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A mathematical analysis of alcoholism*

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Abstract. A deterministic mathematical model for the spread of alcoholism is designed and analysed to gain insights into this growing health and social problem. The reproduction number and equilibria states of the model are determined and their local asymptotic stabilities investigated. Analysis of the reproduction number have shown conditions under which encouraging and supporting moderate drinkers to quit alcohol consumption is more effective in the control of alcoholism than supporting and encouraging alcoholics to quit and vice-versa. Numerical simulations show that targeting moderate drinkers by encouraging and supporting them to quit alcohol consumption will in the long run be more effective in curtailling the spread of alcoholism than singly targeting alcoholics only. However, as shown by the numerics encouraging and supporting all alcohol consumers to quit drinking will be the best strategy.

Keywords: alcohol; mathematical model, reproduction number, equilibria

1 Introduction

Alcoholism, also known as 'alcohol dependence' is a disease that includes alcohol craving and continued drinking despite its negative effect on an individual's health, relationships and social standing[11]. As with all other drug addictions, alcoholism is taken as a treatable disease. The World Health Organisation estimates that about 140 million people throughout the world suffer from alcohol dependence with related problems, such as being sick, losing a job among a host of other things[7, 15]. Alcoholism has a higher prevalence among men, although in recent decades, the proportion of female alcoholics has increased[19].

The biological mechanisms underpinning alcoholism are not known, however, risk factors include social environment, stress, mental health, genetic predisposition, age, ethnic group and sex[1, 6, 8, 9]. It has been reported that about one in seven of the United Kingdom armed forces who saved in Iraq and Afghanistan abused alcohol as a way of blotting out the horror of war[8]. Long-term alcohol abuse produces physiological changes in the brain such as tolerance and physical dependence. The changes make it difficult for alcoholics to stop drinking and result in alcohol withdrawal symptoms upon discontinuation of alcohol consumption[10]. Alcohol damages almost every part of the body and contribute to a number of human diseases including but not limited to liver cirrhosis, pancreatitis, heart disease, sexual dysfunction and can eventually be fatal[3, 19]. Damage to the central and peripheral nervous systems can occur from sustained alcohol consumptionu[14, 17]. Additionally, heavy drinking in women have been found to have a negative effect on the reproductive functioning[3].

Not much work have been done in the mathematical modelling of alcoholism as growing health problem, although some studies offered some mathematical approaches to understand the growing burdern of alcoholism[2, 13, 16]. Howewer, it is worth noting that the work presented here differs from Benedict [2] in

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that in this a more realistic model is presented dividing people who drink alcohol into moderate drinkers and alcoholics. This, then allows comparison of alcohol control approaches targeting different alcohol drinking populations. Given some other alcohol related diseases which kill people (liver cirrhosis, pancreatitis, etc) attributed to alcohol consumption this model also captures that important phenomenon through disease induced death "alcohol induced death" for the alcoholics. Furthermore, the work presented here extends the work done by Mubayi et al. [13] by including "alcohol related death" as well as recovery and relapse, although the later two have been partially addressed elsewhere[16].

The rest of the manuscript is presented as follows. In the next section, a SEIR-type model of alcoholism is presented. Stability analysis of the model is carried out in Section 3. Numerical simulations are carried out in Section 4 and finally a discussion is presented.

2 Model formulation

The model subdivides the human population into susceptibles (those who do not consume alcohol and have never consumed it) $(S(t))$, those who consume alcohol but have not become alcohol dependent, $(D(t))$, alcohol consumers now dependent on alcohol, $(A(t))$ and those recovered with or without treatment, $(R(t))$. It is worth mentioning that individuals in $D(t)$ also include casual drinkers that is those who drink only on social accassions as well as regular drinkers not yet alcoholics and are at times called moderate drinkers. The total human population is given by:

$$N(t) = S(t) + D(t) + A(t) + R(t). \tag{1}$$

Humans are recruited through births at a rate Λ into the susceptible class. Susceptibles acquire alcohol consuming habits through peer pressure (social pressure), social influence (people are surrounded by acceptance and encouragement of alcohol in many different forms: from television advertisement to host of other things where alcohol consumption appears normal and accepted), stress relief and escapism (using alcohol as a mechanism to cope with (i) some memories like battlefield memories like seeing close comrades killed or severely injured[8], (ii) depression and boredom among a host of other factors), looser inhibitions (many people feel shy, awkward or afraid in certain situations and alcohol causes people to feel more relaxed and less shy). It is against this background susceptibles spending time with people who consume alcohol are likely to consume it. Thus, non-alcoholic drinkers acquire alcohol drinking habits through social contacts at a rate λ with:

