue and the other eigenvalues are real and negative. Denote by $\mathbf{v} = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)$ and $\mathbf{w} = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T$, a left and a right eigenvectors associated with

the zero eigenvalue, respectively, such that $\mathbf{v}.\mathbf{w} = 1$. It follows that:

$$\mathbf{v} = \begin{pmatrix} v_1 = v_2 = v_6 = v_7 = 0, \quad v_3 = 1, \quad v_4 = \dfrac{\eta_1\Phi_3\Phi_5}{\sigma(\mu+\tau_1) + \alpha_1\eta_1\Phi_5 + \Phi_5(\delta+\sigma_2+\mu)}, \\ \\ v_5 = \tau_2 v_4. \end{pmatrix}$$

$$\mathbf{w} = \begin{pmatrix} w_1 = -\mu\Phi_1\Phi_6\left(\dfrac{\theta_2\Lambda\Phi_5\psi\left(\alpha_1+\theta_1\Lambda\psi\right)}{\Phi_5\left(\delta+\mu+\sigma_2\right)+\sigma_1\left(\mu+\tau_1\right)} + \Phi_1\Phi_6\left(\alpha_1+\alpha_2+\theta_1\Lambda\psi+\mu\right)\right), \\ w_2 = \dfrac{\Lambda\psi\left(\alpha_1\theta_2\Phi_5 + \theta_1\left(\Phi_5\left(\delta+\mu+\sigma_2\right)+\sigma_1\left(\mu+\tau_1\right)\right)\right)}{\mu\Phi_1\Phi_6\left(\Phi_5\left(\delta+\mu+\sigma_2\right)+\sigma_1\left(\mu+\tau_1\right)\right)}, \quad w_3 = 1, \\ w_4 = \dfrac{\alpha_1\Phi_5}{\Phi_5\left(\delta+\mu+\sigma_2\right)+\sigma_1\left(\mu+\tau_1\right)}, \quad w_5 = \dfrac{\alpha_1\sigma_1}{\Phi_5\left(\delta+\mu+\sigma_2\right)+\sigma_1\left(\mu+\tau_1\right)}, \\ w_6 = \dfrac{\alpha_2\left(\Phi_5\left(\delta+\mu+\sigma_2\right)+\sigma_1\left(\mu+\tau_1\right)\right)+\alpha_1\left(\sigma_1\tau_1+\sigma_2\Phi_5\right)}{\mu\Phi_5\left(\delta+\mu+\sigma_2\right)+\mu\sigma_1\left(\mu+\tau_1\right)}, \\ w_7 = \dfrac{\alpha_1\theta_2\Phi_5+\theta_1\left(\Phi_5\left(\delta+\mu+\sigma_2\right)+\sigma_1\left(\mu+\tau_1\right)\right)}{\rho\left(\Phi_5\left(\delta+\mu+\sigma_2\right)+\sigma_1\left(\mu+\tau_1\right)\right)}. \end{pmatrix}^T$$

The system (4.1) can be written in the form $\frac{dx}{dt} = F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7)^t$. From (Castillo-Chavez and Song, 2004), the coefficients a and b are defined as

$$a = \sum_{k,i,j=1}^{3} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_1 \partial x_j}(E_0, \beta^*), \quad b = \sum_{k,i=1}^{3} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta^*}(E_0, \beta^*).$$

If we let $S = x_1, S_a = x_2, L = x_3, H = x_4, T = x_5, Q = x_6$ and $M = x_7$, It can be checked that:

$$\frac{\partial^2 f_3}{\partial x_1 \partial x_3} = \beta^*, \quad \frac{\partial^2 f_3}{\partial x_1 \partial x_4} = \eta_1\beta^*, \quad \frac{\partial^2 f_3}{\partial x_2 \partial x_3} = \Phi_2\beta^*, \quad \frac{\partial^2 f_3}{\partial x_2 \partial x_4} = \Phi_2\eta_1\beta^*,$$
$$\frac{\partial^2 f_3}{\partial x_3 \partial \beta^*} = \frac{\Lambda}{\mu}, \quad \frac{\partial^2 f_3}{\partial x_4 \partial \beta^*} = \frac{\eta_1\Lambda}{\mu}.$$

Taking into account of system (4.1) and considering only the nonzero components

of the left eigenvector $\mathbf{v}$, it follows that

$$
\begin{aligned}
a &= v_3 w_1 w_3 \frac{\partial^2 f_3}{\partial x_1 \partial x_3} + v_3 w_1 w_4 \frac{\partial^2 f_3}{\partial x_1 \partial x_4} + v_3 w_2 w_3 \frac{\partial^2 f_3}{\partial x_2 \partial x_3} + v_3 w_2 w_4 \frac{\partial^2 f_3}{\partial x_2 \partial x_4}, \\
&= -\Phi_3 \\
&\left(\frac{\alpha_1 + \alpha_2 + \mu}{\Lambda} + \frac{\beta_m (2\omega + \mu(2-\epsilon)) \left(\alpha_1 \theta_2 \Phi_5 + \theta_1 \left(\Phi_5 \left(\delta + \mu + \sigma_2\right) + \sigma_1 \left(\mu + \tau_1\right)\right)\right)}{\mu \Phi_1 \Phi_6 \left(\Phi_5 \left(\delta + \mu + \sigma_2\right) + \sigma_1 \left(\mu + \tau_1\right)\right)} \right) \\
&< 0, \\
b &= v_3 w_3 \frac{\partial^2 f_3}{\partial x_3 \partial \beta^*} + v_3 w_4 \frac{\partial^2 f_3}{\partial x_4 \partial \beta^*} = \frac{\Lambda}{\mu} \left(1 + \frac{\eta_1 \alpha_1 \Phi_5}{\Phi_5 \left(\delta + \mu + \sigma_2\right) + \sigma_1 \left(\mu + \tau_1\right)} \right) > 0.
\end{aligned}
$$

