2021-08

Mathematical models for the dynamics of health related risks associated with alcoholism and its control strategy in Tanzania

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NM-AIST

https://doi.org/10.58694/20.500.12479/1369

MATHEMATICAL MODELS FOR THE DYNAMICS OF HEALTH RELATED RISKS ASSOCIATED WITH ALCOHOLISM AND ITS CONTROL STRATEGY IN TANZANIA

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A Thesis Submitted in Fulfilment of the Requirements for the Degree of PhD Degree in Mathematical and Computer Science and Engineering of the Nelson Mandela African Institution of Science and Technology

Arusha, Tanzania

August, 2021

ABSTRACT

Alcoholic behavior has continually posed health challenges in many communities for decades. Referring to Tanzanian situation, this study presents a more realistic model for the dynamics of health risks associated with alcoholism. The model considered a population proportion that has social cultural protection from alcohol consumption. In the context of this study, such protection emanated from religious beliefs practiced in the country. Three versions of the model were analyzed considering different model analysis scenarios: the basic model, fuzzy logic model, and optimal control model. The equilibria of the basic model were obtained and their stability analysis was performed. The Next Generation Matrix (NGM) approach was used to compute the basic risk reproduction number of the basic model. The risk free equilibrium point of the basic model was proved to be globally asymptotically stable whenever the basic risk reproduction ratio was less than unit and unstable otherwise. The sensitivity analysis of the basic risk reproduction number of the basic model and numerical simulation were carried out. Their results revealed that deliberate intervention strategies and policies focused on discouraging alcoholic behaviors on their onset during initiation stage were more effective than dealing with alcoholic population proportions. The fuzzy logic based model analysis have confirmed this result where uncertainty conditions were assumed in the measure of influence of alcoholic individual β and the additional death rate α. Nevertheless, the cost-effectiveness analysis for the model with two control options provided some useful insights. Based on analytical results, it was clear that although strategy B appeared to be ineffective when applied alone. This strategy worked much more better when applied together with strategy A. Whereas application of control option u_1 increases the level of protection to the susceptible population by implementing public health education campaign; the control option u_2 increases removal rate of the moderate risky individuals into recovered population. Thus, the health risks associated with alcoholism may be effectively eradicated when both controls, u_1 and u_2 are applied together as the combination.

APPROVAL

I, **Maranya Makuru Mayengo**, do hereby declare to the Senate of Nelson Mandela African Institution of Science and Technology that this thesis is my own original work and that it has neither been submitted nor being concurrently submitted for degree award in any other institution.

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DECLARATION

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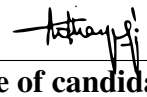
Maranya Makuru Mayengo  06.08.2021

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CERTIFICATION

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ACKNOWLEDGEMENTS

To my life-coach, my forever interested, encouraging and always enthusiastic, my dear mom Mrs. Justine Mkami Makuru: because I owe it all to you. Many Thanks mom!

I am grateful to my young family: my wife Grace and children, who have provided me thorough moral and emotional support in my life. I am also grateful to my other family members and friends for their timely support whenever they were needed.

A very special gratitude goes out to Nelson Mandela African Institution of Science and Technology (NM−AIST) for offering me the DAAD In-Country Scholarships to cover my program expenses; African Centre for Research, Agricultural advancement, Teaching Excellence and Sustainability (CREATES) for their partial sponsorship to attend training on Modeling Dynamics, Statistical Inferences and Prediction of Infectious Deceases (MoDSIP−ID) in India; Research and Training Fellowship Program for Developing Countries Scientists (RTF−DCS) for their partial financial support to cover my research expenses in India.

With a special mention to Prof. Gabriel Shirima (NM−AIST, Tanzania), Prof. Moatlhodi Kgosimore (BUANR, Botswana) and Prof. Padmanabhan Seshaiyer (GMU, USA) for their precious guidance; Dr. Michael Winkler − MathCom and Prof. Till Bärnighausen − Institute for Global Health, Universität Heidelberg for their host supervision during my research visit in Universität Heidelberg, Germany; Prof. Snehashish Chakraverty − Department of Mathematics, National Institute of Technology (NIT), Rourkela, India for the host supervision in NIT−Rourkela. It was fantastic to have the opportunity to work majority of my research in your facilities under your guidance.

I am also grateful to the following: Prof. Karoli Njau (NM−AIST, Tanzania); Prof. Luis Grilo − Polytechnic Institute of Tomar, Portugal; Dr. Carmen Caisseda − Inter American University of Puerto Rico, Bayamón Campus, Puerto Rico; and Mr. Christian Bwaya − Mwenge Catholic University, Moshi Tanzania for their unfailing support and assistance every time I reached them for help.

And finally, last but by no means least, my former employer, The University of Dodoma (UDOM); my colleagues − staff and students of NM−AIST, and its surrounding society. They had important roles in providing social, care and cooperation during the entire period of my

studies at NM-AIST. To them all, my sincere thanks.

Thanks for all your encouragement!

DEDICATION

This work is dedicated to my brother and a dear friend, the late Chunus Kelisa Mayengo (1960−2013)

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LIST OF ABBREVIATIONS, ACRONYMS AND SYMBOLS

ADA	Alcohol Drinking Attitude
ADF	Alcohol Drinking Frequency
ADP	Alcohol Drinking Patterns
AMCSE	Applied Mathematics and Computational Sciences and Engineering
CoCSE	Graduate School of Computational and Communication Sciences and Engineering
DAAD	Deutscher Akademischer Austauschdienst (German Academic Exchange Service)
DGC	Directorate General Communication
DI	Demographic Information
EEP	Endemic Equilibrium Point
FEV	Fuzzy Expected Value
FSU	Former Soviet Union
HED	Heavy Episodic Drinking
HRAA	Health Risks Associated with Alcoholism
IAR	Infection Averted Ratio
ICER	Incremental Cost Effective Ratio
IRB	Influence of Religious Beliefs
G.A.S	Globally Asymptotically Stable
L.A.S	Locally Asymptotically Stable
LHS	Left hand side
NCETA	National Center for Education and Training on Addiction
NHMRC	National Health and Medical Research Council
NM-AIST	Nelson Mandela - African Institution of Science and Technology
ODE	Ordinary Differential Equations
PMP	Pontryagin's Maximum Principle
RK4	Runge-Kutta Method of order four
RTF-DCS	Research and Training Fellowship for Developing Countries Scientists
TAB	Types of Alcoholic Beverages
WHO	World Health Organization

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CHAPTER ONE

INTRODUCTION

1.1 Background of the Problem

Alcoholic beverages are an integral part of cultures around the world (Ellison *et al.*, 2007; WHO, 2014). The consumption of alcoholic beverages is linked to alcoholism, a state of addiction which eventually accelerates to alcohol dependency. Many factors, internally and externally motivated, influence alcohol drinking habits. While culture acts as a powerful tool in promoting drinking practices that are positive, it discourages practices attributed to health risks. In some studies, it has been established that age, genetic disposition, ethnic groups, sex, mental health, social environment and stress contribute to risk factors encouraging alcoholic behaviors in the community (Bhunu, 2012). However, in other studies the volume of alcohol consumed, patterns of drinking and quality of alcohol consumed are key determinants of alcohol related harms to its recipients (WHO, 2014; NHMRC-Australia, 2001).

Common alcoholic beverages available in Tanzania include standard bottled beers, spirits, wines and locally made brews and spirits. Although locally made spirits are not legally accepted, they have long been in the markets. While it is illegal to sell alcoholic beverages to persons under 18 years of age in Tanzania, studies consistently revealed that alcohol use in the country is extended to secondary school children (Francis *et al.*, 2015) and young children in many families. Early exposure and easy access to alcoholic beverages is common to children raised in poor families in communities where some families depend on selling locally brewed alcohol and spirits to make a living (Francis *et al.*, 2015). In most cases, homes in which local brews and spirits are produced are turned out into local bars jeopardizing young family members raised in such households. The survey conducted by Francis *et al.* (2015) confirmed that, at least six out ten secondary school students had access to alcohol whenever they needed. The locally produced alcoholic beverages are brewed in a considerably poor hygienic conditions with varying quality and sometimes sold in premature fermentation (Tusekwa *et al.*, 2000). Similarly, alcohol drinking has been the major concern in public health discussions, categorized as among the leading preventable cause of death in some parts of the world (Giraldo *et al.*, 2017). Local communities have different brands of alcohols depending on ingredients available in the area. Actually, this goes down to tribal culture where each tribe has its alcohol type

and flavor. Availability of the raw materials needed for production of alcohol makes it readily available at a lower price as compared to modern beers and spirits (Tusekwa *et al.*, 2000). This may lead to an increase of individuals who proudly identify themselves with alcohol drinking. Many social events in Tanzania, for instance, are considered a failure or uninteresting if they do not make alcohol beverages available. As a result, drinking behaviors are likely to spread in the society in a contagious manner. In that way, the spread of alcohol drinking behavior can be similar to communicable diseases where a drinker acts as an agent in recruiting others into the behavior.

According to WHO, alcoholism is associated to injury and violence with its negative impacts spreading throughout a community, influencing levels and patterns of alcohol consumption (WHO, 2011). Health effects associated with alcohol intake in large amounts include malnutrition, chronic pancreatitis, alcoholic liver disease and cancer. In addition, damage to the central and peripheral nervous system can occur from chronic alcohol abuse (WHO, 2004, 2009; Bhunu, 2012; WHO, 2014; Huo *et al.*, 2016). At times, the prevalence of drug use in the community may be measured by the frequency of occurrences of social problems and crimes in the society (Wiessing *et al.*, 2001). Scientific evidence supported an direct association between alcohol consumption and the risk of cardiovascular diseases, diabetes, cognitive decline, and mortality (Ellison *et al.*, 2007).

Locally and/or illegally produced alcohol may contain some unwanted contents in which some are very toxic and dangerous to human health (WHO, 2014). Research have consistently revealed that, nearly every organ of the human body can be damaged by over consumption of alcohol (WHO, 2009; Bhunu, 2012; WHO, 2014; Huo *et al.*, 2016). WHO indicates at least three million deaths and 132.6 disabilities due to harmful use of alcohol annually (WHO, 2018). This translates to six deaths every minute and five disabilities occurring fortnightly. This serves as an indication that alcohol, if not taken responsibly, can have serious consequences to human health. At least 20% of total deaths attributable to harmful use of alcohol is due to cardiovascular diseases (CVDs) which accounts for more than half a million deaths each year globally. CVDs contributes 10% of the total disease burden attributable to alcohol (WHO, 2009). Hypertension, cardiac arrhythmias and heart failure are all adversely affected by effects of over consumption of alcohol, with a more complex picture for ischemic heart disease (WHO, 2009; Bhunu, 2012).

Studies further underscore the age related harmful effects of alcohol consumption. In 2004, for instance, it was estimated that 58.8 million deaths occurred globally with at least 50% of the total global alcohol related deaths involved people of 60 years and above. This largely differs with statistics from African countries where alcohol related deaths took more younger people than older population. It was reported that only 20% of alcohol related deaths occurred in Africa involved people of 60 years and above. The remaining 80% of the total deaths involved youths and children (WHO, 2004). This is to say, 80% of human resource of the continent is lost prematurely every year. Comparatively, over-consumption of alcohol causes more harm to human health than tuberculosis, HIV/AIDS and diabetes (WHO, 2018). At least 40% of all alcohol-related deaths are due to cancer and cardiovascular diseases (CVDs) combined, which accounts for more than one million deaths each year globally (WHO, 2004). Furthermore, alcoholism is linked to liver cirrhosis (WHO, 2014; Huo *et al.*, 2016) and accounts for 10% of the total disease burden attributable to harmful use of alcohol (WHO, 2009). The literature suggests that alcohol consumption increases risks of cancer infections, attributable to 20% of alcohol related deaths. Common types of cancers related to alcohol over consumption are cancers of the mouth, oropharynx, esophagus, colorectal, liver and breast (Huo *et al.*, 2016; WHO, 2009; Bhunu, 2012). Since these health risks are directly attributed to alcohol drinking behaviors, the spread of drinking behaviors implies the spread of health related risks. Health risks mitigation efforts may be successful if alcohol drinking behaviors mitigation efforts are taken into consideration.

Nevertheless, alcoholic beverages has been shown to have beneficial effects on health such as prevention of thrombosis of the heart (Grönbaek, 2009). For instance, it was revealed that some families use locally made alcoholic beverages as food to improve their nutritional status (Tusekwa *et al.*, 2000). However, a broader public health message of the beneficial effects of alcohol does not seem to be of interest in some societies, where only a small fraction of the population are non-drinkers. The acceptable threshold for alcohol intake is estimated to be 21 standard bottles per week for men and 14 drinks per week for women (Grönbaek, 2009; WHO, 2014; NHMRC-Australia, 2001). Alcohol related health risks may be classified into different levels depending on the amount of alcohol consumed. In view of this, NHMRC-Australia (2001) identifies multiple levels of health related risks by defining low risk regular drinking as at most four standard drinks per day for men and not more than two standard drinks for women. Tolerable limit is six standard drinks for males and four standard drinks for females.

In the context of Tanzania, the volume of standard bottle of a beer carries between 250mls to 500mls with concentrantion range of $3\%v/v$ to $6\%v/v$ while the specific volumes for spirits and wines depend on the package. Occasionally, higher level of alcohol intake can be tolerated provided safety precautions and restrictions are observed. These include drinking less when pregnant and not drinking and driving, just to mention a few. However, the current practice reveals that, a good proportion of drinking population violate this standard. For instance, studies reported that 46% of Austrarian males and 32% of females violate the minimum acceptable standard of alcohol drinking, at least once a month (Chikritzhs *et al.*, 2000). However, the anecdotal survey shows that, in Tanzania, both moderate and higher alcohol drinkers would take more than the accepted standard called low risk drinking whenever they go out for a drink. While it may be true that individuals can control alcohol intake, this could, however, facilitate the spread of alcohol drinking behaviors and the resulting dependency. In this regard, the need to shape drinking behaviors should be underscored.

The influence of religious based organizations in shaping drinking habits have been considered and appreciated in some studies (European Commission, 2007; Francis *et al.*, 2015). For instance, despite the highest per capita alcohol consumption in Europe, where individual countries' results about alcohol consumers varied between 60% for Italy to 93% for Denmark, 53% of the Turkish population abstained from using any type of alcohol (European Commission, 2007). Turkish statistics on alcohol use suggest that being a Muslim majority country could be the major reason (European Commission, 2007). This argument has gained support from the technical report by WHO (2014), where most of Islamic States recorded low per capita alcohol consumption in records compared to the non-Islamic States. Afghanstan, for instance, had no record of per capita alcohol consumption for its population of age 15 years or above; but it was estimated to be 0.7 liters of pure alcohol. In Iraq, Kuwait and Iran per capita alcohol consumption are 0.5, 0.1 and 1.0 liters of pure alcohol respectively (WHO, 2014).

Despite the fact that, for some cultural reasons in some countries, respondents tend to under report their consumptions (European Commission, 2007), Islamic States have maintained low scores of Heavy Episodic Drinking (HED) prevalence for the population of age 15 years or above. For instance, WHO (2014) records the HED prevalence score of less than 0.1 for both Iraq and Islamic Republic of Iran, while Afghanistan and Kuwait scored 0.1 and 0.4 respectively. The low HED prevalence scores in these countries may not have happened coinciden-

tally. Chances are that, low HED scores in these countries have been influenced by the religious beliefs these countries identify with. Similarly, the prevalence of Heavy Episodic Drinking among drinkers population of age 15 years or above for these countries range from 0.4 to 4.3 (WHO, 2014). Again this serve as an evidence that, religious belief play a great role in regulating the country's alcohol consumption. Most of these countries have also recorded the least score on alcohol attributable years of life lost (WHO, 2014).

Contrary to religious beliefs, most people have continually been influenced by pressure groups and companions in shaping their drinking behaviors in the face of prestige and other factors associated to drinking behaviors (European Commission, 2007; Francis *et al.*, 2015). Parental influence has also been identified as shaping individual's behaviors including alcohol consumption (Francis *et al.*, 2015). Problems caused by irresponsible drinking of alcohol often affect entire families and communities. Similarly, in economic perspective, a significant portion of the family budget is often spent on alcoholic beverages. For instance, in 2002, the global cost of harmful use of alcohol was estimated to be between US$ 210 – 665 billion, accounting for between 0.6% and 2.0% of global gross domestic product (WHO, 2009). It appears, therefore, that the recruitment and spread of alcohol drinking behavior is escalated when an individual is subjected to social groups such individual identifies with. In view of this, models that try to address the behavioral dynamics should incorporate peer influence in a way *force of infection* is used in the infectious diseases models.

Tanzania's population, as in other countries, suffers from the challenges of overconsumption of alcohol. In 2014, for instance, WHO reported an average per capita consumption to be 6.8 liters for Tanzanian population of people aged 15 years or above (WHO, 2014). This is arguably large compared to the global average of 6.2 liters and 6.0 liters in Africa. In the other hand, among Tanzanian drinkers population, alcohol per capita consumption was as high as 19.5 liters (WHO, 2014). The report further revealed that, the prevalence of heavy alcohol drinking regardless of sex is 14.2% of Tanzanian population aged 15 years or above (WHO, 2014). It is worth noting that, these consumed at least 60 grams of pure alcohol on at least one occasion in a month. Similarly, 34% of Tanzanian drinkers population were reported to have consumed at least 60 grams of pure alcohol on at least one occasion in a month (WHO, 2014). In view of the situation, the Tanzanian government has made deliberate measures to mitigate alcohol consumption levels of its citizens. Of the recent, for instance, the government issued a total

ban on liquor packed in plastic sachet popularly known as *viroba* (singular kiroba), with effect from 1st March, 2017 (Daily News, 2017). The government challenged the packaging method of using the convenient plastic sachets which increased their use down to school children.

The successful part of the ban is that, it reduced the wide range in which viroba was used, whereas viroba were used by pupils in class, teachers at school, drivers and conductors in their buses/vehicles just to mention a few. Presently, this is no longer happening. However, the inefficient part of the ban appears as alcohol producers in the country came up with the marketing strategy in which banned products in sachets are being repacked in big bottles and made available in tots with the same price and taste as of viroba. It is also worth noting that the government's intervention came following long due lamentations from the citizen on potential heath risks in connection to over consumption of such alcohol. In view of the failed mitigation strategies employed in Tanzania, it is reasoned that key components governing the spread of alcoholism, its recruitment mechanisms, and other important aspects of population dynamics were not properly featured. Therefore, mathematical modeling which involves the key components affecting the consumption patterns and dynamics of alcohol related health risks is required. The model that incorporates multiple levels of alcohol related health risks and virtually protected population proportion.

1.2 Statement of the Problem

Alcoholism has continually posed health challenges in Tanzanian population in decades. A number of studies have been conducted to examine the etiology of high-risk drinking and to design prevention strategies to reduce unhealthy alcohol-consumption and related harms. Despite the available literature, at least to the best of my knowledge, the role of socio-cultural beliefs has not been factored in the reviewed mathematical studies. The gap therefore, remain in identifying and investigating key components that affect consumption patterns during drinking events and their association to health risks. Informed by the *Theory of Reasoned Action*, this study integrates the aspects of religious beliefs and peer pressure as parts of socio-cultural beliefs in the control of alcoholism.

1.3 Rationale of the Study

Tanzanian population has continually suffered from the challenges of overconsumption of alcohol for decades. While the global per capita consumption for the population of people aged at least 15 years is 6.2 liters, the consumption rate for Tanzanian population of the same age group is reported to be 6.8 liters (WHO, 2014). This is arguably large compared to the global average and the consumption of African population which is 6.0 liters in Africa (WHO, 2014).

In the other hand, among Tanzanian drinkers population, alcohol per capita consumption was as high as 19.5 liters (WHO, 2014). The report further revealed that, the prevalence of heavy alcohol drinking regardless of sex is 14.2% of Tanzanian population aged 15 years or above (WHO, 2014). This calls for deliberate interventions to rescue the situation from getting in more worse situations. In view of the situation, the Tanzanian government has made deliberate measures to mitigate alcohol consumption levels of its citizens. Of the recent, for instance, the government issued a total ban on liquor packed in plastic sachet popularly known as *viroba* (singular kiroba), with effect from 1st March, 2017 (Daily News, 2017).

The effects of alcohol on health and social well-being can be observed through two major perspectives: the effects due to diseases and injuries associated with alcohol use; and the impact of alcohol on an individual and the extended effects beyond the individual to the family and community through violence, accidents and the risk behaviors associated with alcohol. At least 60% of total global deaths are due to non-communicable diseases/conditions including alcoholism (WHO, 2004). In 2002 for instance, alcohol use was reported to be responsible for about 2.3 million premature deaths worldwide, accounting for 3.7% of global deaths. It is the fifth leading contributor to the global disease burden (WHO, 2009).

1.4 Objectives

1.4.1 General objective

The aim of this study is to develop a mathematical model, analyze and simulate it to investigate the dynamics of health risks associated with alcoholism for best bet intervention approaches with reference to Tanzania. The specific objectives of this study are as follows:

1.4.2 Specific objectives

(i) To develop a mathematical model and analyze it for dynamics of health related risks associated with alcoholism.

(ii) To develop a mathematical model and analyze it for dynamics of alcohol related health risks with interventions.

(iii) To evaluate the cost-effectiveness in the controlled model for dynamics of alcohol related health risks with interventions.

(iv) To develop a fuzzy mathematical model and analyze it for dynamics of health related risks associated with alcoholism.

1.5 Research Questions

This study seek to answer two key questions as listed bellow:

(i) Is there any social psychology based theory which can best describe alcoholism behavior and its spreading nature?

(ii) Can the health risks associated with alcoholism in the community be modeled?

1.6 Significance of the Study

Alcoholism has been seen to have both benefits (economically and Health-wise) and detrimental effects (health-wise and socially). Hence, there is need to have a balance between these two fronts. But to establish the trade-off it is important to understand the transmission dynamics to alcoholism and consequently the health risks associated or tied to drinking behaviors, as well as ways to mitigate against risks and consequences. This thesis in an attempt to address these issues formulated mathematical models for conversion of individuals to alcoholism (Chapter 3, Section 3.2), accounted for uncertainties conversion characteristics using the fuzzy logic theory (Chapter 3, Section 3.4), discussed interventions to manage alcoholism (Chapter 3, Section 3.5) and finally carried out cost-benefit analysis to establish the trade-off (Chapter 3, Section 3.6). In the nutshell, the significance of this study were gain insight of the evolution of alcoholism,

understand the critical factors or behaviors propelling alcoholism, establishing migration measures for the spread of alcoholism and finally to quantify the benefits and risks associated with alcoholism to inform policy.

1.7 Delineation of the Study

Similar to other social behaviors, most people tend to hide their drinking status especially when they belongs to social groups with controversial opinions on alcohol drinking. Consequently, most people are likely to compromise when their drinking status and loyalty to their religious doctrines are questioned. There were complexities resistant and complex behaviors making it difficult in collecting sufficient and reliable data, as also reported by O'Dwyer *et al.* (2019). The complexity of the model owes to and attempt to capture reality and insufficient data to fit the model and estimate relevant parameters. However, the similarities in drinking behaviors, patterns, frequencies, and drinking cultures in public venues observed in the survey and anecdotal survey made it easier. Apart from under reporting one's drinking status, alcohol drinking being a behavioral issue is not properly recorded.

CHAPTER TWO

LITERATURE REVIEW

2.1 Introduction

The field of mathematical modeling and its applications has become the domain of solution based research in communities. Scholars have attempted to address alcoholism in different ways. A number of studies have been conducted on alcoholism, socio-cultural beliefs such as religiosity, and modeling perspectives especially in developed countries.

Mathematical approach by means of model development, simulation and analysis regarding the question of alcohol epidemic has been employed in an attempt to provide insight on effects of alcohol consumption on health and socio-economic aspects of the society. This chapter provides reference of the current state of modeling health risks epidemic associated with alcoholic behavior. It discusses and identifies current open problems reflecting on realistic community. It also proposes new research perspectives in the mathematical modeling of alcoholism and its related risks.

2.2 The Dynamics of Alcoholism

2.2.1 The effects of religious beliefs

Religions have continuously acted as a control agent of addictive substances including alcoholism. Where religiosity is defined as organized set of beliefs and measurable practices within a community of people who accept an authoritative doctrine (Koenig *et al.*, 2001). Religiosity has positive effects on both physical and mental health (Koenig *et al.*, 2001). This fact has propelled researches regarding direct or indirect role of religion on health (Miller *et al.*, 2003). However, an increased number of individuals who actively participate in religious activities might have contributed to the increased interest of research on the relationship between religiosity and health (Creel, 2007). Francis *et al.* (2015), for example, among other reasons, loyalty to religiosity is attributed to people abstaining from alcohol drinking. Since the definition of religiosity is debatable, this study, uses the definition by Koenig *et al.* (2001), which views religiosity as organized set of beliefs and measurable practices within a community of people who accept an authoritative doctrine. Motivated by the above studies, this study considers religiosity as an important aspect in the model formulation where religious individuals form

a population proportion in the community. The peculiarity of this group is that, its members receive a non-permanent virtual protection from alcohol drinking behaviors. Since members engage actively in religious activities, religiosity, in this regard, helps members replace their previous drinking habits with other socially acceptable behaviors.

2.2.2 Effects of peer influence

Peer influence has been closely associated with spread of alcoholism in the community (Benedict, 2007). Different cultures in Tanzania, for instance, promote alcohol drinking habits through cultural songs and other cultural practices. Advancement of science and technology and vast increase of social media applications available in both android smartphones and iOS phones has narrowed the world into a small village as far as social interactions is concerned. The physical separation and distance is no longer a barrier for interaction among the people. The social media platforms for instance, facilitate the social interactions. These platforms provide useful interfaces where people can interact both individually and in designated social groups. The digital interactions ranges from all sorts of jokes to serious discussions on matters pertaining a variety of disciplines.

Everyday, a number of alcohol based online created memes are pumped in various social media platforms, most of them carrying positive connotations to alcoholic beverages and consumers. These types of social media contents have become influential to the young generation, persuading them to use the products. On the other hand, peer influence and social pressure is recorded as the main agent to the increase of alcohol abuse in University communities (Benedict, 2007). This fact is supported by Walsh *et al.* (2014) who examined parental, peer and school predictors of alcohol drinking among Israeli-born adolescents. The study involved first and second generation adolescent immigrants from the Former Soviet Union (FSU) and Ethiopians in Israel. By using Pearson's Chi square and logistic regression models, the differences between the groups for drinking and group specific predictors of drinking was examined. The study observed high levels of binge drinking and drunkenness among first generation FSU and Ethiopian groups who had greater time with friends while maintaining lower levels of parental monitoring than Israeli-born adolescents. Group specific logistic regression models proved that, time spent with peers consistently predicted immigrant alcohol use. The study suggested further that, drinking patterns must be understood in relation to country of origin and immigration experience of a particular group.

Recently, a study conducted in Tanzania revealed that, most of alcohol drinkers had their first drinking experience at the social event (Francis *et al.*, 2015). This initiation experience may be closely associated with the influence of peer pressures and provision of free and easy access to the alcoholic beverages in exciting moments. It was further reported that, between 31 - 66% of the drinkers population took standard bottled beers during their first time drinking while 36 - 45% of the school girls had the locally made brews as their first experience (Francis *et al.*, 2015). This implies that exposure to drinking context is yet another important pulling factor to alcoholic behaviors. In Tanzania and many other African countries, most local brewery activities are performed by women with the assistance from their daughters. The practice exposes girls to drinking environment than boys. Generally, drinking habits at younger ages, is unacceptable in many communities. In 2007, for instance, The Directorate General Communication (DGC) reported that, at least seven out of ten (76%) of the European population opined against alcohol advertisements targeted the young citizens in all member states (European Commission, 2007).

While both Benedict (2007) and Walsh *et al.* (2014) insist on the importance of peer pressures in influencing behaviors, Francis *et al.* (2015) and European Commission (2007) underscore the role of early exposures and parenting as an important in the initiation of unwanted behaviors including alcoholism. In view of this, the present study considers peer influence as an important behavioral recruitment agent playing the vital roles of force of infection in the context of infectious diseases modeling.

2.2.3 Application of the Theory of Reasoned Action (TRA)

The Theory of Reasoned Action (TRA) was pioneered by Azjen (1980) to describe the influence of one's beliefs and social pressure in determining individual's course of action. The theory details the mechanism of an individual's intention to initiate a new behavior as a result of their change of attitude. According to Azjen (1980), intention variable which ultimately determines whether an individual is likely to engage with the behavior or not is determined by their attitude toward the behavior in question. Social psychology defines attitude as one's evaluation or assessment towards an object, situation or phenomenon. In the context of this study, an attitude towards alcohol drinking can be positive or negative. If the attitude is negative, there is less chance that an individual will engage with the alcohol. But if an individual is in favor of alcohol, even if a particular individual does not drink, there is higher chances that such individual

will eventually engage with alcohol drinking in the near future. The question is, therefore, how does one's attitude form? What makes the difference between individuals who are in favor of alcohol drinking and those who hold negative attitude?

According to the theory, it is the beliefs and subjective norms that influence individual's attitude. Belief comprises of all that an individual thinks regarding the object, situation or phenomenon. In the context of this study, individual's beliefs on alcoholism could be positive or negative depending on the information that the person has regarding alcohol. If, for instance, an individual believes that alcohol is sinful and undesirable to health, it is unlikely that this person will hold positive attitude toward drinking. But if the person believes that alcohol is good for health, nutritious and desirable, it is unlikely that such individual will hold negative attitude towards drinking.

The theory argues that our beliefs are influenced by subjective norm, a second variable that pressures the people to change their opinion towards an object, situation or phenomenon in question. Subjective norm comprises of all that a target believes that the people around him/her are thinking about the object, situation or phenomenon. In the context of this study, subjective norm includes the opinion and the actual behavior of the close associates and acquaintances of the target. If, for instance, the target believes that the friends or the social groups and individual identifies with are in favor of alcohol drinking behavior, one's attitude towards alcohol is likely to be positive. This explains why individuals who associates with drinkers are likely to be recruited into drinking due to the social pressure to conform with group identity. Similarly, individuals who associate with friends and acquaintances with not only negative attitude towards drinking, but refraining from drinking, are less likely to be recruited into drinking. In this regard, individuals who associate with conservative religious groups are less likely to start drinking unless they break their identity with their religious groups.

In view of the theory, it can be argued that no one drinks alcohol accidentally. An individual will consciously consume alcohol if such an individual expects that the positive affective consequences of drinking will outweigh those of not drinking and vise versa is true (Cox *et al.*, 1988). An individual's decision on whether or not to take an alcoholic drink is determined by various factors including past experiences with drinking and current life situation. These factors will help to form expectations of affective change from drinking, these factors always modulated by a individual's neurochemical reactivity to alcohol (Cox *et al.*, 1988). In Francis *et al.* (2015),

parental influence is featured as one among the main reasons some people did not engage in the drinking behaviors. If alcohol drinking is not something the parents feel proud of, then no one under their guidance would be expected to drink while hunting the approval of the parents.

2.3 Alcoholic Models

In epidemiological studies, transmission of infectious agents in the host population is a key process that requires descriptive analysis when the model compartments is used to study a particular infectious disease (Mandal *et al.*, 2011). Mathematical models may be extended to describe behavioral dynamics and transmission where people already in the behavior may act as transmission agent in the host population provided a desired amount of interactions between them is allowed. When a behavior associated with health risk factors emerge in any community, the total population in the community can be partitioned into a number of categories depending on the risk levels or defined patterns individuals exhibit. Mathematical modeling of alcohol drinking epidemic and its consequences on human health has been an interesting topic for many researchers. The similarity between the spreading nature of alcoholism behavior and that of infectious diseases has attracted modelers to use mathematical modeling as an essential tool for simulating the behavior and provide valuable control analysis. Most models developed in relation to alcoholism and its consequences fall in the category of SIR with or without significant modification (Bhunu, 2012; Huo *et al.*, 2016; Wang *et al.*, 2014; Mushayabasa, 2015).

The extension basic SIR model approach were appreciated in modeling the spread of alcoholism in the community (Bhunu, 2012; Huo *et al.*, 2016). While remaining conventional to SIR model, Bhunu considered deterministic models by splitting alcoholic population into two classes based on different consumption levels and recovered individuals relapsed into alcoholic class (Bhunu, 2012). The study divides human population into four population proportions: susceptible, drinkers $-$ consisting alcohol consumers who have not become alcohol dependent, alcohol dependent, and recovered. Bhunu (2012) aimed at gaining insight on the growth of alcoholism as a health and social problem. The study establishes that, it is easy for moderate drinkers to quit alcohol drinking than the alcoholics. Thus, any effort geared to encourage and support moderate drinkers to quit drinking will be effective than doing the same to the alcoholics. Owing to this study, it is clear that severity of alcohol induced harms can be associated with drinking status of individuals. However, with reference to Tanzania, two population com-

partments may not exhaust drinking communities. In that regards, the present model considers alcohol related health risks as a staged process involving three risk stages depending on their alcohol consumption levels, making it appropriate to most developing countries.

Wang *et al.* (2014) investigated the optimal control strategies in alcoholism using deterministic $SATQ$ type mathematical model. This is another SIR like model with slight modification to accommodate treatment intervention. In this study, the spread of alcoholism is studied with two control strategies u_1, and u_2, to gain insights about health and social phenomenon. Where u_1 indicates the proportion of susceptible population which successfully avoids to stay off the alcoholism; u_2 represents the proportion of alcoholic population which takes part in treatment. The model considered the closed environment with the total population in four compartments: the susceptible compartment, $S(t)$ −with individuals who either do not drink or drink moderately without affecting the physical health; the alcoholism compartment, $A(t)$ −with individuals who binge drink and affect the physical health seriously; the treatment compartment, $T(t)$ −with individuals who have been receiving alcohol related treatments after alcoholism; and the quitting compartment, $Q(t)$ −which refers to the individuals who recover from alcoholism after treatment and stay off alcohol hereafter. The study proposes two equally important effective control strategies measured by reduction in the number of alcoholics and increase in the number of susceptible population. However, grouping moderate drinkers and susceptible populations together is challenged by recently published scientific study affirming that alcohol consumption at whatever level poses health challenges and that there is no healthy drinking of alcohol (Griswold *et al.*, 2018). In addition, Wang *et al.* (2014) ignored the contribution of social cultural practices in the control of alcoholism in the community, the present study considers it an important component in the model formulation.

Nevertheless, Huo *et al.* (2016) considered a relapse alcoholic model on weighted network by dividing the total population into susceptible, S, infectious, I, and recovered, R, making a simple SIR model. They studied the peer influence on individual's drinking dynamics. The analytical results looked at interaction between susceptible and alcoholism, and recuperator recurrence drinking alcohol as the determinants of alcoholic problem. Huo *et al.* (2016) suggested two effective controls to eliminate alcoholic problem in the community. The controls are namely, reduction of interaction between susceptible and alcoholic individuals and ending reculperator recurrence. While both Bhunu (2012) and Huo *et al.* (2016) suggested some work-

able intervention programs, they did not consider the effects of social cultural beliefs which plays an influencing roles in molding peoples' behaviors (Francis *et al.*, 2015; Rosmarin *et al.*, 1998). Also, they partially agree with the multilevel risk of alcohol consumption and its relationship with health and social outcomes as suggested by Rehm *et al.* (2004). The multilevel risks of alcohol consumption can be meaningful when extended beyond two classes.

Similarly, Mushayabasa (2015) used an SIR-like-mathematical model to investigate the role of optimal intervention strategies on controlling excessive alcohol drinking and its related adverse health effects in the community. The study considered the transmission process as the social contract between heavy and light alcohol consumers within an unchanging shared drinking context. Two models −an autonomous system with constant parameters in relevant alcohol drinking components and alcohol related treatment model− incorporating peer influence were proposed and analyzed qualitatively. The model development considered the total drinking population in four different population compartments depending on individuals' alcohol consumption level. The population segments include Susceptible, $S(t)$ − consisting of light alcohol consumers; heavy, $H(t)$ − consisting of heavy alcohol consumers; $A(t)$ − consisting of individuals receiving alcohol related treatment and consume alcohol occasionally; and recovered, $R(t)$ − consisting of both individuals on treatments and those who have successfully completed treatment and permanently quit alcohol consumption. The study suggests that, effective control of high-risk alcohol drinking can be achieved if more resources and efforts are devoted on weakening the intensity of social interactions between light and heavy drinkers. It further suggests that, time dependent interventions have the potential to eliminate the problem of excessive alcohol use. Having alcoholic population in three classes depending on their consumption level answers the multilevel risks challenges appeared in the previous literature. However, the study considered only drinkers population and left non drinkers population out of the system. As observed in the previous studies, Mushayabasa (2015) also ignored the contribution on social cultural beliefs in controlling the problem.

Likewise, Xiang and collaborators worked on global property of a drinking model with public health campaigns (Xiang *et al.*, 2016). To describe the problem, drinking population was divided into five subgroups: susceptible drinkers, $S(t)$ −moderate alcohol consumers with no symptoms of alcohol related problems and refuses public health education; educated drinkers, $E(t)$ −moderate alcohol consumers who accepts public health education; alcoholics, $A(t)$

$-$alcohol consumers with drinking problems or addictions; and temporarily recovered drinkers, $R(t)$ $-$former alcoholics who have entered treatment and are abstaining from alcohol; quit drinkers, $Q(t)$ $-$who permanently quit drinking. With the help of Lyapunov function, global stability of equilibria of the model is derived. The basic reproduction number, $\mathcal{R}_0$, was obtained by means of the next generation matrix and the global stability of model has been proved by using the Lyapunov function. The study analysis revealed that, public health educational campaigns to drinking individuals can slow down the drinking dynamics. Some numerical simulations were also used to support this conclusions. Again this work is not free from the challenges of ignoring the influence of social cultural beliefs existing in the society.

A non-linear $SHTR$ model presented by Adu *et al.* (2017) studied the dynamics of drinking epidemics. The model compartments were susceptible (S) which took non drinkers population and the compartment (H) recruited heavy alcohol drinkers. The other population compartments are (T) which recruited population proportion going through treatment, and the recovered (R). In this study, the conditions for existence and stability of drinking free and endemic equilibria points were established by using Lassalle's invariance principle of Lyapunov function. Later, analytical results were confirmed by some numerical simulations putting forward three useful methods of combating the drinking epidemics. These include: reducing the contact rate between non drinkers and heavy drinkers; increasing the number of drinkers that go into treatment; and educating to refrain from drinking. The authors generalized the concept of alcoholism without considering multilevel risks phenomenon based on one's drinking patterns and frequency.