$$\lambda_h = \frac{\beta c(D + \theta A)}{N}, \tag{2}$$

with β being the probability of becoming a drinker following prolonged contacts with an individual who consumes alcohol and c is the number contacts between the people who drink and those who do not drink necessary to convince the non-drinker to drink, $\theta > 1$ accounts for the increased chances of becoming an alcohol drinker for those staying with alcoholics owing to easy access of alcohol around alcoholics. Upon starting to consume alcohol individuals in $S(t)$ move into $D(t)$ at a rate λ. With increase in drinking habits, moderate drinkers progress to the alcoholic stage of alcohol consumption at a rate ρ. Some moderate drinkers quit drinking at a rate γ to get into the class, $R(t)$. Alcoholics quit drinking at a rate δ, due to combined effect of treatment and just voluntarily stopping to move into the $R(t)$ class. Some of individuals in $R(t)$ relapse back into the class $A(t)$ at a rate σ, due to failure to defeat alcohol withdrawal symptoms like shakes, sweats, headache among a host of other symptoms. Here, we further assume that alcoholics who quit drinking do not become drinkers due to peer pressure but due to failure to control alcohol withdrawal symptoms (this is the author's assumption). Given that alcohol withdrawal symptoms for moderate drinkers are not severe, we assume moderate drinkers who quit drinking are able to control alcohol withdrawal symptoms. Thus, individuals in $R(t)$ do not relapse back into $D(t)$. All individuals experience natural death at a rate μ which is proportional to the number in each class. Alcoholics experience alcohol related diseases like liver cirrhosis, pancreatitis, heart disease among other alcohol induced diseases and die at a rate ν because of these diseases. The structure of the model is given Fig. 1.

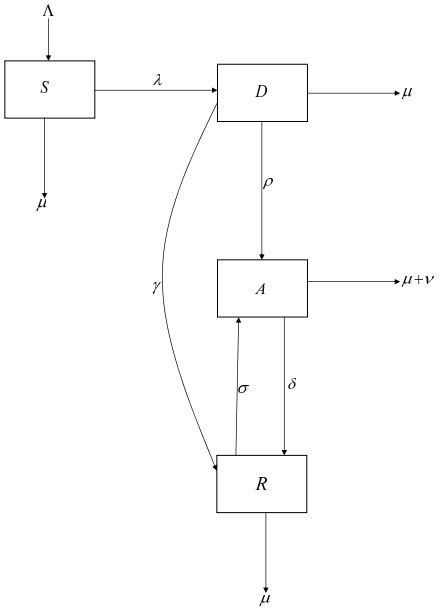

Fig. 1. Structure of model

Based on these assumptions, the following system of differential aligns describe alcoholism in a community:

$$\begin{aligned} S'(t) &= \Lambda - (\lambda + \mu)S, \quad D'(t) = \lambda S - (\mu + \rho + \gamma)D, \\ A'(t) &= \rho D - (\mu + \nu + \delta)A + \sigma R, \quad R'(t) = \gamma D + \delta A - (\sigma + \mu)R. \end{aligned} \tag{3}$$

Since model system Eq. (3) monitors human population all associated parameters and state variables are non-negative for all $t \geq 0$. Now we show that the state variables of the model remain non-negative for all non-negative initial conditions. Thus, we claim the following result.

Lemma 1. *The closed set:*

$$\Omega = \left\{ (S, D, A, R) \in \mathbb{R}^4_+ : \ N \leq \frac{\Lambda}{\mu} \right\},$$

is positively invariant and attracting with respect to model system Eq. (3).

Proof. Adding all the aligns in model system Eq. (3) gives:

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \Lambda - \mu N - \nu A, \quad N = S + D + A + R.$$

Since $\frac{dN}{dt} \leq \Lambda - \mu N$, it follows that $\frac{dN}{dt} < 0$ if $N(t) > \frac{\Lambda}{\mu}$. Thus, a standard comparison theorem[21] can be used to show that $N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu}(1 - e^{-\mu t})$. Also, $N(t) \leq \frac{\Lambda}{\mu}$ if $N(0) \leq \frac{\Lambda}{\mu}$. Thus, Ω is positively invariant. Furthermore, if $N(t) > \dfrac{\Lambda}{\mu}$, then either solution enters Ω in finite time or $N(t)$ approaches $\dfrac{\Lambda}{\mu}$. Hence, Ω is attracting that is all solutions in $\mathbb{R}^4_+$ eventually approach, enter or stay in Ω.

Therefore, the model is mathematically well-posed and epidemiologically reasonable since all variables remain non-negative for all $t \geq 0$. Hence, it is sufficient to consider the dynamics of model system Eq. (3) in Ω[22].

3 Equilibria states and stability analysis

In this section we compute and analyse the equilibria states of the model starting with alcohol-free equilibrium which in this manuscript shall be called disease free equilibrium point.

3.1 Alcohol-free equilibrium

The alcohol-free equilibrium (disease-free equilibrium) of model system Eq. (3) is given by:

$$E^0 = \left(S^0, D^0, A^0, R^0\right) = \left(\frac{\Lambda}{\mu}, 0, 0, 0\right). \tag{4}$$

Following Van and Watmough [18], we have:

$$R_H = \frac{\beta c}{\mu+\gamma+\rho}\left(1+\frac{\theta(\mu\rho+(\gamma+\rho)\sigma)}{\delta\mu+(\mu+\nu)(\mu+\sigma)}\right). \tag{5}$$

Theorem 1 follows from Theorem 2 [18].

