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MATHEMATICAL MODEL FOR THE DYNAMICS OF ALCOHOL-MARIJUANA CO-ABUSE

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ABSTRACT. A mathematical model for the dynamics of alcohol-marijuana coabuse is presented in this work. In the past years legalization of recreational marijuana in several states in the United States has added a new layer to alcohol addiction. Much research has been done for alcohol addiction or drug abuse independently, but few include the incidence of marijuana use for alcohol users. A compartmental epidemiological model is used, and results such as the existence and boundedness of solutions, the basic reproduction number using the next-generation method, the disease-free equilibrium, and an analytical expression for the endemic equilibrium are included. Numerical simulations with parameters obtained from data in the United States are performed for different compartments of the population as well as the reproduction number for the alcohol and marijuana sub-models. The model can be adapted for different regions worldwide using appropriate data. This work contributes to understanding the dynamics of the co-abuse of addictive substances. Even though alcohol and marijuana are both legal, they can be of great harm to the brain of the individual when combined, having tremendous consequences for society as a whole. Creating awareness of a public health concern with facts based on scientific research is the ultimate goal of this work.

1. INTRODUCTION

Alcohol consumption is a widely accepted social practice between friends and family and sometimes in work environments. Despite its status as an intoxicating substance, classification as a central nervous system depressant [16], and its risk of addiction for one in ten who try alcohol, networking events, business meetings,

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and social gatherings normalize the use of this substance. Since alcohol is socially acceptable, many users disregard the consequences that come with its use [16]. Currently, alcohol use can be placed on a spectrum. Drinking habits can be classified as occasional, such as casual drinking at social events, as moderate, including binge drinking, or as heavy, which is the most dangerous and can lead to many health problems and even death.

The behavior becomes a public health crisis when someone cannot cut back on consumption despite efforts to control consumption habits. This leads to physical health issues, possible driving while intoxicated, and the less quantified suffering that abusers experience with out-of-control behavior when intoxicated [12]. In addition to drinking alcohol, cigarette smoking, marijuana, and other drug use while drinking alcohol is common, as studies show that using one often involves using the other in the same event/occurrence [30].

Studies have shown that peer pressure in adolescence plays a significant role in young adults starting to consume alcohol and/or marijuana [28]. Furthermore, college students are constantly participating in activities that involve the use of alcohol and/or marijuana [20]. Some studies have shown that certain conditions in individuals make them more vulnerable to becoming addicted to alcohol and/or marijuana [14].

Researchers from NIH:NIAAA present a comprehensive study on risk factors periodically, [19], where several studies have shown the severity of alcoholism in modern society. Another study [27] examines alcohol abuse using a mathematical model with recovery and relapse from epidemiology. Neurologists have studied the brain during addictions and concluded that when trauma happens at an early age, the brain structure of an individual may change predisposing someone to be more likely to be addicted than others [14]. Additionally, the use of alcohol and/or marijuana during pregnancy causes "impaired neuro-development" [17].

From well-known studies on rats in the 1950's where pleasure in the brain was identified when certain areas were electronically stimulated and rats would seek that sensation despite negative consequences, scientists observed something that is now termed "hijacking" of the brain [23]. This is where the brain confuses the pleasurable results of the drug with survival such as eating for nutrients or procreating to perpetuate the species [23].

Similar studies have yet to be done for the use of marijuana and the nature of each differs in that alcohol is a depressant but marijuana has other properties, [11]. In the United States the use of medical and recreational marijuana is fully legal and decriminalized in several states [33]. However not enough research has been done on the effects of the use of marijuana, or the co-abuse of alcohol and marijuana. Many teenagers are engaging in the use of alcohol and marijuana at an early age, without knowing the side effects. Unfortunately for many of them, the recreational use of this combination ends in a disorder they cannot control, affecting their health, their family environment, and their future as productive members of society. In the majority of cases, treatment plans are not affordable for most families, and numbers as few as one out of ten of those who have a substance use disorder attend treatment with approximately 13% attending alcohol treatment, 50% drugs only, and 30% both [22].

There is plenty of literature using mathematical models to analyze the dynamics of alcohol addiction or marijuana addiction individually, see for example [9, 13, 25, 27, 29]. However, the literature surrounding the co-abuse of alcohol and marijuana, to the authors' knowledge, is sparse. As the use of marijuana becomes socially acceptable and legalized in many states, related data for the co-abuse is not totally available. Since multiple health organizations show several studies where the co-abuse of alcohol and marijuana is at the top of health concerns in the United States [1, 5, 6], the necessity of developing mathematical models to contribute to the analysis of alcohol-marijuana co-abuse is imperative.