A risk-structured model for the spread of drug abuse is appreciated in estimation of an epidemic abuse reproduction number (Musanyu *et al.*, 2018). Musanyu *et al.* (2018) divide human population into six population compartments namely, $S_H(t)$ for population at high risk of initiating drug abuse; $S_L(t)$ for those at low risk of initiating drug abuse; $U(t)$ denotes drug users not in rehabilitation; $T(t)$ for clients of rehabilitation services and treatment; $R_H(t)$ for recovered individuals at high risk of relapsing; and $R_L(t)$ for recovered individuals at low risk of relapsing. In this complex model, the question of multilevel risks has been addressed and the model solution revealed that public health education on skills to handle the risky situation may be the best approach to protect individuals from initiating or re-initiating into drug abuse. However, the study did not consider the importance of social cultural influences in molding human behavior.

Mundt *et al.* (2012), formulated and analyzed a stochastic actor-based model for the effects of

peer selection and influence on adolescent alcohol use. The model was developed based on the assumptions that, changes in the network take place in accordance to continuous-time Markov chain with stationary transition distribution where the future state of the network depends on the present state only. The study aimed at disentangling selection associated with the dynamic interplay of adolescent friendship and alcohol use. By analyzing data from Add Health, a longitudinal survey of selected students enrolled between 1995 and 1996 in the U.S revealed that, peer selection plays a major role in alcohol use behavior among adolescent friends.

2.4 Conclusion

In light of the above literature, it is clear that mathematical modeling approach towards solving different problems have continually attracted most researchers. This is also true for health problems and its associated risks such as the consequences of alcoholism. However, the modeling largely depends on behavioral criteria than physical and biological personalities. The challenges includes defining, quantifying and measuring the phenomena of studies (Sloboda, 2002).

Reviewing and improving available models from time to time for the purpose of increasing its usefulness is important since even the best models have some imperfections in one way or another (Box, 1979). In view of the above literature, the open problem which comes out clearly includes: developing complex and more relevant models for the dynamics of health related risks associated with alcoholism and its control strategy addressing the main aspects raised above as the challenges to most of the studies. The models presented in the next chapters address the influence of external motivations on the spreading of alcohol abuse by introducing different population segments with distinct expositions, towards and resistance to the influence of drugs. Since different levels of alcohol consumption have different health effects (Griswold *et al.*, 2018), the present study has considered drinking population in three categories depending on their alcohol drinking habits as follows;

(i) **Low risk population:** a population proportion drinking alcohol once in a while, for instance, in special events. It is also known as occasional drinkers.

(ii) **Moderate risk population:** a population proportion drinking alcohol on regular basis but has not developed symptoms of alcohol dependency.

(iii) **High risk population:** a population proportion consuming alcohol frequently and in

large amount, and may or may not have developed symptoms of alcohol dependency but has crossed the threshold of alcoholism.

Inclusion of the influence of external motivations with positive and negative influential effects, and a clearly defined alcohol drinking population distinguishes this study from the rest of existing models over the subject.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Introduction

In this study, ethnographic approach were used to collect useful information in the model development. The use of ethnographic methods is appreciated in research studies related to substance use and have provided desirable insights into social and cultural practices of different social groups (Elijah, 1990; Becker, 1953; Philippe, 1995; Briggs, 2013; Griffin *et al.*, 2009; Maher, 2002; Preble *et al.*, 1969; Rubin *et al.*, 1975; Ward, 2010; Young, 1971; Briggs *et al.*, 2015; Barnes, 2017). However, substance abuse remains to be one of the sensitive topics or issues to discuss (O'Dwyer *et al.*, 2019). The validity of information supplied by the respondents largely depends on the approach and assurance of confidentiality. Alcohol drinkers like other drug users are likely to discuss their drinking behavior to their drinking fellows than acquaintances. In some situation, alcohol drinkers tend to hide or give compromised information related to their involvement in alcohol drinking behaviors (O'Dwyer *et al.*, 2019).

This study considers two main aspects, the psycho-social and mathematical aspects. In the psycho-social aspect, alcohol drinking and group cultures are considered, especially in the shared drinking venues. On the other hand, mathematical aspect was exhibited by the use of Ordinary Differential Equations (ODEs) in the formulation of mathematical models. The study also considered religiosity as one of the powerful social cultural beliefs affecting human behaviors. It examined the combined effects of group cultures and religiosity in the dynamics of health related risks in connection to alcoholic behaviors with reference to Tanzanian population.

3.2 Formulation of the Basic Models

Informed by ethnographic survey results in which five latent variables were examined, the model was formulated by using Ordinary Differential Equations (ODEs). The ethnographic survey results were supplemented by anecdotal survey and researcher's participatory observations regarding the subject matter.

3.2.1 Latent Variables

The survey collected data related to six different aspects; influence of religious beliefs in abstaining alcohol intake, personal alcohol drinking attitude, frequency, patterns, types of alcoholic beverages used, and diagnosed health related risks associated with alcohol consumption.

3.2.2 Ethnographic Survey

In this approach, with verbal consents, two groups were involved in a series of observational events and interrogations during the first year of this study. While the first group consisted of alcohol drinkers, the second group involved non-drinkers. Selection of the group participants were done considering the readiness and personal interests of an individual to take part in the study. Information were collected by means of interacting with drinking group and interrogations. Natural participatory observation was used, where the researcher interacted with drinking individuals. This is an efficient approach since respondents were free to reveal their information (with verbal consents) without fear of being interrogated by acquaintances. The secondary data were drawn from related scholarly works. Other information which could not be captured by literature and ethnographic survey were estimated for simulation purposes.

3.2.3 Model Descriptions

The basic model, mathematical models for the dynamics of alcohol related health risks $(SPLMAR)$, is formulated following a set of latent variables regarding to Tanzanian multicultural drinking context. Adopting the framework of Xiang *et al.* (2016), the model considered a total population in six compartments based on individuals' risk levels defined by their drinking habits.

Let $S(t)$ be the risk susceptible population which carries individuals who are not drinking and/or never drank alcohol but are susceptible to the habit. Their abstinence from alcohol drinking is not founded on social or moral obligations restricting them from alcohol drinking. $P(t)$ is the protected population consisting of individuals who have either quited alcohol drinking or never drank alcohol due to restrictions set by their denominations/religions. The low risk compartment, $L(t)$ is consisting of individuals with low risk drinking habit, low and occasional alcohol consumers who drink responsibly. The model has medium risk compartment, $M(t)$ $-$consisting of individuals with moderate risk drinking habit, regular alcohol consumers$-$ and

Alcoholic addicted population, $A(t)$, consisting of individuals with harmful or high risk alcohol drinking habit. Nevertheless, the recovered population, $R(t)$, consisting of individuals recovering from risky alcohol drinking habit through receiving treatment of diseases attributable to alcohol drinking, where quitting alcohol becomes an essential part of treatment.

A non-drinker acquires alcohol drinking habits through social contacts (Bhunu, 2012; Sánchez *et al.*, 2007) at the force of peer influence

$$\lambda = c\beta\left(\frac{L + \theta_1 M + \theta_2 A}{N}\right) \tag{1}$$

where β is the probability of initiation to alcohol drinking, θ_1 and θ_2 are the modification parameters ($\theta_1 \leq \theta_2$), c is the effective contact to influence one into alcohol drinking, and N is the total population.

An individual in the protected compartment assumes the same alcohol drinking status as the recovered individual since they are not drinking as long as they remain loyal to their respective religious teachings and principles. The only difference between individuals in the two compartments is that, a proportion of the protected population, $P(t)$, never drank alcohol in their life. The religious individual (here referred to as protected population) acquires the virtual immunity from joining any of the drinking compartment available. The total population is assumed to be non – constant, with its members having varying social interaction degrees depending on the social classes they belong.

Let π be the constant recruitment rate to the age of 15 years or above where both susceptible and protected individuals are recruited; μ the natural fatality rate of individuals of 15 years or above, which is time constant and occurs naturally at each compartment; α is the fatality rate due to alcohol related diseases –alcohol induced deaths; and that, upon recovery, there is a possibility that recovered individual will re–join a susceptible population at a rate of ω. A proportion ϕ of the recruit enters the religious group while the remaining $(1 - \phi)$ joins the susceptible population.

The susceptible population joins a protected subgroup restricting alcohol taking and hence avoids or reduces significantly the frequency of regular interactions with low, moderate or high risk alcohol drinkers. The rate at which susceptible population joins protected population is γ_1. We assume that some religious individuals will, at times, lose their religious faith and backslide to join susceptible group at a rate of γ_2.

Susceptible individuals enter drinking compartments at the rate λ which is a force of peer influence to induce drinking habit. The proportion ρ of peer influence recruits susceptible individuals into low risk drinking compartment, while the remaining proportion $(1-\rho)$ enters the moderate drinking compartment. Individuals at low risk population proportion consume alcohol to the generally accepted standard on occasional basis. However, after a period of time, the low risk alcohol drinking individuals enter the moderate risk compartment, denoted by $M(t)$; and the protected group, $P(t)$, at rates of σ and ν, respectively.

Similarly, in the course of time, the moderate risk drinkers enter the high risk drinking compartment, $A(t)$; protected compartment, $P(t)$, and recovered compartment, $R(t)$, at rates of δ, τ and ξ respectively. The high risk drinking individual may change behavior by responding to the religious preaching/teachings or public health education. It is therefore assumed that, high risk alcohol consumers recover at a rate η by responding to either, public health education or attending the psychotherapy clinic as the deliberate efforts to change their drinking behaviors. Some high risk consumers join the religious group at a rate ψ by responding to religious based preaching/teachings and hence quits alcohol taking as a result of changing beliefs. It is assumed further that, an individual in the high risk compartment becomes vulnerable to alcohol related diseases. In essence the social cultural beliefs and practices serve as agents of behavior change both in the control and intervention perspectives to the current problem.

3.2.4 Model Assumptions

The alcohol related health risk model was formulated based on the following assumptions:

(i) Drinking behavior is a staged process progressing from low risk drinking to higher risk drinking.

(ii) Drinking habit is a behavioral change which obeys the theory of reasoned action.

(iii) Religious beliefs and other social cultural beliefs influences individuals' behaviors (Rosmarin *et al.*, 1998; Miller *et al.*, 2003; Francis *et al.*, 2015).

(iv) Peer pressure influences individuals' behavior (Benedict, 2007; Walsh *et al.*, 2014; Francis *et al.*, 2015; European Commission, 2007).

(v) Alcohol related health risks is proportional to the amount of alcohol uptake of an individual. The higher alcohol consumption, the greater the alcohol related risks.

(vi) A non-drinker acquire alcohol drinking habits through social interaction with drinkers (Bhunu, 2012; Sánchez *et al.*, 2007; Musanyu *et al.*, 2018; Mushayabasa, 2015; Mushanyu *et al.*, 2016).

(vii) Each population is homogeneously mixed.

(viii) There is no direct progression from susceptible to high risk compartment.

(ix) The natural mortality rate in each of the population states is constant.

(x) The recruitment rate for each population is greater than natural mortality.

(xi) Virtual protection for alcohol drinking acquired by the protected population is not permanent.

The model parameters and state variables used in the formulation of alcohol related health risks model are summarized in Tables 1 and 2 respectively. The description of alcohol related health

Table 1: Model state variables and their descriptions

Variable	**Description**
$S(t)$	Risk susceptible population
$P(t)$	Protected population
$L(t)$	Low risk drinkers
$M(t)$	Medium risk drinkers
$A(t)$	Alcohol addicts
$R(t)$	Recovered population

risks model in the community can be summarized by compartmental diagram presented in Fig. 1.

Table 2: Model parameters and their descriptions

Parameters	Descriptions
π	Per capita recruitment rate
ϕ	The proportion of recruitment joining the protected population
μ	Natural mortality rate
λ	Force of peer influence to induce drinking
β	The probability that a susceptible individual will drink alcohol after prolonged contact with drinking individuals
c	The contact rate between a susceptible member and a drinker necessary to convince him/her to drink.
θ_1	The chances of becoming alcoholic after successful influence of a moderate risk drinker
θ_2	The chances of becoming alcoholic after successful influence of a high risk drinker
ρ	The proportion of susceptible individual recruited to the low risk drinking population
ω	The rate at which recovered individuals join susceptible compartment
γ_1	The rate at which susceptible population joins protected compartment
γ_2	Protection wane rate
ν	The rate at which low risk population joins protected compartment
τ	The rate at which moderate risk population joins protected compartment
ψ	The rate at which high risk population joins protected compartment
σ	Progressive rate from low to moderate risk compartments
δ	Progressive rate from moderate to high risk compartments
ξ	Recovery rate for moderate risk population
α	Alcohol induced fatality rate
η	Recovery rate for high risk population

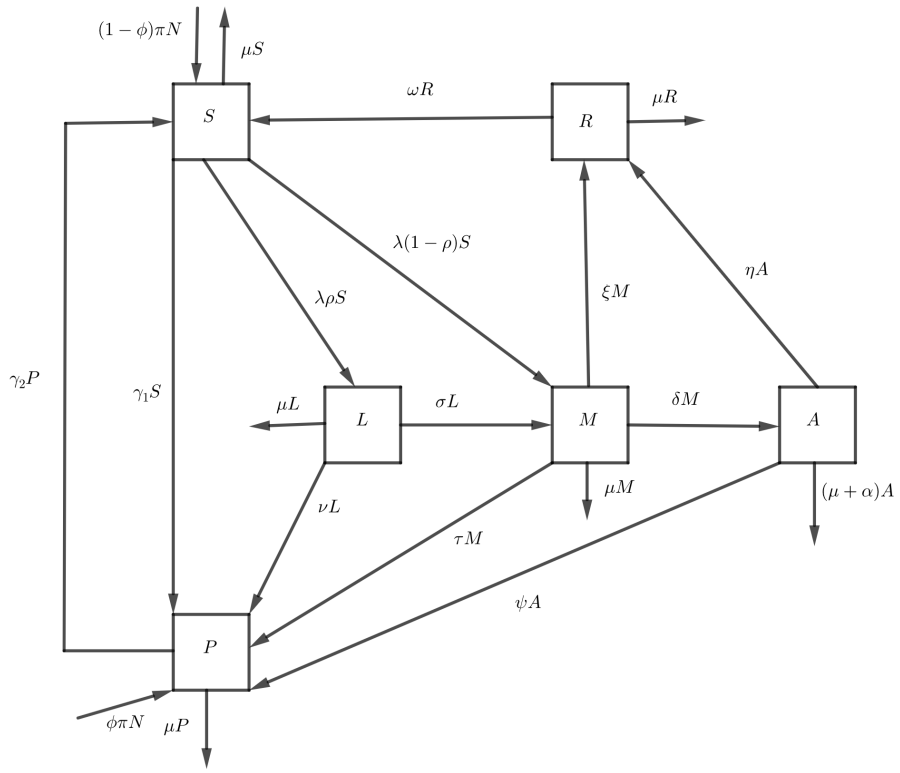


Figure 1: The structure of the dynamics of alcohol related health risks model

3.2.5 Model Equations

Based on the dynamics of model compartment in Fig. 1, and the model descriptions discussed in the previous subsections, the following set of equations are formulated.

$$\begin{cases} \dot{S} &= -(\mu+\gamma_1+\lambda)S + \gamma_2 P + \omega R + (1-\phi)\pi N \\ \dot{P} &= \gamma_1 S - (\mu+\gamma_2)P + \nu L + \tau M + \psi A + \phi\pi N \\ \dot{L} &= \lambda\rho S - (\nu+\mu+\sigma)L \\ \dot{M} &= \lambda(1-\rho)S + \sigma L - (\tau+\mu+\delta+\xi)M \\ \dot{A} &= \delta M - (\mu+\alpha+\eta+\psi)A \\ \dot{R} &= \xi M + \eta A - (\omega+\mu)R \end{cases} \tag{2}$$

where $S \geq 0, P \geq 0, L \geq 0, M \geq 0, A \geq 0$ and $R \geq 0$.

Let N be the total population size given by;

$$N = S + P + L + M + A + R \tag{3}$$

Then the equation describing changes in the total population is given by,

$$\frac{dN}{dt} = (\pi - \mu)N - \alpha A \tag{4}$$

3.3 Model Analysis

3.3.1 Basic properties of the model

In this section, the basic solutions for model system (2) is discussed. The following properties lay down a foundation of proofs of stability analysis results of the model. Following Bhunu *et al.* (2012), let $x \in \mathbb{R}^n_+$ denote the set of state variables $x = (x_1, x_2, \ldots, x_n)$ with the positive components, the following results may be established.

Lemma 3.1

Let $f : \mathbb{R}^n_+ \to \mathbb{R}^n, f(x) = (f_1(x), f_2(x), \ldots, f_n(x))$ with $x \in \mathbb{R}^n_+$, be continuous and there exists a continuous partial derivatives $\dfrac{\partial f_j}{\partial x_i}$ in $\mathbb{R}^n_+$ for $i, j = 1, 2, \ldots, n$. Then f is locally Lipschitz continuous in $\mathbb{R}^n_+$.

Theorem 3.1

Let $f : \mathbb{R}^n_+ \to \mathbb{R}^n$ be a locally Lipschitz continuous and for each $i = 1, 2, \ldots, n$ satisfy $f_i(x) \geq 0$ whenever $x \in \mathbb{R}^n_+, \;\; x_i = 0$. Then, for every $x_0 \in \mathbb{R}^n_+$, there exists a unique solution of $\dot{x} = f(x), \;\; x(0) = x_0$ with values in $\mathbb{R}^n_+$ defined in some intervals such that $0 < x_0 \leq b$ with $0 < b \leq \infty$. If $b < \infty$, then $\sup \sum_{i=1}^n x_i(t) = \infty$.

Theorem 3.2

For all $S(0), P(0), L(0), M(0), A(0), R(0) > 0$, there exists $S, P, L, M, A, R : (0, \infty) \to (0, \infty)$ which solve the model system (2) with initial conditions $S = S(0), P = P(0), L = L(0), M = M(0), A = A(0)$, and $R = R(0)$.

Proof. Applying Theorem 3.1, we define $f_1(x) = \dot{S}, f_2(x) = \dot{P}, f_3(x) = \dot{L}, f_4(x) = \dot{M}, f_5(x) = \dot{A}$, and $f_6(x) = \dot{R}$ where $x = (S, P, L, M, A, R)$. By the properties of continuity over operations, there is a continuity of f_i for all $i = 1, 2, ..., 6$. Furthermore,

$$\frac{\partial f_j}{\partial x} = \frac{\partial f_j}{\partial x_1} + \frac{\partial f_j}{\partial x_2} + \cdots + \frac{\partial f_j}{\partial x_6}, \; \forall j \in \{1, 2, \ldots, 6\} \tag{5}$$

where the derivatives are continuous. It follows that,

$$\frac{\partial f_1}{\partial x} = \left(6\lambda - c\beta\left(1 + \theta_1 + \theta_2\right)\right)\frac{S}{N} - \left(\mu + \gamma_1 + \lambda\right) + 6\left(1 - \phi\right)\pi + \gamma_2 + \omega \tag{6}$$

By Lemma 3.1, f is locally Lipschitz continuous. Let $x_1 = S = 0$ with $x_{i \neq 1} > 0$. Then

$$\frac{\partial f_1}{\partial x} = -\left(\mu + \gamma_1 + \lambda_1\right) + 6\left(1 - \phi\right)\pi + \gamma_2 + \omega > 0 \tag{7}$$

where $N_1 = P + L + M + A + R$ and $\lambda_1 = c\beta\left(\frac{L + \theta_1 M + \theta_2 A}{N_1}\right)$. Similarly, repeating the procedures for the rest of state variables, the following conditions are established

$$\frac{\partial f_2}{\partial x} = (6\phi\pi + \gamma_1 + \nu + \tau + \psi) - (\mu + \gamma_2) > 0 \tag{8}$$

$$\frac{\partial f_3}{\partial x} = \rho\lambda_3\left(1 - 6\frac{S}{N_3}\right) + c\beta\rho\left(1 + \theta_1 + \theta_2\right)\frac{S}{N_3} - (\mu + \nu + \sigma) > 0 \tag{9}$$

$$\frac{\partial f_4}{\partial x} = (1-\rho)\left(1 - 6\frac{S}{N_4}\right)\lambda_4 + c\beta\left(1-\rho\right)\left(1 + \theta_1 + \theta_2\right)\frac{S}{N_4} + \sigma - (\tau + \mu + \delta + \xi) > 0 \tag{10}$$

$$\frac{\partial f_5}{\partial x} = \delta - (\mu + \alpha + \eta + \psi) > 0 \tag{11}$$

$$\frac{\partial f_6}{\partial x} = (\xi + \eta) - (\mu + \omega) > 0 \tag{12}$$

where $N_3 = S{+}P{+}M{+}A{+}R,\ N_4 = S{+}P{+}L{+}A{+}R$, consequently $\lambda_3 = c\beta\left(\frac{\theta_1 M + \theta_2 A}{N_3}\right)$, and $\lambda_4 = c\beta\left(\frac{L + \theta_2 A}{N_4}\right)$. By Theorem 3.2, for every $x(0) \in \mathbb{R}^6_+$ defined in some intervals such that $0 < x_0 \leq b$ with $0 < b \leq \infty$, there exist a unique solution of $\dot{x} = f(x)$.

$$\sup_{0 \leq t \leq b}[N] = \infty$$

provided that $b < \infty$.

3.3.2 Invariant Region

Since the model (2) represents human population, it is assumed that all the state variables and parameters of the model are non-negative for all $t \geq 0$. Now, equation (4) can be written as, $\frac{dN}{dt} \leq (\pi - \mu)\, N$ which upon integration yields

$$N \leq N_0 e^{(\pi - \mu)t} \tag{13}$$

where N_0 is the initial population obtained by evaluating equation (3) at initial conditions of respective state variables. It follows that, for $\mu < \pi$ as $t \to \infty, N_0 \leq N \leq \infty$, therefore $N(t)$ is bounded, posing contradictions to Theorem 3.2. As a result, $b = \infty$, suggesting that feasible solutions of components of system (2) are positive and are defined on the interval $0 < b < \infty$. □

It can be concluded that, the region governing solutions of the model is positively-invariant under the flow induced by system (2). Following the boundedness of model solutions, the following theorem is established:

Theorem 3.3

All solutions of model system (2) are bounded and the components of system enter the region Ω such that $\Omega = \left\{(S, P, L, M, A, R) \in \mathbb{R}^6_+,\ N_0 \leq N \leq \infty\right\}$.

Hence, the region Ω is positively-invariant under the flow induced by system (2), that it is well posed mathematically and epidemiologically, sufficiently for the dynamics of the flow generated by system (2) to be considered in Ω.

3.3.3 Existence of Steady State Solutions

The steady state $\Phi^*(S^*, P^*, L^*, M^*, A^*, R^*)$ of the model system (2) is obtained by setting the LHS to zero and solving the resulting system of equations (14) simultaneously for the state variables.

$$\begin{cases} 0 = & (1-\phi)\pi N^* + \omega R^* + \gamma_2 P^* - \kappa_1 S^* - \lambda^* S^* \\ 0 = & \phi\pi N^* + \gamma_1 S^* + \nu L^* + \tau M^* + \psi A^* - \kappa_2 P^* \\ 0 = & \lambda^* \rho S^* - \kappa_3 L^* \\ 0 = & \lambda^*(1-\rho) S^* + \sigma L^* - \kappa_4 M^* \\ 0 = & \delta M^* - \kappa_5 A^* \\ 0 = & \xi M^* + \eta A^* - \kappa_6 R^* \end{cases} \tag{14}$$

where $\kappa_1 = \mu + \gamma_1,\ \kappa_2 = \mu + \gamma_2,\ \kappa_3 = \mu + \nu + \sigma,\ \kappa_4 = \mu + \tau + \delta + \xi,\ \kappa_5 = \mu + \alpha + \eta + \psi$, and $\kappa_6 = \mu + \omega$ are simplifying constants; and

$$\lambda^* = c\beta \left(\frac{L^* + \theta_1 M^* + \theta_2 A^*}{N^*} \right) \tag{15}$$

It can be observed that $L^* = Q_0 \lambda^* S^*,\ M^* = Q_1 \lambda^* S^*$, and $A^* = Q_2 \lambda^* S^*$ where $Q_0 = \frac{\rho}{\kappa_3},\ Q_1 = \frac{(1-\rho)\,\kappa_3 + \rho\sigma}{\kappa_3 \kappa_4}$, and $Q_2 = \frac{((1-\rho)\,\kappa_3 + \rho\sigma)\,\delta}{\kappa_3 \kappa_4 \kappa_5}$. Substituting for $L^*,\ M^*$, and A^* in the equation (15), yields

$$(N^* - \mathcal{R}_H S^*)\,\lambda^* = 0 \tag{16}$$

giving, either

$$\lambda^* = 0 \tag{17}$$

or

$$N^* - \mathcal{R}_H S^* = 0 \tag{18}$$

where

$$\mathcal{R}_H = c\beta \left(\frac{\rho\kappa_4\kappa_5 + (\theta_1\kappa_5 + \theta_2\delta)\,((1-\rho)\,\kappa_3 + \rho\sigma)}{\kappa_3\kappa_4\kappa_5} \right). \tag{19}$$

3.3.4 Existence of Risk Free Equilibrium

The risk free equilibrium point is obtained by eliminating all the risky classes from the system, setting $L = M = A = 0$ results into $\lambda^* = 0$. Consequently, equation (17) leads to the following risk free equilibrium

$$E_0 = (S_0^*, P_0^*, L_0^*, M_0^*, A_0^*, R_0^*) = \left(\frac{\pi\left(\gamma_2 + \mu\left(1-\phi\right)\right) N_0^*}{\mu\left(\mu+\gamma_1+\gamma_2\right)}, \frac{\pi\left(\phi\mu+\gamma_1\right) N_0^*}{\mu\left(\mu+\gamma_1+\gamma_2\right)}, 0, 0, 0, 0 \right). \tag{20}$$

3.3.5 Existence of Risk Endemic State

The solution $N^* - \mathcal{R}_H S^* = 0$ from equation (18) leads to endemic state. From system (14) noting that $R^* = Q_3 \lambda^* S^*$ with

$$Q_3 = \frac{\left(\xi\kappa_5 + \eta\delta\right)\left(\left(1-\rho\right)\kappa_3 + \rho\sigma\right)}{\kappa_3\kappa_4\kappa_5\kappa_6}.$$

The total population is given by

$$N^* = S^* + P^* + Q\lambda^* S^* \tag{21}$$

where $Q = \sum_{i=0}^{3} Q_i$. Substituting for N^*, the following result is obtained

$$P^* = \left(\left(\mathcal{R}_H - 1\right) - Q\lambda^*\right) S^*$$

Re-writing the first two equations of the model system (2) in terms of λ^* and S^* with appropriate substitution of other state variables. Eliminating S^* from the subsystem, equation (22) is established with an implication of forward bifurcation.

$$\lambda^* = \frac{\left(1-\mathcal{R}_H\right)\left(\left(1-\phi\right)\mu + \gamma_2\right) - \left(\phi\mu+\gamma_1\right)}{\left(\mu\left(1-\phi\right)+\gamma_2\right)Q + \left(1-\phi\right)\left(\nu Q_0 + \tau Q_1 + \psi Q_2\right) - \omega Q_3 \phi} \tag{22}$$

It follows that the establishment of non-trivial solution E_1 for the risk endemic as

$$E_1 = \left(S^*, \left(\left(\mathcal{R}_H - 1\right) - Q\lambda^*\right) S^*, Q_0\lambda^* S^*, Q_1\lambda^* S^*, Q_2\lambda^* S^*, Q_3\lambda^* S^*\right) \tag{23}$$

3.3.6 Basic Risk Reproduction Number

In this section, the basic risk reproduction number of the model is computed using Next Generation Matrix (NGM) method as appreciated by different studies (van den Driessche *et al.*, 2002;

Diekmann *et al.*, 2010). The basic risk reproduction number, denoted by $\mathcal{R}_0$, may be defined as an average number of secondary risk individuals produced by a single risk individual in an entirely susceptible population for the entire risky life. It serves as an indicator used to predict the possibility of the occurrence of risk epidemic. Based on Diekmann *et al.* (2010), the basic risk reproduction number is given as the dominant eigenvalue or spectral radius of the next generation matrix. Considering three risky states, $L(t), M(t)$ and $A(t)$; and three non-risky states, $S(t), P(t)$ and $R(t)$ of the system of equations (2). The system can be composited into risk subsystem stated as

$$g(X,Z) = \begin{cases} \dot{L} = & \lambda\rho S - \kappa_3 L \\ \dot{M} = & \lambda(1-\rho)S + \sigma L - \kappa_4 M \\ \dot{A} = & \delta M - \kappa_5 A \end{cases} \tag{24}$$

and non-risky subsystem as

$$f(X,Z) = \begin{cases} \dot{S} &= -(\kappa_1 + \lambda)\, S + \gamma_2 P + \omega R + (1-\phi)\pi N \\ \dot{P} &= \gamma_1 S - \kappa_2 P + \nu L + \tau M + \psi A + \phi\pi N \\ \dot{R} &= \xi M + \eta A - \kappa_6 R \end{cases} \tag{25}$$

Following van den Driessche *et al.* (2002), new risk cases are distinguished from all other changes in population. Let $\mathcal{F}_i$ be the rate of appearance of new risk cases in compartment i, $\mathcal{V}_i^+$ be the rate of transfer of individuals into compartment i by all other means, and $\mathcal{V}_i^-$ be the rate of transfer of individuals out of compartment i. Assuming that each function is continuously differentiable at least twice in each variable. The risk transmission model consists of non-negative initial conditions together with the following system of equations:

$$\dot{Z} = \mathcal{F}(Z) - \mathcal{V}(Z) = \begin{bmatrix} \lambda\rho S \\ \lambda(1-\rho)S \\ 0 \end{bmatrix} - \begin{bmatrix} \kappa_3 L \\ -\sigma L + \kappa_4 M \\ -\delta M + \kappa_5 A \end{bmatrix} \tag{26}$$

where $\mathcal{V}(Z) = \mathcal{V}^-(Z) - \mathcal{V}^+(Z)$, $X = \{S, P, R\}^T \in \mathbb{R}^3$, $Z = \{L, M, A\}^T \in \mathbb{R}^3$ and $(\cdot)^T$ denotes transpose.

The matrix F corresponds to *"transmissions"* and the matrix V to *"transitions"* of risks factors through different states with different risk levels. Following van den Driessche *et al.* (2002) and Diekmann *et al.* (2010), where indices i and j are used to refer the risk states, with $i, j \in \{1, 2, 3\}$, the entry F_{ij} is the rate at which individuals in a risk state j give rise to individuals in

risk state i, in the linearlized system. Thus, $F_{ij} = 0$ occurs only when there are no new cases in risk state i can be produced by an individual in risk state j. All epidemiological events that lead to new risks are incorporated in the model via matrix F, and all other events via matrix V.

The linearlization of risk system (24) at the risk free state E_0 yields the following F and V matrices,

$$F = \frac{S_0^*}{N_0^*}\begin{bmatrix} \beta c\rho & \beta c\theta_1\rho & \beta c\theta_2\rho \\ \beta c(1-\rho) & \beta c\theta_1(1-\rho) & \beta c\theta_2(1-\rho) \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \kappa_3 & 0 & 0 \\ -\sigma & \kappa_4 & 0 \\ 0 & -\delta & \kappa_5 \end{bmatrix} \tag{27}$$

where $S_0^* = \dfrac{\pi\left(\gamma_2 + \mu\left(1-\phi\right)\right)N_0^*}{\mu\left(\mu+\gamma_1+\gamma_2\right)}$. Thus by direct computation, it gives

$$V^{-1} = \begin{bmatrix} \dfrac{1}{\kappa_3} & 0 & 0 \\ \dfrac{\sigma}{\kappa_3\kappa_4} & \dfrac{1}{\kappa_4} & 0 \\ \dfrac{\sigma\delta}{\kappa_3\kappa_4\kappa_5} & \dfrac{\delta}{\kappa_4\kappa_5} & \dfrac{1}{\kappa_5} \end{bmatrix} \tag{28}$$

In epidemiological sense, the interpretation of the entry V_{ij}^{-1} describes the expected time that an individual with risk state j will spend in a risk state i for the rest of his/her life. For instance, in a matrix V^{-1} above, individuals who are presently in state L will spend, on average, an amount of time $\dfrac{1}{\kappa_3}$ in that state. Similarly, the same individuals will spend on average an amount of time $\dfrac{\sigma}{\kappa_3\kappa_4}$ in state M. While $\dfrac{\sigma}{\kappa_3}$ is the probability that an individual will successfully progress from L to M −instead of leaving state L by either dying or changing behavior under the influence of religion, an individual in state M, spends on average amount of time $\dfrac{1}{\kappa_4}$ in the state. The individuals in state M will spend no time at all in state L leading to zeros for appropriate elements (Diekmann *et al.*, 2010). Now, the following Next Generation Matrix is formulated

$$FV^{-1} = \frac{S_0^*}{N_0^*}\begin{bmatrix} \dfrac{c\beta\rho}{\kappa_3}\left(1+\dfrac{\theta_1\sigma}{\kappa_4}+\dfrac{\theta_2\delta\,\sigma}{\kappa_4\kappa_5}\right) & \dfrac{c\beta\rho}{\kappa_4}\left(\theta_1+\dfrac{\theta_2\delta}{\kappa_5}\right) & \dfrac{c\beta\theta_2\rho}{\kappa_5} \\ \dfrac{c\beta\left(1-\rho\right)}{\kappa_3}\left(1+\dfrac{\theta_1\sigma}{\kappa_4}+\dfrac{\theta_2\delta\,\sigma}{\kappa_4\kappa_5}\right) & \dfrac{c\beta\left(1-\rho\right)}{\kappa_4}\left(\theta_1+\dfrac{\theta_2\delta}{\kappa_5}\right) & \dfrac{c\beta\theta_2\left(1-\rho\right)}{\kappa_5} \\ 0 & 0 & 0 \end{bmatrix} \tag{29}$$

To interpret the entries of the Next Generation Matrix FV^{-1} and develop a meaningful definition of $\mathcal{R}_0$ we note that entry FV_{ij}^{-1} is the expected number of secondary risk individuals

in compartment i produced by individuals initially in compartment j assuming that the drinking environment shared by individuals remains homogeneous (Padmanabhan *et al.*, 2017). The basic risk reproduction number, $\mathcal{R}_0$, is given by the dominant eigenvalue of matrix FV^{-1}. Therefore,

$$\mathcal{R}_0 = c\beta \left(\frac{\rho\kappa_4\kappa_5 + (\theta_1\kappa_5 + \theta_2\delta)\left((1-\rho)\kappa_3 + \rho\sigma\right)}{\kappa_3\kappa_4\kappa_5} \right) \left(\frac{S_0^*}{N_0^*} \right) \tag{30}$$

It is important to note that

$$\mathcal{R}_0 = \left(\frac{S_0^*}{N_0^*} \right) \mathcal{R}_H < \mathcal{R}_H \tag{31}$$

3.3.7 Local Stability of the Risk Free Equilibrium

For the system of differential equations presented in (2) the following theorem can be established (Bhunu *et al.*, 2012; Chavez *et al.*, 2002) .

Theorem 3.4

The risk-free equilibrium E_0 is locally asymptotically stable (L.A.S) for $\mathcal{R}_0 < 1$ and unstable otherwise.

Proof. Consider the Jacobian matrix evaluated at the risk free equilibrium point bellow Observe that

$$\begin{aligned}
\left[\frac{\partial(\lambda S)}{\partial L}\right]_{\mathcal{E}_0} &= \left[\frac{\partial}{\partial L}\left(c\beta\left(\frac{L+\theta_1 M+\theta_2 A}{N}\right)S\right)\right]_{\mathcal{E}_0} = c\beta\frac{S_0}{N_0} \\
\left[\frac{\partial(\lambda S)}{\partial M}\right]_{\mathcal{E}_0} &= \left[\frac{\partial}{\partial M}\left(c\beta\left(\frac{L+\theta_1 M+\theta_2 A}{N}\right)S\right)\right]_{\mathcal{E}_0} = c\beta\theta_1\frac{S_0}{N_0} \\
\left[\frac{\partial(\lambda S)}{\partial A}\right]_{\mathcal{E}_0} &= \left[\frac{\partial}{\partial A}\left(c\beta\left(\frac{L+\theta_1 M+\theta_2 A}{N}\right)S\right)\right]_{\mathcal{E}_0} = c\beta\theta_2\frac{S_0}{N_0}
\end{aligned}$$

.

Now, defining

$$\epsilon = \frac{S_0}{N_0} = \frac{\gamma_2 + (1-\phi)\mu}{\mu + \gamma_1 + \gamma_2},$$

the Jacobian matrix evaluated at the risk free equilibrium point, $\mathcal{E}_0$, which decomposed into block matrices for simplifications as

$$J_0 = \begin{bmatrix} B_{11} & B_{12} & B_{13} \\ \mathbf{0} & B_{22} & \mathbf{0} \\ \mathbf{0} & B_{32} & B_{33} \end{bmatrix} \tag{32}$$

where

$$B_{11} = \begin{bmatrix} -\kappa_1 + (1-\phi)\,\pi & \gamma_2 + (1-\phi)\,\pi \\ \phi\,\pi + \gamma_1 & \phi\,\pi - \kappa_2 \end{bmatrix}, \tag{33}$$

$$B_{12} = \begin{bmatrix} -c\beta\,\epsilon + (1-\phi)\,\pi & -\epsilon\,c\beta\,\theta_1 + (1-\phi)\,\pi & -\epsilon\,c\beta\,\theta_2 + (1-\phi)\,\pi \\ \phi\,\pi + \nu & \phi\,\pi + \tau & \phi\,\pi + \psi \end{bmatrix}, \tag{34}$$

$$B_{13} = \begin{bmatrix} \omega + (1-\phi)\,\pi \\ \phi\,\pi \end{bmatrix}, \tag{35}$$

$$B_{22} = \begin{bmatrix} c\beta\,\rho\,\epsilon - \kappa_3 & c\beta\,\theta_1\,\rho\,\epsilon & c\beta\,\theta_2\,\rho\,\epsilon \\ c\beta\ (1-\rho)\,\epsilon + \sigma & c\beta\,\theta_1\ (1-\rho)\,\epsilon - \kappa_4 & c\beta\,\theta_2\ (1-\rho)\,\epsilon \\ 0 & \delta & -\kappa_5 \end{bmatrix}, \tag{36}$$

$$B_{32} = \begin{bmatrix} 0 & \xi & \eta \end{bmatrix}, \tag{37}$$

$$B_{33} = \begin{bmatrix} -\kappa_6 \end{bmatrix}, \tag{38}$$

and $\mathbf{0}$ are zero matrices.