The value of $a < 0$ and $b > 0$, hence the system (4.1) exhibits backward bifurcation at $R_{0a} = 1$ (Theorem 8). □

3.2.10 Sensitivity Analysis of Model 2

We apply the method of (Bidah et al., 2020) ,(Pennington, 2015) and (Gomero, 2012) to analyze the sensitivity analysis of our model. They used PRCC to test the sensitivity analysis and LHS to sample the parameters.

Model 2 has seventeen parameters and determined the most influential parameters. We performed 1000 runs in our simulations. From Table 3.4 the parameters with the sensitivity values close to -1 and $+1$ are more significant than the parameters close to zero. Figure 3.3 shows the sensitivity analysis using bars on the left and right. The bars on the left have a negative sensitivity analysis index and are inversely proportional to the reproduction number. The parameters on the right have a positive sensitivity analysis index which implies that if their values are increased the reproduction number increases. It means that these values are more significant in increasing alcohol consumption in the community.

Table 3.6: Sensitivity analysis index of model 2

Serial No	Parameter	300 Run
1	Λ	0.0219
2	μ	-0.6111
3	β	0.5671
4	β_m	-0.0119
5	α_1	0.0629
6	α_2	-0.2923
7	σ_1	0.9492
8	σ_2	-0.0874
9	τ_1	-0.7389
10	τ_2	-0.7182
11	δ	-0.9058
12	Θ_1	-0.0291
13	Θ_2	-0.0507
14	ρ	-0.0080
15	ω	-0.0252
16	η_1	-0.0385
17	ϵ	-0.4499

The parameters with large negative sensitivity analysis index greater than -0.5 are $\alpha_1, \tau_1, \tau_2, \delta$ and ϵ. These parameters contribute greatly in reduction alcohol consumption in the community. The parameters with positive sensitivity analysis index are β and σ_1.

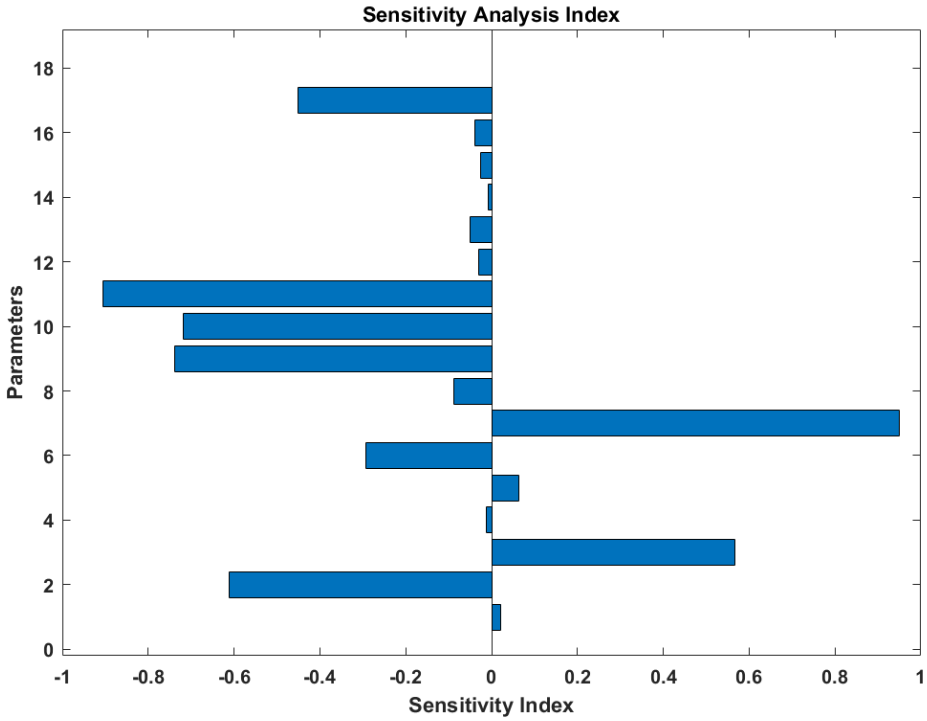

Figure 3.4: Tornado Plots for Model 2

CHAPTER FOUR: RESULT AND DISCUSSION

4.1 Parameters

In this chapter, we look at the numerical simulations and discussions of each model and then consider the comparative analysis of what is already done by other authors comparing to our work. For both models, we use the values in Table 4.1.