Theorem 1. *The disease-free equilibrium E^0 is locally asymptotically stable whenever $R_H < 1$, and unstable otherwise.*

3.1.1 Analysis of the reproduction number

Here we analyse different scenarios for the reproduction number R_H.

Case 1: All people who consume do not quit

In this case $\gamma = \delta = \sigma = 0$ so that R_H becomes R_{0_H} which is given by:

$$R_{0_H} = \lim_{(\gamma,\delta,\sigma)\to(0,0,0)} R_H = \frac{\beta c}{\mu+\rho}\left(1+\frac{\theta\rho}{\mu+\nu}\right).$$

Case 2: Only alcoholics quit

If only the alcoholics do quit alcoholic consumption voluntarily or due to treatment, then R_H becomes R_{H_A} which given as:

$$R_{H_A} = \lim_{\gamma\to 0} R_H = \frac{\beta c(\delta\mu+(\sigma+\mu)(\mu+\nu+\theta\rho))}{(\mu+\rho)[\delta\mu+(\mu+\nu)(\mu+\sigma)]}. \tag{6}$$

Expressing in R_{H_A} in terms of R_{0_H} we have:

$$R_{H_A} = H_1 R_{0_H},\ H_1 = \frac{(\mu+\nu)(\delta\mu+(\mu+\sigma)(\mu+\nu+\theta\rho))}{(\mu+\nu+\theta\rho)(\delta\mu+(\mu+\nu)(\mu+\sigma))}.$$

We are only able to conclude that in the presence of failure to control alcohol withdrawal symptoms, alcoholics quitting alcohol consumption with achieve the desired outcome provided,

$$H_1 < 1,\ \frac{(\mu+\nu)(\delta\mu+(\mu+\sigma)(\mu+\nu+\theta\rho))}{(\mu+\nu+\theta\rho)(\delta\mu+(\mu+\sigma)(\mu+\nu))} < 1 \text{ (since } \delta\mu > 0).$$

Thus, voluntary or due to treatment quitting of alcohol will help in reducing the number of alcoholics, even when some fail to control alcohol withdrawal symptoms and revert to drinking habits. Under what conditions does alcohol relapse (failure to defeat alcohol withdrawal symptoms) lead to an increase of alcoholism in a society? To answer this seemingly naive question requires one to find the critical relapse rate σ^c which is given by:

$$\sigma^c = \frac{\mu(R_{H_A}(\mu+\rho)(\delta+\mu+\nu)-(\delta+\mu+\nu+\theta\rho)\beta c)}{\beta c(\mu+\nu+\theta\rho)-(\mu+\rho)(\mu+\nu)R_{H_A}},$$

and exists for $\frac{(\mu+\nu)(\delta+\mu+\nu+\theta\rho)R_{0_H}}{(\delta+\mu+\nu)(\mu+\nu+\theta\rho)} < R_{H_A} < R_{0_H}$. If $\sigma^c > \sigma$, then alcohol relapse will not lead to an increase of alcoholism. However, if $\sigma^c < \sigma$, the story is different as relapse will significantly contribute to alcoholism cases. This happens typically when a large number of people who have quitted alcohol

consumption fail to defeat alcohol withdrawal symptoms and relapse back into alcohol consumption habits. Against this background there is strong need to offer support to those who have quitted so that they will not relapse back.

Case 3: Only moderate drinkers quit

In this case $\delta = \sigma = 0$ and R_H becomes R_{H_D} which is given by:

$$R_{H_D} = \lim_{(\delta,\sigma)\to(0,0)} R_H = \frac{\beta c(\mu+\nu+\theta\rho)}{(\mu+\nu)(\mu+\gamma+\rho)}.$$

Expressing R_{H_D} in terms of R_{0_H} we have:

$$R_{H_D} = H_2 R_{0_H},\ H_2 = \frac{\mu+\rho}{\mu+\gamma+\rho} < 1.$$

The fact that $H_2 < 1$, suggests increase in moderate drinkers quitting alcoholic consumption results in a decrease of the number of alcoholics. This will also have a positive impact on the control alcohol related health issues (liver cirrhosis, heart disease, etc.) Now comparing R_{H_D} and R_{H_A}, is just the same as comparing H_1 and H_2 resulting in Lemma 2.