To study the stability of the risk-free, it suffices to investigate the signs of real parts of the eigenvalues of of B_{11}, B_{22} and B_{33}. Clearly, the eigenvalue of B_{33} has negative real parts. The trace of B_{11}, is

$$Tr(B_{11}) = -(2\mu + \gamma_1 + \gamma_2) + \pi$$

and the determinant of B_{11}, is

$$Det(B_{11}) = (\mu - \pi)\,(\mu + \gamma_1 + \gamma_2) > 0.$$

It is clear that $\mu > \pi$ satisfies the inequalities $Tr(B_{11}) < 0$ and $Det(B_{11}) > 0$. Now, it can be see clearly that the stability of the model $\mathcal{E}_0$ is solely determined by the signs of the real parts of the eigenvalues of B_{22}. The characteristic equation arising from B_{22} is a cubic equation

$$b_0\lambda^3 + b_1\lambda^2 + b_2\lambda + b_3 = 0,$$

$$
\begin{aligned}
b_3 &= \kappa_3\kappa_4\kappa_5\left(1-\mathcal{R}_0\right)+\left(\left(c\beta\epsilon+1\right)\rho-\kappa_3\right)\kappa_4\kappa_5 \\
b_2 &= -c\beta\epsilon\left(\delta\theta_2\left(1-\rho\right)-\rho\left(2\mu+2\nu+\sigma\right)\theta_1+\rho\left(\kappa_4+\kappa_5\right)\right) \\
&\quad +\kappa_3\,\kappa_4+\kappa_3\,\kappa_5+\kappa_4\,\kappa_5 \\
b_1 &= -c\beta\epsilon\left(\rho+\theta_1\left(1-\rho\right)\right)+\kappa_3+\kappa_4+\kappa_5 \\
b_0 &= 1.
\end{aligned}
\tag{39}
$$

Using the Routh-Hurwitz stability criteria

$$
b_0>0,\ b_1>0,\ \text{and}\ b_2b_1>b_0b_3,
$$

it is clear that $b_1 = 1 > 0$, $b_1 > 0$ whenever $\left(\kappa_3+\kappa_4+\kappa_5\right) > c\beta\epsilon\left(\rho+\theta_1\left(1-\rho\right)\right)$ and $b_1b_2 > b_3$ for appropriate values of parameters. □

3.3.8 Global Stability Conditions for the Risk Free Equilibrium

The roles of $\mathcal{R}_0$ in global stability analysis can be traced back from Chavez *et al.* (2002) and Castillo-Chavez *et al.* (2002) whereby the satisfaction of two well established axioms ((H1) and (H2)), guarantee the global asymptotic stability of the risk free state. Following Chavez *et al.* (2002), the system (2) can be we presented in the form:

$$
\begin{aligned}
\frac{dX}{dt} &= f(X,Z) \\
\frac{dZ}{dt} &= g(X,Z),\ g(X,0)=0
\end{aligned}
\tag{40}
$$

where $X=(S,P,R)^T\in\mathbb{R}^3$ whose components denote the number of individuals at risk free (non alcoholic individuals) and $Z=(L,M,A)^T\in\mathbb{R}^3$ whose components denote the number of alcoholic individuals at different risk levels. The coordinate $(X_0^*,0)\in\mathbb{R}^6$ denotes the risk free equilibrium for the model and $(\cdot)^T$ denotes a vector transpose. Now with the system (2) in terms of X and Z, the following axioms combined guarantee globally asymptotically stability.

(H1) For $\dfrac{dX}{dt}=f(X,0)$, X_0^* is globally asymptotically stable (G.A.S).

(H2) $g(X,Z)=BZ-\tilde{g}(X,Z);\ \ \tilde{g}(X,Z)\geq 0,\ \ \forall X,Z\in\Omega$

$B=D_Zg(X_0^*,0)$ is defined as a Metzler matrix and Ω is the region where model (2) makes biological sense. The following theorem hold true provided that the system (2) satisfies the above two conditions (H1) and (H2).

Theorem 3.5

The risk free equilibrium E_0 of the model (2) is globally asymptotically stable (G.A.S) provided that axioms (H1) and (H2) are satisfied.

Proof. Re-writing the model equation (2) into $f(X,Z)$ and $g(X,Z)$ such that $X = (S,P,R)\,,\ Z = (L,M,A)$. The following subsystems can be formulated

$$f(X,Z) = \begin{cases} \dot{S} &= -(\kappa_1+\lambda)S + \gamma_2 P + \omega R + (1-\phi)\pi N \\ \dot{P} &= \gamma_1 S - \kappa_2 P + \nu L + \tau M + \psi A + \phi\pi N \\ \dot{R} &= \xi M + \eta A - \kappa_6 R \end{cases} \tag{41}$$

$$g(X,Z) = \begin{cases} \dot{L} = & \lambda\rho S - \kappa_3 L \\ \dot{M} = & \lambda(1-\rho)S + \sigma L - \kappa_4 M \\ \dot{A} = & \delta M - \kappa_5 A \end{cases} \tag{42}$$

Evaluation of the subsystem (41) at $(X_0^*, 0)$ gives

$$f(X_0^*,0) = \begin{bmatrix} -\kappa_1 S_0^* + \gamma_2 P_0^* + (1-\phi)\pi N_0^* \\ \gamma_1 S_0^* - \kappa_2 P_0^* + \phi\pi N_0^* \\ 0 \end{bmatrix}$$

which satisfies axiom (H1). Now, re-writing susbsystem (42) such that

$$g(X,Z) = BZ - \tilde{g}(X,Z),$$

defining B and $\tilde{g}(X,Z)$ as

$$B = \begin{bmatrix} -\kappa_3 + \dfrac{c\beta\rho}{N} & \dfrac{c\beta\theta_1\rho}{N} & \dfrac{c\beta\theta_2\rho}{N} \\ \sigma + \dfrac{c\beta(1-\rho)}{N} & -\kappa_4 + \dfrac{c\beta\theta_1(1-\rho)}{N} & \dfrac{c\beta\theta_2(1-\rho)}{N} \\ 0 & \delta & -\kappa_5 \end{bmatrix} \text{ and } \tilde{g}(X,Z) = \begin{bmatrix} \lambda\rho(S-1) \\ \lambda(1-\rho)(S-1) \\ 0 \end{bmatrix}.$$

Since B is a Metzler matrix and $\tilde{g}(X,Z) \geq 0$ provided that $S(t) \geq 1$ and $\rho \leq 1$, then axioms (H1) and (H2) are satisfied. It is clear that the equilibrium point $E_0 = \left(\dfrac{\pi(\gamma_2+\mu(1-\phi))}{\mu(\mu+\gamma_1+\gamma_2)}, \dfrac{\pi(\phi\mu+\gamma_1)}{\mu(\mu+\gamma_1+\gamma_2)}, 0, 0, 0, 0\right)$ is a globally asymptotically stable equilibrium. □

3.4 Fuzzy model for the dynamics of alcohol related health risks

3.4.1 Fuzzy Model Descriptions

The fuzzy model for health risks challenges associated with alcoholism is proposed by assuming uncertainty condition in the measure of influence of the risky individual β, and the additional death rate α. The two fuzzy numbers are defined function of the degree of social/peer influence x held by individuals in the community. The effects of variation of the two fuzzy numbers $\alpha(x)$ and $\beta(x)$ in the fuzzy model is then studied through numerical simulations.

An application of fuzzy set theory in solving dynamical systems have recently been an interesting research area (Massad *et al.*, 2009; Mishra *et al.*, 2010; Verma *et al.*, 2018; Nandi *et al.*, 2018) where the approach is used in solving different problems in the community. Using fuzzy set theory approach, the basic *SPLMAR* model developed in (2) is modified into fuzzy model. As the case was in the basic model, the fuzzy model has considered two main aspects of model formulation. Firstly, the social cultural beliefs considered as an integral part of the society (Kendler *et al.*, 1997; Rosmarin *et al.*, 1998; Haber *et al.*, 2012). Secondly, is the staged process in which alcoholism behaviors take in the spread of health risks (Bhunu, 2012; Huo *et al.*, 2016; Rehm *et al.*, 2004; Mushayabasa, 2015; Xiang *et al.*, 2016; Musanyu *et al.*, 2018).

Owing to the model (2) developed by considering multi-risk levels established referring to Tanzanian population with active religious beliefs. The fuzzy model is proposed with the variable x describing the degree of peer influence of a susceptible individual to initiate drinking behavior. The model development is made under the assumption that both β and α are the functions of x. The physical meaning of parameters used in the model influenced the choice of the two parameters. While β is attributed to the spread of health risks associated with alcoholism in the community, α translates the consequences of health risks by means of additional death rate.

The fuzzy numbers $\beta = \beta(x)$, and $\alpha = \alpha(x)$ represent the likelihood that a susceptible individual will drink alcohol after prolonged contact with drinking individuals, and the additional death rate induced by alcoholism respectively.

Definition 3.1

A fuzzy number may be defined as a generalization of a real number referring to a connected set of possible values, rather than referring to one single value, where each possible value is weighted between 0 and 1.

3.4.2 Fuzzy Model Equations

Enlightened by the above descriptions, the fuzzy system (43) of differential equations is established

$$\begin{cases} \dot{S} &= (1-\phi)\pi N - (\mu+\gamma_1+\lambda(x))\,S + \gamma_2 P + \omega R \\ \dot{P} &= \phi\pi N + \gamma_1 S - (\mu+\gamma_2)P + \nu L + \tau M + \psi A \\ \dot{L} &= \lambda(x)\rho S - (\nu+\mu+\sigma)\,L \\ \dot{M} &= \lambda(x)(1-\rho)S + \sigma L - (\tau+\mu+\delta+\xi)\,M \\ \dot{A} &= \delta M - (\mu+\alpha(x)+\eta+\psi)A \\ \dot{R} &= \xi M + \eta A - (\omega+\mu)R \end{cases} \tag{43}$$

with non-negative initial conditions of the state variables: $S(0) > 0, P(0) \geq 0, L(0) \geq 0, M(0) \geq 0, A(0) \geq 0$ and $R(0) \geq 0$ where

$$\frac{dN}{dt} = (\pi - \mu)\,N - \alpha(x)A \tag{44}$$

and the new force of peer influence is redefined into

$$\lambda(x) = c\beta(x)\left(\frac{L + \theta_1 M + \theta_2 A}{N}\right). \tag{45}$$

3.4.3 Analysis of the Fuzzy System

Following Verma *et al.* (2017) and Nandi *et al.* (2018), the fuzzy *SPLMAR* model (43) is studied and model analysis and interpretations are presented. The two parameters $\beta(x)$ and $\alpha(x)$ can be described through two different fuzzy membership functions.

For example, considering a situation where a drinking individual interacts with a susceptible member, the minimum amount of the degree of peer influence, $x = x_{min}$ is required to have an impact on a susceptible member. That is to say, when $x < x_{min}$, the impact of behavioral influence is considered negligible. The quantity x_{min} is therefore considered as a parameter whose exact value depends upon both attitude $-$public opinions towards the drinking behavior or the drinking individual, and willingness of a susceptible individual to conform with the peer pressures. As x increases, the behavior inducement rate increases to the maximum which is equal to unity at $x \geq x_0$. Further, it is also assumed that the degree of peer influence is bounded above where $x = x_{max}$ marks an upper bound. Therefore, the values of x with an effect to the system lies in the interval of $x_{min} \leq x \leq x_{max}$. The fuzzy membership function for the fuzzy

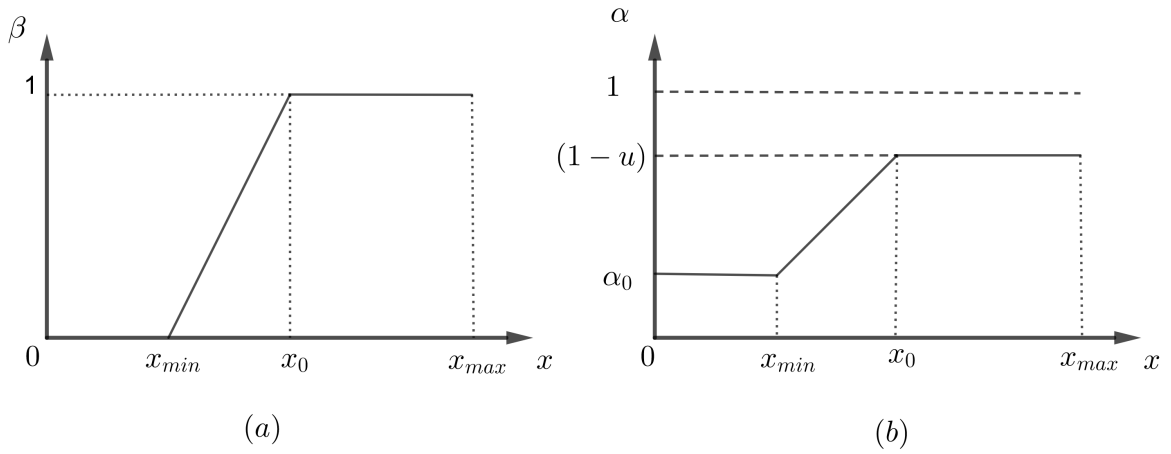
number $\beta(x)$ is given by

$$\beta(x) = \begin{cases} 0 & \text{if } x < x_{min} \\ \dfrac{x - x_{min}}{x_0 - x_{min}} & \text{if } x_{min} \leq x \leq x_0 \\ 1 & \text{if } x_0 < x < x_{max} \end{cases} \tag{46}$$

Similarly, we also assume that the addition death rate, $\alpha(x)$, is a fuzzy number as it occurs due to the risks "transmission". The set of values $x < x_{min}$ imply negligible amount of risk transmissions setting minimum values of additional death rate, $\alpha(x)$, say $\alpha(x < x_{min}) = \alpha_0$. As x increases, the additional death rate increases and gets the highest value at $x = x_0$. Since the additional death rate may not reach $\alpha(x) = 1$ as its highest score due to several limitations. Let the maximum value $\alpha(x) = (1 - u)$ for some real number u such that $0 < u < (1 - \alpha_0)$. The fuzzy membership function of $\alpha(x)$ may be established as follows

$$\alpha(x) = \begin{cases} \alpha_0, & \text{if } 0 \leq x < x_{min} \\ \alpha_0 + \left(\dfrac{1 - u - \alpha_0}{x_0 - x_{min}}\right) x, & \text{if } x_{min} \leq x < x_0 \\ (1 - u), & \text{if } x_0 \leq x \leq x_{max} \end{cases} \tag{47}$$

The graphs of membership functions $\beta(x)$ and $\alpha(x)$ are presented in Fig. 2 (a) and (b) respectively. Let Π be the linguistic variable with varying classification whose degree of peer influence



(a) (b)

Figure 2: The graph of membership functions β and α

differs from among drinking individuals depending on their social status in the community. Using x_c and d as, respectively, the central value and dispersion of each one of the fuzzy sets assumed by Π. Each classification can be modeled by using a triangular fuzzy number whose

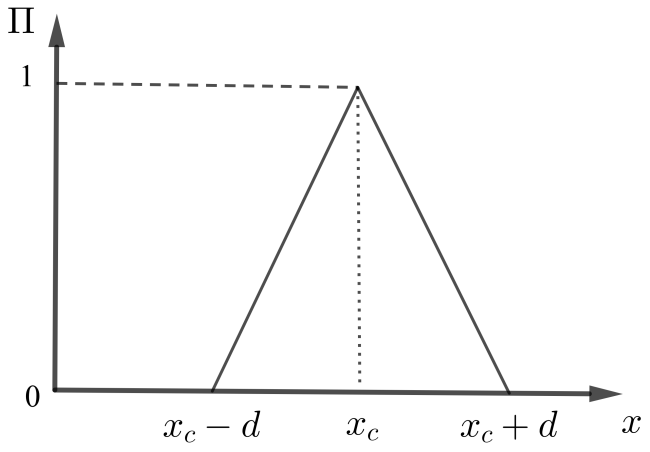


Figure 3: The graph of membership function Π

membership function is given in (48) and the graph of membership function Π is presented in Fig. 3.

$$\Pi(x) = \begin{cases} 0, & \text{if } x < x_c - d \\ \dfrac{x - x_c + d}{d}, & \text{if } x_c - d \leq x \leq x_c \\ -\dfrac{x - x_c - d}{d}, & \text{if } x_c < x \leq x_c + d \\ 0, & \text{if } x_c + d < x \end{cases} \tag{48}$$

Let $L(x,t),\ M(x,t)$, and $A(x,t)$ be the family solutions of the fuzzy model system (43). These are the numbers of risky population proportions created as the result of social interactions between the susceptible members and risky individuals with social influence x at time t. Now, $L(x,t),\ M(x,t)$, and $A(x,t)$ are fuzzy numbers which lie in the interval $[0,1]$.

3.5 Alcohol related health risks model with intervention

Using a non-autonomous approach, the model *SPLMAR* developed in (2) can be extended to accommodate control functions and analyze the mathematical model for optimal control strategies. The following control functions are introduced to cater for the purpose: $u_1(t)$, and $u_2(t)$ to extend the basic *SPLMAR* health risk model (2). Time dependent variable $u_1(t)$ is introduced as a control function representing the public health education campaigns. It aims at increasing public awareness on health risks associated with alcoholism to reduce the number of risky susceptible population. On another hand, $u_2(t)$ represents an organized treatment and rehabilitation programs aiming at speeding up the rate of recovery from the medium risk sub-population. The rehabilitation programs and sober houses are meant to rescue affected population from drinking

addictiveness. When the programs are run on client-voluntary bases, the medium risk population are more likely to respond positively than the rest of risk sub-populations (Bhunu, 2012). While low risk drinkers will not feel as targeted population by considering themselves healthy, the main opposition should be expected from the higher risk sub-population. Thus, $u(t)$ is likely to be more efficient and probably more effective when applied to the medium risk population. The flow diagram for the dynamics of alcohol related health risks subject to the control variables u_1 and u_2, is presented in Fig. 4.

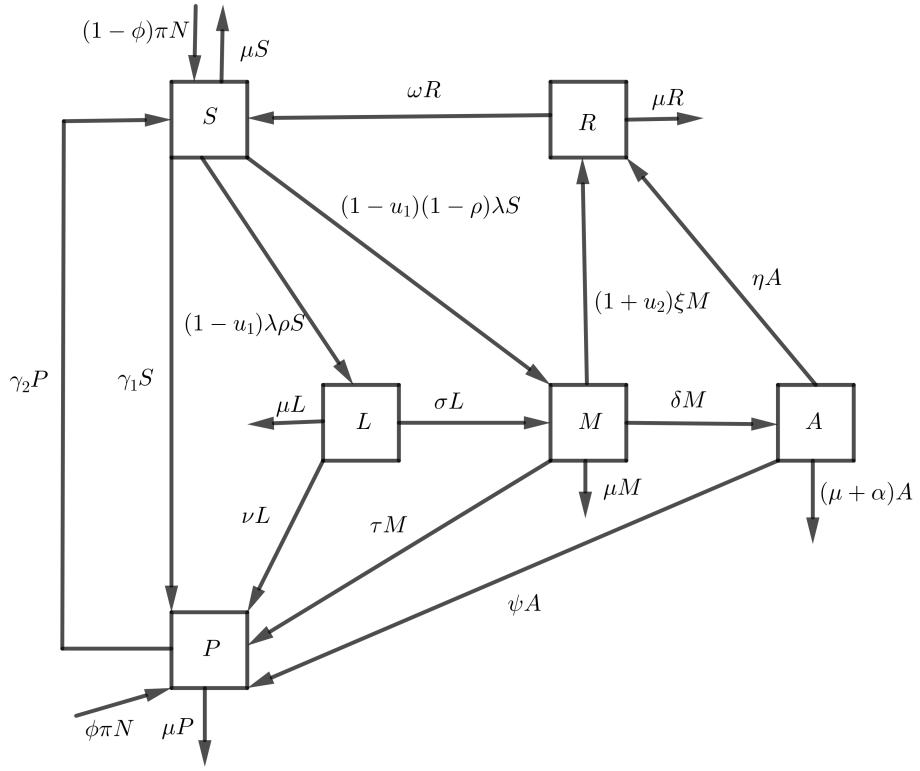


Figure 4: The model compartment for the population dynamics of alcohol related health risks with controls

Based on the descriptions given above, the controlled model for the population dynamics of alcohol related health risks (49) is formulated.

$$\begin{cases} \dot{S} &= -\left(\mu+\gamma_1+(1-u_1)\lambda\right)S+\gamma_2 P+\omega R+(1-\phi)\pi N \\ \dot{P} &= \gamma_1 S-(\mu+\gamma_2)P+(\nu L+\tau M+\psi A)+\phi\pi N \\ \dot{L} &= (1-u_1)\lambda\rho S-(\nu+\mu+\sigma)\,L \\ \dot{M} &= (1-u_1)(1-\rho)\lambda S+\sigma L-(\tau+\mu+\delta+(1+u_2)\xi)\,M \\ \dot{A} &= \delta M-(\mu+\alpha+\eta+\psi)A \\ \dot{R} &= (1+u_2)\xi M+\eta A-(\omega+\mu)R \end{cases} \tag{49}$$

where $N > 0, S > 0, P \geq 0, L \geq 0, M \geq 0, A \geq 0$ and $R \geq 0$.

3.5.1 Normalization of the model with control

Following Mushayabasa (2015), the controlled model (49) is normalized for easy computations. Let s, p, l, m, a, and r be the dimensionless variables such that $s = \frac{S}{N},\ p = \frac{P}{N},\ l = \frac{L}{N},\ m = \frac{M}{N},\ a = \frac{A}{N}$, and $r = \frac{R}{N}$. Substituting these values into system (49), the following re-scaled system is

$$\begin{cases} \dot{s} &= -\left(\mu + \gamma_1 + (1-u_1)\,\Lambda\right) s + \gamma_2 p + \omega r + (1-\phi)\pi \\ \dot{p} &= \gamma_1 s - (\mu + \gamma_2) p + (\nu l + \tau m + \psi a) + \phi\pi \\ \dot{l} &= (1-u_1)\,\Lambda\rho s - (\nu + \mu + \sigma) l \\ \dot{m} &= (1-u_1)\,(1-\rho)\Lambda s + \sigma l - (\tau + \mu + \delta + (1+u_2)\,\xi) m \\ \dot{a} &= \delta m - (\mu + \alpha + \eta + \psi) a \\ \dot{r} &= (1+u_2)\,\xi m + \eta a - (\omega + \mu) r \end{cases} \tag{50}$$

where $\Lambda = \beta c\,(l + \theta_1 m + \theta_2 a)$ and $s + p + l + m + a + r = n$ such that $0 \leq n \leq 1$. It is observed that the total population in system (50) satisfies the equation

$$\frac{dn}{dt} \leq \pi - \mu n. \tag{51}$$

Thus, $n(t) \leq n_0 e^{-\mu t}$ where n_0 defines the constant initial population proportion at $t = 0$. Therefore, the feasible region of system (50) is given by the closed set:

$$\Gamma = \{(s, p, l, m, a, r) \in n | 0 \leq n \leq 1\} \tag{52}$$

3.5.2 Formulation of the Optimal Control Problem

Consider the time-varying control function set $U(t)$ whose components represent the deliberate efforts geared to reduce the level of health risks associated with alcoholism behavior in the community targeting different population compartments. Suppose that the components $u_i \in U(t)$ are Lebesque measurable, we define

$$U(t) = \{u_i(t)\, \forall i \in \{1, 2\} : u_i \in [0, 1]\,; 0 \leq t \leq T \tag{53}$$

To investigate the optimal level of efforts that would be required to control the spread of health risks in the community. Following Lee *et al.* (2010), Mushayabasa (2015), Hugo *et al.* (2017),

Berhe *et al.* (2018) and Nyerere *et al.* (2020), the objective function, $\mathcal{J}$ can be formulated as follows,

$$\mathcal{J} = \min_{u_i \in U} \int_0^T \left(B_1 l + B_2 m + B_3 a + \frac{1}{2}\left(C_1 u_1^2 + C_2 u_2^2\right) \right) dt \tag{54}$$

The values B_j, and C_i are respectively positive balancing constants of the risky population and cost factors associated with control strategies u_i for $i \in \{1, 2\}$ and $j \in \{1, 2, 3\}$. Assuming non-linearity in the cost of each control strategy, using quadratic form that is: $\frac{C_1 u_1^2}{2}$ is the cost of control strategy associated with public health education campaign; and $\frac{C_2 u_2^2}{2}$ is the cost associated with running rehabilitation and sober houses. The goal is to minimize both the total number of the population at risk and the cost of controls, $u_1(t)$, and $u_2(t)$, by choosing appropriate positive balancing constants B_j's and C_i's aiming at minimizing the risky population at the minimum cost of the control.

3.5.3 Characterization of the Optimal Control

By using Pontryagin's Maximum Principle, the necessary conditions for optimal control and corresponding states are derived (Pontryagin, 1964; Lee *et al.*, 2010; Joshi *et al.*, 2015; Hugo *et al.*, 2017; Nyerere *et al.*, 2020). The Hamiltonian function in (55) is formulated.

$$\begin{aligned}
\mathcal{H} \;=\; & B_1 l + B_2 m + B_3 a + \frac{1}{2}\left(C_1 u_1^2 + C_2 u_2^2\right) \\
& + \lambda_1 \left(-\left(\mu + \gamma_1 + (1-u_1)\,\Lambda\right) s + \gamma_2 p + \omega r + (1-\phi)\pi \right) \\
& + \lambda_2 \left(\gamma_1 s - (\mu + \gamma_2) p + (\nu l + \tau m + \psi a) + \phi\pi \right) \\
& + \lambda_3 \left((1-u_1)\,\Lambda\rho s - (\nu + \mu + \sigma) l \right) \\
& + \lambda_4 \left((1-u_1)\,(1-\rho)\Lambda s + \sigma l - (\tau + \mu + \delta + (1+u_2)\,\xi) m \right) \\
& + \lambda_5 \left(\delta m - (\mu + \alpha + \eta + \psi) a \right) \\
& + \lambda_6 \left((1+u_2)\,\xi m + \eta a - (\omega + \mu) r \right)
\end{aligned} \tag{55}$$

Given the optimal control $U^* = (u_1^*, u_2^*)$, there exists adjoint functions, $\lambda_i \forall i \in \{1, 2, \cdots, 6\}$ corresponding to the states x_i such that $\lambda_i' = -\frac{\partial \mathcal{H}}{\partial x_i}, \forall x_i \in \{s, p, l, m, a, r\}$. It follows that, the adjoint system in (56) is established

$$
\begin{aligned}
\frac{d\lambda_1}{dt} &= -\left(1-u_1\right)\left(\left(1-\rho\right)\lambda_4+\rho\,\lambda_3-\lambda_1\right)\Lambda-\gamma_1\left(\lambda_2-\lambda_1\right)+\mu\lambda_1 \\
\frac{d\lambda_2}{dt} &= \gamma_2\left(\lambda_2-\lambda_1\right)+\mu\lambda_2 \\
\frac{d\lambda_3}{dt} &= -B_1+c\beta\left(1-u_1\right)\left(\lambda_1-\rho\,\lambda_3-\left(1-\rho\right)\lambda_4\right)s+\nu\left(\lambda_3-\lambda_2\right)+\mu\lambda_3+\sigma\left(\lambda_3-\lambda_4\right) \\
\frac{d\lambda_4}{dt} &= -B_2+c\beta\,s\left(1-u_1\right)\theta_1\left(\left(\lambda_1-\rho\,\lambda_3\right)-\left(1-\rho\right)\lambda_4\right)+\xi\,\left(1+u_2\right)\left(\lambda_4-\lambda_6\right) \\
&\quad +\delta\left(\lambda_4-\lambda_5\right)+\mu\lambda_4+\tau\left(\lambda_4-\lambda_2\right) \\
\frac{d\lambda_5}{dt} &= -B_3+c\beta\left(1-u_1\right)\left(\lambda_1-\rho\,\lambda_3-\lambda_4\,\left(1-\rho\right)\right)\theta_2 s+\left(\mu+\alpha\right)\lambda_5+\eta\left(\lambda_5-\lambda_6\right) \\
&\quad +\psi\left(\lambda_5-\lambda_2\right) \\
\frac{d\lambda_6}{dt} &= \mu\lambda_6+\omega\left(\lambda_6-\lambda_1\right)
\end{aligned}
\tag{56}
$$

with transversality conditions

$$
\lambda_i(T)=0;\ \forall i\in\{1,2,\cdots,6\} \tag{57}
$$

Now, the Hamiltonian function, $\mathcal{H}$ is minimized with respect to the control function, U in order to obtain an optimal control U^* such that

$$
J(u_1^*,u_2^*)=\min_{\Omega}J(u_1,u_2) \tag{58}
$$

where $\Omega=\{(u_1(t),u_2(t))\in U|a\leq u_1(t),u_2(t)\leq b,t\in[0,T]\}$

3.5.4 Existence of optimal controls

Theorem 3.6

Given $\mathcal{J}(U)$ subject to system (50) with non-negative initial conditions of the state variables, that is $s(0)\geq 0,\ p(0)\geq 0,\ l(0)\geq 0,\ m(0)\geq 0,\ a(0)\geq 0$, and $r(0)\geq 0$, there exists an optimal control $U^*=(u_1^*,u_2^*)$ that minimizes $\mathcal{J}(U)$ over U, and the corresponding adjoint variables $\lambda_i\forall i\in\{1,2,\cdots,6\}$ satisfying the following equations

$$
\begin{cases}
\dfrac{\partial\mathcal{H}}{\partial u_1} &= C_1u_1-s\left(\left(1-\rho\right)\lambda_4+\rho\,\lambda_3-\lambda_1\right)\Lambda \\
\dfrac{\partial\mathcal{H}}{\partial u_2} &= C_2u_2-\xi\left(\lambda_4-\lambda_6\right)m
\end{cases}
\tag{59}
$$

The control set $U^* = (u_1^*, u_2^*)$ gives $\dfrac{\partial \mathcal{H}}{\partial U} = 0$, in particular, $\dfrac{\partial \mathcal{H}}{\partial u_1^*} = 0$, and $\dfrac{\partial \mathcal{H}}{\partial u_2^*} = 0$. Thus, solving the system (59) for u_1^*, and u_2^* gives

$$\begin{aligned} u_1^* &= \frac{\Lambda}{C_1}\left(-\lambda_1 + \rho\lambda_3 + (1-\rho)\,\lambda_4\right) s \\ u_2^* &= \frac{\xi}{C_2}\left(\lambda_4 - \lambda_6\right) m \end{aligned} \tag{60}$$

The characterization (61) holds on the interior of the control set U^*.

$$\begin{aligned} u_1^* &= \max\left\{0, \min\left(1, \frac{\Lambda}{C_1}\left(-\lambda_1 + \rho\lambda_3 + (1-\rho)\,\lambda_4\right) s\right)\right\} \\ u_2^* &= \max\left\{0, \min\left(1, \frac{\xi}{C_2}\left(\lambda_4 - \lambda_6\right) m\right)\right\} \end{aligned} \tag{61}$$

Proof. The adjoint system (56) obtained by taking the negative partial derivatives of Hamiltonian function $(\mathcal{H})$ with respect to each state variables under the transversality condition (57) are standard results from Pontryagin Maximum Principle (Pontryagin, 1964). Also, the partial derivatives of the Hamiltonian equation (55) with respect to each of the control variables u_i is observed where the optimal solution (60) is obtained by setting $\dfrac{\partial \mathcal{H}}{\partial u_i} = 0$, the system is then solved for u_i^* subject to constraints to establish the characterization equation. Hence, using the bonds $a \leq u_1$, and $u_2 \leq b$ the optimality equation (61) is formed. □

3.6 Cost-effective analysis

Using the method of Cost-Effectiveness, the costs and benefits of implementing the control strategies are compared. The total cost of implementing the control strategies presented in the previous sections was given by,

$$\Phi(U) := \int_0^T \left(\frac{C_1 u_1^2}{2} + \frac{C_2 u_2^2}{2}\right) dt \tag{62}$$

CHAPTER FOUR

RESULTS AND DISCUSSIONS

4.1 Introduction

This chapter presents the discussions of results emanating from different approaches of model analyses. The discussions vary from numerical simulations for the basic, fuzzy, and the controlled *SPLMAR* model. The ethnographic survey results confirming the latent variables involved in the study are also featured. The values of some model parameters were obtained from scholarly literature review. Numerical simulations and graphical presentations of the analytical results were done using the MATLAB software.

4.2 Ethnographic survey results for the latent variables

In this study, ethnographic survey was used to collect information related to alcohol drinking in Tanzanian context. Guided by an ethnographic survey results, important factors to be featured in model formulation were determined reflecting the true life style of drinking community in the country. Six areas were covered by the survey, that is, influence of religious beliefs; alcohol drinking attitudes/behaviors; alcohol drinking frequency; alcohol drinking patterns; types of alcoholic beverages consumed; and health risks associated with alcoholism.

The ethnographic data also provided alcohol associated behaviors such as daily consumption rate and factors which contributed to conversion into alcohol drinking. Observational studies provide important information in the model development, particularly if the study is relevance to a particular society, Tanzania in this context.

4.2.1 Influence of Religious Beliefs

The study examined the roles of religious beliefs in molding the behaviors of believers in the community. In this topic it was observed that, the community had the population proportion which identified with one of religious beliefs operating in the country. It was observed further, that a proportion of religious population were raised in religious families. An individual were considered an active religious member based on commitment of such individual dedicated in religious activities.

The knowledge of participants about the official doctrine of their religious group regarding to alcohol drinking behaviors was also the topic of concerned. In that regard, it was observed that a population proportion believed in responsible drinking and that it was okay to consume alcohol moderately and responsibly as one's belief allows. On contrary the other population proportion believed that, the behavior is strictly not allowed under any circumstances.

It was further observed that, sharing the same drinking status does not guarantee that individuals share the same perceptions about alcohol drinking behaviors. For instance, among non-drinkers population, there existed non-drinkers who associated drinking habit with sinful act, while others did not associate alcohol drinking with sin despite their stand against it. One interviewee would say this regarding to this topic:

> *Despite the fact that my religious beliefs is not strictly regarding to alcohol drinking, I have never took an alcoholic beverages in my life* (Anonymous interviewee, personal communication, 2018).

The surprising results on the relationship between alcohol drinking and religiosity is that, despite the fact that most religions support the inverse relationship between religiosity and alcohol consumption, a serious breach of religious doctrine on alcohol drinking were observed among religious members. An anonymous interviewee who identified to be the follower of a religious belief which is strictly against alcohol drinking said this:

> *You know very well that in [. . .] we have zero tolerance to alcohol drinkers by regarding the behavior sinful, but I used to drink whenever I go out to discuss business deals with my business partners, and I do this secretly, away from my [. . .] leaders or fellow believers* (Anonymous interviewee, personal communication, 2019).

This shows that, the confidence level of religious individuals to take alcoholic beverage openly, in the presence of their religious leaders or other fellow members sharing beliefs did not match with the breaching level of doctrine. The observation proves the powerful roles of religious beliefs in shaping individuals' behavior as supported by both European Commission (2007) and Francis *et al.* (2015) in different studies. It also suggests that, the virtual immunity received by religious population is non-permanent. Thus, the survey revealed existence of non-drinkers, consisting of individuals who do not consume alcoholic beverages as a result of associating themselves to a religious beliefs which regard drinking as a sinful behavior to be avoided by all means possible.

4.2.2 Alcohol Drinking Attitude

Initiation of drinking behaviors to the susceptible members was one of the interesting area of this survey where it was revealed that people acquired drinking behaviors for different reasons. Peer pressures and influences of close friends were strongly associated with initiation of drinking behavior.

> *My close friends and/or family members were drinking and insisted that I need to drink whenever we went out together, I believed that drinking was good idea because we had funny time together and I didn't see anything wrong with they drinking behaviors* (Anonymous interviewee, personal communication, 2018).

This result support the work of Mundt *et al.* (2012) in which peer selection was cited as the major cause for adolescents to engage in binge drinking. It was observed further that some individuals started drinking out of their own curiosity.

> *My parents would never let me drink, this increased my curiosity on how it feels like getting high. So when I joined the university, I was free from parental over-protection, it was my time to experience the world. I went out with my friends, that's when I had my first drinking experience. I honestly don't blame any of my friends to have pressurized me, I started drinking just to feed my own curiosity* (Anonymous interviewee, personal communication, 2019).

Apart from peer pressures, early exposure to alcohol drinking behavior was associated to both initiation and progression at the pace of drinking. This also agrees with the study findings presented by Francis *et al.* (2015) advocating early exposure being responsible for initiation of alcohol drinking behaviors. Wrong role modeling was also mentioned to be responsible to initiation of alcohol drinking. In this case, people were recruited into drinking since they identified with significant individuals around them and those whom the community considered important persons based on their social lives. It was also observed that, a proportion of the drinking population believed that, the nature of their job facilitated their recruitment into drinking. This applies both ways, either the nature of their job required them to drink or they had to drink as a way of releasing stresses emanated from the pressures of their job.

> *See here bro (brother), I work in alcohol production company, we have the company's pub in the production premises and workers are allowed to drink from four*

> *to twelve bottles of beers depending on one's title. For my position I am entitled eight beers every day, my boss gets twelve and only four bottles for the casual workers. Remember, these beers can not be accumulated, meaning if you don't drink today you can not claim them tomorrow.* (Anonymous interviewee, personal communication, 2019).

This also may partly be explained as early exposure in the event where an individual works in brewery.

4.2.3 Alcohol Drinking Frequency

Alcohol drinking frequency is another aspect that was examined where probing questions were designed to assess the frequency of alcohol drinking in the community. It was observed that non-drinkers, occasional drinkers, regular drinkers, alcoholics and recovered population all existed in the community. However, recovering from alcohol drinking practices was not seem to be a popular phenomenon in the community. This could be explained in quantitative terms that, there existed insignificant number of recovered population. The low rate of individuals who voluntarily opt to find intervention programs at their disposal may explain the height of the resistance expected from the alcoholic victims against the intervention efforts. While the roles of intervention programs cannot be overemphasized, the low recovery rate underscores the importance of control intervention as the key subject in the reduction of alcohol related risks in the community.

4.2.4 Alcohol Drinking Patterns

For the purposes of this survey, one unit of alcohol consumption is defined as equivalent volume of one bottle of standard beer, a glass of wine, or a tot of spirit. Assessing the quantified amount of alcohol consumption as per one drinking occasion among the population of drinkers. It was observed that exceeding standard drinking limit according to acceptable standards of WHO was commonly practiced in the community. The Word Health Organization recommends not more than 21 standard bottles of alcoholic beverages per week for men and 14 drinks per week for women (WHO, 2014), where majority of drinkers crossed this line. However, a number of reasons were given in justification of drinking habit above the acceptable standards. Among the popular justifications cited regularly are: meeting drinking friends in a drinking venue; attending social events where alcohol was served freely; being emotionally charged; and night

club attendance. Since a group of friends may not share drinking capacity, when friends meet for drinks no one wishes to leave the rest, as a result over-drinking cases may occur.