Table 4.1: Parameter Values and their Sources

Parameter	Value	Source
Λ	1674000	KKNB (2010)
μ	0.025	Assumed
β, β_2	0.00000002	Misra et al. (2011)
β_m	0.00005	Assumed
α_1	0.031	Assumed
α_2	0.07	Assumed
σ_1	0-0.09	Assumed
σ_2	0.05	Assumed
τ_1	0.1	Assumed
τ_2	0.2	Assumed
δ	0.2	Assumed
θ_1	0.0005	Misra et al. (2011)
θ_2	0.0001	Assumed
ρ	0.06	Misra et al. (2011)
ω	0.0002	Assumed
η_1	0.01	Assumed
ϵ	0-1	Assumed

4.2 Numerical Simulation of Model 1

To study the dynamics of system (3.1), we use MATLAB (ode45) software. We use parameters in Table 4.1. The initial population of S, S_a, L, H and Q are from (United Nations and Social Affairs, 2019). We collected secondary data from rehabilitation centers in Kenya and data from (NACADA, 2017) website to identify the initial population of the treatment class.

To estimate the initial conditions of the steady states we used the Kenyan population which is estimated to be approximately 52 million people according to United Nation Estimate population (2019) (United Nations and Social Affairs, 2019). 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 (NACADA, 2012). This translates to 15.6 million people in the classes $L + H + T + Q$ and about 2.028 million people addicted to alcohol. So we use the initial conditions are: $S(0) = 7280000$, $S_a(0) = 29100000$, $L(0) = 11570000$, $H(0) = 2028000$, $T(0) = 2000$, $Q(0) = 2000000$.

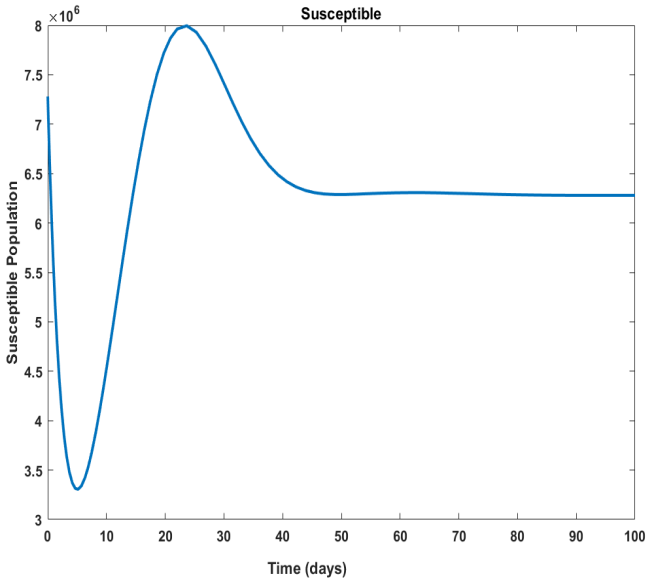

Figure 4.1: Susceptible population

Figure 4.1 represent a susceptible population which reduces sharply for the first

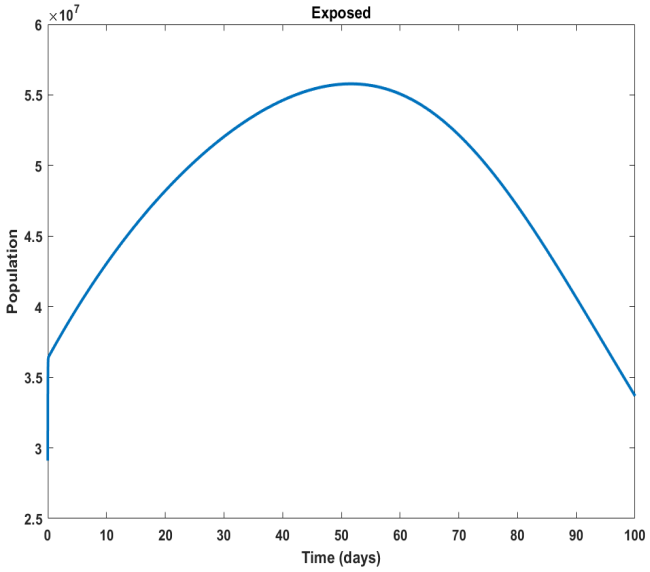

Figure 4.2: Exposed Population.

five days. This is due to the movement to the exposed after contact with media and others initiated to light drinking class. The population then increases for the next forty days and reduces again. This is because the population from the exposed get back to the susceptible classes after an effective media campaign.

Figure 4.2 represent the exposure to media class which increase for the first thirty days. This is due to the inflow from the susceptible class. The population then reduces as the exposed individual gradually move back to the susceptible class. These two classes have never taken alcohol in their lifetime.