Lemma 2. *If, (1)* $\gamma = \frac{\mu\delta\theta(\mu+\rho)}{(\mu+\nu)(\delta\mu+(\mu+\sigma)(\mu+\nu+\theta\rho))}$*, then the effect of moderate drinkers only quitting is just the same as the effect of alcoholics only quitting on the control of alcoholism.*
(2) $\gamma > \frac{\mu\delta\theta(\mu+\rho)}{(\mu+\nu)(\delta\mu+(\mu+\sigma)(\mu+\nu+\theta\rho))}$*, then supporting an increase in the number of moderate drinkers only to quit drinking habits will more beneficial when compared to supporting alcoholics only to quit.*
(3) $\gamma < \frac{\mu\delta\theta(\mu+\rho)}{(\mu+\nu)(\delta\mu+(\mu+\sigma)(\mu+\nu+\theta\rho))}$*, then supporting an increase in the number of alcoholics only to quit drinking habits will more beneficial when compared to moderate drinkers only to quit.*

From Lemma 2 (2) encouraging more people who are moderate drinkers to quit drinking result in less people progressing to the alcoholic stage. This in turn results in less cases of serious alcohol related withdrawal symptoms as well as alcohol related diseases. This may be the most ideal strategy in a community in which there is a small population of alcoholics and a large growing population of moderate drinkers. However, from Lemma 2 (3) encouraging alcoholics to quit will be more beneficial to the community, as in doing so alcohol related deaths are averted. This may be more ideal in a population where there is a growing number of alcoholics. However, should resources be available it may be best to target encouraging all alcohol drinking people to quit and be put on treatment if they cannot defeat alcohol related withdrawal symptoms. With this in mind it may be necessary to step up public health campaigns to make people aware of dangers associated with alcohol consumption and putting necessary support groups for those quitting alcoholic consumption.

3.2 Existence of the endemic equilibrium state

Solving the aligns of the model Eq. (3) at steady state in terms of the force infection λ^* gives $Q^* = (S^*, D^*, A^*, R^*)$ where

$$\begin{aligned} S^* &= \frac{\Lambda}{\mu+\lambda^*},\ D^* = \frac{\Lambda\lambda^*}{(\mu+\lambda^*)(\mu+\rho+\gamma)}, \\ A^* &= \frac{\Lambda\lambda^*(\mu\rho+(\gamma+\rho)\sigma)}{(\mu+\lambda^*)(\mu+\rho+\gamma)(\delta\mu+(\mu+\sigma)(\mu+\nu))}, \\ R^* &= \frac{\Lambda\lambda^*(\gamma(\mu+\nu+\delta)+\delta\rho)}{(\mu+\lambda^*)(\mu+\rho+\gamma)(\delta\mu+(\mu+\sigma)(\mu+\nu))} \end{aligned} \tag{7}$$

Substituting align Eq. (7) into Eq. (2) we obtain:

$$\begin{aligned} &\lambda^* h(\lambda^*) = \lambda^*(A\lambda^* + (1-R_H)) = 0, \\ &A = \frac{\delta(\mu+\rho)+(\mu+\nu)(\mu+\sigma)+\mu\rho+(\gamma+\rho)\sigma+\gamma(\mu+\nu+\delta)}{(\mu+\rho+\gamma)(\delta\mu+(\mu+\nu)(\mu+\sigma))}. \end{aligned} \tag{8}$$

From align Eq. (8) $\lambda^* = 0$ corresponds to the disease-free equilibrium and $\lambda^* = \dfrac{R_H - 1}{A}$ which exists for $R_H > 1$ corresponding the endemic equilibrium. To check on the local stability of this equilibrium point we apply the Centre Manifold theory [4] as outlined in Theorem 4.1 [5]. To apply the said Theorem 4.1 [5] in order to establish the local asymptotic stability of the endemic equilibrium, it is convenient to ake the following change of variables: $S = x_1,\ D = x_2,\ A = x_3, R = x_4$ so that $N = \sum_{n=1}^{4} x_n$. We now use the vector notation $X = (x_1, x_2, x_3, x_4)^T$. Then, model system Eq. (3) can be written in the form $\frac{dX}{dt} = F = (f_1, f_2, f_3, f_4)^T$, where

$$\begin{aligned} x_1'(t) = f_1 = \Lambda - \frac{\beta c(x_2 + \theta x_3)}{\sum_{n=1}^{4} x_n} x_1 - \mu x_1, \quad x_2'(t) = f_2 = \frac{\beta c(x_2 + \theta x_3)}{\sum_{n=1}^{4} x_n} x_1 - (\mu + \rho + \gamma) x_2, \\ x_3'(t) = f_3 = \rho x_2 - (\mu + \nu + \delta) x_3 + \sigma R, \quad x_4'(t) = f_4 = \gamma x_2 + \delta x_3 - (\mu + \sigma) x_4. \end{aligned} \tag{9}$$

The Jacobian matrix of system Eq. (9) at E^0 is given by:

$$J(E^0) = \begin{bmatrix} -\mu & -\beta c & -\theta\beta c & 0 \\ 0 & \beta c - (\mu + \rho + \gamma) & \theta\beta c & 0 \\ 0 & \rho & -(\mu + \nu + \delta) & \sigma \\ 0 & \gamma & \delta & -(\mu + \sigma) \end{bmatrix}, \tag{10}$$

from which it can be shown that the reproduction number of the model is:

$$R_H = \frac{\beta c}{\mu + \gamma + \rho} \left(1 + \frac{\theta(\mu\rho + (\gamma + \rho)\sigma)}{\delta\mu + (\mu + \nu)(\mu + \sigma)} \right), \tag{11}$$

as shown earlier. If β is taken as a bifurcation parameter and if we consider the case $R_H = 1$ and solve for β we obtain:

$$\beta^* = \frac{(\mu + \gamma + \rho)(\delta\mu + (\mu + \nu)(\mu + \sigma))}{c[\delta\mu + (\mu + \nu)(\mu + \sigma) + \theta(\rho\mu + (\gamma + \rho)(\mu + \sigma))]}. \tag{12}$$