4.2.5 Common types of alcoholic beverages consumed

The most common alcoholic beverages consumed in Tanzania includes: standard beers, wines, spirits, and unlabeled locally made alcohol. Except for unlabeled, the other alcoholic beverages served are available in local brands and imported international brands.

4.3 Sensitivity analysis of the basic model

Sensitivity analysis was used to determine the best solution in reduction of health risks and mortality associated to alcoholism. It was used to understand the relative importance of the different parameters responsible for risk transmission dynamics and prevalence (Chitnis *et al.*, 2008). The sensitivity analysis of parameters was performed in order to determine the input parameters with the most contribution to the output variability of $\mathcal{R}_0$ (Huo *et al.*, 2016; Hamby, 1994; Rosa *et al.*, 2018). This was done based on the fact that, the initial spread of health risk associated to alcoholism in the model presented in (2) is directly related to the basic risk reproduction number $\mathcal{R}_0$ (Chitnis *et al.*, 2008).

Using normalized forward sensitivity index method $-$a partial differential technique$-$ the sensitivity coefficients, $\Upsilon_{y_i}^{\mathcal{R}_0}$, for each of the input parameters $y_i \in y$ for $\mathcal{R}_0$ in equation (30) to the output variable $\mathcal{R}_0$ were calculated (Hamby, 1994; Chitnis *et al.*, 2008). Given a vector y $-$a set of parameters$-$ with independent input for the corresponding dependent output $\mathcal{R}_0$. The sensitivity coefficient, defined as the measure of the relative change in the dependent variable when the independent variables change one at a time (Chitnis *et al.*, 2008; Hamby, 1994; Rosa *et al.*, 2018), explains the impact of each parameter value in the health risk transmission threshold. Researchers conduct sensitivity analysis for a number of reasons in an attempt to answer their research questions. However, the robustness of the model predictions to parameter values is the motivation of performing sensitivity analysis to many modelers (Chitnis *et al.*, 2008; Rosa *et al.*, 2018).

Considering an explicit formula for $\mathcal{R}_0$ given in equation (30), to each of the input parameters we derive an analytical expression for the sensitivity coefficient of $\mathcal{R}_0$, with respect to the

parameter y_i as

$$\Upsilon_{y_i}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial y_i} \times \frac{y_i}{\mathcal{R}_0} \tag{63}$$

where y_i is the i^{th} parameter as shown in Table 3. For example, the sensitivity index of $\mathcal{R}_0$ with respect to β is given by

$$\Upsilon_{\beta}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \beta} \times \frac{\beta}{\mathcal{R}_0} = +1.0000 \tag{64}$$

Following the similar procedure, the sensitivity coefficients of the rest of parameters were calculated and presented their results in Table 3. Learning from the sensitivity coefficients in Table 3, the most sensitive parameters which make significant changes in $\mathcal{R}_0$ include: the natural mortality rate, μ; recruitment rate, π; measure of influence of risky individuals, β; and the necessary contact rate between a susceptible member and a drinker required to convince the susceptible member to drink, c. The rest of the parameters have smaller sensitivity indices which may not require as much attention to estimate since small perturbation in those parameters lead to insignificant changes in the output variable (Rosa *et al.*, 2018).

The sensitivity coefficients $\Upsilon_{\mu}^{\mathcal{R}_0} = -1.6901$, and $\Upsilon_{\pi}^{\mathcal{R}_0} = \Upsilon_{\beta}^{\mathcal{R}_0} = \Upsilon_{c}^{\mathcal{R}_0} = +1.0000$ imply that, μ is negatively correlated with $\mathcal{R}_0$ while $\pi,\ c$, and β are positively correlated with $\mathcal{R}_0$. For instance, increasing (or decreasing) μ by 10%, the resultant $\mathcal{R}_0$ is also expected to decrease (or increase) by 16.901% of its original value. Similarly, increasing (or decreasing) either $\pi,\ \beta$ or c by 10%, the value of $\mathcal{R}_0$ also increases (or decreases) by 10%. However, despite the significant sensitivity coefficient of π, and μ, they may not be the suitable decision variables in this case. Any deliberate efforts to increase the natural death rate, μ or decrease the recruitment rate, π for the purpose of reducing the intensity of the spread of the health risks in the community defeats ethical requirements thus the two are removed from the list of targeted parameters.

According to the sensitivity analysis results of input parameters for $\mathcal{R}_0$, intervention strategies focusing on discouraging the drinking behaviors at its initiation stage is more effective than targeting the population proportions at any risky stages. This result supports research findings presented by Bhunu (2012) which advocates efforts to rescue moderate drinkers than alcoholics. Discouraging drinking habits at its initiation stage can be effectively done when the focus is on reducing β, and c values to desired levels. Reduction of β value implies that lowering down the influence level of the risky population over the susceptible individuals. From social psychology point of view, the influencing capacity of an individual depends on individual's social status in the community. Reduction of β value can be effective when influential people in the community

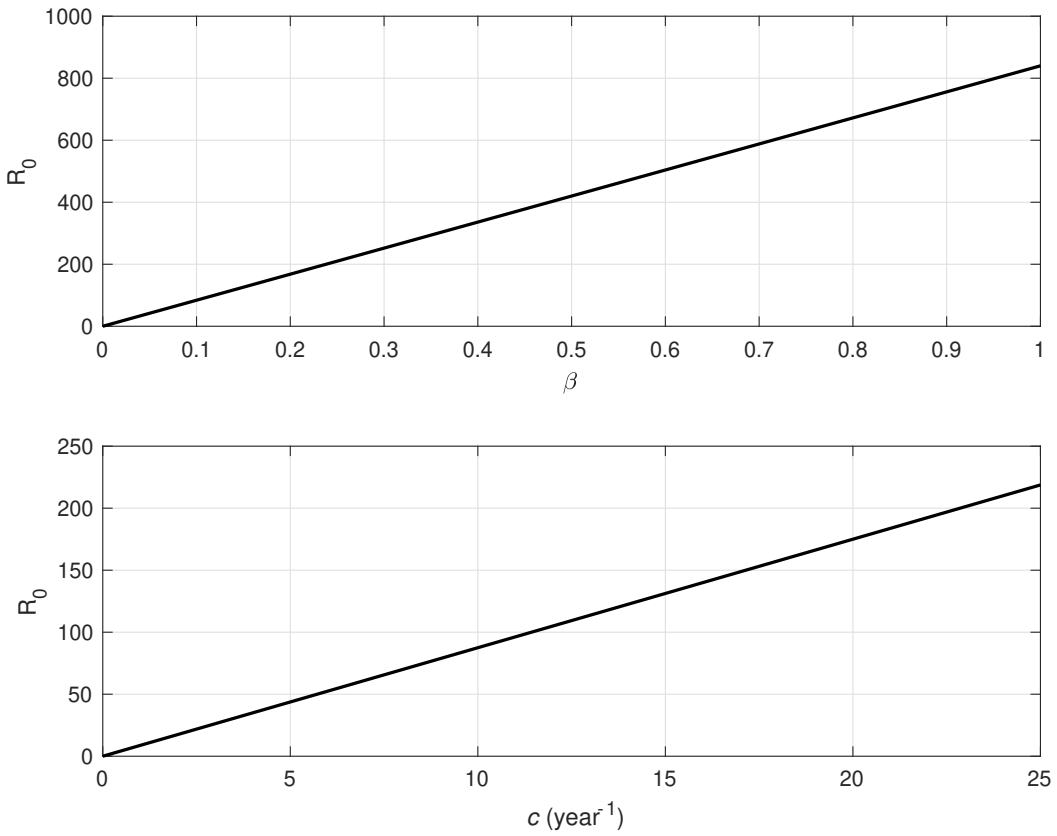


Figure 5: Simulation for the effects of β, and c on $\mathcal{R}_0$, using the other parameter values in Table 3

are involved in campaigning against unhealthy alcohol drinking habits. Similarly, it is also observed that $\mathcal{R}_0$ may be reduced significantly by reducing the number of times, c, in which alcoholic and non-alcoholic individuals interact. However, reduction of c greatly depends on both individual's willingness and desire to change, and the personal efforts dedicated towards the desirable change. Figure 5 presents the effects of variation of β, and c values on $\mathcal{R}_0$ using parameter values in Table 3 where the particular parameter is not considered as independent variable of $\mathcal{R}_0$.

Table 3: Description of parameters for model (2) and their sensitivity indices on $\mathcal{R}_0$

Parameters (y)	**Parameter values**	**Source**	**Sensitivity Index** $\Upsilon_{y_i}^{\mathcal{R}_0}$
π	$0.0310yr^{-1}$	Assumed	$+1.0000$
μ	$0.02yr^{-1}$	Bhunu (2012)	-1.6901
α	$0.0350yr^{-1}$	Bhunu (2012)	-0.0456
δ	$0.0075yr^{-1}$	Bhunu (2012)	-0.1325
σ	$0.0100yr^{-1}$	Thamchai (2014)	-0.0041
ν	$0.0020yr^{-1}$	Assumed	-0.0048
τ	$0.0016yr^{-1}$	Assumed	-0.0477
ψ	$0.0100yr^{-1}$	Assumed	-0.0130
ξ	$0.0025yr^{-1}$	Bhunu (2012)	-0.0746
η	$0.0050yr^{-1}$	Bhunu (2012)	-0.0065
ω	$0.0010yr^{-1}$	Thamchai (2014)	0
γ_1	$0.1300yr^{-1}$	Mushanyu *et al.* (2016)	-0.3333
γ_2	$0.2400yr^{-1}$	Mushanyu *et al.* (2016)	$+0.3524$
θ_1	1.0002	Assumed	$+0.8515$
θ_2	1.0005	Bhunu (2012)	$+0.0913$
ϕ	0.6000	Assumed	-0.0484
ρ	0.06500	Assumed	$+0.0131$
β	0.2500	Bhunu (2012)	$+1.0000$
c	$24yr^{-1}$	Assumed	$+1.0000$

4.4 Numerical analysis of the basic model

The parameter values in Table 3 and conditions of state variables, $S(0) = 350$, $P(0) = 80$, $L(0) = 20$, $M(0) = 100$, $A(0) = 30$ and $R(0) = 0$ were used for the purpose of simulating the risky population for system (2) against time in years. For avoidance of the danger of confusion, it is important to note that when one parameter took a variable nature for simulation purposes, the rest of parameters were considered constant with the value as indicated in Table 3.

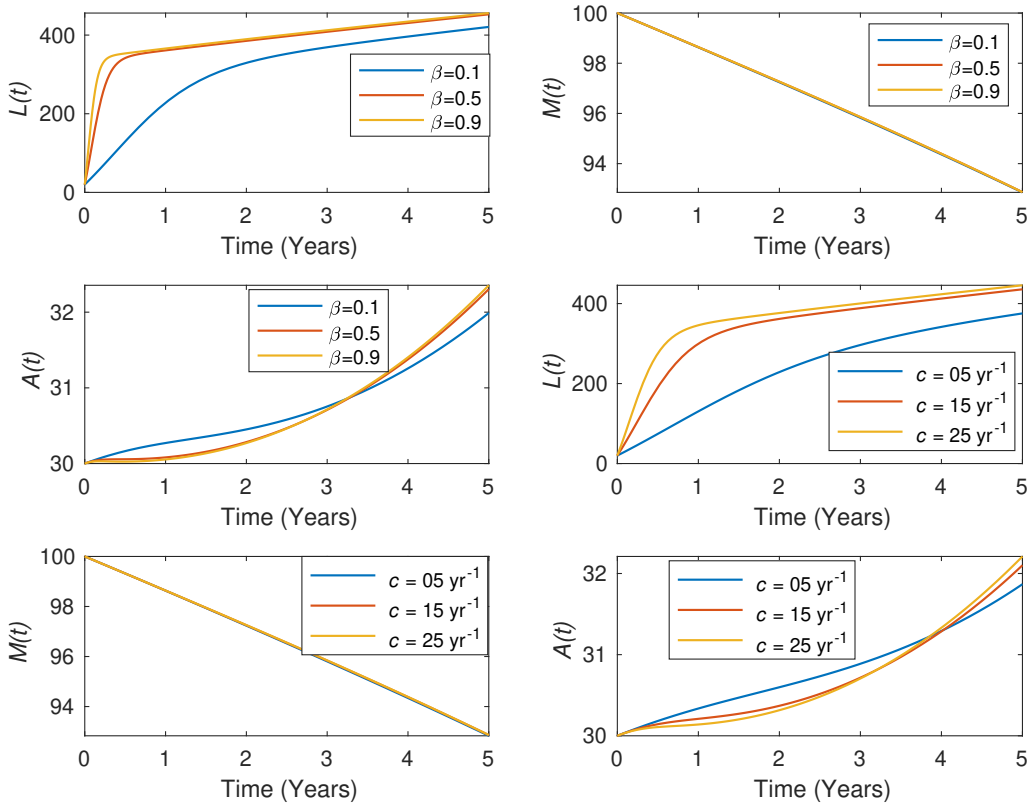


Figure 6: Simulation for the effects of β and c on risky populations using the other parameter values in Table 3.

Figure 6 shows the effects of β, and c values to the risky classes. The values of β was set to increase from 0.1 to 0.9 allowing the increase of step size of 0.4 while c was set to increase from $c = 5$ to $c = 25$ with the step size of 10. The increase in β values translates into the increase of the influencing level of the risky individuals over non risky individuals. Consequently, it increases the recruitment of susceptible population proportion into low risk compartment (see Fig. 6). The variation of β shows no significant effects on the medium risk population compartment,

so is the variation of c.

Considering the fact that alcohol related risks is a staged process depending on the consumption patterns and frequencies among others factors, as β increases $L(t)$ will increase quantitatively. However, the population proportion of the low risk class converges to a common value after the period of one year. Similarly, we observe that the number of high risk drinkers decreases with the increase of β values for the first three years and later it increases with the increase of β. The effects of variation of the contact rates between risky and non risky individuals giving easy access to alcohol beverages follows similar patterns as that of β variation with β being more efficient in terms of time. That is to say, any efforts targeting to reduce β value will yield the desired results in considerably shorter time than the case may be when c is targeted.

4.5 Analysis of fuzzy modeling

4.5.1 Bifurcation and Fuzzy Basic Risk Reproduction number

Owing to the basic health risk reproduction number, $\mathcal{R}_0$, presented in (30). The new function $\mathcal{R}_0(x)$ can be written as

$$\mathcal{R}_0(x) = c\beta(x)\left(\frac{\rho\kappa_4\left(\bar{\kappa}_5+\alpha(x)\right)+\left(\theta_1\left(\bar{\kappa}_5+\alpha(x)\right)+\theta_2\delta\right)\left(\left(1-\rho\right)\kappa_3+\rho\sigma\right)\left(1-\kappa\right)}{\kappa_3\kappa_4\left(\bar{\kappa}_5+\alpha(x)\right)}\right) \tag{65}$$

where $\bar{\kappa}_5 = \mu+\eta+\psi$, and $\kappa = \dfrac{\pi\left(\phi\mu+\gamma_1\right)}{\mu\left(\mu+\gamma_1+\gamma_2\right)}$. The value of $\mathcal{R}_0$ may vary significantly de-

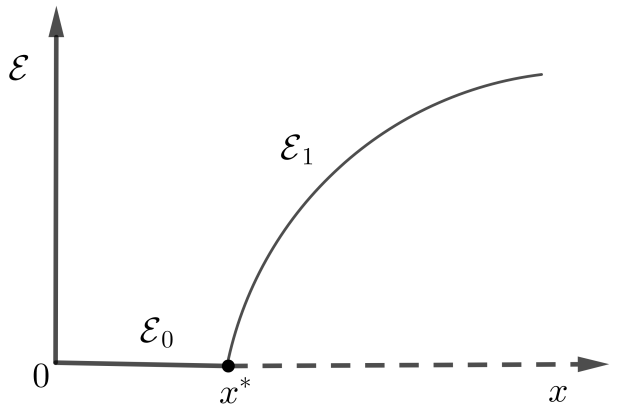


Figure 7: Bifurcation diagram

pending on the type of risk dynamics studied in the population or different populations involved in similar studies (Verma *et al.*, 2018). Similarly, the stability of the risk-free equilibrium also changes from stable to unstable when $\mathcal{R}_0$ increases to 1. The system acquires a bifurcation at

the risk-free equilibrium when $\mathcal{R}_0 = 1$. Suppose that the bifurcation value occurs at x^*, (see Fig. 7) where x^* is given as

$$x^* = \frac{a_4 - \sqrt{a_2 - 2a_3 + (-1 + u + \alpha_0)^2 (x_0 - x_{min})^2}}{2a_1 (-1 + u + \alpha_0)(a_5\theta_1 + \rho\kappa_4)} \tag{66}$$

provided that $x_{min} \leq x^* \leq x_0$ and

$$\begin{aligned}
a_1 &= \frac{c(1-\kappa)}{\kappa_3\kappa_4}, \\
a_2 &= (((-1 + u + \alpha_0) x_{min} + (\alpha_0 + \kappa_5)(x_{min} - x_0))(\theta_1 a_5 + \rho\kappa_4) + \theta_2\delta a_5 (x_{min} + x_0))^2 {a_1}^2, \\
a_3 &= (x_0 - x_{min})(-1 + u + \alpha_0)(((-1 + u + \alpha_0) + (\alpha_0 + \kappa_5)(x_0 + x_{min}))(\theta_1 a_5 + \rho\kappa_4) \\
&\quad - (x_0 + x_{min})\theta_2\,\delta a_5)\, a_1, \\
a_4 &= (((u-1) x_{min} + x_0\alpha_0)(\theta_1 a_5 + \rho\kappa_4) + ((\theta_1 a_5 + \rho\kappa_4)\kappa_5 + \theta_2\delta a_5)(x_0 - x_{min}))\, a_1 \\
&\quad + (-1 + u + \alpha_0)(\alpha_0 - x_{min}), \\
a_5 &= ((1-\rho)\kappa_3 + \rho\sigma)
\end{aligned}$$

The basic risk reproduction number presented in (65) is the function of x, that is $\mathcal{R}_0 = \mathcal{R}_0(x)$ but it can not be referred to as the fuzzy number since it is open to values exceeding unity. It is clear that $\mathcal{R}_0(x)$ is a the function of the degree of social influence in the spread of the behavior. However, both $\beta(x)$ and $\alpha(x)$ incline to their maximum level whenever $x \geq x_0$ and as such gives

$$\mathcal{R}_0(u) = c\left(\frac{(\rho\kappa_4(\kappa_5 + (1-u)) + (\theta_1(\kappa_5 + (1-u)) + \theta_2\delta)((1-\rho)\kappa_3 + \rho\sigma))(1-\kappa)}{\kappa_3\kappa_4(\kappa_5 + (1-u))}\right) \tag{67}$$

Introducing a positive number ϵ_0 such that $\epsilon_0\mathcal{R}_0(x) \leq 1$, with an appropriate choices of ϵ_0, a fuzzy set $\epsilon_0\mathcal{R}_0(x)$ whose fuzzy expected value, $FEV[\epsilon_0\mathcal{R}_0(x)]$ can be well defined. Therefore, the fuzzy basic risk reproduction number $\mathcal{R}_0^f$, which can be defined as the average number of secondary risk cases caused by one alcohol drinker introduced into entirely susceptible population (Mishra *et al.*, 2010) is given by

$$\mathcal{R}_0^f = \frac{1}{\epsilon_0} FEV[\epsilon_0\mathcal{R}_0(x)]. \tag{68}$$

The definition of fuzzy expected value leads to

$$FEV[\epsilon_0\mathcal{R}_0(x)] = \sup_{0\leq y\leq 1} \inf[y, k(y)], \tag{69}$$

where $k(y) = \Phi\{z \in \Omega : \epsilon_0\mathcal{R}_0(x) \geq y\} = \Phi(\Omega)$ is a fuzzy measure. For the purpose of this study, the possibility measure is given by

$$\Phi(\Omega) = \sup_{x\in X} \Pi(x), \quad \Omega \subset \mathbb{R}. \tag{70}$$

The linguistic variable $\Pi(x)$ was used to define different levels of influence, that is, low, medium, and high influence. From $FEV\left[\epsilon_0\mathcal{R}_0(x)\right]$, for a monotonic function $\mathcal{R}_0(x)$, the set X given as an interval $[x^*, x_{max}]$, we let $x^* \in X$ be the solution of the following equation

$$\epsilon_0 c\beta(x)\left(\frac{\left(\rho\kappa_4\left(\kappa_5+\alpha(x)\right)+\left(\theta_1\left(\kappa_5+\alpha(x)\right)+\theta_2\delta\right)\left(\left(1-\rho\right)\kappa_3+\rho\sigma\right)\right)\left(1-\kappa\right)}{\kappa_3\kappa_4\left(\kappa_5+\alpha(x)\right)}\right)=y. \tag{71}$$

Thus,

$$k(y)=\Phi\left[x^*, x_{max}\right]=\sup_{x^*\le x\le x_{max}}\Pi(x), \tag{72}$$

where $k(0)=1$ and $k(1)=\Pi(x_{max})$.

Now, $FEV(\epsilon_0\mathcal{R}_0)$ can be determined by considering the linguistic variable Π in three classes: 'weak Π_-', 'medium Π_-^+', and 'strong Π^+'. Each of these classification is a fuzzy number based on based on x_{min}, x_0, and x_{max} as appeared in Fig. 8. The classification of the degree of

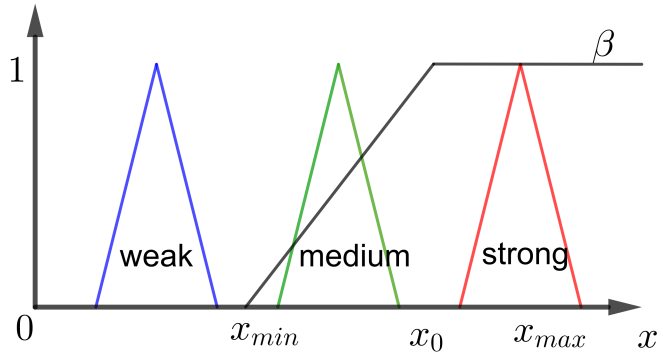


Figure 8: Classification of linguistic variable Π

social influences in the community can be explained in three different cases for positive values of d as follows.

4.5.2 Case One

In this case the weak degree of social influence (Π_-) is considered where $x_c + d < x_{min}$. Suppose that there exists $\bar{x}$ such that $\mathcal{R}_0(\bar{x}) = \mathcal{R}_0^f$, if $x_c + d < \bar{x}$ then

$$k(y)=\sup_{\bar{x}\le x\le x_{max}}\Pi(x)=0,\ \ \forall y\in[0,1] \tag{73}$$

Establishing $FEV\left[\epsilon_0\mathcal{R}_0\right] = 0 < \epsilon_0$ which can be translated as fuzzy basic risk reproduction number $\mathcal{R}_0^f < 1$ and hence the extinction of the health risk associated with alcoholism.

4.5.3 Case Two

In this case the medium degree of social influence (Π^{+}_{-}) is considered with $x_c - d > x_{min}$ and $x_c + d < x_0$ giving

$$k(y) = \begin{cases} 1, & \text{if } 0 \leq y < \epsilon_0 \mathcal{R}_0(x_c) \\ \Pi(\bar{x}), & \text{if } \epsilon_0 \mathcal{R}_0(x_c) \leq y \leq \epsilon_0 \mathcal{R}_0(x_c + d) \\ 0, & \text{if } \epsilon_0 \mathcal{R}_0(x_c + d) < y \leq 1 \end{cases} \tag{74}$$

For $d > 0$, $k(y)$ is the continuous function giving $k(0) = 1$ and $k(1) = 0$. This translates $\mathcal{R}_0^f$ as the fixed point k and $\mathcal{R}_0(x_c) < \mathcal{R}_0^f < \mathcal{R}_0(x_c + d)$. Since $\mathcal{R}_0(x)$ is a continuous monotonic (increasing) function, the Intermediate Value Theorem suggests that there exists $\bar{x}$ with $x_c < \bar{x} < x_c + d$ in which the values of $\mathcal{R}_0(\bar{x})$ and $\mathcal{R}_0^f$ coincide such that $\mathcal{R}_0^f = \mathcal{R}_0(\bar{x}) > \mathcal{R}_0(x_c)$. Furthermore, the average number of fuzzy basic risk reproduction number $\mathcal{R}_0^f$ is higher than the number of secondary risk cases $\mathcal{R}_0(x_c)$ due to the medium level of social influence implying that the health risk associated with alcoholism is endemic.

4.5.4 Case Three

In this case a strong degree of social influence (Π^{+}) was considered by defining $x_c - d > x_0$ and $x_c + d < x_{max}$. The results in (75) can be established.

$$k(y) = \begin{cases} 1, & \text{if } 0 \leq y < \epsilon_0 \mathcal{R}_0(x_c) \\ \Pi(\bar{x}), & \text{if } \epsilon_0 \mathcal{R}_0(x_c) \leq y < \epsilon_0 \mathcal{R}_0(x_c + d) \\ 0, & \text{if } \epsilon_0 \mathcal{R}_0(x_c + d) \leq y \leq 1 \end{cases} \tag{75}$$

For any given $d > 0$, $k(y)$ is a monotonically decreasing and continuous function with $k(0) = 1$ and $k(1) = 0$. Therefore $FEV[\epsilon_0 \mathcal{R}_0]$ is established as a fixed point such that

$$\epsilon_0 \mathcal{R}_0(x_c) < FEV[\epsilon_0 \mathcal{R}_0] < \epsilon_0 \mathcal{R}_0(x_c + d) \tag{76}$$

By dividing by ϵ_0 throughout, we have the following results

$$\mathcal{R}_0(x_c) < \mathcal{R}_0^f < \mathcal{R}_0(x_c + d) \tag{77}$$

Now, since $\mathcal{R}_0^f > 1$ translates into endemic health risks associated with alcoholism.

4.5.5 Analysis of risk control in fuzzy epidemic system

The control analysis of the risk estimation in the population was performed by using the fuzzy basic risk threshold $\mathcal{R}_0^f = \mathcal{R}_0(\bar{x})$. The spread of health risk in the proposed fuzzy *SPLMAR*

model (43) depends on the degree of the social influence x as an input value of the transmission factor $\beta(x)$. The description of existence and stability of the risk in the system is case-wise presented hereunder. Since the proposed fuzzy system (43) represents a family of systems depending on the variable x, these family systems can be simplified by a unique system of equations with the same results. It is shown that the bifurcation occurs at $x = x^*$, that is $\mathcal{R}_0(x^*) = 1$.

(i) **Weak influence:** In this case we have $x < x_{min}$ giving $\mathcal{R}_0 = 0$ suggesting the extinction of the health risks associated with alcoholism in the community.

(ii) **Medium influence:** In this case, three possibilities may arise as follows:

- If $x < x^*$, then $\mathcal{R}_0 < 1$ suggesting the risk free community.
- If $x = x^*$, then $\mathcal{R}_0 = 1$ an indication of risk bifurcation.
- If $x > x^*$, then $\mathcal{R}_0 > 1$ implying the risk endemic in the community.

(iii) **Strong influence:** In this case we have $x \in [x_0, x_{max}]$ giving

$$\mathcal{R}_0(u) = c\left(\frac{(\rho\kappa_4(\kappa_5 + (1-u)) + (\theta_1(\kappa_5 + (1-u)) + \theta_2\delta)((1-\rho)\kappa_3 + \rho\sigma))(1-\kappa)}{\kappa_3\kappa_4(\kappa_5 + (1-u))}\right)$$

The spread of the health risks depends upon the parameter u. Let u^* be an improved value of u, three distinct possibilities can be established in which the spread of health risks takes as follows

(i) If $0 \leq u < u^*$, then $\mathcal{R}_0(u) < 1$ suggesting the health risks would be cleared in the community.

(ii) If $0 \leq u = u^*$, then $\mathcal{R}_0(u) = 1$ implying that the system passes through a bifurcation state.

(iii) If $0 \leq u^* < u$, then $\mathcal{R}_0(u) > 1$ suggesting that the health risk problem would spread out in the system.

4.6 Numerical methods and simulations of the optimal control model

4.6.1 Effects of variation of control strategies

The optimal control strategies for the alcohol related health risks transmission model system (50) were analyzed numerically focusing on public health education campaign, and rehabilitation program. For effective reduction of health risks associated with alcoholism in the community, impacts of individual control strategies or in combination were investigated and simulated in a period of five years. Choosing arbitrarily the values $B_1 = 10, B_2 = 15, B_3 =$

$15, C_1 = 10, C_2 = 0.05$ and using parameter values from Table 3 with initial conditions, $s(0) = 0.5833$, $p(0) = 0.1333$, $l(0) = 0.0333$, $m(0) = 0.1667$, $a(0) = 0.0500$ and $r(0) = 0.0333$. The following strategies were implemented and their cost-effectiveness were examined. The simulation results showing the effects of variation of control strategies on the risky classes were presented graphically in Fig. 9 - 11 where red dashed lines represented specific population dynamics without control, the blue solid lines represented population dynamic under specific control. However, in the graphs representing the control profiles during the implementation of a particular control strategy, the blue and red lines represented the control option u_1, and u_2 respectively.

4.6.2 Strategy A: implementing public health education campaign (u_1)

Implementation of this strategy involved one control option u_1, to minimize the objective function $(\mathcal{J})$, while keeping $u_2 = 0$. Simulation results from Fig. 9 show a significant differences between risky classes with controls and those without controls. Observing from Fig. 9(a)

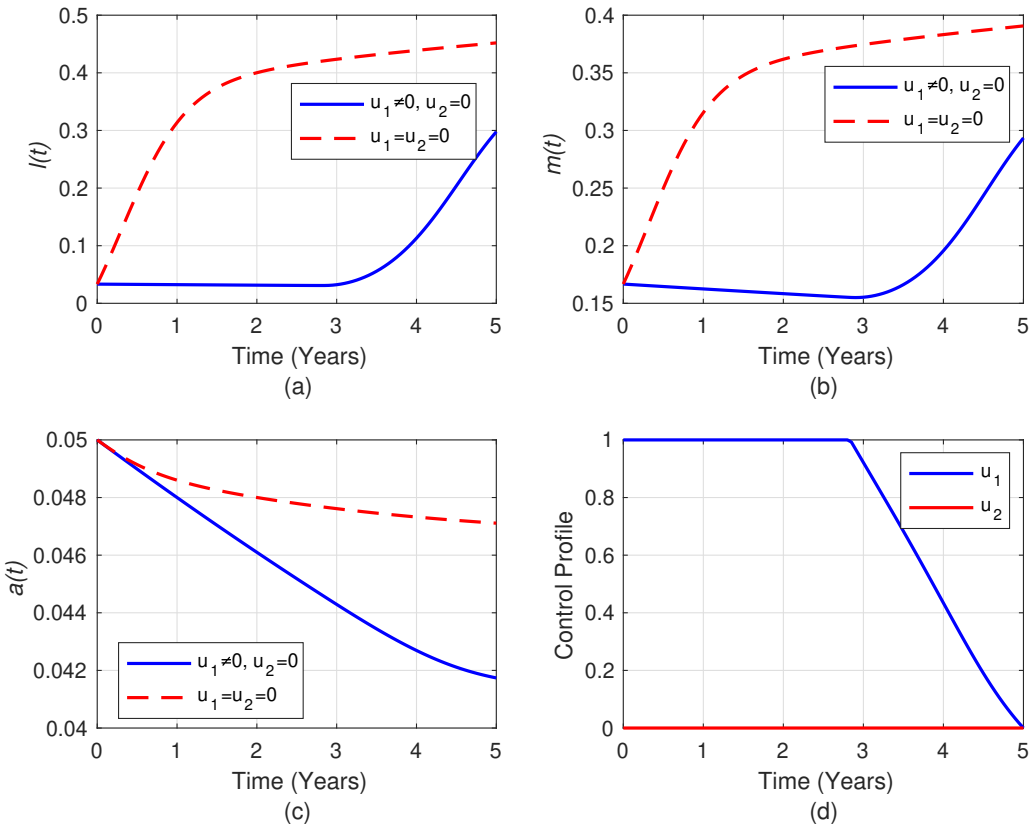


Figure 9: Time series of different risky population proportions with their corresponding Control profile for implementation of strategy A

showed that in the absence of the control, the population proportion $l(t)$ grew monotonically during the first year and slowed down afterwards. On another hand, with an implementation of the control strategy, the population proportion $l(t)$ remained stable for at least three years before it started increasing afterwards. The positive effects of the application of control strategy A in the population proportions $m(t)$ and $a(t)$ could be observed in Fig. 9(b) and (c) respectively. The total risk averted by implementing strategy A is 45.8773. This was achieved when the control profiles u_1 was implemented at a maximum level for the first three years before it started decreasing to zero by the end of fifth year.

4.6.3 Strategy B: implementing rehabilitation program (u_2)

Under this strategy u_2 was implemented while maintaining $u_1 = 0$ whose simulation results were recorded in Fig. 10. In this case, no significant changes of the risk averts in all risky population proportions was observed. Insignificant changes may be translated as the expected resistance from the victims in using the rehabilitation program as the control option when the programs are run on voluntary bases. However, the role of rehabilitation programs may not be ignored especially to the victims who have accepted the method. In this regard, strategy C is proposed, where rehabilitation program is implemented along with the implementation of public health education campaign.

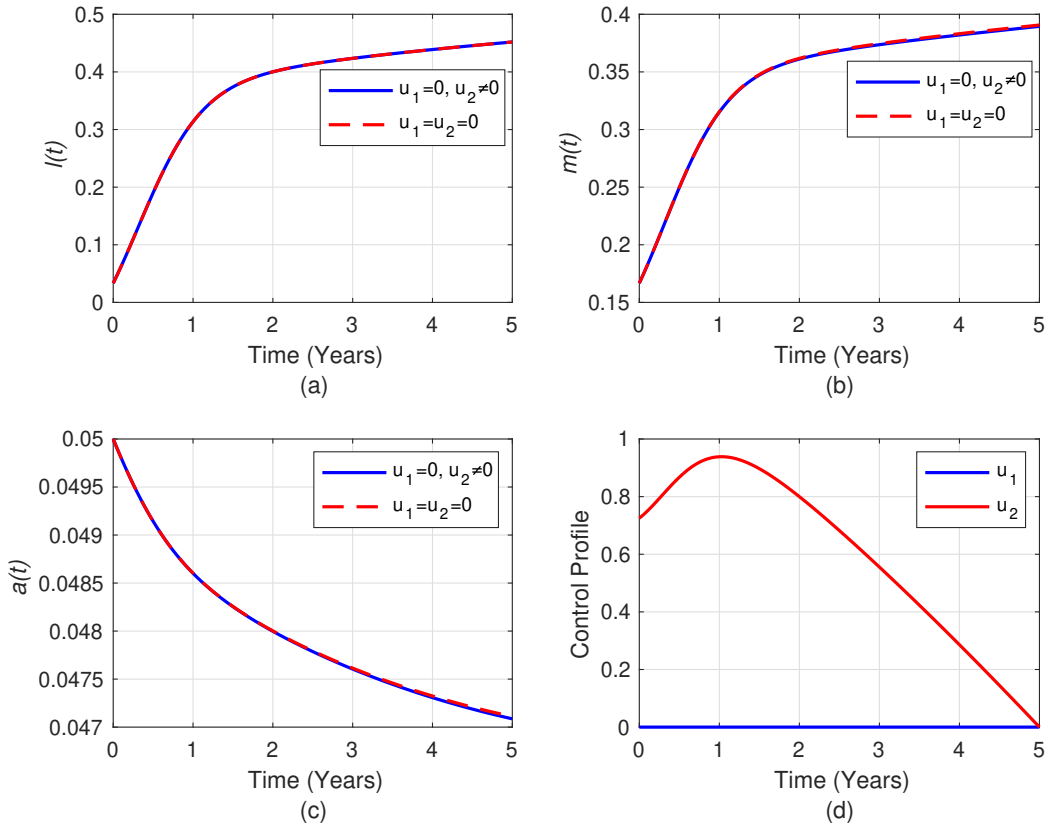


Figure 10: Time series of different risky population proportions with their corresponding control profile for implementation of strategy B

4.6.4 Strategy C: implementing both public health education campaign (u_1), and rehabilitation program (u_2)

Strategy C was made by implementing a combination of the two control options, that is, u_1 and u_2 where $u_i \neq 0, \forall i \in \{1, 2\}$. The simulation results presented in Fig. 11 are similar to results presented in strategy A. Figures 11(a)-(d) showed that in the absence of the control, the risky population proportion $l(t)$ grew monotonically during the first year before it changed the growth rate into some lower values. On the other hand, with an implementation of the control strategy, population proportion $l(t)$ remained stable for the first three years and increased its growth rate into higher values. For the period of five years, the total risky averts produced by implementation of strategy C amounted to 45.9176. The positive effects of the application of control strategy C in the population proportions $l(t)$, $m(t)$ and $a(t)$ could be observed in Fig. 11. This was achieved when the control profiles u_1 was implemented at a maximum level for the first three years before decreasing to zero by the end of fifth year; and u_2 was implemented

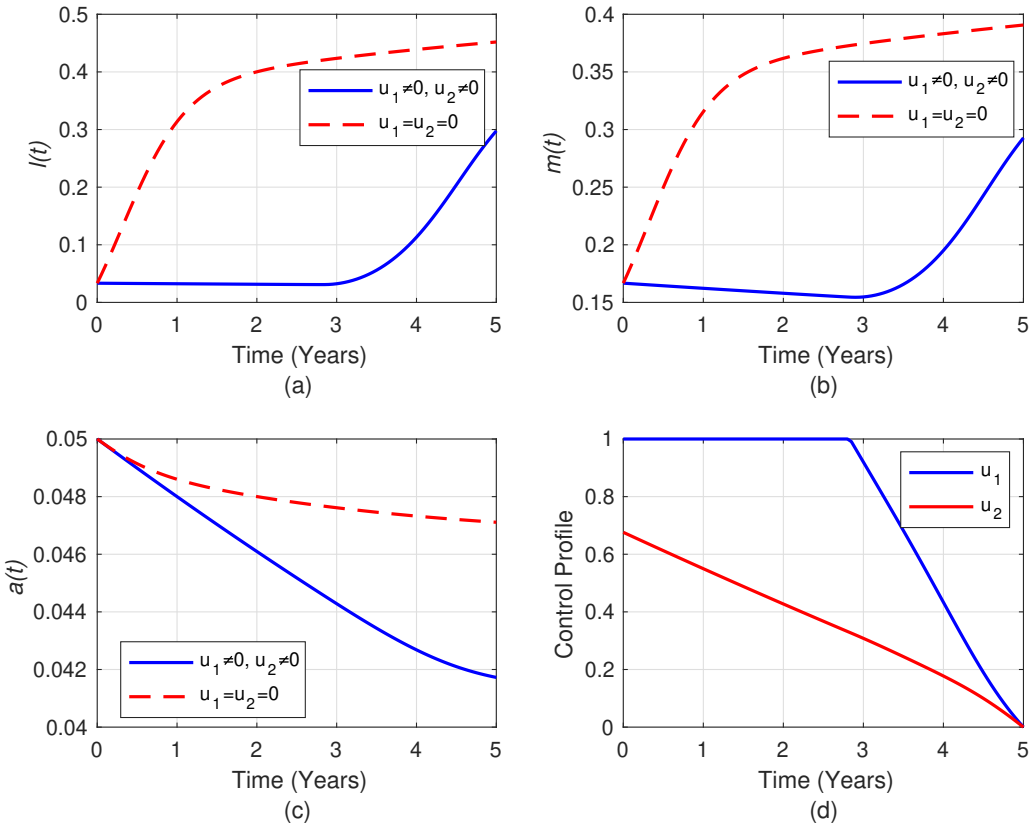


Figure 11: Time series of different risky population proportions with their corresponding control profile for implementation of strategy C

initial value of 0.6761 and slowly decreased to zero for the period of five years.