A mathematical model for the dynamics of the co-abuse of alcohol and marijuana is presented in this work, by using a system of ordinary differential equations under certain assumptions for the whole population. In the first part of this work, the analysis of the model is carried out. The system was divided in two subsystems, one corresponding to the dynamics of alcohol, and the second to the dynamics of marijuana, the evaluation of the basic reproduction number was performed for each sub-system by using the next generation method [31]. The basic reproduction number for the entire system is evaluated in terms of the population parameters. Stability results for the **disease-free** equilibrium are included. Furthermore a section with the analytic solution for the endemic equilibrium is included. The last section of this work includes multiple simulations for different compartments of the population using parameters for the population and simulations for the reproduction number for the sub-systems as well as the entire system. Most of these parameters were gathered from health organizations in the United States, [1,4,6].

By using mathematical modeling, the ultimate goal of this work is to contribute to understanding the co-abuse of alcohol and marijuana as it is now a public concern of the 21st century and to create awareness in teenagers, young-adults, and adults of the consequences of co-abuse. Public health reports indicate that even though both alcohol and marijuana are legal, this does not mean they are good for consumption together. This model can be used for different geographic regions, by changing the parameter values in the simulations.

2. Model Formulation

A compartmental epidemiological model is used to describe the dynamics of the co-abuse of alcohol and marijuana in the population. The total population is divided in 13 compartments, S(t) – Susceptible, $E_a(t)$ – latent alcohol consumers, $E_m(t)$ – latent marijuana consumers, $E_{am}(t)$ – latent alcohol-marijuana consumers, $U_a(t)$ – alcohol users, $U_m(t)$ – marijuana users, $U_{am}(t)$ – alcohol-marijuana users, $T_a(t)$ – alcohol users in treatment, $T_m(t)$ – marijuana users in treatment, T_{am} – alcohol-marijuana users in treatment, $Q_a(t)$ – alcohol quitters (recovery from alcoholism), $Q_m(t)$ – marijuana quitters (recovery from marijuana abuse), $Q_{am}(t)$ – alcohol-marijuana quitters (recovery from alcohol-marijuana co-abuse). For ease of exposition, we will simply write *compartments*, rather than list each compartment individually. With the above assumptions in place, we have

$$N(t) = S(t) + E_a(t) + E_m(t) + E_{am}(t) + U_a(t) + U_m(t) + U_{am}(t) + T_a(t) + T_m(t) + T_{am}(t) + Q_a(t) + Q_m(t) + Q_{am}(t).$$

Our model draws inspiration from a co-abuse model for alcohol and methamphetamine presented in [26]. We build on this model by introducing a latent compartment E consisting of users who are not yet addicted. It is assumed that susceptible individuals become alcohol/marijuana users after an effective contact with alcohol/marijuana users. In this model, latent classes represent individuals who use alcohol/marijuana moderately, user classes represent individuals who use alcohol/marijuana on a regular basis (these are the infected individuals in general epidemiological terminology, meaning they are alcoholic individuals or marijuana addicted individuals or both). In Table 1, the symbol * indicates that the range for those parameters were estimated for numerical simulation purposes. Those values are still a very good approximation following the literature. Data collection for marijuana use is still in process due to the fact that legalization of recreational marijuana is pretty recent in many states in the United States. The main public health organizations are making a great effort to collect data as mentioned in [18, 32].

We assume a homogeneous mixing of populations. A complete analysis for the theory that human social networks may exhibit a "three degrees of influence" property was included in [15], which suggests that individuals acquire habits of alcohol use, marijuana use, or both, based on interactions with different populations. In this model we also assume that individuals who consume alcohol at any level, including during treatment (rehabilitation), contribute to the new alcohol user population. Individuals from T_{am} relapsing during treatment from abusing multiple drugs [4,21] also have the potential to influence susceptible individuals to drink alcohol. Therefore, individuals adopt the habit of alcohol consumption at the rate λ_1 given by the following expression:

$$\lambda_1 = \beta_1 \left(\frac{E_a + \theta_1 U_a + \theta_2 T_a + E_{am} + \theta_3 U_{am} + \theta_4 T_{am}}{N} \right)$$

where β_1 denotes the effective contact rate (the contact with an alcoholic drinker that will result in one taking alcohol). Similarly, individuals acquire the habit of smoking marijuana at the rate λ_2 given by

$$\lambda_2 = \beta_2 \left(\frac{E_m + \epsilon_1 U_m + \epsilon_2 T_m + E_{am} + \epsilon_3 U_{am} + \epsilon_4 T_{am}}{N} \right).$$

where β_2 denotes the effective contact rate (the contact with a marijuana user that will result in one smoking marijuana).