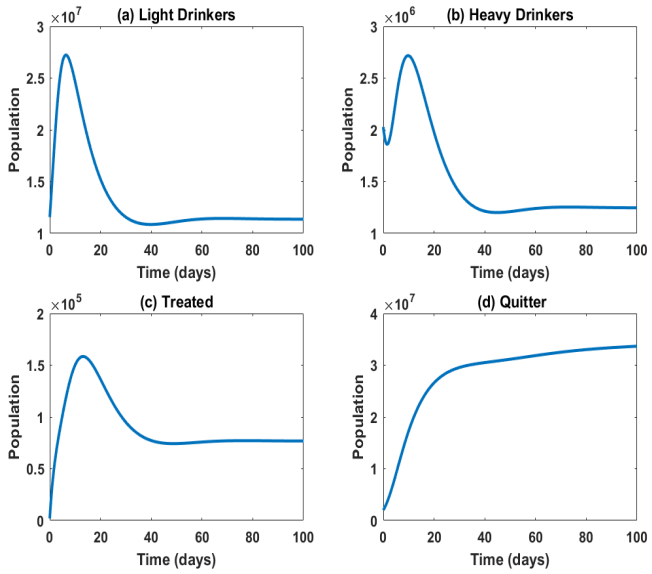

Figure 4,3: Alcoholic Classes for model 1

Figure 4.3 represent classes that have ever consumed alcohol in their lifetime. These are light drinkers (Figure 4.3 (a)), heavy drinkers (Figure 4.3 (b)), treated (Figure 4.3 (c))and the quitters (Figure 4.3 (d)). The number of light drinkers decreases for the first ten days. This is because they either move to heavy drinking class or quit drinking alcohol.

The heavy drinking class decrease with time due to the individuals being encouraged to go for treatment and others die due to alcohol-related complications.

The people under treatment increase sharply for the first ten days. This is because of the population from the heavy drinking class joining rehabilitation centres. The population then decreases due to effective treatment rate and others relapse back to heavy drinking class. The population then increases as the individuals get back to the class from heavy drinking class.

The quitters increase for the given time. This is because individuals quit or stop drinking from the light drinking class, heavy drinking class and mostly from the treatment class. This shows that when individuals quit drinking, alcohol abuse is greatly reduced in the community.

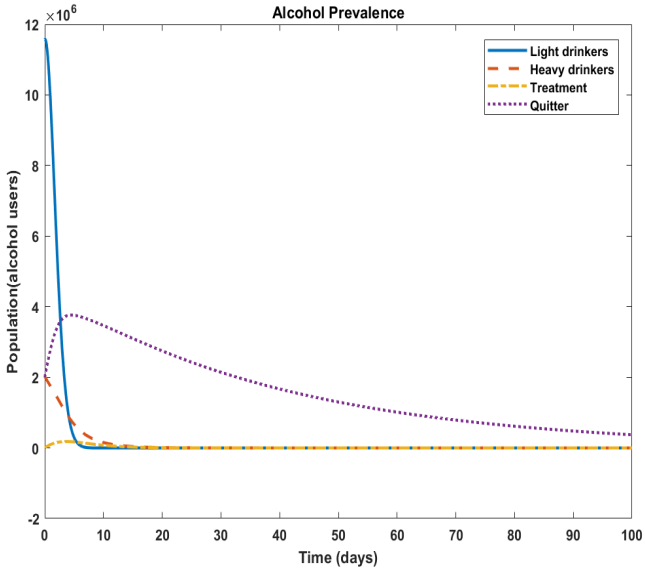

Figure 4.4: Alcohol Prevalence classes

Figure 4.4 represent the alcohol prevalence classes plotted in the same axes. The light drinkers decrease in the first few days because some individuals move to the heavy drinking class while many others move to quitters class. Light drinkers have a higher probability of quitting alcohol since they are not addicted to alcohol. The quitters class population increase in the first few days then decrease but the number in the class is more than the number in the heavy drinking class and also in the treatment class.

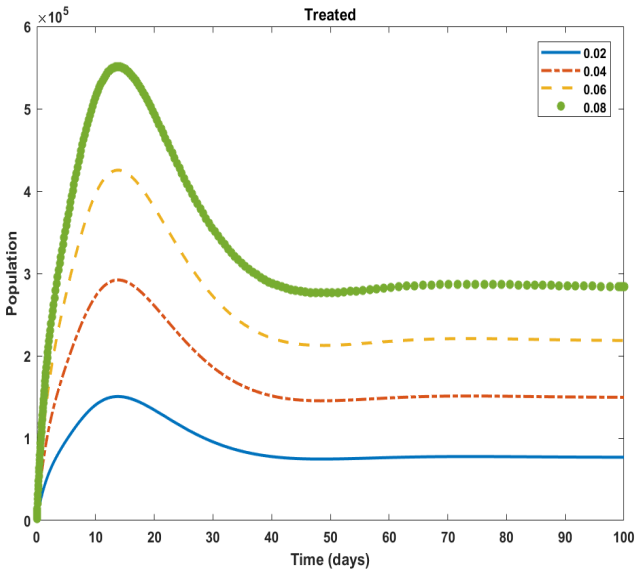

Figure 4.5: Varying alcohol treatment

In Figure 4.5, the treatment rate increases from $0.02 - 0.08$ where these numbers represent the rate at which the individuals join the treatment class. The treatment offered in the rehabilitation centres includes medication and counselling. When the rate of treatment is increased, the number in the treatment class increases then decrease and later stabilizes as they settle in the rehabilitation centres. The treatment time ranges from one month to three months. From the graph, we conclude that as we increase the rate of treatment the number of individuals under treatment increases.