4.7 Incremental Cost-Effectiveness Ratio (ICER)

Following Okosun *et al.* (2013); Hugo *et al.* (2017); Berhe *et al.* (2018), and Nyerere *et al.* (2020) the cost-effectiveness analysis was performed by using Increamental Cost Effectiveness Ratio (*ICER*) approach. This is the ratio of the difference in cost between two possible interventions to the difference in their outcomes. It provides the comparison results for interventions options while incorporating two important features which are cost of implementing the intervention and its outcome (or benefit). For instance, when two interventions options u_i and u_j were available, where $\Phi(u_i)$ and $\Phi(u_j)$ represented the cost and benefit of interventions u_i and u_j respectively. The *ICER* value was computed as follows,

$$ICER = \frac{\Phi(u_i) - \Phi(u_j)}{E(u_i) - E(u_j)}, \tag{78}$$

where $E(u_i)$ is the total risks averted for implementing interventions u_i.

Based on the *ICER* functional equation (78), while the numerator translates the differences in the cost of implementation of intervention strategies; the denominator translates the differences in health outcomes by means of the total number of risk cases averted as the consequences of implemented strategies (Hugo *et al.*, 2017; Berhe *et al.*, 2018; Nyerere *et al.*, 2020).

The health benefits obtained when intervention strategy (or some combinations) applied, were measured in terms of disability-adjusted life years (DALYs) gained or lost thereafter. The approach requires that most costly alternatives with less effective to be excluded while accepting more efficient and less costly strategies.

Simulation results for the implementation of three intervention strategies were ranked in order of increasing control effectiveness in terms of the number of risk cases averted. These results are presented in the Table 4.

Table 4: Incremental Cost-Effective Ratio in increasing order of total risks averted

Strategies	Total cost in $	Total risk averted	$ICER_i$
B	1120.4	-0.6857	-1633.9507
A	1169.3	45.8773	1.0502
C	1168.3	45.9176	-24.8139

Where strategy A represented implementation of public health campaign alone; strategy B represented implementation of rehabilitation programs alone; and strategy C occurred when public health campaign and rehabilitation programs were implemented simultaneously. The $ICER$ values in Table 4 were calculated as follows;

$$ICER_B = \frac{1120.4}{-0.6857} = -1633.9507$$

$$ICER_A = \frac{1169.3 - 1120.4}{45.8773 - (-0.6857)} = 1.0502$$

$$ICER_C = \frac{1168.3 - 1169.3}{45.9176 - 45.8773} = -24.8139$$

where $ICER_i$ was the $ICER$ value corresponding to strategy i. Owing to simulation results presented in Table 4, it was clear that strategy B was less effective compared to the rest of strategies implemented. Although it was not the most costly strategy, since it produced negative total risk averted, it was rejected right away and removed from the list of alternatives.

The *ICER* values were re-culculated for the rest of alternatives and its simulation results were presented in Table 5. In comparison, strategy C showed lower *ICER* value as compared to strategy A suggesting strong dominance of strategy A. This implied that, strategy C was less costly and more effective than strategy A. Since strategy A was likely to consume limited resources available, it was removed from the set of alternative solutions. Consequently, strategy C was accepted as the best cost-effective combination.

Table 5: Incremental Cost-Effective Ratio in increasing order of total risks averted

Strategies	Total cost in $	Total risk averted	$ICER_i$
A	1169.3	45.8773	25.4876
C	1168.3	45.9176	−24.8139

With these results, it could be observed that implementation of both public health education campaign (u_1) and rehabilitation program (u_2) presented as strategy C in this study had the least $ICER$ value and more cost-effective than the rest of strategies presented. This result gains literature support from Wang *et al.* (2014) besides the challenge of grouping moderate and susceptible population together. Similarly, while Huo *et al.* (2016), Mushayabasa (2015), Adu *et al.* (2017), Musanyu *et al.* (2018), Xiang *et al.* (2016) and Mundt *et al.* (2012) support weakening the intensity of social interaction between susceptible population and drinkers (of whatever level) as effective method to eradicate alcoholism in the community. The present study showed that implementation of such a method through providing public health education by itself was not cost-effective unless it was supplemented by installation of rehabilitation and sober house programs. The difference between the present study findings and those of reviewed models might be due to broad consideration of health risks in multiple stages and some social cultural beliefs in the current model.

CHAPTER FIVE

CONCLUSION AND RECOMMENDATIONS

5.1 Conclusion

In this study, the dynamics of health risks associated with alcoholism in a community is studied with reference to Tanzania. The aim was to develop, analyze and simulate the model and its controls, which was guided by the achievement of the following specific objectives: (1) To develop and analyze mathematical model for the dynamics of health related risks associated with alcoholism; (2) To develop and analyze mathematical model for the dynamics of alcohol related health risks with interventions; (3) To develop and analyze fuzzy mathematical model for the dynamics of health related risks associated with alcoholism by assuming uncertainty condition to selected parameters of the model; and (4) To evaluate the cost-effectiveness in the controlled model for the dynamics of alcohol related health risks with interventions.

The study considered a situation in which a proportion of susceptible population receives a social cultural protection acquired from religious beliefs. Except for occasional drinkers, it considered further that a proportion of drinkers population quit alcohol drinking voluntarily or upon medical grounds. Three different model analyses approaches were used to solve the formulated model with different objectives and the following conclusions were reached.

(i) The sensitivity analysis and numerical simulation results for $\mathcal{R}_0$ suggested that discouraging alcohol drinking behavior at its initiation stage is more effective in the control and reduction of the health related risks associated with alcoholism than targeting alcoholic population. The optimal benefit of education campaigns may be reached if the influential characters are involved in the campaigns. When a campaign targets particular group of people, the choice of characters to be involved should consider the interest of such a group. That is, the character should be viewed as a role model to the group in such a way that the people belongs to such a group would like to identify themselves with the character.

(ii) Implementation of Strategy B, where rehabilitation programs was implemented alone as a way of fighting against alcoholism, had less significant effects in the system. The same was true when the strategy was featured in Strategy C in the combination. Since there

was a close matching of the results between Strategy A and C, this shows that the significant effects detected in strategy C were influenced more by the control strategy A, public health education campaign. This implies further that, different from other infectious scenarios, intervention strategy by means of *"vaccination"* works better than that of *"treatment"* when it comes to health related risks associated with alcoholism as contagious behavior.

(iii) Implementation of strategy C, where both public health education campaign and rehabilitation programs are implemented together, make the best cost-effective combination in reduction of health risks associated with alcoholism in the community. For optimal cost effective benefit, health risks associated with alcoholism may be effectively eradicated by increasing the level of protection to the susceptible population by implementing public health education campaign (u_1); and increase the removal rate of alcoholics by implementing rehabilitation program (u_2) presented as Strategy C.

5.2 Recommendations

Considering both ethnographic and analytical results, it was clear that alcohol related health risks might be reduced significantly by keeping $\mathcal{R}_0 < 1$. This could be made possible by raising protection level of the susceptible population against societal (peer) influence. A great deal of this could be achieved through personal efforts of individuals towards behavioral change when there is substantial amount of public health awareness. However, I recommend the following to individuals, the government, and to the policy makers:

(i) Implement regular public health education campaign. When the influential characters are involved in the public education campaign, better results are expected since societal influence is the key factor of recruiting people into any particular behavior. In order to increase public health awareness in the community, the Government should initiate health education campaign programs targeting different age based groups to increase awareness to its citizens. In that regards, the influence of characters vary in different age based groups.

(ii) The choice of friends is as important as approving behavioral changes. A wise choice and a personal commitment to hang around with friends whose behaviors match with an

individual's desired behavior is needed. The future characters of an individual may be predicted by thorough character assessment of friends associated with such an individual. In this regards, if an individual is not strong enough to resist peer pressure when surrounded with drinking friends, one should avoid such drinking environment.

(iii) Although the laws of the land restrict alcoholic beverages to be sold to children under 18 years, this law is not effectively enforced. Policy makers should impose tight penalties to those breaching this law especially when alcoholic beverages are sold in essential goods local glossaries.

(iv) Recommended future work shall include the following problems:

- Carry out robust study to determine factors contributing to uncertainties in the environment.
- Optimal control studies targeting specific or different population proportions.
- Parameter estimations targeting specific or different parameters.

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APPENDICES

MATLAB Codes for Simulation of the Basic Model

MATLAB codes for sensitivity indices for input parameters to $\mathcal{R}_0$

```
clc;
clear all;
close all;
%List of input parameters
syms mu c phi sigma nu rho beta gamma1 gamma2 delta theta1...
 theta2 tau xi alpha eta psi

%Input parameters in vector form
v=[mu c phi sigma nu rho beta gamma1 gamma2 delta theta1 ...
theta2 tau xi alpha eta psi];
kappa3=mu+nu+sigma; kappa4=mu+tau+delta+xi; ...
kappa5=mu+alpha+eta+psi;
R0=(beta*c*(rho*kappa4*kappa5+(delta*theta2+theta1*kappa5)...
*((1-rho)*kappa3+rho*sigma))*(pi*(gamma2+mu*(1-phi))))/...
(kappa3*kappa4*kappa5*mu*(mu+gamma1+gamma2));
P=jacobian(R0,v);
%Parameter initial values
%mu  c  phi sigma nu   rho  beta gamma1 gamma2 delta theta1 ...
theta2 tau   xi   alpha   eta    psi
v0=[0.02 24 0.6 0.01 0.002 0.065 0.25 0.13 0.24 0.0075 ...
1.0002 1.0005 0.0016 0.0025 0.0350 0.0050 0.0100];
sensi= subs(P.*v/R0,v,v0);
sensitivityInd=double(sensi)
```

MATLAB Codes for Simulation of the Normalized Controlled Model

Function 1

```
function ydot = kims(t,yy,U,Constant)

s=yy(1);
p=yy(2);
l=yy(3);
m=yy(4);
a=yy(5);
r=yy(6);
%% Parameters
pi      = Constant(1);
phi     = Constant(2);
mu      = Constant(3);
beta    = Constant(4);
c       = Constant(5);
theta1  = Constant(6);
theta2  = Constant(7);
rho     = Constant(8);
omega   = Constant(9);
gamma1  = Constant(10);
gamma2  = Constant(11);
nu      = Constant(12);
tau     = Constant(13);
psi     = Constant(14);
sigma   = Constant(15);
delta   = Constant(16);
xi      = Constant(17);
eta     = Constant(18);
alpha   = Constant(19);
%---------------------
B1    = Constant(20);
B2    = Constant(21);
B3    = Constant(22);
C1    = Constant(23);
C2    = Constant(24);

%% CONTROL VARIABLES
u1 = U(1); u2=U(2);
%% ODE
ydot1 = -(mu+gamma1+(1-u1)*beta*c*(l+theta1*m+theta2*a))*s...
+gamma2*p+omega*r+(1-phi)*pi;
ydot2 = gamma1*s-(mu+gamma2)*p+nu*l+tau*m+psi*a+phi*pi;
ydot3 = (1-u1)*beta*c*(l+theta1*m+theta2*a)*rho*s ...
-(mu+nu+sigma)*l;
ydot4 = (1-u1)*beta*c*(l+theta1*m+theta2*a)*(1-rho)*s...
```

```
+sigma*l-(tau+mu+delta+(1+u2)*xi)*m;
ydot5 = delta*m-(mu+alpha+eta+psi)*a;
ydot6 = (1+u2)*xi*m+eta*a-(mu+omega)*r;
ydot  = [ydot1; ydot2; ydot3; ydot4; ydot5; ydot6];
```

Function 2

```
function ydot = kims_costate(t,p,U,X,Constant);
%this function solves a MU infection on human type three
% equations

L1=p(1);
L2=p(2);
L3=p(3);
L4=p(4);
L5=p(5);
L6=p(6);
%% Parameters
pi      = Constant(1);
phi     = Constant(2);
mu      = Constant(3);
beta    = Constant(4);
c       = Constant(5);
theta1  = Constant(6);
theta2  = Constant(7);
rho     = Constant(8);
omega   = Constant(9);
gamma1  = Constant(10);
gamma2  = Constant(11);
nu      = Constant(12);
tau     = Constant(13);
psi     = Constant(14);
sigma   = Constant(15);
delta   = Constant(16);
xi      = Constant(17);
eta     = Constant(18);
alpha   = Constant(19);
%---------------------
B1    = Constant(20);
B2    = Constant(21);
B3    = Constant(22);
C1    = Constant(23);
C2    = Constant(24);

%% Controls
u1 = U(1); u2=U(2);

s = X(1,:);p = X(2,:);l = X(3,:);m = X(4,:);a = X(5,:);
```

```
r=X(6,:);

ydot1 = -(1-u1)*((1-rho)*L4+rho*L3-L1)*beta*c*...
(l+theta1*m+theta2*a) -gamma1*(L2-L1)+mu*L1;

ydot2 = gamma2*(L2-L1)+mu*L2;

ydot3 = -B1+(1-u1)*(L1-rho*L3-(1-rho)*L4)*c*beta*s...
+mu*L3+nu*(L3-L2) +sigma*(L3-L4);

ydot4 = -B2+beta*c*(1-u1)*((L1-rho*L3)-(1-rho)*L4)*...
theta1*s+xi*(1+u2)*(L4-L6)+delta*(L4-L5)+mu*L4+tau*(L4-L2);

ydot5 = -B3+c*beta*(1-u1)*(L1-rho*L3-(1-rho)*L4)*theta2*s...
+(mu+alpha)*L5+eta*(L5-L6)+psi*(L5-L2);

ydot6 = mu*L6+omega*(L6-L1);

ydot  = [ydot1; ydot2; ydot3; ydot4; ydot5; ydot6];
```

MATLAB codes for Figure 9

```
clc
clear all
close all
t0 = 0; tf=5; n=100;
time =linspace(t0,tf,n);
% initial condition for STATE SYSTEM
y0=[0.5833 0.1333 0.0333 0.1667 0.0500  0.0333];
%% parameter values
Constant(1)  = 0.031;   % pi
Constant(2)  = 0.686;   % phi
Constant(3)  = 0.016;   % mu
Constant(4)  = 0.25;    % beta
Constant(5)  = 12;      % c
Constant(6)  = 1.0002;  % theta1
Constant(7)  = 1.0005;  % theta2
Constant(8)  = 0.65;    % rho
Constant(9)  = 0.001;   % omega
Constant(10) = 0.13;    % gamma1
Constant(11) = 0.24;    % gamma2
Constant(12) = 0.002;   % nu
Constant(13) = 0.0016;  % tau
Constant(14) = 0.01;    % psi
Constant(15) = 0.01;    % sigma
Constant(16) = 0.0075;  % delta
Constant(17) = 0.0025;  % xi
Constant(18) = 0.005;   % eta
```

```
Constant(19) = 0.0350;  % alpha
%----------------------
Constant(20) = 10;      % B1
Constant(21) = 15;      % B2
Constant(22) = 15;      % B3
Constant(23) = 10;      % C1
Constant(24) = 0.05;    % C2

%% Parameters
pi      = Constant(1);
phi     = Constant(2);
mu      = Constant(3);
beta    = Constant(4);
c       = Constant(5);
theta1  = Constant(6);
theta2  = Constant(7);
rho     = Constant(8);
omega   = Constant(9);
gamma1  = Constant(10);
gamma2  = Constant(11);
nu      = Constant(12);
tau     = Constant(13);
psi     = Constant(14);
sigma   = Constant(15);
delta   = Constant(16);
xi      = Constant(17);
eta     = Constant(18);
alpha   = Constant(19);
%--------------------
B1    = Constant(20);
B2    = Constant(21);
B3    = Constant(22);
C1    = Constant(23);
C2    = Constant(24);

lf  = [0 0 0 0 0 0];
%%
init = y0;
init2 =lf;
h = (tf-t0)/n;
u = linspace(0,0,n+1);
u1=u'; u2=u';
U = [u1 u2];

%% IMPLIMENTATION OF THE ALGORITHM
%Test 1 stoping condition 1
epsilon = 0.01;
X=init;
```

```
i=0; %Initialize iteration counter
mm=size(X);
NumXX =10e10;
Xnew = rand(n+1,mm(2)).*(repmat(X,n+1,1));
DenXnew=norm(Xnew);
while  NumXX/DenXnew>epsilon
Xold =  Xnew;
oldu = U;
%FORWARD RUNGE KUTTA  FOR STATES
[Tx, X]=rk4foward(@kims,t0, tf,n, init,U,Constant);

% BACKWARD RUNGEKUTA FOR COSTATES
[Tp, P]=rk4back(@kims_costate,t0,tf,n,init2,U,X,Constant);

%UPDATE THE CONTROLS
s = X(1,:); p = X(2,:); l = X(3,:); m = X(4,:);
a = X(5,:); r=X(6,:);
L1 = P(1,:); L2 = P(2,:); L3 = P(3,:); L4 = P(4,:);
L5 = P(5,:); L6 = P(6,:);


%% Graph ploting
 % Case1: u1/=0, u2=0
     u1 = max(0, min(1, beta.*c.*(l+theta1.*m+theta2.*a)...
     .*(-L1+rho.*L3+(1-rho).*L4).*s./C1));
     u2 = zeros(1,n+1);

Uu=[u1' u2'];
U = 0.5*Uu + 0.5*oldu; % Convex combination of the controls


Xnew =  X';
NumXX =abs(norm(Xnew-Xold));
DenXnew =norm(Xnew);
i=i+1; %Update iteration counter
end
%% PLOTING
X=X';
Tx =Tx';
SS=X(:,1); PP=X(:,2); LL=X(:,3); MM=X(:,4); AA=X(:,5);
RR=X(:,6);

Up = [0 0];
[T,Y] = ode45(@kims,time,y0,[],Up,Constant);

J =sum((B1*LL(end)+B2*MM(end)+B3*AA)+((C1/2)*...
Uu(:,1).*Uu(:,1)+(C2/2)*Uu(:,2).*Uu(:,2)))
```

```
%% Averts
l1 =  sum(Y(:,3));
l2 =  sum(X(:,3));
avert_l=l1-l2;
%%
m1 =  sum(Y(:,4));
m2 =  sum(X(:,4));
avert_m = m1-m2;
%%
a1 =  sum(Y(:,5));
a2 =  sum(X(:,5));
avert_a = a1-a2;
Total_risk_averts = avert_l+avert_m + avert_a
%%
figure(1)
hold on
subplot(2,2,1)
plot(Tx,X(:,3),'-b',T, Y(:,3),'--r', 'LineWidth',1.5);
ylabel('{\it l(t)}');
xlabel({'Time (Years)','(a)'});
  legend('u_1\neq0, u_2=0','u_1=u_2=0') %case1
grid on
hold on
%xlim([0 5]);
%%
subplot(2,2,2)
plot(Tx,X(:,4),'-b',T, Y(:,4),'--r', 'LineWidth',1.5);
ylabel('{\it m(t)}');
xlabel({'Time (Years)','(b)'});
  legend('u_1\neq0, u_2=0','u_1=u_2=0') %case1
grid on
hold on

%%
subplot(2,2,3)
plot(Tx,X(:,5),'-b',T, Y(:,5),'--r', 'LineWidth',1.5);
ylabel('{\it a(t)}');
xlabel({'Time (Years)','(c)'});
  legend('u_1\neq0, u_2=0','u_1=u_2=0') %case1
grid on
hold on
%%
subplot(2,2,4)
plot(Tx,Uu(:,1),'-b',Tx,Uu(:,2),'-r', 'LineWidth', 1.5);
ylabel('Control Profile');
xlabel({'Time (Years)','(d)'});
legend('u_1','u_2')
box
```

```
grid on
hold off
box
```

MATLAB codes for Figure 10

```
clc
clear all
close all
t0 = 0; tf=5; n=100;
time =linspace(t0,tf,n);
% initial condition for STATE SYSTEM
y0=[0.5833 0.1333 0.0333 0.1667 0.0500  0.0333];
%% parameter values
Constant(1)  = 0.031;   % pi
Constant(2)  = 0.686;   % phi
Constant(3)  = 0.016;   % mu
Constant(4)  = 0.25;    % beta
Constant(5)  = 12;      % c
Constant(6)  = 1.0002;  % theta1
Constant(7)  = 1.0005;  % theta2
Constant(8)  = 0.65;    % rho
Constant(9)  = 0.001;   % omega
Constant(10) = 0.13;    % gamma1
Constant(11) = 0.24;    % gamma2
Constant(12) = 0.002;   % nu
Constant(13) = 0.0016;  % tau
Constant(14) = 0.01;    % psi
Constant(15) = 0.01;    % sigma
Constant(16) = 0.0075;  % delta
Constant(17) = 0.0025;  % xi
Constant(18) = 0.005;   % eta
Constant(19) = 0.0350;  % alpha
%----------------------
Constant(20) = 10;      % B1
Constant(21) = 15;      % B2
Constant(22) = 15;      % B3
Constant(23) = 10;      % C1
Constant(24) = 0.05;    % C2

%% Parameters
pi      = Constant(1);
phi     = Constant(2);
mu      = Constant(3);
beta    = Constant(4);
c       = Constant(5);
theta1  = Constant(6);
theta2  = Constant(7);
```

```
rho     = Constant(8);
omega   = Constant(9);
gamma1  = Constant(10);
gamma2  = Constant(11);
nu      = Constant(12);
tau     = Constant(13);
psi     = Constant(14);
sigma   = Constant(15);
delta   = Constant(16);
xi      = Constant(17);
eta     = Constant(18);
alpha   = Constant(19);
%--------------------
B1    = Constant(20);
B2    = Constant(21);
B3    = Constant(22);
C1    = Constant(23);
C2    = Constant(24);

lf  = [0 0 0 0 0 0];
%%
init = y0;
init2 =lf;
h = (tf-t0)/n;
u = linspace(0,0,n+1);
u1=u'; u2=u';
U = [u1 u2];

%% IMPLIMENTATION OF THE ALGORITHM
%Test 1 stoping condition 1
epsilon = 0.01;
X=init;
i=0; %Initialize iteration counter
mm=size(X);
NumXX =10e10;
Xnew = rand(n+1,mm(2)).*(repmat(X,n+1,1));
DenXnew=norm(Xnew);
while  NumXX/DenXnew>epsilon
Xold =  Xnew;
oldu = U;
%FORWARD RUNGE KUTTA  FOR STATES
[Tx, X]=rk4foward(@kims,t0, tf,n, init,U,Constant);

% BACKWARD RUNGEKUTA FOR COSTATES
[Tp, P]=rk4back(@kims_costate,t0,tf,n,init2,U,X,Constant);

%UPDATE THE CONTROLS
s = X(1,:); p = X(2,:); l = X(3,:); m = X(4,:); a = X(5,:);
```

```
r=X(6,:);
L1 = P(1,:); L2 = P(2,:); L3 = P(3,:); L4 = P(4,:);
L5 = P(5,:); L6 = P(6,:);


%% Graph ploting
% Case2:u1=0, u2/=0,
     u1 =zeros(1,n+1);
     u2 =max(0, min(1, xi.*(L4-L6).*m./C2));

Uu=[u1' u2'];
U = 0.5*Uu + 0.5*oldu; % Convex combination of the controls

Xnew =  X';
NumXX =abs(norm(Xnew-Xold));
DenXnew =norm(Xnew);
i=i+1; %Update iteration counter
end
%% PLOTING
X=X';
Tx =Tx';
SS=X(:,1); PP=X(:,2); LL=X(:,3); MM=X(:,4); AA=X(:,5);
RR=X(:,6);

Up = [0 0];
[T,Y] = ode45(@kims,time,y0,[],Up,Constant);

J =sum((B1*LL(end)+B2*MM(end)+B3*AA)+((C1/2)*Uu(:,1).*...
Uu(:,1)+(C2/2)*Uu(:,2).*Uu(:,2)))

%% Averts
l1 =  sum(Y(:,3));
l2 =  sum(X(:,3));
avert_l=l1-l2;
%%
m1 =  sum(Y(:,4));
m2 =  sum(X(:,4));
avert_m = m1-m2;
%%
a1 =  sum(Y(:,5));
a2 =  sum(X(:,5));
avert_a = a1-a2;
Total_risk_averts = avert_l+avert_m + avert_a
%%
figure(1)
hold on
subplot(2,2,1)
plot(Tx,X(:,3),'-b',T, Y(:,3),'--r', 'LineWidth',1.5);
```

```
ylabel('{\it l(t)}');
xlabel({'Time (Years)','(a)'});
  legend('u_1=0, u_2\neq0','u_1=u_2=0') %case2
grid on
hold on
%xlim([0 5]);
%%
subplot(2,2,2)
plot(Tx,X(:,4),'-b',T, Y(:,4),'--r', 'LineWidth',1.5);
ylabel('{\it m(t)}');
xlabel({'Time (Years)','(b)'});
  legend('u_1=0, u_2\neq0','u_1=u_2=0') %case2
grid on
hold on

%%
subplot(2,2,3)
plot(Tx,X(:,5),'-b',T, Y(:,5),'--r', 'LineWidth',1.5);
ylabel('{\it a(t)}');
xlabel({'Time (Years)','(c)'});
  legend('u_1=0, u_2\neq0','u_1=u_2=0') %case2
grid on
hold on
%%
subplot(2,2,4)
plot(Tx,Uu(:,1),'-b',Tx,Uu(:,2),'-r', 'LineWidth', 1.5);
ylabel('Control Profile');
xlabel({'Time (Years)','(d)'});
legend('u_1','u_2')
box
grid on
hold off
box
```

MATLAB codes for Figure 11

```
clc
clear all
close all
t0 = 0; tf=5; n=100;
time =linspace(t0,tf,n);
% initial condition for STATE SYSTEM
y0=[0.5833 0.1333 0.0333 0.1667 0.0500  0.0333];
%% parameter values
Constant(1)  = 0.031;   % pi
Constant(2)  = 0.686;   % phi
Constant(3)  = 0.016;   % mu
Constant(4)  = 0.25;    % beta
```

```
Constant(5)  = 12;      % c
Constant(6)  = 1.0002;  % theta1
Constant(7)  = 1.0005;  % theta2
Constant(8)  = 0.65;    % rho
Constant(9)  = 0.001;   % omega
Constant(10) = 0.13;    % gamma1
Constant(11) = 0.24;    % gamma2
Constant(12) = 0.002;   % nu
Constant(13) = 0.0016;  % tau
Constant(14) = 0.01;    % psi
Constant(15) = 0.01;    % sigma
Constant(16) = 0.0075;  % delta
Constant(17) = 0.0025;  % xi
Constant(18) = 0.005;   % eta
Constant(19) = 0.0350;  % alpha
%----------------------
Constant(20) = 10;      % B1
Constant(21) = 15;      % B2
Constant(22) = 15;      % B3
Constant(23) = 10;      % C1
Constant(24) = 0.05;    % C2

%% Parameters
pi      = Constant(1);
phi     = Constant(2);
mu      = Constant(3);
beta    = Constant(4);
c       = Constant(5);
theta1  = Constant(6);
theta2  = Constant(7);
rho     = Constant(8);
omega   = Constant(9);
gamma1  = Constant(10);
gamma2  = Constant(11);
nu      = Constant(12);
tau     = Constant(13);
psi     = Constant(14);
sigma   = Constant(15);
delta   = Constant(16);
xi      = Constant(17);
eta     = Constant(18);
alpha   = Constant(19);
%---------------------
B1    = Constant(20);
B2    = Constant(21);
B3    = Constant(22);
C1    = Constant(23);
C2    = Constant(24);
```

```
lf  = [0 0 0 0 0 0];
%%
init = y0;
init2 =lf;
h = (tf-t0)/n;
u = linspace(0,0,n+1);
u1=u'; u2=u';
U = [u1 u2];

%% IMPLIMENTATION OF THE ALGORITHM
%Test 1 stoping condition 1
epsilon = 0.01;
X=init;
i=0; %Initialize iteration counter
mm=size(X);
NumXX =10e10;
Xnew = rand(n+1,mm(2)).*(repmat(X,n+1,1));
DenXnew=norm(Xnew);
while  NumXX/DenXnew>epsilon
Xold =  Xnew;
oldu = U;
%FORWARD RUNGE KUTTA  FOR STATES
[Tx, X]=rk4foward(@kims,t0, tf,n, init,U,Constant);

% BACKWARD RUNGEKUTA FOR COSTATES
[Tp, P]=rk4back(@kims_costate,t0,tf,n,init2,U,X,Constant);

%UPDATE THE CONTROLS
s = X(1,:); p = X(2,:); l = X(3,:); m = X(4,:); a = X(5,:);
 r=X(6,:);
L1 = P(1,:); L2 = P(2,:); L3 = P(3,:); L4 = P(4,:);
L5 = P(5,:); L6 = P(6,:);


%% Graph ploting

% Case3:u1/=0, u2/=0
u1 = max(0, min(1, beta.*c.*(l+theta1.*m+theta2.*a)...
.*(-L1+rho.*L3+(1-rho).*L4).*s./C1));
u2 = max(0, min(1, xi.*(L4-L6).*m./C2));


Uu=[u1' u2'];
U = 0.5*Uu + 0.5*oldu; % Convex combination of the controls


Xnew =  X';
```

```
NumXX =abs(norm(Xnew-Xold));
DenXnew =norm(Xnew);
i=i+1; %Update iteration counter
end
%% PLOTING
X=X';
Tx =Tx';
SS=X(:,1); PP=X(:,2); LL=X(:,3); MM=X(:,4); AA=X(:,5);
RR=X(:,6);

Up = [0 0];
[T,Y] = ode45(@kims,time,y0,[],Up,Constant);

J =sum((B1*LL(end)+B2*MM(end)+B3*AA)+((C1/2)*Uu(:,1).*...
Uu(:,1)+(C2/2)*Uu(:,2).*Uu(:,2)))

%% Averts
l1 =  sum(Y(:,3));
l2 =  sum(X(:,3));
avert_l=l1-l2;
%%
m1 =  sum(Y(:,4));
m2 =  sum(X(:,4));
avert_m = m1-m2;
%%
a1 =  sum(Y(:,5));
a2 =  sum(X(:,5));
avert_a = a1-a2;
Total_risk_averts = avert_l+avert_m + avert_a
%%
figure(1)
hold on
subplot(2,2,1)
plot(Tx,X(:,3),'-b',T, Y(:,3),'--r', 'LineWidth',1.5);
ylabel('{\it l(t)}');
xlabel({'Time (Years)','(a)'});
legend('u_1\neq0, u_2\neq0','u_1=u_2=0') %case3
grid on
hold on
%xlim([0 5]);
%%
subplot(2,2,2)
plot(Tx,X(:,4),'-b',T, Y(:,4),'--r', 'LineWidth',1.5);
ylabel('{\it m(t)}');
xlabel({'Time (Years)','(b)'});
legend('u_1\neq0, u_2\neq0','u_1=u_2=0') %case3
grid on
hold on
```

```
%%
subplot(2,2,3)
plot(Tx,X(:,5),'-b',T, Y(:,5),'--r', 'LineWidth',1.5);
ylabel('{\it a(t)}');
xlabel({'Time (Years)','(c)'});
legend('u_1\neq0, u_2\neq0','u_1=u_2=0') %case3
grid on
hold on
%%
subplot(2,2,4)
plot(Tx,Uu(:,1),'-b',Tx,Uu(:,2),'-r', 'LineWidth', 1.5);
ylabel('Control Profile');
xlabel({'Time (Years)','(d)'});
legend('u_1','u_2')
box
grid on
hold off
box
```

RESEARCH OUTPUT

Published papers

(i) **Mayengo, M. M.**, Shirima, G. M., Chakraverty, S., Kgosimore, M., Seshaiyer, P., & Caiseda, C. (2020). Mathematical modeling of the dynamics of health risks associated with alcoholism in Tanzania: a literature review. *Commun. Math. Biol. Neurosci.*, 2020, http://scik.org/index.php/cmbn/article/view/4749

(ii) **Mayengo, M. M.**, Kgosimore, M., & Chakraverty, S. (2020). Fuzzy Modeling for the Dynamics of Alcohol-Related Health Risks with Changing Behaviors via Cultural Beliefs. *Journal of Applied Mathematics*, 2020. https://doi.org/10.1155/2020/8470681

(iii) **Mayengo, M. M.**, Kgosimore, M., Chakraverty, S., Seshaiyer, P., Caiseda, C., & Shirima, G. (2020). Mathematical models for the dynamics of alcohol related health risks with changing behavior via cultural beliefs in Tanzania. *Commun. Math. Biol. Neurosci.*, 2020, http://scik.org/index.php/cmbn/article/view/4788.

Poster presentation

Author: **Mayengo, M. M.**, Shirima, G., Kgosimore, M., Seshaiyer, P. and Chakraverty, S.

Title: Fuzzy Modeling for the Dynamics of Alcohol-Related Health Risks with Changing Behaviors via Cultural Beliefs.

Available online at http://scik.org
Commun. Math. Biol. Neurosci. 2020, 2020:39
https://doi.org/10.28919/cmbn/4749
ISSN: 2052-2541

MATHEMATICAL MODELING OF THE DYNAMICS OF HEALTH RISKS ASSOCIATED WITH ALCOHOLISM IN TANZANIA: A LITERATURE REVIEW

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Abstract. In this paper, a number of alcohol related studies have been reviewed in light of the strength and challenges of the models developed for their applicability in Tanzanian drinking context. The aim of this paper is to provide a meaningful reference of the current state of the art regarding to modeling the health risks epidemics associated with alcoholic behavior in the community. It also discusses and identify current open problem reflecting on more realistic community and suggesting new research perspectives in the mathematical modeling of alcoholism and its related risks. The peer influence and social cultural practices appear to be two equally important influential aspects of model formulation with opposing effects on the spread of alcoholism. The study also reveals that, different levels of alcohol consumption has differing health effects. In this regard, the study proposes development of an advanced infection models to represent the influence of external motivations on the spreading of alcohol abuse by introducing different population segments with distinct exposition towards and resistance to the influence of drugs as an open relevant problem.

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Received June 3, 2020

Keywords: alcoholism; mathematical models; health risks.

2010 AMS Subject Classification: 93A30.

1. INTRODUCTION

Alcoholic beverages have long been used and considered as integral part of most cultures around the world [1, 2]. They have been used as part of entertainment by means of bringing people together in so many ways. Unfortunately, alcohol drinking is an addictive behavior in which a drinking individual accelerates to the state physical alcohol dependency after using the drink for sometimes without personal control initiatives. Unless stated otherwise, this state of addiction to the consumption of alcoholic drinks which eventually accelerate to alcohol dependency is referred to as alcoholism throughout this paper.

Alcohol drinking behaviors is one of the health risk factors for some lifestyle diseases [3, 4]. It is one among the major global risk factors in the Global Burden of Diseases (GBD) [4, 5]. Health challenges associated with alcoholism include malnutrition, chronic pancreatitis, liver cirrhosis, different types of cancer, and damage to the central and peripheral nervous system [2, 6, 7, 8, 9]. In support of this, Wiessing and collaborators identified social problems and crimes as direct and indirect indicators of prevalence of drugs and substance abuse in the community [10].

The effects of alcohol on human health and social well-being can be addressed in two perspectives: the health risks and injuries associated with alcohol use; and its extended impacts to the family and community through violence, and economy instability. Cancer and cardiovascular diseases (CVDs) combined contribute at least 40% of all alcohol-related deaths that accounting to more than one million deaths per year globally [6]. This statistics has continually been increasing from time to time. For instance, recently WHO [11] reported that, alcohol associated health risks resulting from harmful use of alcoholic beverages is responsible for three million deaths every year. This may be translated into six deaths occurring every single minute daily.

Alcoholism in the community may be influenced by several factors including; age, genetic disposition, ethnic groups, sex, mental health, social environment, and stress [8]. The key

determinants of alcohol related health risks are: the volume of alcohol consumed, the patterns of drinking and quality of alcohol consumed [2, 12].

Despite the health challenges emanating from alcoholism, most cultures promote drinking practices that are positive while discouraging those associated with adverse behaviors. This idea gets some literature supports in which many alcohol drinkers borrow a lief. According to Grönbaek [13], alcohol consumed at a desired level has some health benefits such as prevention of thrombosis. The study also identify alcohol as a source of income in rural communities [13]. However, the recent studies put this stand into challenges, it is finally established that there are health challenges regardless of the amount of alcohol consumed [4]. That is to say, there is no safe level of alcohol consumption which is free from health risks as it was believed for quite sometimes.

In Tanzanian settings, alcoholic beverages are available in terms of standard bottled beers, spirits, wines and locally made brews. Most of locally made illegal spirits are not freely sold in the market but they have been long existed in the free black markets in different communities. Since local brews are produced from the locally available ingredients, they are readily available at a comparatively lower prices than modern/standard beers and spirits, posing a greater health risks to low earned income population [14]. Although, the legally acceptable age for alcohol use in Tanzania is 18 years, it was observed that in many places of the country people of underage have access to alcohol especially the locally made brews and spirit [15]. This is supported by a recent survey conducted among secondary school students that revealed at least 64% had easy access to alcohol [15].

The drinking threshold to be referred to as an alcoholic is estimated to be a maximum of 21 standard bottles per week for men and 14 drinks per week for women [2, 12, 13]. In Tanzanian context, the standard bottle of a beer carries between 250mls to 500mls while spirits and wines takes different volumes depending on the packages applied. However, a very small fraction of the Tanzanian population are non-drinkers while majority endure in drinking. This implies that a broader public health message of the beneficial effects regarding to alcohol use does not seem to be interesting topic in most societies.