It is assumed that individuals under alcohol/marijuana treatment tend to have lower recruitment rates relative to alcoholics without treatment or marijuana addicts. Then the following relations hold: $\theta_1 > 1$, $\theta_3 > 1$, $\theta_2 < 1$, $\theta_4 < 1$, $\epsilon_1 > 1$, $\epsilon_3 > 1$, $\epsilon_2 < 1$, $\epsilon_4 < 1$.

Epidemiological models for co-abuse or co-infections are of tremendous interest in recent research [8]. For example in [7] a complete study for the co-infection between HIV and HCV was developed.

The following system of ordinary differential equations captures the dynamics of alcohol-marijuana co-abuse:

$$\frac{dS}{dt} = \Lambda - (\lambda_1 + \lambda_2 + \mu)S \tag{1}$$

$$\frac{dE_a}{dt} = \lambda_1 S + \rho_1 E_{am} - (\eta_a \lambda_2 + \sigma_a + \mu) E_a$$
(2)

$$\frac{dE_{am}}{dt} = \eta_a \lambda_2 E_a + \eta_m \lambda_1 E_m - (\rho_1 + \rho_2 + \sigma_{am} + \mu) E_{am}$$
(3)

$$\frac{dE_m}{dt} = \lambda_2 S + \rho_2 E_{am} - (\sigma_m + \eta_m \lambda_1 + \mu) E_m \tag{4}$$

$$\frac{dU_a}{dt} = \sigma_a E_a + \rho_3 U_{am} + \psi_a T_a - (\eta_a \lambda_2 + \alpha_a + \xi_a + \delta_a + \mu) U_a$$
(5)
$$\frac{dU_{am}}{dU_{am}} = \nabla_a E_a + \rho_3 U_{am} + \psi_a T_a - (\eta_a \lambda_2 + \alpha_a + \xi_a + \delta_a + \mu) U_a$$
(5)

$$\frac{\partial_{am}}{\partial t} = \sigma_{am} E_{am} + \eta_a \lambda_2 U_a + \eta_m \lambda_1 U_m + \psi_{am} T_{am} - (\rho_3 + \rho_4 + \alpha_{am} + \xi_{am} + \delta_{am} + \mu) U_{am} \quad (6)$$

$$\frac{dU_m}{dt} = \sigma_m E_m + \rho_4 U_{am} + \psi_m T_m - (\eta_m \lambda_1 + \alpha_m + \xi_m + \mu) U_m \tag{7}$$

$$\frac{dT_a}{dt} = \alpha_a U_a - (\psi_a + \gamma_a + \mu)T_a \tag{8}$$

$$\frac{dI_{am}}{dt} = \alpha_{am}U_{am} - (\psi_{am} + \gamma_{am} + \mu)T_{am}$$
⁽⁹⁾

$$\frac{dI_m}{dt} = \alpha_m U_m - (\psi_m + \gamma_m + \mu)T_m \tag{10}$$

$$\frac{dQ_a}{dt} = \xi_a U_a + \gamma_a T_a - \mu Q_a \tag{11}$$

$$\frac{dQ_{am}}{dt} = \xi_m U_{am} + \gamma_{am} T_{am} - \mu Q_{am} \tag{12}$$

$$\frac{dQ_m}{dt} = \xi_m U_m + \gamma_m T_m - \mu Q_m \tag{13}$$

Figure 1 represents the transition between compartments for the alcohol-marijuana co-abuse model.