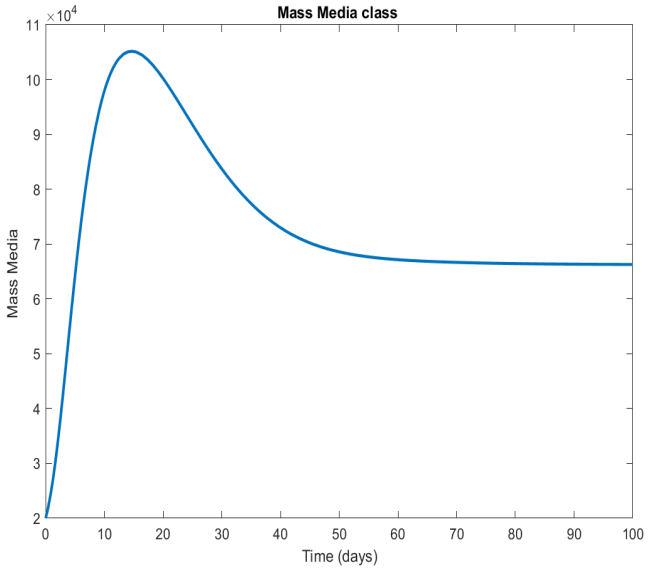

Figure 4.6: Mass media density for model 1

Figure 4.6 shows the rate of increase of media dissemination with time. The mass media campaign increase in the first days and then decrease up to the day thirty when the mass media campaign stabilizes. The y-axis includes all mass media campaigns. This means that this does not involve the human population but the actual mass media.

4.3 Numerical Simulation of Model 2

We carry out numerical simulation of model 2 using the parameters in table 4.1 and the initial conditions: $S = 7.28X10^6, S_a = 2.91X10^7, L = 11.57X10^7, H = 2.028X10^6, T = 2.0X10^3, Q = 2.0X10^6$. We use initial values $S = 7.28X10^6, S_a =$

$2.91X10^7, L = 11.57X10^7, H = 2.028X10^6, T = 2.0X10^3, Q = 2.0X10^6.$

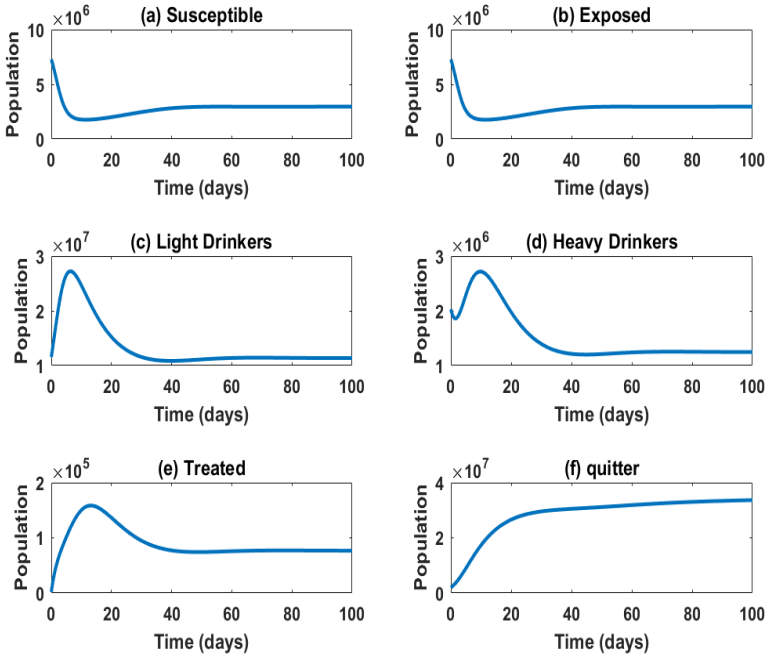

Figure 4.7: All Human Population

Figure 4.7 shows plots of all human population in model 2. The susceptible class population goes down and then stabilizes after the first few days because the populations move to exposed class and light drinking class. The exposed to media population decrease due to movement to the other class but thereafter increase to an equilibrium point. The light and the heavy drinkers' population increase in the first few days, then reduce and then become steady. The treatment class increase as individuals move to other classes then reduces and become steady. The quitters class increase since all the other populations are quitting alcohol abuse.