Different scholars have made an attempt to address the problem of alcoholism in different ways. Mathematical approach by means of model development, simulation and analysis regarding the question of alcohol epidemic have been employed in an attempt to provide insight on effects of alcohol consumption on health and socio-economic aspects of the society. The aim of this review paper is twofold: first is to provide a meaningful reference of the current state of the art regarding to modeling of health risks epidemic associated with alcoholism behavior in the community. Secondly, is to discuss and identify current open problems reflecting on more realistic community and suggesting new research perspectives in the mathematical modeling of alcoholism and its related risks.

2. THE DYNAMICS OF ALCOHOLISM

2.1. The effects of Religious beliefs. Religious beliefs have gained popularity in recent times over different parts of the world. It is one of the popular social cultural practices with such a greater influence which assumes the roles of the control agent of addictive substances including alcoholism. According to Koening and collaborators [16], religiosity has positive effects on both physical and mental health. This fact has propelled researches regarding to the direct or indirect roles of religious on health [18]. However, an increased number of individuals that are actively participating in their religious might have contributed to the increased interest of researching on the relationship between religious and health [17]. In the recent study [15], it was revealed that religiosity is one among the reasons for some people to abstain alcohol drinking. This was also true for some individuals who are non alcohol drinkers. Since the definition of religiosity is debatable, in this study, we borrow the definition of religiosity from [16], that is, any organized set of beliefs and measurable practices within a community of people who accept an authoritative doctrine. Based on the above literature, for the model to be realistic it is clear that religious beliefs and/or any other influential social cultural beliefs make an important component in the formulation of alcohol dynamic models.

2.2. The effects of peer influence. Peer influence has been closely associated with the spread of alcoholic behaviors in the community. Different cultures in Tanzania, for instance, praises alcohol drinking habits through cultural songs and other cultural practices. Available literature

reveals that, at least 64% of American adults consider themselves drinkers and 20% admitted to have occasionally over consumed alcohol. Over consumption and abuse of alcohol among American college and university communities is reported to be as higher as 33%. Peer influence and social pressure is also recorded as the main agent to the increase of the scenes of alcohol abuse in university communities [19].

Mundt and collaborators [20], formulated and analyzed a stochastic actor - based model for peer selection and influence effects on adolescent alcohol use with the aim of disentangling selection associated with the dynamic interplay of adolescent friendship and alcohol use. In this study, they analyzed data from Add Health, a longitudinal survey of selected students enrolled between 1995 and 1996 in the U.S. The study findings revealed that, peer selection plays a major role in alcohol use behavior among adolescent friends.

Walsh and collaborators [21] examined parental, peer, and school predictors of alcohol drinking among Israeli−born adolescents and first and second generation adolescent immigrants from the Former Soviet Union (FSU) and Ethiopia in Israel. In this study, they used Pearson's Chi-Square and logistic regression models to examined the differences between the groups for drinking and group specific predictors of drinking respectively. The influence of both, parents through parenting and peer pressure measured by the amount of time spent together, play greater roles in shaping individuals drinking behaviors. For example, the study found out that, first generation FSU and both Ethiopian groups had greater time with friends with lower levels of parental monitoring and they reported to have higher levels of binge drinking and drunkenness than Israeli - born adolescents. It reveals further that, time spent with peers consistently predicted immigrant alcohol use. It was suggested further that, drinking patterns must be understood in relation to country of origin and immigration experience of a particular group.

A recent study conducted in Tanzania reveals that, most of alcoholic drinkers had their first drinking experience at the social event [15]. This experience in the initiation stage may be closely associated to the influence of peer pressures and provision of free and easy access to the alcoholic beverages in an exciting moments. It was reported further that, between 31% - 66% of the drinkers population took the standard bottled beers during their first time drinking while 36% - 45% of the school girls had the locally made brews as their first alcoholic drink

[15]. This implies that exposure to drinking context is yet another important pulling factor to alcoholic behaviors. In Tanzania and many other African countries, local brewery activities are performed by women with the assistance from their daughters. The practice will automatically set good exposure for girls to the drinking context than the case may be in the boys.

Generally, drinking habits at younger ages, is unacceptable behavior in many communities. For example, the study conducted by DGC in 2007 reported that, at least 76% of the European population were against alcohol advertisements targeted the young citizens in all member states [22]. It is therefore important to consider peer influence in the model formulation. The peer influence inters in the system as a changing agent in molding individuals behavior towards alcohol drinking.

2.3. Application of the theory of reasoned action (TRA). Different studies have indirectly proved that alcohol drinking habit obeys the theory of reasoned action (TRA) rooted in Socio - psychology. TRA explains how and why individual's attitude influences behavior. Darwin [29] is considered to be the father of this phenomenon, by pioneering the study about the attitude towards behavior. The study defines the attitude as the expression of physical and emotion. The great psychologist later looked at attitude as the source of emotion of cognitive with the behavior component, both verbal or non verbal [30].

The theory of reasoned action or behavior is applicable to many kinds of behaviors today, especially in socio-psychology. It focus on intention variable determined by the attitude, subjective norm and other important norms. The individual's behavior is determined by attitude toward behavioral outcome and public opinions on the behavior. According to the theory, every behavior is influenced by intention which is the function of attitude of behavior and subjective norm [31]. A behavior, for that matter, is the transition of intention into an action or practice. On another hand, attitude towards a behavior is result of thorough assessment made by an individual regarding to the belief about cost and benefits of the behavior and its consequences. Similarly, subjective norm is influenced by the the combination of personal belief towards public opinions, and the motivation to conform to the group norms.

Regarding to alcohol drinking behavior, no one drinks alcohol accidentally. An individual will consciously consume alcohol whenever expectations of getting positive consequences of

drinking outweigh those of not drinking, its converse is also true [32]. An individual's decision on whether or not to take an alcoholic drink is determined by various factors including, but not limited to, past experiences with drinking, current life situation. These factors, modulated by individual's neurochemical reactivity to alcohol, will help to form expectations of effective change from drinking alcohol [32]. However, parental influence over individual's behavior may not go unappreciated. For instance, in survey conducted by Francis and collaborators [15], parental influence was mentioned as one among the main reasons some people did not engage in the drinking behaviors. That, if alcohol drinking is not something the parents feel proud of, then no one under their guidance would be expected to drink while hunting the approval of the parents. The same is true for other behaviors. This stand is also backed up by [21] in which parenting and peer pressure proved to be very influential towards behavioral change. People tend to learn or unlearn different behaviors from different individuals around them, especially those who deserve their respect befitting a role model.

3. MODELING BASICS

In epidemiological studies, transmission of infectious agents in the host population is key process deserving the descriptive analysis when the model compartments is used to study a particular infectious disease [33]. Mathematical models may be extended to describe behavioral dynamics and transmission where people already in the behavior may act as transmission agent in the host population provided a desired amount of interactions between them is allowed. When a behavior associated with health risk factors emerge in any community, the total population in the community can be partitioned into a number of categories depending on the risk levels or defined patterns individuals exhibit. Mathematical modeling of alcohol drinking epidemic and its consequences on human health has been an interesting topic for many researchers. The similarity between the spreading nature of alcoholic behavior and that of infectious diseases has attracted modelers to use mathematical modeling as an essential tool for simulating the behavior and provide valuable control analysis. Most models developed in relation to alcoholism and its consequences fall in the category of SIR with or without significant modification.

In both studies Bhunu [8], and Huo & Liu [9] separately, used an extension of the basic *SIR* model approach to model the spread of alcoholism in the community. While remaining

conventional on *SIR* model, Bhunu [8] considered deterministic models by splitting alcoholic population into two classes based on different consumption levels (see system (1)). According to Bhunu [8], the following model reflects the problem of alcoholism epidemic

$$\begin{aligned} S'(t) &= \Lambda - (\lambda + \mu) S \\ D'(t) &= \lambda S - (\mu + \rho + \gamma) D \\ A'(t) &= \rho D - (\mu + \nu + \delta) A + \sigma R \\ R'(t) &= \gamma D + \delta A - (\gamma + \mu) R \end{aligned} \tag{1}$$

with $\lambda = \dfrac{\beta c (D + \theta A)}{N}$ and non-negative associated parameters and state variables at all times. The study aimed at gaining insight on the growth of alcoholism as a health and social problem. Analytical and numerical methods were used for analysis of the model. It was found out that it is easy for moderate drinkers to quit alcohol drinking than alcoholics. Thus, any effort geared to encourage and support moderate drinkers to quit drinking will be effective that using the same efforts to alcoholic population.

Two years later, Wang and collaborators [35] authored a different study with deterministic *SATQ* type mathematical model to investigate the optimal control strategies in alcoholism. This is another *SIR* like model with slight modification to accommodate treatment intervention. In this study, using the model system (2), the spread of alcoholism is studied with two control strategies u_1, and u_2, to gain insights about health and social phenomenon. The following is the model system presented in the said study.

$$\begin{aligned} S'(t) &= \mu N - (1 - u_1) \frac{\beta S A}{N} - \mu S \\ A'(t) &= (1 - u_1) \frac{\beta S A}{N} + \xi T - (u_2 + \mu) A \\ T'(t) &= u_2 A - (\mu + \xi + \delta) T \\ Q'(t) &= \delta T - \mu Q \end{aligned} \tag{2}$$

where $0 \leq u_i \leq 1$, $\{i = 1, 2\}$. It considered the closed environment with the total population in four compartments: the susceptible compartment, $S(t)$, with individuals who either do not drink or drink moderately without affecting the physical health; the alcoholism compartment, $A(t)$, with individuals who binge drink and affect the physical health seriously; the treatment compartment, $T(t)$, with individuals who have been receiving alcohol related treatments after

alcoholism; and the quitting compartment, $Q(t)$, which refers to the individuals who recover from alcoholism after treatment and stay off alcohol hereafter. However, grouping moderate drinkers population and susceptible population together is challenged by the recently published scientific study affirming that alcohol consumption at whatever level poses health challenges and that there is no healthy drinking of alcohol [4]. Just like in Bhunu [8], this study also ignored the contribution of the social cultural practices in the control of alcoholism problem in the community.

In a different study, Huo & Liu [9] considered a relapse alcoholic model on weighted network by dividing the total population into susceptible, S; infections, I; and recovery, R; making a simple *SIRS* model. In that regard, the following model were formulated and analyzed

$$\begin{aligned} S_k'(t) &= b\left(1-S_k(t)-I_k(t)-R_k(t)\right)S - kS_k(t)\Theta(t) + \sigma R_k(t) - \mu S_k(t) \\ I_k'(t) &= kS_k(t)\Theta(t) + \beta R_k(t) - \alpha I_k(t) - \mu I_k(t) \\ R_k'(t) &= \alpha I_k(t) - \beta R_k(t) - \sigma R_k(t) - \mu R_k(t) \end{aligned} \tag{3}$$

with initial conditions

$$\Omega^* = \left\{(S_k(t), I_k(t), R_k(t)) \in \mathbb{R}_+^{3n} | 0 \le S_k(t) \le 1, 0 \le I_k(t) \le 1, 0 \le R_k(t) \le 1, k = 1,2,\ldots,n\right\}$$

and

$$\Theta(t) = \sum_i \lambda_{ik} \frac{\varphi(i)}{i} P(i|k)\, I_i(t).$$

The model system (3) was used to study the peer influence on individual's drinking dynamics. The analysis results looked at interaction between susceptible and alcoholism, and reculperator recurrence drinking alcohol as the determinant of alcoholic problem. Thus, reducing the sequence of interaction between susceptible and alcoholism, and stopping reculperator recurrence drinking alcohol may be an effective control to eliminate alcoholic problem in the community.

While both, Bhunu & Huo suggested some workable intervention programs, they did not seem to have considered the effects of social cultural beliefs which plays an influencing roles in molding peoples' behaviors [15, 34]. Also, they partially agree with the multilevel risk of alcohol consumption and its relationship between health and social outcomes suggested by Jernigan [3]. The multilevel risks of alcohol consumption can be meaningful when extended beyond two classes.

In Mushayabasa [36], an *SIR* like mathematical model to investigate the role of optimal intervention strategies on controlling excessive alcohol drinking and its related adverse health effects in the community was proposed and analyzed. The study considered the transmission process as the social contract between heavy and light alcohol consumers within an unchanging shared drinking context. Two models − an autonomous system with constant parameters in relevant alcohol drinking components and alcohol related treatment model − incorporating peer influence were proposed and analyzed qualitatively. The study considers the model with the total drinking population in four different population compartments depending on individuals' alcohol consumption level as follows.

$$\begin{aligned} S'(t) &= \mu N - g(H)\frac{S}{N} - \mu S + (1-p)\,\psi A \\ H'(t) &= g(H)\frac{S}{N} - (\phi + \varepsilon + \mu)\,H \\ A'(t) &= (1-f)\,\phi H - (\psi + \mu)A \\ R'(t) &= f\phi H + p\phi A - \mu R \end{aligned} \tag{4}$$

Susceptible, $S(t)$, contains light alcohol consumers; heavy, $H(t)$, contains heavy alcohol consumers; $A(t)$ contains individuals receiving alcohol related treatment and consume alcohol occasionally; and recovered, $R(t)$, which contains both individuals on treatments and those who have successfully completed treatment and permanently quit alcohol consumption. In his study, optimal control results suggested that, effective control of high-risk alcohol drinking can be achieved if more resources and efforts are devoted on weakening the intensity of social interactions between light and heavy drinkers. It was suggested further that, time dependent interventions have the potential to eliminate the problem of excessive alcohol use. Having alcoholic population in three classes depending on their consumption level answers the multilevel risks challenges appeared in the previous literature. However, the study considered only drinkers population and left the non drinkers population out of the system. As observed in the previous literature, the study also ignored the contribution on social cultural beliefs in controlling the problem.

Xiang and collaborators [37] worked on global property of a drinking model with the public health campaigns. To describe the problem, the drinking population is subdivided into five groups: susceptible drinkers, $S(t)$, who moderately consume alcohol but may develop alcohol

related problems and refuses public health education; educated drinkers, $E(t)$, who consume alcohol in moderation and accept the public health education; alcoholics, $A(t)$, who have drinking problems or addictions; and temporarily recovered drinkers, $R(t)$, former alcoholics who have entered treatment and are abstaining from alcohol; quit drinkers, $Q(t)$, who permanently quit drinking. The following model was established

$$\begin{aligned} S'(t) &= q\mu\Lambda - \beta SA - \beta SA + (p+\mu)S \\ E'(t) &= (1-q)\mu\Lambda + pS - \beta\sigma EA - (\mu+\varepsilon)E \\ A'(t) &= \beta SA + \beta\sigma EA + \delta R - (\mu + a_1 + \gamma)A \\ R'(t) &= \gamma A - (\delta + \xi + \mu + a_2)R \\ Q'(t) &= \xi R + \varepsilon E - \mu Q \end{aligned} \tag{5}$$

With the help of Lyapunov function, global stability of equilibria of the model system (5) is derived. The basic reproduction number, $\mathscr{R}_0$, ware obtained by means of the next generation matrix and the global stability of model has been proved by using the Lyapunov function. The study analysis revealed that, the public health educational campaigns of drinking individuals can slow down the drinking dynamics. Some numerical simulations are also used to support this conclusions. Again this work is not free from the challenges of ignoring the influence of social cultural beliefs existing in the society.

A non - linear $SHTR$ model [38] studied the dynamics of drinking epidemics where the susceptible compartment (S) of the model took non drinkers population and the compartment (H) recruited heavy alcohol drinkers with no considerations of the light alcohol drinkers.

$$\begin{aligned} S'(t) &= b - \alpha SH - \mu S + \eta R \\ H'(t) &= \alpha SH - (\mu + \delta_1 + \phi)H \\ T'(t) &= \phi H - (\mu + \delta_2 + \gamma)T \\ R'(t) &= \gamma T - (\mu + \eta)R \end{aligned} \tag{6}$$

with initial conditions $S(0) \geq 0, H(0) \geq 0, T(0) \geq 0$, and $R(0) \geq 0$. In this study, the conditions for existence and stability of drinking free and endemic equilibria points were established by using Lassalle's invariance principle of Lyapunov function. Analytical results were later confirmed by some numerical simulations putting forward three useful methods of combating the drinking epidemics. These include: reducing the contact rate between non drinkers and heavy

drinkers; increasing the number of drinkers that go into treatment; and educating to refrain from drinking. The authors also seem to have generalized the concept of alcoholism without considering multilevel risks phenomenon based on one's drinking patterns and frequency.

A risk - structured model for the spread of drug abuse presented by system (7) was used to estimate an epidemic threshold value (abuse reproduction number) [39].

$$
\begin{aligned}
S_H'(t) &= \rho\Lambda - \frac{\beta_1 U S_H}{N} - (\mu+\omega_1) S_H + \omega_2 S_L \\
S_L'(t) &= (1-\rho)\Lambda - \frac{\eta_1\beta_1 U S_L}{N} - (\mu+\omega_2) S_L + \omega_1 S_H,\ 0<\eta_1<1 \\
U'(t) &= \frac{\beta_1 U (S_H+\eta_1 S_L)}{N} + \frac{\beta_2 U (R_H+\eta_2 R_L)}{N} - (\mu+\sigma+\delta+\rho+\rho_h+\rho_l) U \\
T'(t) &= \sigma U - (\mu+\gamma_h+\gamma_l) T \\
R_H'(t) &= \gamma_h T + \rho_h U - \frac{\beta_2 U R_H}{N} - (\mu+\varepsilon_1) R_H + \varepsilon_2 R_L \\
R_L'(t) &= \gamma_l T + \rho_l U - \frac{\eta_2\beta_2 U R_L}{N} - (\mu+\varepsilon_2) R_L + \varepsilon_1 R_H,\ 0<\eta_2<1
\end{aligned} \tag{7}
$$

with initial conditions $S_H(0)=S_{H0}>0, S_L(0)=S_{L0}>0, U(0)=U_0\geq 0, T(0)=T_0\geq 0, R_H(0)=R_{H0}\geq 0, R_L(0)=R_{L0}\geq 0$ and positive model parameters. In this complex model, the question of multilevel risks has been addressed and the model solution revealed that public health education on skills to handle the risky situation may be the best approach to protect individuals from initiating or re-initiating into drug abuse. However, just like any other previous studies, the study did not consider the importance of social cultural influences in molding human behavior.

4. Discussion and Conclusion

In light of the above literature, it is clear that mathematical modeling approach towards solving different problems have continually attracted most researchers. This is also true for health problems and its associated risks as the consequences of alcoholism. However, the field of drugs abuse and substance addiction like other fields is not without challenges. Based on the fact that, the field of drug abuse and addiction largely depend on behavioral criteria than physical and biological personalities: defining, quantifying and measuring the phenomena of studies of drug abuse has been recorded as one among the great challenges facing the drug abuse epidemiologists [40].

It is well known that even the best models have some imperfections in one way or another [41]. It is therefore utmost important to continue reviewing and improving the models available

from time to time, in order to increase their usefulness. Motivated by above literature, the studies reviewed provide the stepping stone towards developing a more relevant model. In light of the above literature, the open problem which comes out clearly includes: developing a complex and more relevant models the dynamics of health related risks associated with alcoholism and its control strategy addressing the main aspects raised above as the challenge to most of the studies. Such models are expected to take into account the influence of external motivations on the spreading of alcohol abuse by introducing different population segments with distinct expositions, towards and resistance to the influence of drugs.

Since different levels of alcohol consumption has different health effects, alcohol drinking population is anticipated to be considered in three categories depending on their alcohol drinking habits as follows;

(1) **Low risk population:** involving the population proportion who drink alcohol once in while mainly when they are involved in some special events, they are also known as occasional drinkers.

(2) **Moderate risk population:** involving the population proportion who drink alcohol on regular basis but have not developed symptoms of alcohol dependency.

(3) **High risk population:** these include the population proportion who consume alcohol frequently and at large amount, they may or may not have developed symptoms of alcohol dependency but they have crossed the threshold of alcoholism.

Inclusion of the influence of external motivations with positive and negative influential effects, and a clearly defined alcohol drinking population will make this study even more peculiar and it will distinguish itself from most of the models developed over the subject.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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Hindawi
Journal of Applied Mathematics
Volume 2020, Article ID 8470681, 9 pages
https://doi.org/10.1155/2020/8470681

Research Article

Fuzzy Modeling for the Dynamics of Alcohol-Related Health Risks with Changing Behaviors via Cultural Beliefs

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Academic Editor: Chongxin Liu

In this paper, we propose and analyze a fuzzy model for the health risk challenges associated with alcoholism. The fuzziness gets into the system by assuming uncertainty condition in the measure of influence of the risky individual and the additional death rate. Specifically, the fuzzy numbers are defined functions of the degree of peer influence of a susceptible individual into drinking behavior. The fuzzy basic risk reproduction number $\mathscr{R}_0^f$ is computed by means of Next-Generation Matrix and analyzed. The analysis of $\mathscr{R}_0^f$ reveals that health risk associated with alcoholism can be effectively controlled by raising the resistance of susceptible individuals and consequently reducing their chances of initiation of drinking behavior. When perceived respectable individuals in the communities are involved in health education campaign, the public awareness about prevailing risks increases rapidly. Consequently, a large population proportion will gain protection from initiation of drinks which would accelerate their health condition into more risky states. In a situation where peer influence is low, the health risks are likely to be reduced by natural factors that provide virtual protection from alcoholism. However, when the perceived most influential people in the community engage in alcoholism behavior, it implies an increase in the force of influence, and as such, the system will be endemic.

1. Introduction

Alcohol consumption that leads to health-related epidemics has been one of the leading causes of mortality of individuals worldwide. In Tanzania, just like in other developing countries, the situation gets worse as alcohol consumption becomes an important part of community social activities. However, available literature cite alcohol consumption behavior is one among the risk factors for various health challenges in Tanzanian population [1–9]. While communities identify themselves with alcohol consumption, a large part of the religious population forbid its uptake. Being religious therefore plays an important role in promoting health and molding an individual's behaviors [10, 11]. Increased health defect cases such as malnutrition, chronic pancreatitis diseases, liver cirrhosis, and some types of cancer have been found to be associated to alcoholic behavior [12–19]. While in some parts of the world individuals believed that a low level of alcohol consumption prevents thrombosis [20], recent studies challenge the belief. For example, Griswold et al. [13] established that any amount of alcohol consumption leads to health risks.

Mathematical modeling has been very useful in providing solutions to epidemiology-related problems for both autonomous and nonautonomous system models [12, 14, 15, 21–29]. While autonomous systems literally mean the use of constant controls, a nonautonomous system occurs when a continuous variable control is used in the system as a

function of time [22]. However, an application of fuzzy set theory in solving dynamical systems has recently been an interesting research area [30–33].

In this paper, we use an application of fuzzy set theory approach to modify the basic *SPLMAR* model developed in system (1) into a fuzzy model. Two main distinguishing aspects of the model are as follows: social cultural beliefs as an integral part of the society [34–36] and the staged process in which alcoholism behaviors take in the spread of health risks [12, 14, 15, 24, 26, 29] are considered.

2. Fuzzy Model Formulation

This paper presents crisp model (1) with six time-dependent state variables described as follows: susceptible population, $S(t)$—comprising of individuals susceptible to alcohol drinking habits; protected population, $P(t)$—comprising of individuals with strong religious beliefs that provide them with virtual protection from alcohol drinking; and low-risk population, $L(t)$—individuals at low risk who drink alcoholic beverages once in a while. Others include medium/moderate-risk population, $M(t)$—individuals at medium/moderate risk who drink alcoholic beverages regularly; alcoholics, $A(t)$—high-risk drinkers or alcohol addicts who have developed physical or psychological dependence on alcoholic beverages; and recovered population, $R(t)$—former drinkers who have voluntarily quit alcohol drinking due to various reasons. The model considers multirisk levels established under the drinking cultures of Tanzanian population with active religious beliefs by using constant model parameters defined in Table 1

$$\begin{cases} \dot{S} = -(\mu + \gamma_1 + \lambda)S + \gamma_2 P + \omega R + (1-\phi)\pi N, \\ \dot{P} = \phi\pi N + \gamma_1 S - (\mu + \gamma_2)P + \nu L + \tau M + \psi A, \\ \dot{L} = \lambda\rho S - (\nu + \mu + \sigma)L, \\ \dot{M} = \lambda(1-\rho)S + \sigma L - (\tau + \mu + \delta + \xi)M, \\ \dot{A} = \delta M - (\mu + \alpha + \eta + \psi)A, \\ \dot{R} = \xi M + \eta A - (\omega + \mu)R, \end{cases} \tag{1}$$

with $S(0) > 0, P(0) > 0, L(0) \geq 0, M(0) \geq 0, A(0) \geq 0, R(0) \geq 0$, and $N = S + P + L + M + A + R$.

The model is developed on the fact that a nonalcoholic drinker acquires alcohol drinking habits through regular social contacts [12, 37] at a rate of λ defined as

$$\lambda = c\beta\left(\frac{L + \theta_1 M + \theta_2 A}{N}\right). \tag{2}$$

We propose a fuzzy model using x as the variable describing the degree of peer influence of a susceptible individual to initiate drinking behavior. The model development is made under the assumption that both β and α are the functions of x. The physical meaning of parameters used in the model determined the choice of the two parameters. While β is attributed to the spread of health risks associated with alcoholism in the community, α translates the consequences of health risks by means of additional death rate. The fuzzy numbers $\beta = \beta(x)$ and $\alpha = \alpha(x)$ represent the likelihood that a susceptible individual will drink alcohol after prolonged contact with drinking individuals and the additional death rate induced by alcoholism, respectively. A fuzzy number may be defined as a generalization of a real number referring to a connected set of possible values, rather than referring to one single value, where each possible value is weighted between 0 and 1. Thus, the fuzzy system (3) of differential equations is established

$$\begin{cases} \dot{S} = (1-\phi)\pi N - (\mu + \gamma_1 + \lambda(x))S + \gamma_2 P + \omega R, \\ \dot{P} = \phi\pi N + \gamma_1 S - (\mu + \gamma_2)P + \nu L + \tau M + \psi A, \\ \dot{L} = \lambda(x)\rho S - (\nu + \mu + \sigma)L, \\ \dot{M} = \lambda(x)(1-\rho)S + \sigma L - (\tau + \mu + \delta + \xi)M, \\ \dot{A} = \delta M - (\mu + \alpha(x) + \eta + \psi)A, \\ \dot{R} = \xi M + \eta A - (\omega + \mu)R, \end{cases} \tag{3}$$

Table 1: Model parameters and their description.

Symbol	Descriptions
π	Per capita recruitment rate
φ	The proportion of recruitment joining the protected population
μ	Natural mortality rate
λ	Force of peer influence to induce drinking
β	The measure of influence of the risky individuals
c	The contact rate between a susceptible member and a drinker necessary to convince the susceptible member to drink
θ_1	The chances of becoming an alcoholic after successful influence of a moderate-risk drinker
θ_2	The chances of becoming an alcoholic after successful influence of a high-risk drinker
ρ	The proportion of susceptible individual recruited to the low-risk drinking population
ω	The rate at which recovered individuals join susceptible compartment
γ_1	The rate at which susceptible population joins protected compartment
γ_2	Virtual protection wane rate
ν	The rate at which low-risk population joins protected compartment
τ	The rate at which moderate-risk population joins protected compartment
ψ	The rate at which high-risk population joins protected compartment
σ	Progressive rate from low to moderate risk compartments
δ	Progressive rate from moderate- to high-risk compartments
ξ	Recovery rate for moderate-risk population
α	Alcohol-induced fatality rate
η	Recovery rate for high-risk population

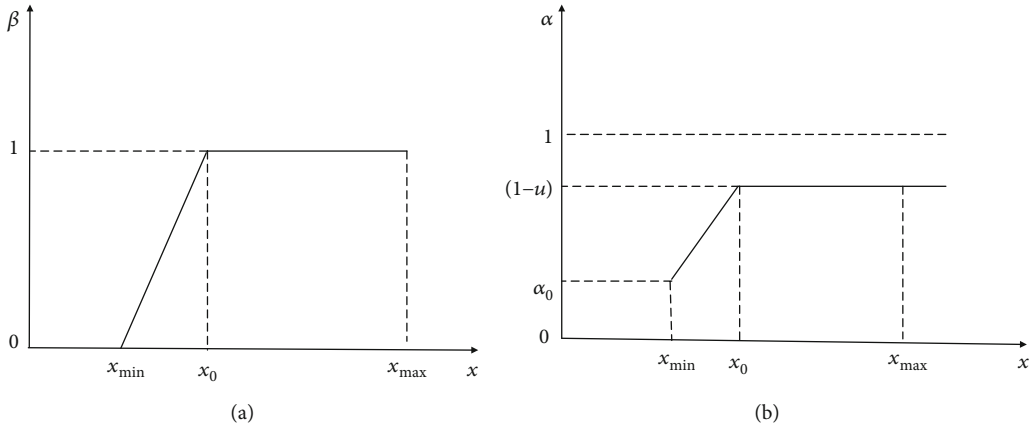

FIGURE 1: The graph of membership functions β and α.

with nonnegative initial conditions of the state variables: $S(0) > 0$, $P(0) \geq 0$, $L(0) \geq 0$, $M(0) \geq 0$, $A(0) \geq 0$ and $R(0) \geq 0$ where

$$\begin{aligned} N &= S + P + L + M + A + R, \\ \dot{N} &= (\pi - \mu)N - \alpha(x)A. \end{aligned} \tag{4}$$

It is further assumed that the population is homogeneous and, at the initial level, drinking behavior is acquired by choice. Further, it is assumed that individuals in the recovered population do not develop a permanent immunity system against alcohol drinking. Similarly, individuals in the protected compartment acquire a nonpermanent virtual protection from alcohol drinking for their entire life in the compartment. On the other hand, a susceptible drinker acquires alcohol drinking habits through prolonged social contacts with drinkers in shared drinking venues. The force of peer influence is redefined into

$$\lambda(x) = c\beta(x)\left(\frac{L + \theta_1 M + \theta_2 A}{N}\right). \tag{5}$$

3. Analysis of the Fuzzy System

Following Verma et al. [38] and Nandi et al. [32], we study the fuzzy *SPLMAR* model (3) and provide the model analysis and interpretations. The parameters $\beta(x)$ and $\alpha(x)$ can be described through two fuzzy membership functions. For example, considering a situation where a drinking individual interacts with a susceptible member, the minimum amount of the degree of peer influence, $x = x_{\min}$, is required to have an impact on a susceptible member. That is to say, when $x < x_{\min}$, the impact of behavioral influence is considered negligible. The quantity $x_{\min}$ is taken as a parameter whose exact value would depend upon both the attitude, public opinions towards the drinking behavior or the drinking individual, and willingness of a susceptible individual to conform with the peer pressures. As x increases, the behavior inducement rate increases to the maximum which is equal to unity at $x \geq x_0$. Further, it is also assumed that the degree of peer influence is bounded above where $x = x_{\max}$ marks an upper bound. Therefore, the values of x with an effect to the system lies in the interval of $x_{\min} \leq x \leq x_{\max}$. The fuzzy membership function for the fuzzy number $\beta(x)$ is given by

$$\beta(x) = \begin{cases} 0, & \text{if } x < x_{\min}, \\ \dfrac{x - x_{\min}}{x_0 - x_{\min}}, & \text{if } x_{\min} \leq x \leq x_0, \\ 1, & \text{if } x_0 < x < x_{\max}. \end{cases} \tag{6}$$

Similarly, we also assume that the addition death rate, $\alpha(x)$, is a fuzzy number as it occurs due to the risk "transmission." When $x < x_{\min}$, we have a negligible amount of risk transmissions implying that the additional death rate, $\alpha(x)$, gets to the minimum level, say $\alpha(x < x_{\min}) = \alpha_0$. As x increases, the additional death rate increases and gets the highest value at $x = x_0$. Since the additional death rate may not reach $\alpha(x) = 1$ as its highest score due to several limitations, we let the maximum value $\alpha(x) = (1 - u)$ for some real number u such that $0 < u < (1 - \alpha_0)$. We therefore establish the fuzzy membership function of $\alpha(x)$ as follows:

$$\alpha(x) = \begin{cases} \alpha_0, & \text{if } 0 \leq x < x_{\min}, \\ \alpha_0 + \left(\dfrac{1 - u - \alpha_0}{x_0 - x_{\min}}\right)x, & \text{if } x_{\min} \leq x < x_0, \\ (1 - u), & \text{if } x_0 \leq x \leq x_{\max}. \end{cases} \tag{7}$$

The graphs of membership functions $\beta(x)$ and $\alpha(x)$ are presented in Figures 1(a) and 1(b), respectively. For the model to be more realistic, we assume that the degree of peer influence of the studied group Π differs from among drinking individuals depending on their social influence in the community. Consequently, Π can be considered a linguistic variable with varying classifications. If we use x_c and d as, respectively, the central value and dispersion of each one of the fuzzy sets assumed by Π, we model each classification by using a triangular fuzzy number whose membership

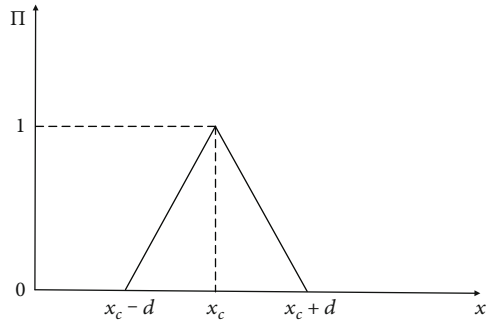

Figure 2: The graph of membership function Π.

function is given in (8) and the graph of membership function Π is presented in Figure 2.

$$\prod(x) = \begin{cases} 0, & \text{if } x < x_c - d, \\ \dfrac{x - x_c + d}{d}, & \text{if } x_c - d \le x \le x_c, \\ -\dfrac{x - x_c - d}{d}, & \text{if } x_c < x \le x_c + d, \\ 0, & \text{if } x_c + d < x. \end{cases} \tag{8}$$

Let $L(x,t)$, $M(x,t)$, and $A(x,t)$ be the family solutions of the fuzzy model system (3). These are the numbers of risky population proportions created as the result of social interactions between the susceptible members and risky individuals with social influence x at time t. Now, $L(x,t)$, $M(x,t)$, and $A(x,t)$ are fuzzy numbers which lie in the interval $[0,1]$.

3.1. Existence of Equilibrium Points. The equilibrium points of the fuzzy model (3) can be obtained by setting the RHS to be equal to zero and solve the system simultaneously. Thus, we have

$$\begin{cases} 0 = (1-\phi)\pi N^* - (\kappa_1 + \lambda^*(x))S^* + \gamma_2 P^* + \omega R^*, \\ 0 = \phi\pi N^* + \gamma_1 S^* - \kappa_2 P^* + \nu L^* + \tau M^* + \psi A^*, \\ 0 = \lambda^*(x)\rho S^* - \kappa_3 L^*, \\ 0 = \lambda^*(x)(1-\rho)S^* + \sigma L^* - \kappa_4 M^*, \\ 0 = \delta M^* - (\kappa_5 + \alpha(x))A^*, \\ 0 = \xi M^* + \eta A^* - \kappa_6 R^*, \end{cases} \tag{9}$$

where $\kappa_1 = (\mu + \gamma_1)$, $\kappa_2 = (\mu + \gamma_2)$, $\kappa_3 = (\nu + \mu + \sigma)$, $\kappa_4 = (\tau + \mu + \delta + \xi)$, $\kappa_5 = (\mu + \eta + \psi)$, and $\kappa_6 = (\omega + \mu)$ are simplifying parameters.

The risk-free equilibrium (RFE) point (ε_0) is obtained when the entire population is free from health risks associated with alcoholism, that is, $L = M = A = 0$ implying that $\lambda(x) = 0$. Solving system (9) by maintaining risky classes at zero, we have

$$\varepsilon_0 = (S_0^*, P_0^*, L_0^*, M_0^*, A_0^*, R_0^*) = ((1-\kappa)N_0^*, \kappa N_0^*, 0, 0, 0, 0), \tag{10}$$

with the positive constant $\kappa = (\pi(\phi\mu + \gamma_1))/(\mu(\mu + \gamma_1 + \gamma_2))$.

Similarly, when L=0, M=0, A=0, and consequently $\lambda^*(x)$=0, the risk endemic equilibrium REE point (ε_1) (11) is established.

$$\varepsilon_1 = (S^*, P^*, L^*, M^*, A^*, R^*), \tag{11}$$

where by P^*, L^*, M^*, A^*, and R^* are defined in

$$P^* = \frac{1}{\gamma_2}((\kappa_1 + (1 - \omega Q_3)\lambda^*(x))S^* - (1-\phi)\pi N^*), \tag{12}$$

$$L^* = Q_0\lambda^*(x)S^*, \tag{13}$$

$$M^* = Q_1\lambda^*(x)S^*, \tag{14}$$

$$A^* = Q_2\lambda^*(x)S^*, \tag{15}$$

$$R^* = Q_3\lambda^*(x)S^*, \tag{16}$$

and $Q_0 = \rho/\kappa_3$, $Q_1 = (\kappa_3 - \rho(\nu + \mu))/\kappa_3\kappa_4$, $Q_2 = ((\kappa_3 - \rho(\nu + \mu))\delta)/(\kappa_3\kappa_4(\kappa_5 + \alpha(x)))$, and $Q_3 = ((\xi(\kappa_5 + \alpha(x)) + \eta\delta)(\kappa_3 - \rho(\nu + \mu)))/(\kappa_3\kappa_4(\kappa_5 + \alpha(x))\kappa_6)$ are the simplifying factors. Thus, the Theorem 1 is established.

Theorem 1. *System (3) has a risk-free equilibrium* $\varepsilon_0 = ((1-\kappa)N_0^*, \kappa N_0^*, 0, 0, 0, 0)$ *and a unique risk endemic equilibrium* $\varepsilon_1 = (S^*, P^*, L^*, M^*, A^*, R^*)$.

3.2. Bifurcation and Fuzzy Basic Risk Reproduction Number. In this section, we first compute the basic health risk reproduction number, denoted as $\mathscr{R}_0$ by using the Next-Generation Matrix method [39, 40]. The value of $\mathscr{R}_0$ serves as the determinant to indicate whether the health risk epidemic is possible or not. Based on Diekmann et al.'s study [39], the risk transmission model consists of nonnegative initial conditions together with the following system of equations:

$$\dot{Z} = \mathscr{F}(Z) - \mathscr{V}(Z) = \begin{bmatrix} \lambda(x)\rho S \\ \lambda(x)(1-\rho)S \\ 0 \end{bmatrix} - \begin{bmatrix} \kappa_3 L \\ -\sigma L + \kappa_4 M \\ -\delta M + (\kappa_5 + \alpha(x))A \end{bmatrix}, \tag{17}$$

where $\mathscr{V}(Z) = \mathscr{V}^-(Z) - \mathscr{V}^+(Z)$, $X = \{S, P, R\}^T \in \mathbb{R}^3$, $Z = \{L, M, A\}^T \in \mathbb{R}^3$, and $(\cdot)^T$ denotes transpose.