The transformed align Eq. (9) with the bifurcation point β^* has a simple zero eigenvalue. This allows us to use the Centre manifold theory to analyze the dynamics of the model system Eq. (9) near $\beta = \beta^*$. The Jacobian of model system Eq. (9) at $\beta = \beta^*$ has a right eigenvector associated with the zero eigenvalue given by $u = [u_1, u_2, u_3, u_4]^T$ where

$$\begin{aligned} u_1 = -\frac{\beta^* c[(\mu + \sigma)(\mu + \nu) + \delta\mu]}{\mu(\sigma\gamma + (\mu + \sigma)\rho)} u_3, \quad u_2 = \frac{(\mu + \sigma)(\mu + \nu) + \delta\mu}{\sigma\gamma + (\mu + \sigma)\rho} u_3, \\ u_3 > 0, \quad u_4 = \frac{\gamma(\mu + \nu + \delta) + \delta\rho}{\sigma\gamma + (\mu + \sigma)\rho} u_3. \end{aligned} \tag{13}$$

The left eigenvector of the Jacobian of model system Eq. (9) at $\beta = \beta^*$ associated the zero eigenvalue is $v = [v_1, v_2, v_3, v_4]^T$ where

$$v_1 = 0,\ v_2 = \tfrac{(\mu+\sigma)(\mu+\nu)+\delta\mu}{(\mu+\sigma)\theta\beta^* c} v_3,\ v_4 = \tfrac{\sigma}{\mu+\sigma} v_3. \tag{14}$$

Now we compute a and b as outlined in Theorem 4.1 [5].

Computations of a and b:

For system Eq. (9) the non-zero partial derivatives of F associated with a are

$$\begin{aligned} \frac{\partial^2 f_2}{\partial x_2^2} = -\frac{2\beta^* c\mu}{\Lambda}, \quad \frac{\partial^2 f_2}{\partial x_2 \partial x_3} = \frac{\partial^2 f_2}{\partial x_3 \partial x_2} = -\frac{(1 + \theta)\beta^* c\mu}{\Lambda}, \quad \frac{\partial^2 f_2}{\partial x_2 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_2} = -\frac{\beta^* c\mu}{\Lambda}, \\ \frac{\partial^2 f_2}{\partial x_3^2} = -\frac{2\theta\beta^* c\mu}{\Lambda}, \quad \frac{\partial^2 f_2}{\partial x_3 \partial x_4} = \frac{\partial^2 f_2}{\partial x_4 \partial x_3} = -\frac{\theta\beta^* c\mu}{\Lambda}. \end{aligned} \tag{15}$$

It follows from expressions in Eq. (15) that

$$a = -\left(\frac{2\mu(B_1B_2+\delta\mu)(B_1(B_2+\theta\rho)+B_3)(B_1(B_2+\rho)+B_4+\gamma(B_1+\nu+\delta))}{\Lambda\theta B_1(\sigma\gamma+B_1\rho)^2}\right)v_3u_3^2<0,$$
$$B_1=\mu+\sigma,\ B_2=\mu+\nu,\ B_3=\delta\mu+\theta\sigma\gamma,\ B_4=\delta(\mu+\rho).$$

For the sign of b, it can be shown that the associated non-zero partial derivatives of F are

$$\frac{\partial^2 f_1}{\partial x_2\beta^*}=-c,\quad \frac{\partial^2 f_1}{\partial x_3\beta^*}=-c\theta,\quad \frac{\partial^2 f_2}{\partial x_2\beta^*}=c,\quad \frac{\partial^2 f_2}{\partial x_3\beta^*}=c\theta. \tag{16}$$

It follows from Eq. (16) that

$$b=\left(\frac{(\mu+\sigma)(\mu+\nu)+\delta\mu}{(\mu+\sigma)\theta\beta^*c}\right)\left(\frac{(\mu+\sigma)(\mu+\nu)+\delta\mu}{\gamma\sigma+(\mu+\sigma)\rho}+\theta\right)cv_3u_3>0.$$

Thus, $a<0$ and $b>0$. So by Theorem 4.1 [5], item (iv) we have established the following result which holds for $R_H>1$ but close to 1.