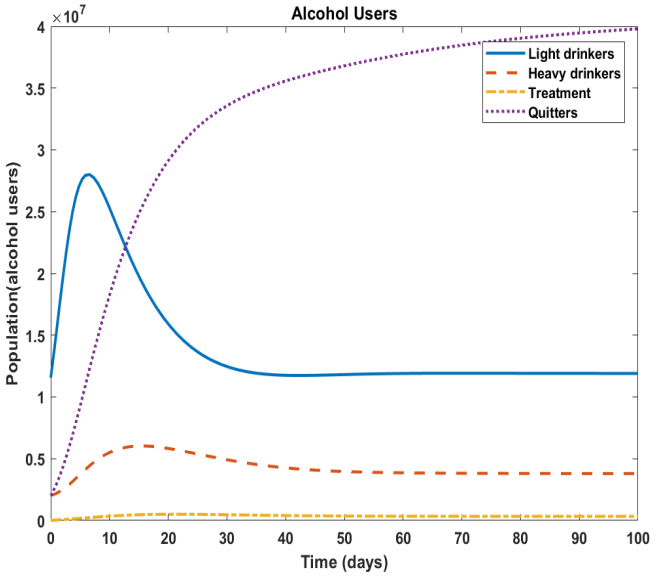

Figure 4.8: Alcohol Users

Figure 4.8 represent the individuals who have ever taken alcohol at any time in their lifetime. The y-axis represents the population of alcohol user. The quitters class increase with time since individuals move from all other classes to quitters class. The light drinkers class increase in the first days but later decrease because some individuals go for treatment and others quit alcohol. The least populated class is the treatment class because few heavy drinkers go for treatment. The rate of change in the treatment class is very low. If the rate of treatment is increased and the number of people under treatment increases then they will a reduction of people in the drinking classes.

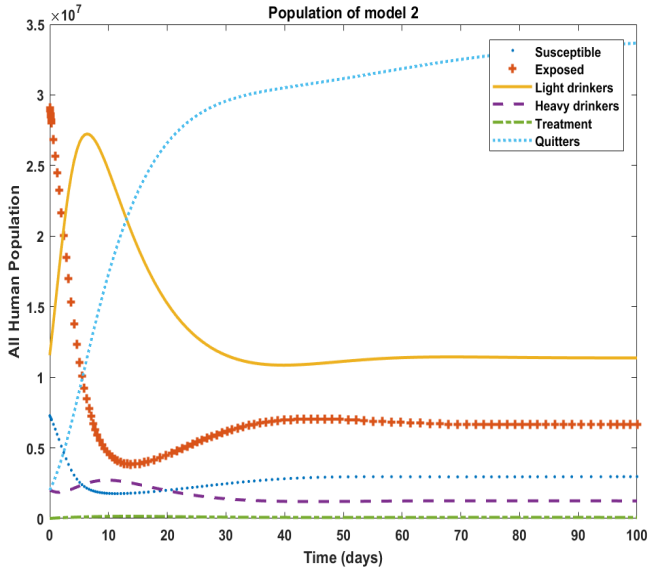

Figure 4.9: All population classes for model 2

Figure 4.9 represent the relationship between all the population classes of our model. The number of people in the treatment class is very small compared to the number in the other classes. The susceptible individuals decreased with time as they join the exposed population and others move to light drinking class. The exposed population increases due to individuals joining the group from the susceptible population. The light drinkers increase in the first few days then decrease. This is because of the individuals moving from the susceptible to this class. The heavy increases slightly then stabilize for the remaining time. The treatment class graph starts from very low because of the number of individuals joining the class and does not increase since some are joining the group and others are quitting. The quitters class is the largest since individuals join it from all the other classes.

Figure 4.10 shows varying treatment rate from 0.02 to 0.08 with $\epsilon = 0.5$. As the treatment rate increases, the number of individuals in the treatment class increases. This is because more individuals are joining the treatment class from the heavy drinking class. The treatment rate of 0.02 represents a smaller number of individuals joining the treatment compared to 0.08. When the rate of treatment is zero, the number of individuals in the treatment class remains constant.

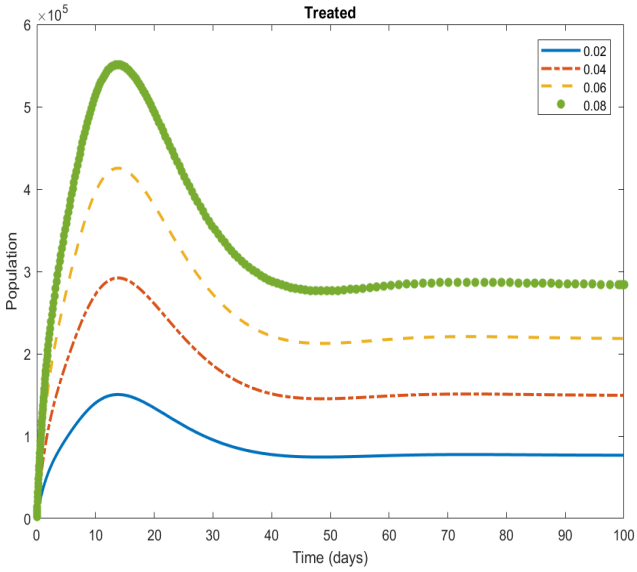

Figure 4.10: Varying treatment rate for model 2

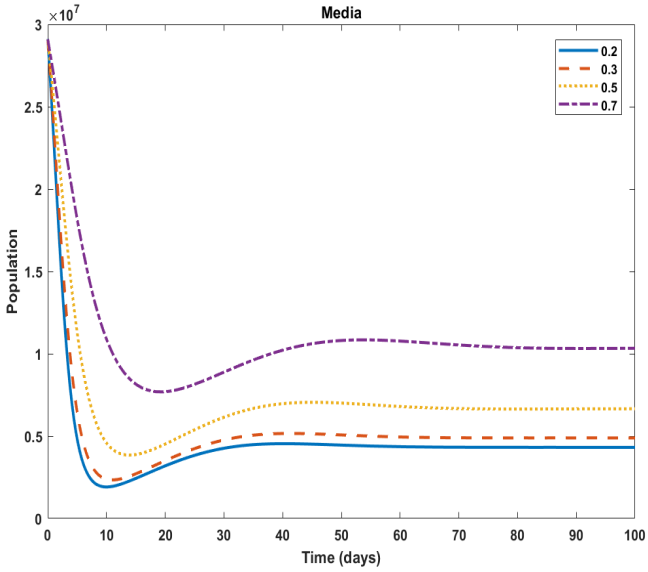

Figure 4.11: Varying mass media exposure for model 2

Figure 4.11 shows how mass media dissemination varies with time from $\epsilon = 0.2$ to $\epsilon = 0.7$. As the rate increases, the populations exposed to media increases. This shows that there will be fewer people joining alcoholic classes as we increase media awareness.