We then formulate the risk "*transmissions*" matrix F and risk "*transitions*" matrix V such that

$$F = (1-\kappa)\begin{bmatrix} c\beta(x)\rho & c\beta(x)\theta_1\rho & c\beta(x)\theta_2\rho \\ c\beta(x)(1-\rho) & c\beta(x)\theta_1(1-\rho) & c\beta(x)\theta_2(1-\rho) \\ 0 & 0 & 0 \end{bmatrix},$$
$$V = \begin{bmatrix} \kappa_3 & 0 & 0 \\ -\sigma & \kappa_4 & 0 \\ 0 & -\delta & \kappa_5+\alpha(x) \end{bmatrix}. \tag{18}$$

Thus, by direct computation, we have

$$V^{-1} = \begin{bmatrix} \frac{1}{\kappa_3} & 0 & 0 \\ \frac{\sigma}{\kappa_3\kappa_4} & \frac{1}{\kappa_4} & 0 \\ \frac{\sigma\delta}{\kappa_3\kappa_4(\kappa_5+\alpha(x))} & \frac{\delta}{\kappa_4(\kappa_5+\alpha(x))} & \frac{1}{(\kappa_5+\alpha(x))} \end{bmatrix}. \tag{19}$$

Now, we have the following Next-Generation Matrix

$$FV^{-1} = (1-\kappa)\begin{bmatrix} \frac{c\beta(x)\rho}{\kappa_3}\left(1+\frac{\theta_1\sigma}{\kappa_4}+\frac{\theta_2\delta\sigma}{\kappa_4(\kappa_5+\alpha(x))}\right) & \frac{c\beta(x)\rho}{\kappa_4}\left(\theta_1+\frac{\theta_2\delta}{(\kappa_5+\alpha(x))}\right) & \frac{c\beta(x)\theta_2\rho}{(\kappa_5+\alpha(x))} \\ \frac{c\beta(x)(1-\rho)}{\kappa_3}\left(1+\frac{\theta_1\sigma}{\kappa_4}+\frac{\theta_2\delta\sigma}{\kappa_4(\kappa_5+\alpha(x))}\right) & \frac{c\beta(x)(1-\rho)}{\kappa_4}\left(\theta_1+\frac{\theta_2\delta}{(\kappa_5+\alpha(x))}\right) & \frac{c\beta(x)\theta_2(1-\rho)}{(\kappa_5+\alpha(x))} \\ 0 & 0 & 0 \end{bmatrix}. \tag{20}$$

The basic risk reproduction number, $\mathscr{R}_0$, is given by the dominant eigenvalue of matrix FV^{-1} Therefore,

$$\mathscr{R}_0(x) = c\beta(x)\left(\frac{(\rho\kappa_4(\kappa_5+\alpha(x))+(\theta_1(\kappa_5+\alpha(x))+\theta_2\delta)((1-\rho)\kappa_3+\rho\sigma))(1-\kappa)}{\kappa_3\kappa_4(\kappa_5+\alpha(x))}\right). \tag{21}$$

With appropriate choices of $\rho, \phi \le 1$, Theorem 2 is established

Theorem 2. *The risk-free equilibrium ε_0 is locally asymptotic stable when $\mathscr{R}_0 < 1$and unstable otherwise.*

Proof. Consider the Jacobian matrix evaluated at the risk free equilibrium point bellow

$$J(\varepsilon_0) = \begin{bmatrix} J_{11} & J_{12} & J_{13} & J_{14} & J_{15} & J_{16} \\ J_{21} & J_{22} & J_{23} & J_{24} & J_{25} & \phi\pi \\ 0 & 0 & J_{33} & J_{34} & J_{35} & 0 \\ 0 & 0 & J_{43} & J_{44} & J_{45} & 0 \\ 0 & 0 & 0 & \delta & J_{55} & 0 \\ 0 & 0 & 0 & \xi & \eta & -k_6 \end{bmatrix}, \tag{22}$$

where $J_{11} = -\kappa_1 + (1-\phi)\pi$, $J_{12} = \gamma_2 + (1-\phi)\pi$, $J_{13} = -c\beta(x)(1-\kappa) + (1-\phi)\pi$, $J_{14} = -c\beta(x)\theta_1(1-\kappa) + (1-\phi)\pi$, $J_{15} = -c\beta(x)\theta_2(1-\kappa) + (1-\phi)\pi$, $J_{16} = \omega + (1-\phi)\pi$, $J_{21} = \phi\pi + \gamma_1$, $J_{22} = \phi\pi - \kappa_2$, $J_{23} = \phi\pi + \nu$, $J_{24} = \phi\pi + \tau$, $J_{25} = \phi\pi + \tau$, $J_{33} = c\beta(x)\rho(1-\kappa) - \kappa_3$, $J_{34} = c\beta(x)\theta_1\rho(1-\kappa)$, $J_{35} = c\beta(x)\theta_1\rho(1-\kappa)$, $J_{43} = (1-\rho)c\beta(x)(1-\kappa) + \sigma$, $J_{44} = c\beta(x)\theta_1(1-\rho)(1-\kappa) - \kappa_4$, $J_{45} = c\beta(x)\theta_1(1-\rho)(1-\kappa) - \kappa_4$, and $J_{55} = -\kappa_5 - \alpha(x)$

Using a trace determinant method, the risk-free equilibrium point ε_0 is locally stable provided that $\mathrm{Tr}(J(\varepsilon_0)) < 0$ and $\mathrm{Det}(J(\varepsilon_0)) > 0$ The trace and determinant of $J(\varepsilon_0)$ are, respectively, given by

$$\mathrm{Tr}(J(\varepsilon_0)) = (\pi - \alpha(x)) + c\beta(x)((1-\rho)\theta_1 + \rho)(1-\kappa) - \sum_{i=1}^{6}\kappa_i,$$
$$\mathrm{Det}(J(\varepsilon_0)) = (1-\mathscr{R}_0)(\pi-\mu)(\mu+\gamma_1+\gamma_2)\kappa_3\kappa_4(\kappa_5+\alpha(x))\kappa_6. \tag{23}$$

Since $\mu < \pi$, it follows that ε_0 is asymptotically stable whenever $\mathscr{R}_0 < 1$ and unstable when $\mathscr{R}_0 > 1$ provided that

$$\frac{(\pi-\alpha(x)) + c\beta(x)((1-\rho)\theta_1 + \rho(1-\kappa)}{\sum_{i=1}^{6}\kappa_i} < 1. \tag{24}$$

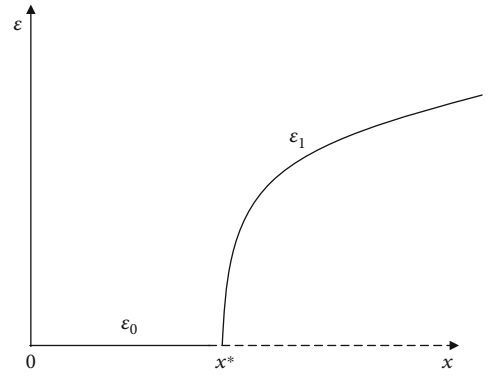

Figure 3: Bifurcation diagram.

The value of $\mathscr{R}_0$ may vary significantly depending on different risk dynamics studied in the population or different populations involved in similar studies [33]. Similarly, the stability of the risk-free equilibrium also changes from unstable to stable when $\mathscr{R}_0$ increases through 1. The system acquires a bifurcation at the risk-free equilibrium when $\mathscr{R}_0 = 1$. Suppose that the bifurcation value occurs at x^* (see Figure 3) where x^* is given as

$$x^* = \frac{a_4 - \sqrt{a_2 - 2a_3 + (-1+u+\alpha_0)^2(x_0 - x_{\min})^2}}{2a_1(-1+u+\alpha_0)(a_5\theta_1 + \rho\kappa_4)}, \quad (25)$$

provided that $x_{\min} \le x^* \le x_0$ and

$$\begin{aligned} a_1 &= \frac{c(1-\kappa)}{\kappa_3\kappa_4}, \\ a_2 &= (((-1+u+\alpha_0)x_{\min} + (\alpha_0+\kappa_5)(x_{\min} - x_0)) \\ &\quad \cdot (\theta_1 a_5 + \rho\kappa_4) + \theta_2\delta a_5(x_{\min} + x_0))^2 {a_1}^2, \\ a_3 &= (x_0 - x_{\min})(-1+u+\alpha_0)(((-1+u+\alpha_0) \\ &\quad + (\alpha_0+\kappa_5)(x_0 + x_{\min}))(\theta_1 a_5 + \rho\kappa_4) \\ &\quad - (x_0 + x_{\min})\theta_2\delta a_5)a_1, \\ a_4 &= (((u-1)x_{\min} + x_0\alpha_0)(\theta_1 a_5 + \rho\kappa_4) \\ &\quad + ((\theta_1 a_5 + \rho\kappa_4)\kappa_5 + \theta_2\delta a_5)(x_0 - x_{\min}))a_1 \\ &\quad + (-1+u+\alpha_0)(\alpha_0 - x_{\min}), a_5 \\ &= ((1-\rho)\kappa_3 + \rho\sigma). \end{aligned} \quad (26)$$

The basic risk reproduction number presented in (21) is the function of x, that is $\mathscr{R}_0 = \mathscr{R}_0(x)$, but it can not be referred to as the fuzzy number since it is open to values exceeding unity. It is clear that $\mathscr{R}_0(x)$ is the function of the degree of social influence in the spread of the behavior. However, both $\beta(x)$ and $\alpha(x)$ incline to their maximum level whenever $x \ge x_0$, and as such, we have

$$\mathscr{R}_0(u) = c\left(\frac{(\rho\kappa_4(\kappa_5 + (1-u)) + (\theta_1(\kappa_5 + (1-u)) + \theta_2\delta)((1-\rho)\kappa_3 + \rho\sigma))(1-\kappa)}{\kappa_3\kappa_4(\kappa_5 + (1-u))}\right). \quad (27)$$

We introduce a positive number ϵ_0 such that $\epsilon_0\mathscr{R}_0(x) \le 1$, with an appropriate choices of ϵ_0; we have a fuzzy set $\epsilon_0\mathscr{R}_0(x)$ whose fuzzy expected value, $\mathrm{FEV}[\epsilon_0\mathscr{R}_0(x)]$, can be well defined. Therefore, the fuzzy basic risk reproduction number $\mathscr{R}_0^f$, which can be defined as the average number of secondary risk cases caused by one infected node introduced into entirely susceptible nodes [31], is given by

$$\mathscr{R}_0^f = \frac{1}{\epsilon_0}\mathrm{FEV}[\epsilon_0\mathscr{R}_0(x)]. \quad (28)$$

We know from the definition of fuzzy expected value that

$$\mathrm{FEV}[\epsilon_0\mathscr{R}_0(x)] = \sup_{0\le y\le 1} \inf\,[y, k(y)], \quad (29)$$

where $k(y) = \Phi\{z \in \Omega : \epsilon_0\mathscr{R}_0(x) \ge y\} = \Phi(\Omega)$ is a fuzzy measure. For the purpose of this study, the possibility measure is given by

$$\Phi(\Omega) = \sup_{x\in X} \prod(x), \quad \Omega \subset \mathbb{R}. \quad (30)$$

From $\mathrm{FEV}[\epsilon_0\mathscr{R}_0(x)]$, for a monotonic function $\mathscr{R}_0(x)$, with set X given as an interval $[x^*, x_{\max}]$, we let $x^* \in X$ be the solution of the following equation:

$$\epsilon_0 c\beta(x)\left(\frac{(\rho\kappa_4(\kappa_5 + \alpha(x)) + (\theta_1(\kappa_5 + \alpha(x)) + \theta_2\delta)((1-\rho)\kappa_3 + \rho\sigma))(1-\kappa)}{\kappa_3\kappa_4(\kappa_5 + \alpha(x))}\right) = y. \quad (31)$$

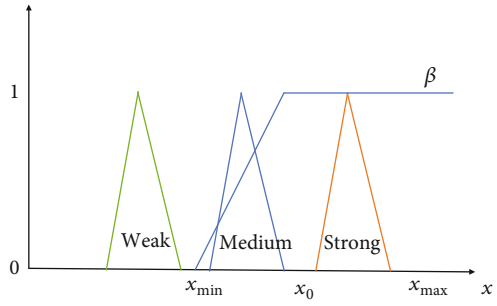

Figure 4: Classification of linguistic variable Π.

Thus,

$$k(y) = \Phi[x^*, x_{\max}] = \sup_{x^* \le x \le x_{\max}} \prod(x), \tag{32}$$

where $k(0) = 1$ and $k(1) = \prod(x_{\max})$.

Now, FEV$(\epsilon_0 \mathscr{R}_0)$ can be determined by considering the linguistic variable Π in three classes: "weak Π_-," "medium Π_-^+," and "strong Π^+." Each of these classification is a fuzzy number based on $x_{\min}$, x_0, and $x_{\max}$ as shown in Figure 4. The classification of the degree of social influences in the community can be explained in three different cases as follows:

Case 1. In this case, we consider the weak degree of social influence (Π_-) where we define that $x_c + d < x_{\min}$. Suppose that there exists $\bar{x}$ such that $\mathscr{R}_0(\bar{x}) = \mathscr{R}_0^f$; if $x_c + d < \bar{x}$c, we have

$$k(y) = \sup_{\bar{x} \le x \le x_{\max}} \prod(x) = 0, \quad \forall y \in [0, 1]. \tag{33}$$

We establish that FEV$[\epsilon_0 \mathscr{R}_0] = 0 < \epsilon_0$. This can be translated that the fuzzy basic risk reproduction number $\mathscr{R}_0^f < 1$, and hence, the extinction of the health risk is associated with alcoholism.

Case 2. In this case, we consider the medium degree of social influence (Π_-^+) where we define that $x_c - d < x_{\min}$ and $x_c + d < x_0$ giving.

$$k(y) = \begin{cases} 1, & \text{if } 0 \le y < \epsilon_0 \mathscr{R}_0(x_c), \\ \prod(\bar{x}), & \text{if } \epsilon_0 \mathscr{R}_0(x_c) \le y \le \epsilon_0 \mathscr{R}_0(x_c + d), \\ 0, & \text{if } \epsilon_0 \mathscr{R}_0(x_c + d) < y \le 1. \end{cases} \tag{34}$$

For $d > 0$, $k(y)$ is the continuous function giving $k(0) = 1$ and $k(1) = 0$. This translates $\mathscr{R}_0^f$ as the fixed point k and $\mathscr{R}_0(x_c) < \mathscr{R}_0^f < \mathscr{R}_0(x_c + d)$. Since $\mathscr{R}_0(x)$ is a continuous monotonic (increasing) function, the Intermediate Value Theorem suggests that there exists $\bar{x}$ with $x_c < \bar{x} < x_c + d$ in which the values of $\mathscr{R}_0(\bar{x})$ and $\mathscr{R}_0^f$ coincide such that $\mathscr{R}_0^f = \mathscr{R}_0(\bar{x}) > \mathscr{R}_0(x_c)$. Further, the average number of fuzzy basic risk reproduction number $\mathscr{R}_0^f$ is higher than the number of secondary risk cases $\mathscr{R}_0(x_c)$ due to the medium level of social influence implying that the health risk associated with alcoholism is endemic.

Case 3. In this case, we consider the strong degree of social influence (Π^+) by defining $x_c - d > x_0$ and $x_c + d < x_{\max}$.

The results in (35) can be established.

$$k(y) = \begin{cases} 1, & \text{if } 0 \le y < \epsilon_0 \mathscr{R}_0(x_c), \\ \prod(\bar{x}), & \text{if } \epsilon_0 \mathscr{R}_0(x_c) \le y < \epsilon_0 \mathscr{R}_0(x_c + d), \\ 0, & \text{if } \epsilon_0 \mathscr{R}_0(x_c + d) \le y \le 1. \end{cases} \tag{35}$$

For any given $d > 0$, $k(y)$ is a monotonically decreasing and continuous function with $k(0) = 1$ and $k(1) = 0$.

Therefore, FEV$[\epsilon_0 \mathscr{R}_0]$ is established as a fixed point such that

$$\epsilon_0 \mathscr{R}_0(x_c) < \text{FEV}[\epsilon_0 \mathscr{R}_0] < \epsilon_0 \mathscr{R}_0(x_c + d). \tag{36}$$

By dividing by ϵ_0 throughout, we have the following results:

$$\mathscr{R}_0(x_c) < \mathscr{R}_0^f < \mathscr{R}_0(x_c + d). \tag{37}$$

Now, since $\mathscr{R}_0^f > 1$, it translates into endemic health risks associated with alcoholism.

3.3. Risk Control in Fuzzy Epidemic System. In this section, we perform the control analysis of the risk estimation in the population by using the fuzzy basic risk threshold $\mathscr{R}_0^f = \mathscr{R}_0(\bar{x})$. The spread of health risk in the proposed fuzzy *SPLMAR* model (3) depends on the degree of the social influence x as an input value of the transmission factor $\beta(x)$. The description of existence and stability of the risk in the system is case-wise presented hereunder. Since the proposed fuzzy system (3) represents a family of systems depending on the parameter x, these family systems can be simplified by a unique system of equations with the same results. It is shown that the bifurcation occurs at $x = x^*$, that is, $\mathscr{R}_0(x^*) = 1$.

(1) Weak influence: in this case, we have $x < x_{\min}$ giving $\mathscr{R}_0 = 0$ suggesting the extinction of the health risks associated with alcoholism in the community.

(2) Medium influence: in this case, three possibilities may arise as follows:

  (i) If $x < x^*$, then $\mathscr{R}_0 < 1$ suggesting the risk free community

  (ii) If $x = x^*$, then $\mathscr{R}_0 = 1$ an indication of risk bifurcation

  (iii) If $x > x^*$, then $\mathscr{R}_0 > 1$ implying the risk endemic in the community

(3) Strong influence: in this case, we have $x \in [x_0, x_{\max}]$ giving

$$\mathscr{R}_0(u) = c\left(\frac{(\rho\kappa_4(\kappa_5 + (1-u)) + (\theta_1(\kappa_5 + (1-u)) + \theta_2\delta)((1-\rho)\kappa_3 + \rho\sigma))(1-\kappa)}{\kappa_3\kappa_4(\kappa_5 + (1-u))}\right). \tag{38}$$

The spread of the health risks depends upon the parameter u. Let u^* be an improved value of u; we can establish three possibilities in which the spread of health risks takes as follows:

(i) If $0 \le u < u^*$, then $\mathscr{R}_0(u) < 1$ suggesting the health risks would be cleared in the community

(ii) If $0 \le u = u^*$, then $\mathscr{R}_0(u) = 1$ implying that the system passes through a bifurcation state

(iii) If $0 \le u^* < u$, then $\mathscr{R}_0(u) > 1$ suggesting that the health risk problem would spread out in the system

4. Discussion and Conclusion

In this work, we propose and analyze the fuzzy *SPLMAR* model with the aim of using fuzzy set theory in the model system. Although all the parameters associated in the model system are important, in an uncertain environment, we consider the selected two most important parameters β and α representing the probability that a susceptible individual will drink alcohol after prolonged contact with drinking individuals and the additional death rate induced by alcoholism, respectively. In particular, the two parameters β and α are considered fuzzy numbers and functions of the degree of force of influence, whose membership functions are well defined. Later, the fuzzy basic risk reproduction number $\mathscr{R}_0^f(x)$ is computed.

Since the basic risk reproduction number $\mathscr{R}_0$ is directly related to varied factors of drinking settings, obtained by considering the classical model system (1) with constant parameters β and α, it implied that the variation of the risky population is always positive. However, when β is considered a fuzzy set, it provide us with an additional information regarding the health risk dynamics. The health risks associated with alcoholism can be effectively controlled by controlling the fuzzy basic risk reproduction number $\mathscr{R}_0^f$. The analysis of the fuzzy model suggests that $\mathscr{R}_0^f$ can be reduced by increasing the value of $x_{\min}$. This may be done through provision of public health education which increases the resistance of susceptible individuals and prevent them from initiation of drinking behavior which would accelerate their health condition into more risky states. It is generally observed that if the amount of degree of peer of influence to an individual is low, then the alcohol-related health risks in the community may not be the most effective. The reason is natural factors in the community provide virtual immunity from deeply engaging into alcoholic behaviors. However, when the perceived most influential people in the community engage in alcoholism, it implies an increase in the degree of force of influence, and as such, the system will be endemic.

In a future work, some other parameters may be considered with different functionalities under the uncertainty environment. However, the present model can be applied to those types of diseases or conditions which spread through direct contact between susceptible and infected individuals.

Data Availability

No data is included.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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Available online at http://scik.org
Commun. Math. Biol. Neurosci. 2020, 2020:54
https://doi.org/10.28919/cmbn/4788
ISSN: 2052-2541

MATHEMATICAL MODELS FOR THE DYNAMICS OF ALCOHOL RELATED HEALTH RISKS WITH CHANGING BEHAVIOR VIA CULTURAL BELIEFS IN TANZANIA

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Abstract. Alcoholism has continually posed health challenges in many communities for decades. In this paper, a more realistic model for health related risks associated with alcoholism is formulated. It considers a population proportion that has social cultural protection from alcohol consumption. In the context of this paper, such protection emanates from religious beliefs. The Next Generation Matrix (NGM) approach is used to compute the basic risk reproduction number. The risk free equilibrium point is proved to be globally asymptotically stable whenever the basic risk reproduction is less than unity and unstable otherwise. The sensitivity analysis of the basic risk reproduction number and numerical simulation results reveal that for effective control of the health risk problem in the community, the deliberate intervention strategies and policies should focus on discouraging alcoholic behaviors on its onset during initiation stage than focusing other population proportions already at risk.

Keywords: health risks; alcoholism; cultural beliefs; religious beliefs; mathematical models.

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Received June 22, 2020

2010 AMS Subject Classification: 92C50, 93A30.

1. INTRODUCTION

Alcoholic beverages are an integral part of cultures across the globe with wide use in rituals, societal artifacts and festivals [1, 2]. In essence alcoholic beverages are important for both social and economic reasons. Precisely, alcohol beverages are a source of income for the livelihood of the rural community and have health benefits such as prevention of thrombosis when consumed at desired levels [3]. However, despite the aforementioned health and economic benefits of alcoholic beverages; when taken to undesired level it can accelerate to alcoholism, a behavior which poses serious health challenges to consumers. Alcoholism may be defined as the state of addiction to the consumption of alcoholic drinks which eventually turns into the state of alcohol dependency. This is a condition in which a person has a physical or psychological dependence on drinks that contain alcohol. It is a precursor to injury and violence and its negative impacts can spread throughout a community or a country, and beyond, influencing levels and patterns of alcohol consumption across borders [4].

The common symptoms of alcoholism include, but not limited to: strong compulsion to drink; inability to limit ones drinking in any given time; physical dependence; increased uptake of alcohol for optimum effects; and problems associated with alcoholism − injuries, receives multiple drunken driving citations, frequent arguments and poor relationships in families.

The drinking limits or threshold to be referred to as an alcoholic is estimated to a maximum of 21 standard bottles per week for men and 14 drinks per week for women [2, 3, 5]. This quantity defines the health tolerable amount of alcohol consumption at which an individual attains the most vulnerable state of exposure to alcohol associated health risks.

According to the World Health Organization report, at least three (3) million deaths and 132.6 disabilities due to harmful use of alcohol reported annually. Comparatively, over-consumption of alcohol causes more harm to human health than tuberculosis, HIV/AIDS and diabetes [6]. Further, alcoholism is linked to liver cirrhosis [2, 7] and accounts for 10% of the total disease burden attributable to harmful use of alcohol [8]. Owing to the available literature, it was observed that alcohol consumption increases risks of cancer infections, which is attributable to

20% of alcohol related deaths. Common types of cancers related to alcohol overconsumption are cancers of the mouth, oropharynx, esophagus, colorectal, liver, and breast [7, 8, 9].

Some cultural practices have promoted positive drinking with the aim of reducing health and socio-economic risks. Part of renown active cultural practices are religious beliefs which have strong influence in people's lives. It plays an important role in promoting the health and molding individual's behaviors [10, 11]. By the fact that religious communities at least discourages irresponsible alcohol drinking by pronouncing it a sinful behavior. It is therefore important to focus on the impacts of religious communities when dealing with the dynamics of health related risks associated with alcoholism because they provide social protection to a proportion of the population from alcohol consumption or alcohol abuse.

In quest to provide insights on effects and the spread of alcoholism, different mathematical alcohol epidemic models have been formulated and analyzed for the purpose of understanding the effects of alcohol consumption on health and socio-economic aspects of the society. In [12], for example, a link between drug addiction and infectious disease is considered and the importance of using dynamic models to predict trends and generate estimates is emphasized especially where data are sparse. Mundt and collaborators in [13] fitted a stochastic actor $-$ based model to Add Health data of 7th through 11th grade U.S students enrolled between 1995 and 1996. The study sought to determine factors that influence the dynamic interplay between adolescent friendships and substance abuse and revealed that peer selection and friendship making has a greater role to play in alcoholic behavior among adolescent. Similarly, [14] presented $SATQ-$ type alcoholic model with two control strategies. Two objective functions for alcohol quitting and cost of controlling alcohol were proposed and analyzed with the help of Pontryagin's Maximum Principle approach. Numerical simulations results recommended reduction in the number of alcoholics and the increase in the number of susceptible as the better control option.

Xiang and collaborators, [15] studied the effects of public health educational campaigns on drinking dynamics. The study explored the use of Lyapunov functional to establish the global stability of the model equilibria. Numerical simulations result proved that public health education campaigns of drinking individuals can slow down the drinking dynamics. In [16] a non $-$ linear $SHTR$ mathematical model for drinking epidemics is presented and analyzed. In this

model, the drinking population were placed in heavy drinkers (H) and drinkers in treatment (T) compartments assuming that after treatment a treated heavy drinker will only become a heavy drinker again after passing through a recovery and susceptible compartments in respective order. The analytical results revealed that drinking epidemic can be controlled by the combined efforts of reducing the contact rate between the non-drinkers and heavy drinkers to refrain from drinking; and increasing the number of drinkers that go into treatment.

In [7], an *SIRS* alcoholism models with relapse on a weighted networks was formulated to study the impact of the fixed weight and adaptive weight on the spread of alcoholism. The study results established that, if the proportion of recuperator to accept treatment is equal to that of susceptible people to refuse alcohol drinking, then preventing the susceptible people from alcohol drinking become more effective. In [9], a deterministic model to study the spread of alcoholism was formulated by dividing the alcohol drinking population into moderate and alcoholic population. The comparison of alcohol control approaches targeting different alcohol drinking populations revealed that encouraging and supporting moderate drinkers to quit alcoholic consumption produces better results than targeting the alcoholic group only.

Mushanyu and Nyabadza in [17] presented a risk − structured model and used it to understand the phenomenon of the spread of drug abuse with in-patients treatment programs. Analysis of the risk abuse and numerical simulations suggested that education and skills to deal with risky situation may better equip individuals to stand against initiating into drug abuse.

Mayengo and collaborators in [18] suggested that for alcohol related health risks modeling to be more relevant, two aspects of the model formulation are proposed: the influence of external motivations with positive and negative influential effects, and a clearly defined alcohol drinking population compartments have to be included. The crisp model developed in [19] have definitely answered this concerns where the fuzzy logic analysis of the model is carried out.

2. Mathematical Model

2.1. Model formulation. Adopting the framework of Mayengo *et al.* [19], we present an analysis of health risk model associated with alcoholism of a population divided into six (6) distinct classes based on the individual's risk level defined by their drinking habits. The distinct classes are: (i) Susceptible, $S(t)$ - comprising individuals at risk of engaging in alcohol

drinking, (ii) Protected class, $P(t)$ - individuals who have virtually gained protection from alcohol drinking through cultural practices, (iii) Low risk drinkers, $L(t)$ - individuals who drink alcoholic drinks responsibly on occasional basis, (iv) Moderate risk drinkers, $M(t)$ - individuals who consume alcoholic drinks regularly, (v) High risk drinkers or Alcohol addicts, $A(t)$ - individuals who have developed high dependence in alcohol, and (vi) Recovered, $R(t)$ - comprising of individuals who have voluntarily quit drinking on health related challenges. New recruits enter the population at a constant rate π. A proportion $\phi \in (0,1)$ of the new recruits are subjected to cultural or religious beliefs and enter a Protected class, while the remaining $1-\phi$ enter a susceptible class. The susceptible class is further increased by individuals backsliding from their religious beliefs at a constant rate γ_2, and recovered individuals at a constant rate ω. The susceptible class is decreased at the rate λ, and cultural (religious) conversion at the rate of γ_1. A non-drinker acquire alcohol drinking habits through social contacts [9, 19, 20] at the force of peer influence

$$\lambda = c\beta\left(\frac{L+\theta_1 M+\theta_2 A}{N}\right) \tag{1}$$

where β is the probability of initiation to alcohol drinking, θ_1 and θ_2 are the modification parameters $(\theta_1 \leq \theta_2)$, c is the effective contact to influence one into alcohol drinking, and N is the total population. The protected class is increased by conversion to cultural and religious beliefs of the susceptible, low risk drinkers, moderate risk drinkers and alcoholics. It is decreased by backsliding at constant rate γ_2. Low and moderate risk drinkers are increased by initiation to alcohol drinking of the susceptible class. Low risk drinkers progress to moderate drinkers at a constant rate σ. Moderate risk drinkers progress to alcohol addiction at a constant rate δ, recover at a constant rate ξ and are protected at a constant rate τ. Alcohol addicts either recover at the rate η or get protected at the rate ψ. The recovered class become susceptible again at the rate ω. All the classes are subjected to reduction due to natural causes at a constant rate μ.

The proportion $\rho \in (0,1)$ of susceptible individuals are recruited via peer influence into low risk drinking class, while the remaining proportion, $(1-\rho)$ enters the moderate risk class. Based on the dynamics on the model compartment in Fig. 1, the following set of equations are

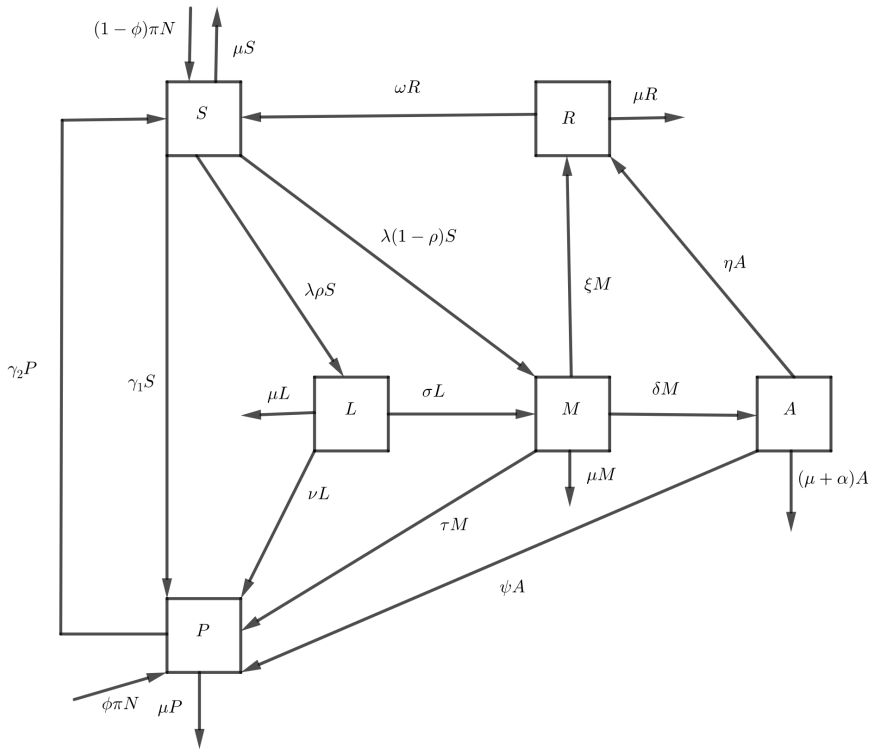

FIGURE 1. The structure of the dynamics of health related risks model

formulated.

$$
\begin{cases}
\dot{S} &= -(\mu+\gamma_1+\lambda)S+\gamma_2 P+\omega R+(1-\phi)\pi N \\
\dot{P} &= \gamma_1 S-(\mu+\gamma_2)P+\nu L+\tau M+\psi A+\phi\pi N \\
\dot{L} &= \lambda\rho S-(\nu+\mu+\sigma)L \\
\dot{M} &= \lambda(1-\rho)S+\sigma L-(\tau+\mu+\delta+\xi)M \\
\dot{A} &= \delta M-(\mu+\alpha+\eta+\psi)A \\
\dot{R} &= \xi M+\eta A-(\omega+\mu)R
\end{cases} \tag{2}
$$

where $S \geq 0, P \geq 0, L \geq 0, M \geq 0, A \geq 0$ and $R \geq 0$.

Let N be the total population size given by;

$$N = S+P+L+M+A+R \tag{3}$$

Then the equation describing changes in the total population is given by,

$$\frac{dN}{dt} = (\pi - \mu) N - \alpha A \tag{4}$$

2.2. Basic properties of the model solution. In this section, the basic results of solutions for model system (2) is discussed. These properties lay down a foundation of proofs of stability analysis results of the model. Following [21], let $x \in \mathbb{R}^n_+$ denote the set of state variables $x = (x_1, x_2, \ldots, x_n)$ with the positive components, the following results may be established.

Lemma 1. *Let $f : \mathbb{R}^n_+ \to \mathbb{R}^n, f(x) = (f_1(x), f_2(x), \ldots, f_n(x))$ with $x \in \mathbb{R}^n_+$, be continuous and there exist a continuous partial derivatives $\dfrac{\partial f_j}{\partial x_i}$ in $\mathbb{R}^n_+$ for $i, j = 1, 2, \ldots, n$. Then f is locally Lipschitz continuous in $\mathbb{R}^n_+$.*

Theorem 2.1. *Let $f : \mathbb{R}^n_+ \to \mathbb{R}^n$ be a locally Lipschitz continuous and for each $i = 1, 2, \ldots, n$ satisfy $f_i(x) \geq 0$ whenever $x \in \mathbb{R}^n_+$, $x_i = 0$. Then, for every $x_0 \in \mathbb{R}^n_+$, there exist a unique solution of $\dot{x} = f(x)$, $x(0) = x_0$ with values in $\mathbb{R}^n_+$ defined in some intervals such that $0 < x_0 \leq b$ with $0 < b \leq \infty$. If $b < \infty$, then $\sup \sum_{i=1}^n x_i(t) = \infty$.*

Theorem 2.2. *For all $S(0), P(0), L(0), M(0), A(0), R(0) > 0$, there exist $S, P, L, M, A, R : (0, \infty) \to (0, \infty)$ which solve the model system (2) with initial conditions $S = S(0), P = P(0), L = L(0), M = M(0), A = A(0)$, and $R = R(0)$.*

Proof. Applying Theorem 2.1, we define $f_1(x) = \dot{S}, f_2(x) = \dot{P}, f_3(x) = \dot{L}, f_4(x) = \dot{M}, f_5(x) = \dot{A}$, and $f_6(x) = \dot{R}$ where $x = (S, P, L, M, A, R)$. By the properties of continuity over operations, we have continuity of f_i for all $i = 1, 2, ..., 6$. Furthermore,

$$\frac{\partial f_j}{\partial x} = \frac{\partial f_j}{\partial x_1} + \frac{\partial f_j}{\partial x_2} + \cdots + \frac{\partial f_j}{\partial x_6}, \ \forall j \in \{1, 2, \ldots, 6\} \tag{5}$$

where the derivatives are continuous. Thus we have,

$$\frac{\partial f_1}{\partial x} = (6\lambda - c\beta (1 + \theta_1 + \theta_2)) \frac{S}{N} - (\mu + \gamma_1 + \lambda) + 6(1 - \phi)\pi + \gamma_2 + \omega \tag{6}$$

By Lemma 1, we know that f is locally Lipschitz continuous. Let $x_1 = S = 0$ with $x_{i \neq 1} > 0$. Then

$$\frac{\partial f_1}{\partial x} = -(\mu + \gamma_1 + \lambda_1) + 6(1 - \phi)\pi + \gamma_2 + \omega > 0 \tag{7}$$

where $N_1 = P+L+M+A+R$ and $\lambda_1 = c\beta\left(\frac{L+\theta_1 M+\theta_2 A}{N_1}\right)$. Similarly, repeating the procedures for the rest of state variables, the following conditions are established

$$\frac{\partial f_2}{\partial x} = 6\phi\pi + \gamma_1 - (\mu+\gamma_2) + \nu + \tau + \psi > 0 \tag{8}$$

$$\frac{\partial f_3}{\partial x} = \rho\lambda_3\left(1-6\frac{S}{N_3}\right) + c\beta\rho\left(1+\theta_1+\theta_2\right)\frac{S}{N_3} - (\mu+\nu+\sigma) > 0 \tag{9}$$

$$\frac{\partial f_4}{\partial x} = (1-\rho)\left(1-6\frac{S}{N_4}\right)\lambda_4 + c\beta\left(1-\rho\right)\left(1+\theta_1+\theta_2\right)\frac{S}{N_4} + \sigma - (\tau+\mu+\delta+\xi) > 0 \tag{10}$$

$$\frac{\partial f_5}{\partial x} = \delta - (\mu+\alpha+\eta+\psi) > 0 \tag{11}$$

$$\frac{\partial f_6}{\partial x} = \xi + \eta - (\mu+\omega) > 0 \tag{12}$$

where $N_3 = S+P+M+A+R$, $N_4 = S+P+L+A+R$, consequently $\lambda_3 = c\beta\left(\frac{\theta_1 M+\theta_2 A}{N_3}\right)$, and $\lambda_4 = c\beta\left(\frac{L+\theta_2 A}{N_4}\right)$. By Theorem 2.2, for every $x(0) \in \mathbb{R}^6_+$ defined in some intervals such that $0 < x_0 \leq b$ with $0 < b \leq \infty$, there exist a unique solution of $\dot{x} = f(x)$. If $b < \infty$ then we have

$$\sup_{0\leq t\leq b}[N] = \infty$$

2.3. Invariant region. Since the above model in (2) represents human population, it is assumed that all the state variables and parameters of the model are non-negative for all $t \geq 0$. Now, from the equation (4) we have, $\frac{dN}{dt} \leq (\pi-\mu)N$ which upon integration yields

$$N \leq N_0 e^{(\pi-\mu)t} \tag{13}$$

where N_0 is the initial population obtained by evaluating equation (3) at the initial conditions of the respective state variables. It follows that, for $\mu < \pi$ as $t \to \infty, N_0 \leq N \leq \infty$, therefore $N(t)$ is bounded, posing contradictions to Theorem 2.2. As a result, $b = \infty$, suggesting that feasible solutions of components of system (2) are positive and are defined on the interval $0 < b < \infty$. □

The region governing the solutions of the model is positively-invariant under the flow induced by system (2). Following the boundedness of the model solutions, the following theorem is established

Theorem 2.3. *All solutions of model system (2) are bounded and the components of system enter the region* Ω *such that* $\Omega = \left\{(S,P,L,M,A,R) \in \mathbb{R}^6_+, N_0 \leq N \leq \infty\right\}$.