Theorem 2. *The unique endemic equilibrium E^* guranteed by Theorem 4.1 [5] is locally asymptotically for $R_H>1$ but close to 1.*

4 Numerical simulations

The life expectancy of most people in developing countries (Zimbabwe, Democratic Republic of Congo etc) is about 50 years, so μ is 0.02 (Central Statistics Office of Zimbabwe(CSOZ)). About 3 million people of Zimbabwe's estimated 12 million people drink alcohol, so the probability one be an alcohol drinker is 0.25 (WH0, 2004) and this allows the use of $\beta=0.25$. The, number of times one is in a company alcohol drinking people enough to let him/ her get the tempation to drink varies with individuals.However, here $c=1$ is used for numerical simulations. The rate at which moderate drinkers become alcoholic again varies depending on a number of factors including one's genetic make up and drinking patterns among a host of other factors, but here $\rho=0.0075$ is used. The rate of quitting for moderate drinkers and alcoholics varies with individuals, so for illustration purposes, $\gamma=0.0025$ and $\delta=0.005$ are used, respectively. Again the rate of relapsing back for alcoholics who have left drinking habits veries depending on individuals so for illustration purposes we use $\sigma=0.001$. Alcohol related death rates are not documented in most developing countries, so we use $\nu=0.035$ which alcohol induced death rates for the United States of America[12] although in developing countries it may be less than that. The recruitment is taken to be $\Lambda=0.029*4500000$ with 0.029 being the birth rate for Zimbabwe and 4.5 million being the number of childbearing women in Zimbabwe (CSOZ). The initial values used in the numerical simulations are: $S(0)=9\times10^6,\ D(0)=3.55\times10^6,\ A(0)=1.45\times10^5,\ R(0)=0.$

Table 1. Model parameters and their interpretations

Parameter	Symbol	Value
Births rate for humans	Λ	$0.029\text{yr}^{-1}*4500000$
Natural mortality for humans	μ	0.02yr^{-1}
Alcohol nduced death rate	ν	0.035yr^{-1}
Rate of becoming alcoholic	ρ	0.0075yr^{-1}
Rate of quitting alcohol for moderate drinkers	γ	0.0025yr^{-1}
Rate of quitting alcohol for alcoholics	δ	0.005yr^{-1}
Rate of relapse	σ	0.001yr^{-1}
Enhancement factor	θ	1.0005
Product of the probability becoming an alcoholic drinker and the effective contact necessary for one to become a drinker	βc	0.25yr^{-1}

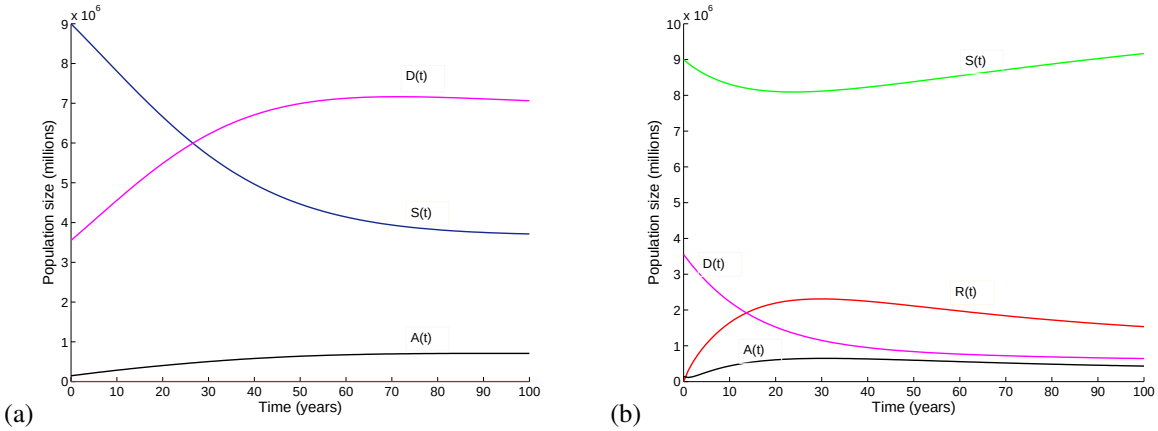

Fig. 2. Simulation results showing the trends the behavior of the population in the absence and presence of voluntary quitting and treatment (Parameter values used are in Tab. 1)

Fig. 2 shows what happens in a population when alcohol consumers quit or do not quit alcohol consuming habits. In the absence alcohol quitting, a decrease of the susceptible population to asymptotically low levels is accompanied by an increase in the population of alcohol drinking population (moderate and alcoholics) as noted in Fig. 2(a). However, in the presence of quitting, the susceptible and recovered populations increase to their corresponding asymptotic states while the drinking population decline to their asymptotic levels. Owing to relapse, the alcoholic population $(A(t))$ do not decrease as much as expected (Fig. 2), as most alcoholics who have voluntarily quitted fail to defeat alcohol withdrawal symptoms. Fig. 3 is a graphical illustration