In the next section a complete mathematical analysis is developed, positiveness and boundedness of solutions are always fundamental properties for a consistent dynamical system in epidemiology. The basic reproduction number is included. The

TABLE 1. Parameters values¹

Symbol	Description	Value
Λ	Recruitment rate for susceptible	.0546 [1]
μ	Natural mortality rate	0.001 [2]
β_1	Alcohol transmission rate	$.2427 \ [3, 10]$
β_2	Marijuana transmission rate	0.169[1]
σ_a	Alcoholism effective rate	0.056~[1]
σ_m	Marijuana users effective rate	0.011 [1]
σ_{am}	Co-abusers effective rate	[0.01-0.06] [1]
α_a	Alcoholism treatment rate	0.131 [1]
α_m	Marijuana users treatment rate	0.09 [1]
α_{am}	Co-abusers treatment rate	0.32 [1]
γ_a	Alcoholism recovery rate after treatment	0.87 [24]
γ_m	Marijuana users recovery rate after treatment	0.45 [1]
γ_{am}	Co-abusers recovery rate after treatment	[0.1-0.4] [1]
θ_1, θ_3	Weight contributions to λ_1 from U_a, U_{am}	[1.01-1.05] $[1]$
$ heta_2, heta_4$	Weight contributions to λ_1 from T_a, T_{am}	[0.01-0.03] $[1]$
ϵ_1, ϵ_3	Weight contributions to λ_2 from U_m, U_{am}	[1.01-1.08] $[1]$
ϵ_2,ϵ_4	Weight contributions to λ_2 from T_m, T_{am}	[0.4-0.7] [1]
η_a	Rate at which alcohol users become marijuana users	[0.5-0.9] [1]
η_m	Rate at which marijuana users become alcohol users	[0.5-0.9] [1]
ψ_a	Relapsing rate from alcoholism	0.13 [24]
ψ_m	Relapsing rate from marijuana use	$[0.4 - 0.6]^*$
ψ_{am}	Relapsing rate from Co-abusers	$[0.4 - 0.6]^*$
ξ_a	Quitting rate from alcohol abusers without treatment	0.36[1]
ξ_m	Quitting rate from marijuana abusers without treatment	$[0.1 - 0.4]^*$
ξ_{am}	Quitting rate from Co-abusers without treatment	$[0.2 - 0.6]^*$
δ_a	Alcohol-induced mortality rate	.000392 [3]
δ_{am}	Co-abusers mortality rate	$[0.0004 - 0.0007]^*$
ρ_1	Rate at which individuals from E_{am} -class back to E_a -class	$[0.4 - 0.7]^*$
ρ_2	Rate at which individuals from E_{am} -class back to E_m -class	$[0.4 - 0.7]^*$
ρ_3	Rate at which Co-abusers back to U_a -class	$[0.4 - 0.8]^*$
ρ_4	Rate at which Co-abusers back to U_m -class	$[0.2 - 0.7]^*$

¹The scenarios used to choose most of the parameters were obtained from statistics found in [1,5,6] for the state of Virginia, United States in 2017. For example, σ_a , σ_m , and α_a are taken from the Behavioral Health Barometer for Virginia which can be found in [1]. Other parameters, in particular those for co-abuse such as α_{am} , were estimated using rates for general drug and alcohol co-abuse or by using compartmental rates as bounds. For example, bounds for σ_{am} were assumed based on σ_a and σ_m . The validity of these bounds, such as σ_{am} can be checked using Crosstab, also from [1]. Individual state level data is not available in Crosstab for general public use. In the case of σ_{am} , this Crosstab tells us that almost 3% of Virginians and Marylanders co-abused alcohol and marijuana in 2017.



FIGURE 1. Visual representation of alcohol-marijuana co-abuse model.

free disease equilibrium and an analytical expression for the endemic equilibrium point are included. Some stability results are proven as well.

3. Mathematical Analysis for the Alcohol-Marijuana Co-Abuse Model

Positiveness and long-term behavior for the solutions of System (1)–(13) are established in this section. Assume that the variables and the parameters are all non-negative for all times $t \ge 0$.

Theorem 1. If each compartment is non-negative at t = 0, then each compartment is non-negative for time t > 0. Moreover,

$$\lim_{t \to \infty} N(t) \le \frac{\Lambda}{\mu}.$$

Proof. Assume that T is the maximum time for the epidemic. That is,

$$T = \sup \{S > 0, E_a \ge 0, E_{am} \ge 0, \dots, Q_m \ge 0\} \in [0, t].$$

Therefore for T > 0, from System (1)–(13), equation (1) is equivalent to

$$\frac{dS}{dt} + (\mu + \lambda_1 + \lambda_2)S = \Lambda,$$

from which it holds

$$S(T) \ge S(0) \exp\left\{-\mu T + \int_0^t (\lambda_1(s) + \lambda_2(s)) ds\right\}.$$

Hence, $S(T) \ge 0$ for all T > 0.