Hence, the region Ω is positively-invariant under the flow induced by system (2), that it is well posed mathematically and epidemiologically, sufficiently for the dynamics of the flow generated by system (2) to be considered in Ω.

2.4. Steady state solutions. To obtain the equilibrium point, we solve a non-linear equations. (14).

$$
\left\{
\begin{array}{rl}
0 = & (1-\phi)\pi N^* + \omega R^* + \gamma_2 P^* - \kappa_1 S^* - \lambda^* S^* \\
0 = & \phi\pi N^* + \gamma_1 S^* + \nu L^* + \tau M^* + \psi A^* - \kappa_2 P^* \\
0 = & \lambda^* \rho S^* - \kappa_3 L^* \\
0 = & \lambda^* (1-\rho) S^* + \sigma L^* - \kappa_4 M^* \\
0 = & \delta M^* - \kappa_5 A^* \\
0 = & \xi M^* + \eta A^* - \kappa_6 R^*
\end{array}
\right. \tag{14}
$$

where

$$
\lambda^* = c\beta \left(\frac{L^* + \theta_1 M^* + \theta_2 A^*}{N^*} \right) \tag{15}
$$

We observe that $L^* = Q_0 \lambda^* S^*$, $M^* = Q_1 \lambda^* S^*$, and $A^* = Q_2 \lambda^* S^*$,
where $Q_0 = \dfrac{\rho}{\kappa_3}$, $Q_1 = \dfrac{(1-\rho)\kappa_3 + \rho\sigma}{\kappa_3 \kappa_4}$, and $Q_2 = \dfrac{((1-\rho)\kappa_3 + \rho\sigma)\delta}{\kappa_3 \kappa_4 \kappa_5}$.
Substituting for L^*, M^*, and A^* in the equation (15), we obtain

$$
(N^* - \mathscr{R}_H S^*)\lambda^* = 0 \tag{16}
$$

giving

$$
\lambda^* = 0 \text{ or } N^* - \mathscr{R}_H S^* = 0 \tag{17}
$$

where

$$
\mathscr{R}_H = c\beta \left(\frac{\rho\kappa_4\kappa_5 + (\theta_1\kappa_5 + \theta_2\delta)((1-\rho)\kappa_3 + \rho\sigma)}{\kappa_3\kappa_4\kappa_5} \right). \tag{18}
$$

2.4.1. *Risk free equilibrium.* The solution $\lambda^* = 0$ from equation (17) leads to the risk free equilibrium

$$E_0 = (S_0^*, P_0^*, L_0^*, M_0^*, A_0^*, R_0^*) = \left(\frac{\pi(\gamma_2 + \mu(1-\phi))N_0^*}{\mu(\mu+\gamma_1+\gamma_2)}, \frac{\pi(\phi\mu+\gamma_1)N_0^*}{\mu(\mu+\gamma_1+\gamma_2)}, 0, 0, 0, 0 \right). \tag{19}$$

2.4.2. *Risk endemic state.* The solution $N^* - \mathscr{R}_H S^* = 0$ from equation (17) leads to endemic state. From system (14) we note that

$$R^* = Q_3 \lambda^* S^*$$

with $Q_3 = \dfrac{(\xi\kappa_5 + \eta\delta)((1-\rho)\kappa_3 + \rho\sigma)}{\kappa_3\kappa_4\kappa_5\kappa_6}$. The total population is given by

$$N^* = S^* + P^* + Q\lambda^* S^* \tag{20}$$

where $Q = \sum_{i=0}^{3} Q_i$. Substituting for N^*, we obtain

$$P^* = ((\mathscr{R}_H - 1) - Q\lambda^*) S^*$$

Re-writing the first two equations of the model system (2) in terms of λ^* and S^* with appropriate substitution of other state variables. Eliminating S^* from the subsystem, equation (21) is established with an implication of forward bifurcation.

$$\lambda^* = \frac{(1-\mathscr{R}_H)((1-\phi)\mu + \gamma_2) - (\phi\mu + \gamma_1)}{(\mu(1-\phi)+\gamma_2)Q + (1-\phi)(\nu Q_0 + \tau Q_1 + \psi Q_2) - \omega Q_3 \phi} \tag{21}$$

It follows the establishment of a non-trivial solution E_1 for the risk endemic as

$$E_1 = (S^*, ((\mathscr{R}_H - 1) - Q\lambda^*)S^*, Q_0\lambda^* S^*, Q_1\lambda^* S^*, Q_2\lambda^* S^*, Q_3\lambda^* S^*) \tag{22}$$

2.5. Basic risk reproduction number. In this section, we compute the basic risk reproduction number of the model using Next Generation Matrix (NGM) method by [19, 23, 24]. The basic risk reproduction number, denoted as $\mathscr{R}_0$, may be defined as an average number of secondary risk individuals produced by a single risk individual in an entirely susceptible population during his/her risk duration. It serves as an indicator used to predict the possibility of the occurrence of risk epidemic. Based on [24], the basic risk reproduction number is given as the dominant eigenvalue or spectral radius of the next generation matrix. Considering the system of

equations (2), this system has three risky states at different risk levels, $L(t), M(t)$ and $A(t)$; and three non-risky states, $S(t), P(t)$ and $R(t)$. Compositing system (2), we state the risk system as

$$g(X,Z) = \begin{cases} \dot{L} = & \lambda\rho S - \kappa_3 L \\ \dot{M} = & \lambda(1-\rho)S + \sigma L - \kappa_4 M \\ \dot{A} = & \delta M - \kappa_5 A \end{cases} \tag{23}$$

and non-risky system as

$$f(X,Z) = \begin{cases} \dot{S} &= -(\kappa_1 + \lambda)S + \gamma_2 P + \omega R + (1-\phi)\pi N \\ \dot{P} &= \gamma_1 S - \kappa_2 P + \nu L + \tau M + \psi A + \phi\pi N \\ \dot{R} &= \xi M + \eta A - \kappa_6 R \end{cases} \tag{24}$$

Following [23], we distinguish new risk cases from all other changes in population. Let $\mathscr{F}_i$ be the rate of appearance of new risk cases in compartment i, $\mathscr{V}_i^+$ be the rate of transfer of individuals into compartment i by all other means, and $\mathscr{V}_i^-$ be the rate of transfer of individuals out of compartment i. Assuming that each function is continuously differentiable at least twice in each variable. The risk transmission model consists of non-negative initial conditions together with the following system of equations:

$$\dot{Z} = \mathscr{F}(Z) - \mathscr{V}(Z) = \begin{bmatrix} \lambda\rho S \\ \lambda(1-\rho)S \\ 0 \end{bmatrix} - \begin{bmatrix} \kappa_3 L \\ -\sigma L + \kappa_4 M \\ -\delta M + \kappa_5 A \end{bmatrix} \tag{25}$$

where $\mathscr{V}(Z) = \mathscr{V}^-(Z) - \mathscr{V}^+(Z)$, $X = \{S,P,R\}^T \in \mathbb{R}^3$, $Z = \{L,M,A\}^T \in \mathbb{R}^3$ and $(\cdot)^T$ denotes transpose.

The matrix F corresponds to *"transmissions"* and the matrix V to *"transitions"* of risks factors through different states with different risk levels. Following [23, 24], referring to the risk states with indices i and j, with $i, j \in \{1,2,3\}$, the entry F_{ij} is the rate at which individuals in a risk state j give rise to individuals in risk state i, in the linearlized system. Thus, $F_{ij} = 0$ occurs only when there are no new cases in risk state i can be produced by an individual in risk state j. All epidemiological events that lead to new risks are incorporated in the model via F, and all other events via V.

The linearlization of risk system (23) at the risk free state E_0 yields the following F and V matrices,

$$F = \frac{S_0^*}{N_0^*}\begin{bmatrix} \beta c\rho & \beta c\theta_1\rho & \beta c\theta_2\rho \\ \beta c(1-\rho) & \beta c\theta_1(1-\rho) & \beta c\theta_2(1-\rho) \\ 0 & 0 & 0 \end{bmatrix} \text{ and } V = \begin{bmatrix} \kappa_3 & 0 & 0 \\ -\sigma & \kappa_4 & 0 \\ 0 & -\delta & \kappa_5 \end{bmatrix} \tag{26}$$

where $S_0^* = \dfrac{\pi(\gamma_2+\mu(1-\phi))N_0^*}{\mu(\mu+\gamma_1+\gamma_2)}$. Thus by direct computation, we have

$$V^{-1} = \begin{bmatrix} \dfrac{1}{\kappa_3} & 0 & 0 \\ \dfrac{\sigma}{\kappa_3\kappa_4} & \dfrac{1}{\kappa_4} & 0 \\ \dfrac{\sigma\delta}{\kappa_3\kappa_4\kappa_5} & \dfrac{\delta}{\kappa_4\kappa_5} & \dfrac{1}{\kappa_5} \end{bmatrix} \tag{27}$$

In epidemiological sense, the interpretation of the entry V_{ij}^{-1} describes the expected time that an individual with risk state j will spend in a risk state i for the rest of his/her life. For instance, in a matrix V^{-1} above, individuals who are presently in state L will spend, on average, an amount of time $\dfrac{1}{\kappa_3}$ in that state. Similarly, the same individuals will spend on average an amount of time $\dfrac{\sigma}{\kappa_3\kappa_4}$ in state M, where $\dfrac{\sigma}{\kappa_3}$ is the probability that an individual actually changes its state from L to M, instead of leaving state L by either dying or changing behavior under the influence of religion, and $\dfrac{1}{\kappa_4}$ is the average amount of time an individual who enters state M spends in state M. The individuals in state M will spend no time at all in state L leading to zeros for the appropriate elements [24]. Now, we have the following Next Generation Matrix

$$FV^{-1} = \frac{S_0^*}{N_0^*}\begin{bmatrix} \dfrac{c\beta\rho}{\kappa_3}\left(1+\dfrac{\theta_1\sigma}{\kappa_4}+\dfrac{\theta_2\delta\sigma}{\kappa_4\kappa_5}\right) & \dfrac{c\beta\rho}{\kappa_4}\left(\theta_1+\dfrac{\theta_2\delta}{\kappa_5}\right) & \dfrac{c\beta\theta_2\rho}{\kappa_5} \\ \dfrac{c\beta(1-\rho)}{\kappa_3}\left(1+\dfrac{\theta_1\sigma}{\kappa_4}+\dfrac{\theta_2\delta\sigma}{\kappa_4\kappa_5}\right) & \dfrac{c\beta(1-\rho)}{\kappa_4}\left(\theta_1+\dfrac{\theta_2\delta}{\kappa_5}\right) & \dfrac{c\beta\theta_2(1-\rho)}{\kappa_5} \\ 0 & 0 & 0 \end{bmatrix} \tag{28}$$

To interpret the entries of the Next Generation Matrix FV^{-1} and develop a meaningful definition of $\mathscr{R}_0$ we note that the entry FV_{ij}^{-1} is the expected number of secondary risk individuals

in compartment i produced by individuals initially in compartment j assuming that the drinking environment shared by the individuals remain homogeneous [25]. The basic risk reproduction number, $\mathscr{R}_0$, is given by the dominant eigenvalue of matrix FV^{-1}. Therefore,

$$\mathscr{R}_0 = c\beta\left(\frac{\rho\kappa_4\kappa_5 + (\theta_1\kappa_5 + \theta_2\delta)((1-\rho)\kappa_3 + \rho\sigma)}{\kappa_3\kappa_4\kappa_5}\right)\left(\frac{S_0^*}{N_0^*}\right) \tag{29}$$

It is important to note that

$$\mathscr{R}_0 = \left(\frac{S_0^*}{N_0^*}\right)\mathscr{R}_H < \mathscr{R}_H \tag{30}$$

3. Model Analysis

3.1. Stability analysis.

3.1.1. *Local stability.* For the differential equations presented in (2), following [21, 26] the following theorem can be established.

Theorem 3.1. *The risk-free equilibrium E_0 is locally asymptotically stable (L.A.S) for $\mathscr{R}_0 < 0$ and unstable otherwise.*

Proof. Consider the Jacobian matrix evaluated at the risk free equilibrium point bellow

$$J(E_0) = \begin{bmatrix} -\kappa_1 + (1-\phi)\pi & \gamma_2 + (1-\phi)\pi & -c\beta\frac{S_0^*}{N_0^*} + (1-\phi)\pi & -c\beta\theta_1\frac{S_0^*}{N_0^*} + (1-\phi)\pi & -c\beta\theta_2\frac{S_0^*}{N_0^*} + (1-\phi)\pi & \omega + (1-\phi)\pi \\ \gamma_1 + \phi\pi & -\kappa_2 + \phi\pi & \nu + \phi\pi & \tau + \phi\pi & \psi + \phi\pi & \phi\pi \\ 0 & 0 & c\beta\rho\frac{S_0^*}{N_0^*} - \kappa_3 & c\beta\theta_1\rho\frac{S_0^*}{N_0^*} & c\beta\theta_2\rho\frac{S_0^*}{N_0^*} & 0 \\ 0 & 0 & c\beta(1-\rho)\frac{S_0^*}{N_0^*} + \sigma & c\beta\theta_1(1-\rho)\frac{S_0^*}{N_0^*} - \kappa_4 & c\beta\theta 2(1-\rho)\frac{S_0^*}{N_0^*} & 0 \\ 0 & 0 & 0 & \delta & -\kappa_5 & 0 \\ 0 & 0 & 0 & \xi & \eta & -\kappa_6 \end{bmatrix}$$

It follows that the trace and determinant of $J(E_0)$ are respectively, given by

$$Tr(J(E_0)) = \pi + c\beta((1-\rho)\theta_1 + \rho)\frac{S_0^*}{N_0^*} - \sum_{i=1}^{6}\kappa_i$$

and

$$Det(J(E_0)) = \kappa_3\kappa_4\kappa_5\kappa_6\left(1 - c\beta\left(\frac{(1-\rho)(\delta\theta_2 + \kappa_5\theta_1)\kappa_3 + ((\sigma\theta_1 + \kappa_4)\kappa_5 + \delta\sigma\theta_2)\rho}{\kappa_3\kappa_4\kappa_5}\right)\frac{S_0^*}{N_0^*}\right)(\pi-\mu)(\mu+\gamma_1+\gamma_2)$$

which upon simplification we get

$$Det(J(E_0)) = (1-\mathscr{R}_0)(\pi-\mu)(\mu+\gamma_1+\gamma_2)\kappa_3\kappa_4\kappa_5\kappa_6 \tag{31}$$

Using trace determinant approach, the risk free equilibrium point E_0 is locally stable if and only if $Tr(J(E_0)) < 0$ and $Det(J(E_0)) > 0$. Since $\mu < \pi$, it follows that, E_0 is asymptotically stable whenever $\mathscr{R}_0 < 1$ and unstable when $\mathscr{R}_0 > 1$ provided that

$$\frac{\pi N_0^* + c\beta\left((1-\rho)\,\theta_1 + \rho\right) S_0^*}{N_0^*\left(\sum_{i=1}^{6} \kappa_i\right)} < 1$$

□

3.1.2. *Global stability conditions for the risk-free equilibrium.* The roles $\mathscr{R}_0$ in global stability analysis can be traced back from [26, 27] who established two axioms ((H1) and (H2)), that need to be satisfied to guarantee the global asymptotic stability of the risk free state. Following [26] we present system (2) in the form:

$$\begin{aligned} \frac{dX}{dt} &= f(X,Z) \\ \frac{dZ}{dt} &= g(X,Z),\ g(X,0) = 0 \end{aligned} \tag{32}$$

where $X = (S,P,R)^T \in \mathbb{R}^3$ whose components denote the number of individuals at risk free (non alcoholic individuals) and $Z = (L,M,A)^T \in \mathbb{R}^3$ whose components denote the number of alcoholic individuals at different risk levels. The coordinate $(X_0^*, 0) \in \mathbb{R}^6$ denotes the risk free equilibrium for the model and $(\cdot)^T$ denotes a vector transpose. Now with the system (2) in terms of X and Z, the following axioms combined guarantee globally asymptotically stability.

(H1) For $\dfrac{dX}{dt} = f(X,0)$, X_0^* is globally asymptotically stable (G.A.S).
(H2) $g(X,Z) = BZ - \tilde{g}(X,Z)$; $\tilde{g}(X,Z) \geq 0$, $\forall X,Z \in \Omega$

we define $B = D_Z g(X_0^*, 0)$ as a Metzler matrix and Ω is the region where model (2) makes biological sense. The following theorem hold true provided that the system (2) satisfies the above two conditions (H1) and (H2).

Theorem 3.2. *The risk free equilibrium E_0 of the model (2) is globally asymptotically stable (g.a.s) of the system (2) provided that axioms (H1) and (H2) are satisfied.*

Proof. We re-write the model equation (2) into $f(X,Z)$ and $g(X,Z)$ such that $X=(S,P,R)$, $Z=(L,M,A)$. We have

$$f(X,Z)=\begin{cases}\dot{S} &= -(\kappa_1+\lambda)S+\gamma_2 P+\omega R+(1-\phi)\pi N\\ \dot{P} &= \gamma_1 S-\kappa_2 P+\nu L+\tau M+\psi A+\phi\pi N\\ \dot{R} &= \xi M+\eta A-\kappa_6 R\end{cases} \tag{33}$$

$$g(X,Z)=\begin{cases}\dot{L}= & \lambda\rho S-\kappa_3 L\\ \dot{M}= & \lambda(1-\rho)S+\sigma L-\kappa_4 M\\ \dot{A}= & \delta M-\kappa_5 A\end{cases} \tag{34}$$

Evaluating the subsystem (33) at $\left(X_0^*,0\right)$ we get

$$f(X_0^*,0)=\begin{bmatrix}-\kappa_1 S_0^*+\gamma_2 P_0^*+(1-\phi)\pi N_0^*\\ \gamma_1 S_0^*-\kappa_2 P_0^*+\phi\pi N_0^*\\ 0\end{bmatrix}$$

which satisfies axiom (H1). Now, from the susbsystem (34) we re-write $g(x,z)$ such that

$$g(X,Z)=BZ-\tilde{g}(X,Z)$$

such that

$$B=\begin{bmatrix}-\kappa_3+\dfrac{c\beta\rho}{N} & \dfrac{c\beta\theta_1\rho}{N} & \dfrac{c\beta\theta_2\rho}{N}\\ \sigma+\dfrac{c\beta(1-\rho)}{N} & -\kappa_4+\dfrac{c\beta\theta_1(1-\rho)}{N} & \dfrac{c\beta\theta_2(1-\rho)}{N}\\ 0 & \delta & -\kappa_5\end{bmatrix} \text{ and } \tilde{g}(X,Z)=\begin{bmatrix}\lambda\rho(S-1)\\ \lambda(1-\rho)(S-1)\\ 0\end{bmatrix}.$$

Since B is a Metzler matrix and $\tilde{g}(X,Z)\geq 0$ provided that $S(t)\geq 1$ and $\rho\leq 1$, then axioms (H1) and (H2) are satisfied. It is clear that the equilibrium point E_0 is a globally asymptotically stable equilibrium. Hence, by the above theorem E_0 is globally asymptotically stable. □

TABLE 1. Description of parameters for model (2) and their sensitivity indices on $\mathscr{R}_0$

S/N	Parameters (y)	Value	Source	Sensitivity Index $\Upsilon_{y_i}^{\mathscr{R}_0}$
1	π	$0.0310yr^{-1}$	-	$+1.0000$
2	μ	$0.02yr^{-1}$	[9]	-1.6901
3	α	$0.0350yr^{-1}$	[9]	-0.0456
4	δ	$0.0075yr^{-1}$	[9]	-0.1325
5	σ	$0.0100yr^{-1}$	[28]	-0.0041
6	ν	$0.0020yr^{-1}$	-	-0.0048
7	τ	$0.0016yr^{-1}$	-	-0.0477
8	ψ	$0.0100yr^{-1}$	-	-0.0130
9	ξ	$0.0025yr^{-1}$	[9]	-0.0746
10	η	$0.0050yr^{-1}$	[9]	-0.0065
11	ω	$0.0010yr^{-1}$	[28]	$-$
12	γ_1	$0.1300yr^{-1}$	[29]	-0.3333
13	γ_2	$0.2400yr^{-1}$	[29]	$+0.3524$
14	θ_1	1.0002	-	$+0.8515$
15	θ_2	1.0005	[9]	$+0.0913$
16	ϕ	0.6000	-	-0.0484
17	ρ	0.06500	-	$+0.0131$
18	β	0.2500	[9]	$+1.0000$
19	c	$24yr^{-1}$	-	$+1.0000$

3.2. Sensitivity analysis. To determine the best solution in reduction of health risks and mortality associated to alcoholism, it is important to understand the relative importance of the different parameters responsible for risk transmission dynamics and prevalence [30]. We perform sensitivity analysis of sensitive parameters in order to determine the input parameters with the most contribution to the output variability of $\mathscr{R}_0$ [7, 31, 32]. We do this based on understanding

that, the initial spread of health risk associated to alcoholism in the model presented in (2) is directly related to the basic risk reproduction number $\mathscr{R}_0$ [30].

Using normalized forward sensitivity index method - a partial differential technique - we calculate the sensitivity coefficients, $\Upsilon_{y_i}^{\mathscr{R}_0}$, for each of the input parameters $y_i \in y$ for $\mathscr{R}_0$ in equation (29) to the output variable $\mathscr{R}_0$ [31, 30]. We therefore have a vector y given as a set of parameters in which some of them serve as independent input for the corresponding dependent output $\mathscr{R}_0$. The sensitivity coefficients, defined as the measure of the relative change in the dependent variable when the independent variables change one at a time [30, 31, 32], explain the impact of each parameter value in the health risk transmission threshold. Researchers conduct sensitivity analyses for a number of reasons in an attempt to answer their research questions. However, the robustness of the model predictions to parameter values is the main impetus of performing sensitivity analysis to many modelers [30, 32].

Consider an explicit formula for $\mathscr{R}_0$ given in equation (29), to each of the input parameters we derive an analytical expression for the sensitivity coefficient of $\mathscr{R}_0$, with respect to the parameter y_i as

$$\Upsilon_{y_i}^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial y_i} \times \frac{y_i}{\mathscr{R}_0} \tag{35}$$

where y_i is the i^{th} parameter as shown in Table 1. For example, the sensitivity index of $\mathscr{R}_0$ with respect to β is given by

$$\Upsilon_{\beta}^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial \beta} \times \frac{\beta}{\mathscr{R}_0} = +1.0000$$

Following the similar procedures we calculate the sensitivity coefficients of the rest of parameters and presents their results in Table 1.

Learning from the sensitivity coefficients in Table 1 we know that the most sensitive parameters in making significant changes in $\mathscr{R}_0$ include: the natural mortality rate, μ; recruitment rate, π; measure of influence of risky individuals, β; and the necessary contact rate between a susceptible member and a drinker required to convince the susceptible member to drink, c. The rest of the parameters have smaller sensitivity indices which may not require as much attention to estimate since small perturbation in those parameters lead to insignificant changes in the output variable [32].

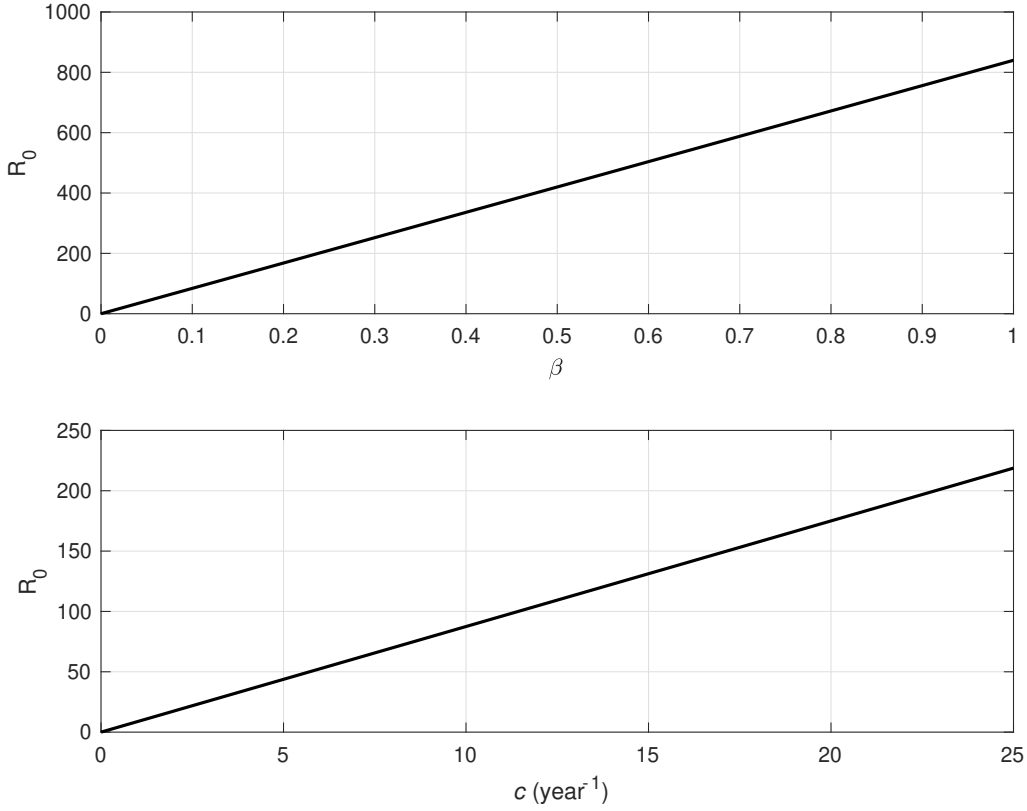

FIGURE 2. Simulation for the effects of β, and c on $\mathscr{R}_0$, using the other parameter values in Table 1

The sensitivity coefficients $\Upsilon_{\mu}^{\mathscr{R}_0} = -1.6901$, and $\Upsilon_{\pi}^{\mathscr{R}_0} = \Upsilon_{\beta}^{\mathscr{R}_0} = \Upsilon_{c}^{\mathscr{R}_0} = +1.0000$ imply that, μ is negatively correlated with $\mathscr{R}_0$ while π, c, and β are positively correlated with $\mathscr{R}_0$. If we increase (or decrease) μ by 10%, the resultant $\mathscr{R}_0$ is also expected to decrease (or increase) by 16.901% of its original value. Similarly, if we increase (or decrease) either π, β or c by 10%, the value of $\mathscr{R}_0$ also increases (or decreases) by 10%. However, despite the significant sensitivity coefficient of π, and μ, they may not be the suitable decision variables in this case. Any deliberate efforts to increase the natural death rate or decrease the recruitment rate in order to reduce the intensity of the spread of the health risks in the community defeats the ethical requirements. We therefore remove π and μ from the list of targeted parameters.

According to the sensitivity analysis results for input parameters for $\mathscr{R}_0$, intervention strategies focusing on discouraging the drinking behaviors at its initiation stage is more effective than

targeting the population proportions at any risky stages. This can be effectively done when we focus on reducing β, and c values to desired level. Reduction of β value implies that lowering down the influence level of the risky population over the susceptible individuals. From social psychology point of view, the influencing capacity of an individual depends on his/her social status in the community. Reduction of β value can be effectively done when influential people in the community are involved in campaigning against unhealthy alcohol drinking habits. Similarly, it is observed that, if the number of times in which risky and non-risky individuals interact socially in an organized setting, c, especially in the events with mixed population spending time together at drinking venues or at social events where alcoholic beverages are freely served is reduced, $\mathscr{R}_0$ will be reduced significantly. However, reduction of c greatly depends on both individual's willingness and desire to change, and the personal efforts dedicated towards the desirable change. Fig. 2 presents the effects of variation of β, and c values on $\mathscr{R}_0$ using parameter values in Table 1 where the particular parameter is not considered as independent variable of $\mathscr{R}_0$.

3.3. Numerical analysis. Where necessary and for the purpose of simulation we use parameter values in Table 1 and initial conditions of state variables, $S(0) = 350,\ P(0) = 80,\ L(0) = 20,\ M(0) = 100,\ A(0) = 30$ and $R(0) = 0$ to simulate the risky population proportions for system (2) against time in years. For avoidance of the danger of confusion, it is important to note that when one parameter takes a variable nature for simulation purposes, the rest of parameters are considered constant with the value as indicated in Table 1.

Figure 3 shows the effects of variation of β, and c values to the risky classes. The values of β was set to increase from 0.1 to 0.9 allowing the increase of step size of 0.1 while c was set to increase from $c = 5$ to $c = 25$ with the step size of 10. The increase in β values translates into the increase of the influencing level of the risky individuals over non risky individuals. Consequently, it increases the recruitment of susceptible population proportion into low risk compartment (see Fig. 3). The variation of β shows no significant effects on the medium risk population compartment, so is the variation of c.

Since alcohol related risks is a staged process depending on the patterns and frequencies of alcohol consumption among others factors, as β increases it is expected that the population size

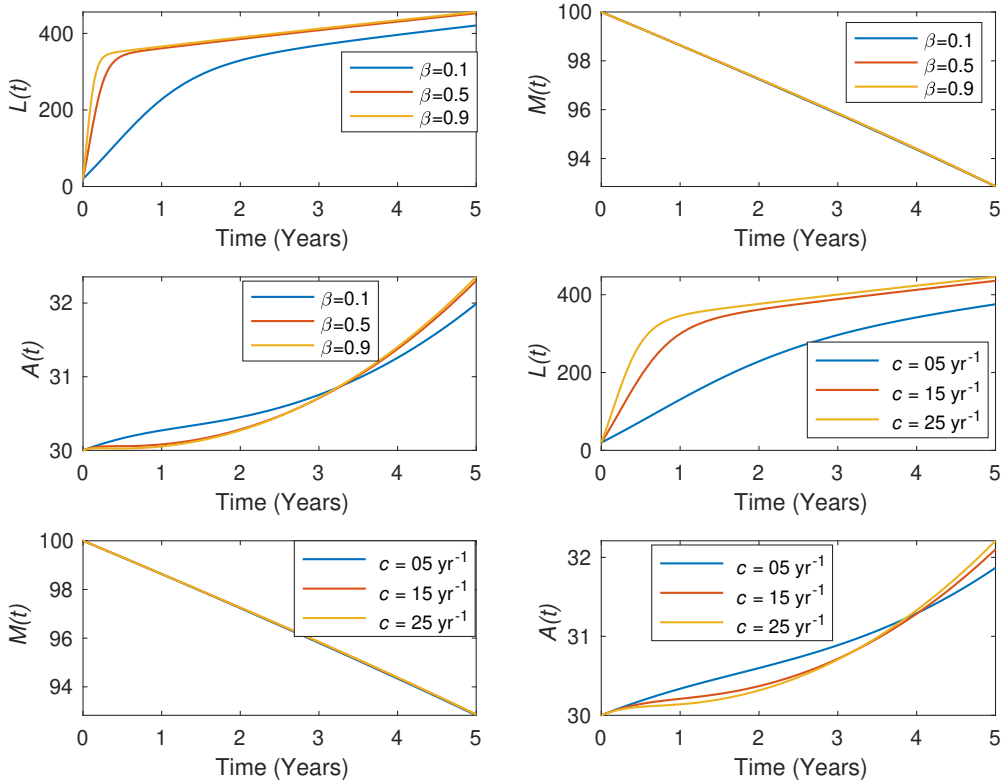

FIGURE 3. Simulation for the effects of varying β and c on risky populations using the other parameter values in Table 1.

of $L(t)$ will increase quantitatively. However, at higher values of β the population proportion of the low risk class converges to a common value within the period of one year. Similarly, we observe that the number of high risk drinkers decreases with the increase of β values for the first three years and later it increases with the increase of β. The effects of variation of the contact rates between risky and non risky individuals giving easy access to alcohol beverages follows similar patterns as that of β variation with β being more efficient in terms of time. That is to say, β based decisions will yield the desired results inn considerably shorter time than is the case for c based decisions.

4. CONCLUSION

This paper studies the dynamics of health risks associated with alcoholism in a community in which a proportion of susceptible population receives a social cultural protection acquired from the religious beliefs. Except for occasional drinkers, this work considers that some people voluntarily quit alcoholic consumption as their personal efforts to change their drinking status or upon medical grounds. The equilibria of the model system are found and their stability are analyzed. The basic risk reproduction number of the model is computed and the sensitivity analysis for its input parameters is established. The sensitivity analysis of $\mathscr{R}_0$ suggested that discouraging alcohol drinking behavior at its initiation stage is more effective in the control and reduction of the health related risks associated with alcoholism than targeting other populations already at risk.

Numerical simulations are performed to illustrate the effects of the most sensitive parameters to various risky classes. The numerical simulations results are used to confirm the sensitivity analysis findings. For effective control of the health related risks associated with alcoholism in the community, public educational campaigns would do a better job. When the influential characters are involved in the public education campaign, better results are expected since societal influence is the key factor recruiting people into the behavior. Since alcohol drinking compromises the quality of decision making to its consumers, it can be closely associated with other irresponsible behaviors such as irresponsible sexual behaviors, the combination of alcohol related risks and the risk of contacting sexually transmitted diseases in one model will make an interesting study.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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Fuzzy Modeling for the Dynamics of Alcohol-Related Health Risks with Changing Behaviors via Cultural Beliefs

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***Abstract**: This study proposes and analyzes a fuzzy model for the health risk challenges associated with alcoholism. The fuzziness gets into the system by assuming uncertainty condition in the measure of influence of the risky individual and the additional death rate. Specifically, the fuzzy numbers are defined functions of the degree of peer influence of a susceptible individual into drinking behavior. The fuzzy basic risk reproduction number, $\mathscr{R}_0^f(x)$, is computed by means of Next-Generation Matrix and analyzed. The analysis reveals that health risk associated with alcoholism can be effectively controlled by raising the resistance of susceptible individuals and consequently reducing their chances of initiation of drinking behavior.*

1. Introduction

Alcohol consumption that leads to health-related epidemics has been one of the leading causes of mortality of individuals worldwide. In Tanzania, just like in other developing countries, the situation gets worse as alcohol consumption becomes an important part of community social activities. Available literature cite alcohol consumption behavior is one among the risk factors for various health challenges in Tanzanian population [1–9]. While communities identify themselves with alcohol consumption, a large part of the religious population forbid its uptake [10, 11].

Application of fuzzy set theory in solving dynamical systems has recently been an interesting research area [12–15]. In this study, we use an application of fuzzy set theory approach to analyze SPLMAR model. Two main distinguishing aspects of the model are as follows: social cultural beliefs as an integral part of the society and the staged process in which alcoholism behaviors take in the spread of health risks are considered.

2. Fuzzy Model Formulation

This paper presents fuzzy model with six time-dependent state variables described as follows: susceptible population, $S(t)$; protected population, $P(t)$; and low-risk population, $L(t)$. Others include moderate-risk population, $M(t)$; alcoholics, $A(t)$; and recovered population, $R(t)$. The model considers multi-risk levels established under the drinking cultures of Tanzanian population with active religious beliefs.

$$\begin{aligned}
\dot{S} &= -(\mu+\gamma_1+\lambda)S+\gamma_2 P+\omega R+(1-\phi)\pi N,\\
\dot{P} &= \phi\pi N+\gamma_1 S-(\mu+\gamma_2)P+\nu L+\tau M+\psi A,\\
\dot{L} &= \lambda\rho S-(\mu+\nu+\sigma)L,\\
\dot{M} &= \lambda(1-\rho)S+\sigma L-(\mu+\tau+\delta+\xi)M,\\
\dot{A} &= \delta M-(\mu+\alpha(x)+\eta+\psi)A,\\
\dot{R} &= \xi M+\eta A-(\mu+\omega)R
\end{aligned} \tag{1}$$

with $S(0)>0, P(0)>0, L(0)\geq 0, M(0)\geq 0, A(0)\geq 0, R(0)\geq 0$ and $N=S+P+L+M+A+R$.

We propose a fuzzy model using x as the variable describing the degree of peer influence of a susceptible individual to initiate drinking behavior. While $\beta(x)$ is attributed to the spread of health risks associated with alcoholism in the community, $\alpha(x)$ translates the consequences of health risks by means of additional death rate.

3. Analysis of Fuzzy System

Let x_{min} be the minimum amount of the degree of peer influence required to have an impact on a susceptible member. In view of this, the impact of behavioral influence is considered negligible whenever $x < x_{min}$. The quantity x_{min} is considered as a parameter whose exact value would depend upon both the attitude, public opinions towards the drinking behavior or the drinking individual, and willingness of a susceptible individual to conform with the peer pressures. As x increases, the behavior inducement rate increases to the maximum which is equal to unity at $x \geq x_0$. Further, it is also assumed that the degree of peer influence is bounded above at $x = x_{max}$ forming an upper bound. Therefore, the values of x with an effect to the system lies in the interval of $x_{min} \leq x \leq x_{max}$.

The fuzzy basic risk reproduction number R_0^f, which can be defined as the average number of secondary risk cases caused by one alcohol drinker introduced into entirely susceptible population is given by $\mathscr{R}_0^f(x) = \frac{1}{\epsilon_0} FEV[\epsilon_0 \mathscr{R}_0(x)]$. We use the linguistic variable $\Pi(x)$ to define different levels of influence, that is, low, medium, and high influence.

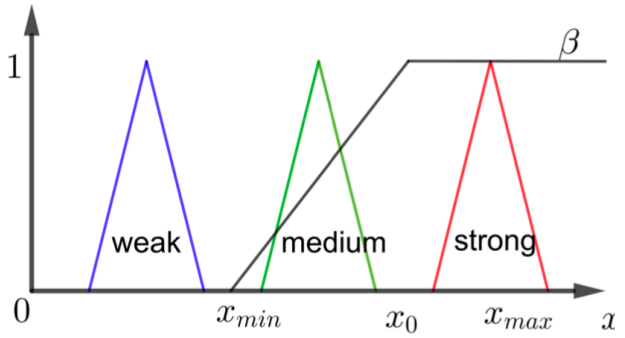

Figure 1: Classification of linguistic variable $\Pi(x)$

4. Results and Conclusion

The fuzzy model analysis suggests that, health risks associated with alcoholism can be effectively controlled by controlling $\mathscr{R}_0^f(x)$ which can be reduced by increasing the value of x_{min}. This may be done through provision of public health education which increases the resistance of susceptible individuals. If the amount of degree of peer of influence to an individual is low, the alcohol-related health risks in the community will be reduced since natural factors in the community provide virtual immunity.

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