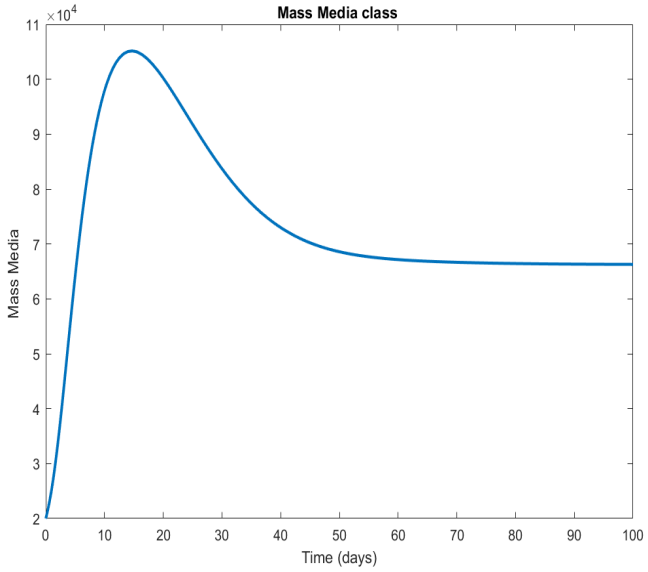

Figure 4.12: Mass media density

Figure 4.12 represent mass media density for model 2. The mass media dissemination increase in the first days then decrease up to a point where the dissemination is steady. The increase in mass media dissemination is due to campaign on against alcohol abuse that is emphasized.

4.4 Comparative Analysis Results

Sharma and Samanta (2013), developed a mathematical model of alcohol abuse and used four population classes. Their findings stressed the need for controlling and preventing habitual drinking as a means of reducing alcohol intake than increasing the number of individuals undergoing treatment. In our model, we stressed the need for increasing the treatment of alcohol as a means of reducing the heavy drinkers and controlling the spread of alcohol use by using mass media campaign.

Mancuso (2016), developed SIR model of alcoholism where the infected classes were divided into moderate and occasional drinkers and heavy drinkers. They concluded that it is better to prevent individuals from susceptible moving to infected classes than it is to treat individuals in already infected or under treatment. In our model we emphasis on reduction of heavy drinkers by recruiting them to

the treatment class.

A study by (Misra et al., 2011), considered non-linear SIS model which studied the effect of awareness programs driven by media on the spread of infectious diseases with migration. The model analysis shown that awareness programs through media campaigns are helpful in decreasing the spread of infectious diseases. In our model we used mass media campaign as a whole and also treatment of alcohol as means of reducing alcoholism in the community.

A research by (Ma et al., 2015), modeled alcoholism as contagious disease. They divided the population into three classes. Their aim was to analyze the impact of awareness programs and time delays on alcohol consumption behavior. Their study showed that awareness programs are an effective measure in reducing the heavy drinking class. In our research, we incorporate the awareness programs together with treatment as a measure to reduce heavy drinking class where thee campaign played a bigger role in reducing alcoholism than treatment.

Huo and Zhang (2016), modeled the positive and the negative role of Twitter on alcoholism. They concluded that the number of tweets affected the reproduction number. They also concluded that reducing the number of negative tweets helped reduce alcohol abuse and controlling the number of tweets posted by moderate drinkers reduce alcohol consumption. In our research, we considered the effects of all types of mass media campaigns on alcohol consumption and not just tweeter.

A study (Dubey et al., 2016), considered the role of media and treatment on SIR model. They assumed that S formed another class S_a which represent the aware population through social/electronic media density M. They showed that the infected population decreased as they increased the information dissemination rate. They concluded that if there is enough media awareness of the susceptible and treatment to the affected then the disease could be eradicated. In our research,

we considered the impact of the mass media campaign against alcohol abuse and treatment of alcohol abuse and not contagious diseases.

CHAPTER FIVE: CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusion of Model 1

Model 1 was formulated using non linear differential equations generated from Figure 3.1. An effective mass media campaigns were incorporated where individuals exposed to media campaigns did not join the drinking class.