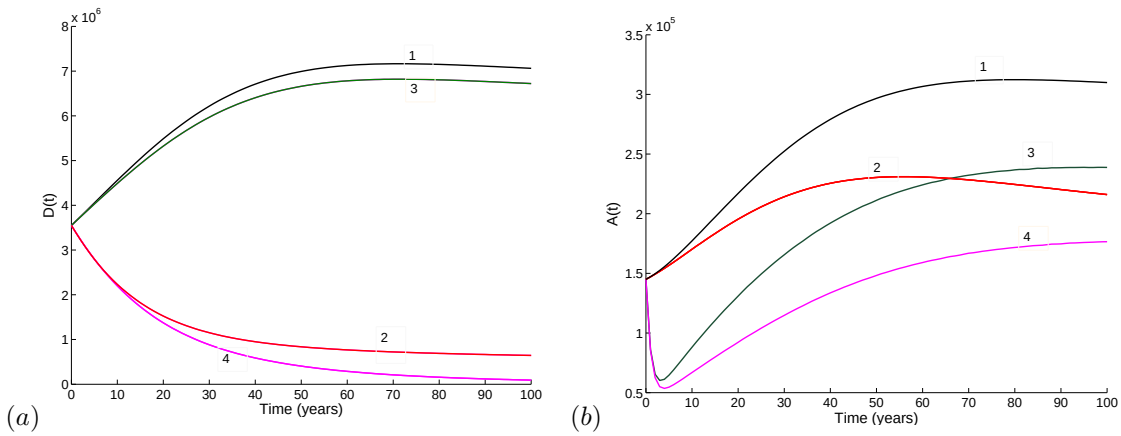

Fig. 3. Simulation results showing effects of targeting one population or both populations to control alcoholism: 1 denotes no intervention strategy, 2 denotes only moderate drinkers voluntarily quitting through encouragement, 3 denotes only alcoholics quitting voluntarily or through treatment, 4 denotes where both populations are targeted (Parameter values used are in Tab. 1)

comparing various ways of controlling alcoholism. It is noted in both graphs in Fig. 3 encouraging moderate drinkers to quit coupled with encouragement of alcoholics to quit voluntarily or as through treatment will be the best approach to control alcoholism in a community as can be noted by trend 4 in both graphs of Fig. 3. Comparing of the outcome of moderate drinkers only quitting against alcoholics only quitting (trends 2 and 3) we note that in the long term in only more moderate drinkers quit, there is a greater advantage than only in only alcoholics quit. However, it should be noted that in the short term quitting of alcoholics only will achieve some control of alcohol related problems. It is shown in Fig. 4 that an increase in θ, translates to an increase in the number of alcohol drinkers $(D(t),\ A(t))$, as an increase in the easy access of alcohol increase the temptation to drink it. Results in Fig. 5 show that people who have quitted alcohol consumption habits should

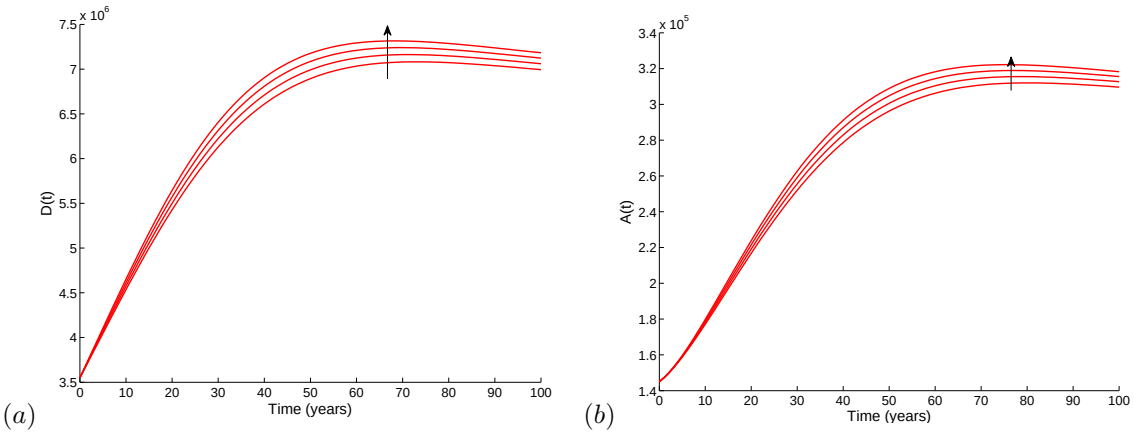

Fig. 4. Simulation results showing the effect of varying θ from 1.0 with a step with a step size of 0.25 on the population of alcoholic drinkers. The direction of the arrow shows the direction of the increase (Parameter values used are in Tab. 1)

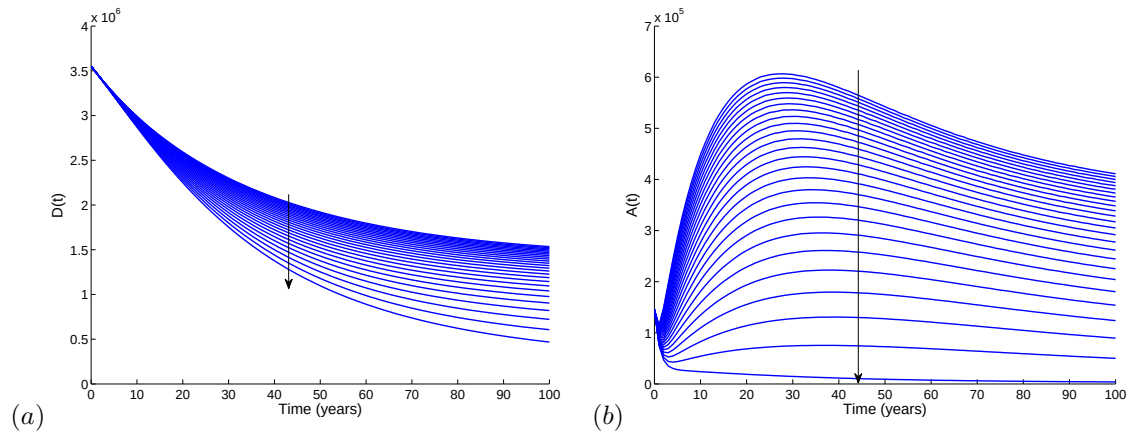

Fig. 5. Effects of varying the relapse rate, σ starting with 0.5 with a step size of 0.02. The direction of the arrow shows the direction of the decrease in σ (Parameter values used are in Tab. 1)

be encouraged and supported so that they will not revert back to old habits. Left alone, most will fail to defeat the alcohol withdrawal systems. Defeating alcohol withdrawal symptoms translate to a decrease of alcohol consuming individuals. Fig. 6, show simulations for various initial conditions when $R_H < 1$ $(R_H = 0.732)$ and when $R_H > 1$ $(R_H = 1.567)$. It shows the alcohol free (disease-free) equilibrium (Fig. 6 (a)) and the endemic equilibrium state (Fig. 6 (b)).

5 Discussion

This paper studies the dynamics of alcoholism in a community taking into account that some people voluntarily quit alcoholic consumption and some as a result of being on treatment. The reproduction number of the model is computed and analysed. Conditions under which failure to defeat alcohol withdrawal symptoms will contribute meaningfully to an increase in the number of alcoholics have been explored. Additionally, analysis of the reproduction number has shown conditions under which supporting the encouragement of moderate drinkers to quit alcoholic consumption leads to decrease in alcoholism better than alcoholics only quitting and vice versa. As in all epidemiological models, the equilibria states are found and stabilities analysed. With the aid of the Centre manifold theory the endemic equilibrium point is shown to be locally asymptotically stable when the corresponding reproduction number is less than unity. Numerical simulations are performed to illustrate various scenarios. It is shown from the numerical simulations in the long term encouraging and

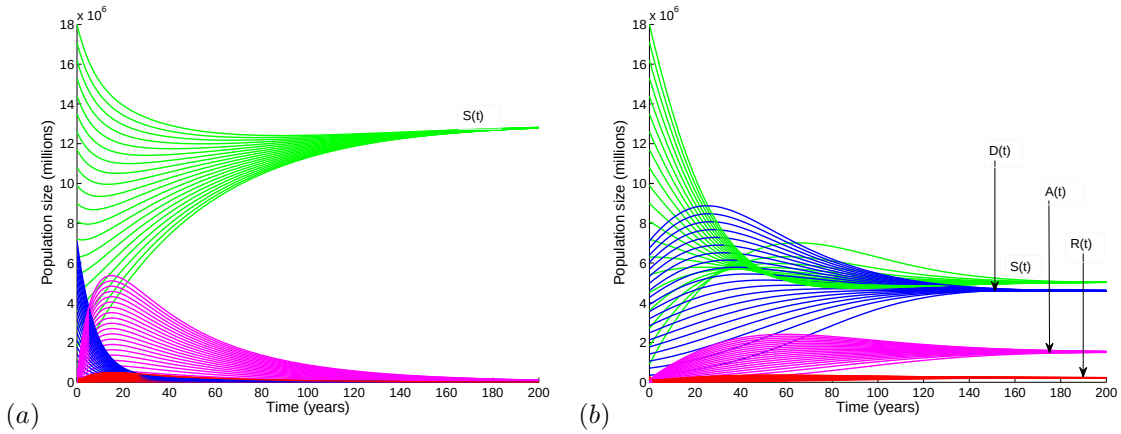

Fig. 6. Simulation results showing the effect of varying initial conditions when $R_H = 0.732$ and when $R_H = 1.567$ (Parameter values used are in Tab. 1)

supporting more moderate drinkers to quit alcoholic consumption will achieve a better result than supporting and encouraging alcoholics only to quit. However, it is worth mentioning supporting and encouraging real alcoholics to quit is of immediate benefit ot the community, as alcoholics are more likely to experience alcohol related problems than moderate drinkers. For effective control of alcoholism it may be best to step up public educational campaigns targeting all the alcohol drinking populations. Given that most people drink alcohol initially due to peer pressure, it may be best for countries with growing numbers of alcoholic to create and support anti-alcoholism peer and support groups. This model is not exhaustive and has some limitations. For instance moderate drinkers while they do not experience severe alcoholic withdrawal symptoms, they can relapse back into the old drinking habits. Also given that most alcohol drinkers also smoke looking into combined alcoholism and smoking model is another avenue to be explored.

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