In section 3.1 we analyzed the model considering the invariant region where we concluded that the solutions sets of solutions enter and remain in the region for all future time and the model is well-posed. The positivity of the model was analyzed and it was concluded that all state variables are positive for all time t. The reproduction number R_0 of the model was determined, and it represented the sum of the reproduction of the light drinkers R_L and the reproduction number of the heavy drinking class R_H. We analyzed the local and global stabilities of AFE. Using the theorem by Martin Jr (1974), we found that AFE is local asymptotically stable if and only if $R_0 < 1$. We also used the theorem by Castillo-Chavez et al. (2002) to analyze global stability of AFE. We concluded that model 1 is global asymptotically stable since it satisfies the conditions in Theorem 2. Conditions for existence of EEP of the model were satisfied since endemic equilibrium exist and is positive if $R_0 > 1$. The bifurcation of the model which studies the nature of the stability of the model at $R_0 = 1$ shown that there exists a backward bifurcation at $R_0 = 1$.

The main objective of model 1 was to analyze the impact of treatment on alcohol abuse hence, sensitivity analysis indicates that increase in the treatment rate reduces alcohol prevalence in the community. Sensitivity analysis also confirms that relapse from the treatment class to heavy drinking class increase alcohol prevalence in the community.

Numerical simulation confirms the sensitivity analysis. The rate of treatment

is varied as in Figure 4.5 and as the rate of treatment increases then alcohol prevalence decrease hence treatment may reduce alcoholism in the community. If no treatment is administered then no increase in population in the treatment class hence more individuals remain in heavy drinking class. We concluded that treatment is the best strategy in reducing individuals in the heavy drinking class.

5.2 Conclusion of Model 2

The local and global stability of AFE for this model were satisfied when $R_{0a} < 1$. For EEP we used the Center Manifold theory to analyze the bifurcation of the model. The analysis identified that, β_2^* changes from negative to positive, 0 changes stability from stable to unstable. The negative unstable equilibrium becomes positive and locally asymptotically stable.

We analyzed the effect of media campaign using numerical simulation, where we varied the mass media campaign rate from zero to 0.8 in Figure 4.11. We concluded that as the rate of mass media campaign increases, the rate of recruitment to alcohol drinking classes decreases. Hence mass media campaign can be used to reduce alcohol consumption in the community.

Lack of sufficient data from rehabilitation centres made it difficult to achieve the last objective in our model. We used the assumed data in our simulations. Otherwise, all other objectives were met in our study.

5.3 Recommendations from the Study

Most of the rehabilitation centres in Kenya are not run by the government. Some are privately owned while others are run by Non-Government Organizations (NGO). Hence the data of individuals in the rehabilitation centres is not available. We were able to visit a few and we estimated the data of individuals

under treatment.

We recommend the following:

1. The data of the individuals on alcohol treatment should be available to the public for research purposes.

2. The Kenya government should construct rehabilitation centers and manage them. This will make the centers available and cheap for all.

3. The Kenya government should regulate mass media campaign on alcohol consumption. The government should encourage the campaign that discourage alcohol consumption and discourage campaign that advertise alcohol mostly to the youth.

5.4 Recommendation for Further Research

Further research to be carried out on the impact of mass media campaign on other drugs abused in Kenya and also on the impact of rehabilitation centers in selected counties where treatment is more emphasized. Further research can also be carried on the prevalence of alcohol and drug abuse per gender and per region.

The main challenge of our study was availability of organized data for fitting in the model. If data was available then we would have carried out validation of the results.

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APPENDIX A

Theorem 9. *(Castillo-Chavez and Song, 2004)*

Consider the following general systems of ordinary differential equations with parameter β_2, $\frac{dx}{dt} = f(x, \beta_2), R^n \times R \to R$ *and* $f \in C^2(R^n \times R)$,

Where 0 *is the equilibrium point of the system (that is,*$f(x, \beta_2) \equiv 0$ *for all* β_2*) 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* (4.1) *around the equilibrium* 0 *with* β_2 *evaluated at* 0*. Zero is a simple eigenvalue of A and other eigenvalues of A have negative real parts:* ***A2****: Matrix a has a nonnegative right eigenvector w and a left eigenvector v corresponding to the zero eigenvalue. let*f_k *be the* k^{th} *component of f and*

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

$b = \sum_{k,i=1}^{n}(0,0,0,0) v_k w_i \frac{d^2 f_k}{d_{xi} d_{\beta_2}}(0,0,0,0)$

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

i. $a > 0, b > 0$*. When* $\beta_2 < 0$ *with* $|\beta| << 1$*, 0 is locally asymptotically stable and there exist a positive unstable equilibrium: when* $0 < \beta_2 << 1.0$ *is unstable and there exists a negative, locally asymptotically stable equilibrium:*

ii. $a < 0, b < 0$*. When* $\beta_2 < 0$ *with* $\phi << 1, 0$ *is unstable, when* $0 < \beta_2 << 1, 0$ *is locally asymptotically stable equilibrium, and there exist a positive unstable equilibrium.*

iii. $a > 0, b < 0$*. When* $\beta_2^* < 0$ *with* $|\beta_2^*| << 1, 0$ *is unstable, and there exist a locally asymptotically stable negative equilibrium: when* $0 < \beta_2^* << 1, 0$ *is stable, and a positive unstable equilibrium appears.*

iv. $a < 0, b > 0$*. When* β_2^* *changes from negative to positive,* 0 *changes its stability from stable to unstable. Corresponding a negative unstable equilibrium becomes positive and locally asymptotically stable.*