From System (1)-(13) equation (2),

$$\begin{aligned} \frac{dE_a}{dt} &= \lambda_1 S + \rho_1 E_{am} - (\eta_a \lambda_2 + \sigma_a + \mu) E_a \\ &\geq -(\eta_a \lambda_2 + \sigma_a + \mu) E_a. \end{aligned}$$

Then

$$E_a(T) \ge E_{a0} \exp\left\{-\left((\sigma_a + \mu)t + \int_0^t \eta_a \lambda_2(s)ds\right)\right\}.$$

Hence, $E_a(T) \ge 0$ for all T > 0. The positiveness of the remaining compartments can be shown in a similar way.

The evolution change in the population is given by

$$\frac{dN}{dt} = \Lambda - \mu N - \delta_a - \delta_{am} U_{am}.$$

Then

$$\frac{dN}{dt} \le \Lambda - \mu N,$$

from which it holds

$$\frac{dN}{dt} + \mu N \le \Lambda.$$

Then

$$N(t) \le \frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right) \exp(-\mu t)$$

Since $(N_0 - \Lambda/\mu)$ is a constant and $\mu > 0$,

$$\frac{\Lambda}{\mu} + \left(N_0 - \frac{\Lambda}{\mu}\right) \exp(-\mu t) \to \frac{\Lambda}{\mu} \text{ as } t \to \infty.$$

So $\lim_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}$ as desired.

The feasible region D, for System (1)–(13) is therefore

$$D = \left\{ (S, E_a, E_{am}, E_m, \dots, Q_a, Q_{am}, Q_m) \in \mathbb{R}^{13}_+ \mid N \le \frac{\Lambda}{\mu} \right\}.$$

3.1. **Basic reproduction number.** The basic reproduction number is the number of secondary infections. In the context of this model, if an individual is an alcoholic or a marijuana user or both, after an effective contact with susceptible individuals, the basic reproduction number corresponds to how many susceptible individuals become alcoholics or marijuana codependent or alcohol-marijuana co-abusers. The next generation matrix method is used to find the basic reproduction number for the co-abuse model System (1)–(13) [31]. First the basic reproduction number is found for the alcohol model, denoted \mathscr{R}_{a0} . Second the basic reproduction number \mathscr{R}_{am} is the larger of \mathscr{R}_{a0} and \mathscr{R}_{m0} . So one only needs to calculate the reproduction number of the co-abuse model, see [7] for a detailed calculation of the basic reproduction number of the co-abuse model.

In the next sub-sections, System (1)-(13) is sub-divided into two models, one corresponding to the dynamics of alcohol use, and the other to the marijuana use.

3.2. Alcohol abuse model. Taking S together with the first column of Figure 1, one can see that the alcohol abuse model is given by

$$\frac{dS}{dt} = \Lambda - (\mu + \tilde{\lambda}_1)S \tag{14}$$

$$\frac{dE_a}{dt} = \tilde{\lambda}_1 S - (\sigma_a + \mu) E_a \tag{15}$$

$$\frac{dU_a}{dt} = \sigma_a E_a + \psi_a T_a - (\alpha_a + \xi_a + \delta_a + \mu)U_a$$
(16)

$$\frac{dT_a}{dt} = \alpha_a U_a - (\psi_a + \gamma_a + \mu)T_a \tag{17}$$

$$\frac{dQ_a}{dt} = \xi_a U_a + \gamma_a T_a - \mu Q_a \tag{18}$$

where

$$\tilde{\lambda}_1 = \beta_1 \bigg(\frac{E_a + \theta_1 U_a + \theta_2 T_a}{N} \bigg).$$

The corresponding matrices to apply to the next generation method to are

$$\mathscr{F} = \begin{bmatrix} \lambda_1 S \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad \mathscr{V} = \begin{bmatrix} (\sigma_a + \mu)E_a \\ -\sigma_a E_a - \psi_a Ta + (\alpha_a + \xi_a + \delta_a + \mu)U_a \\ -\alpha_a U_a + (\psi_a + \gamma_a + \mu)T_a \\ -\Lambda + (\mu + \tilde{\lambda}_1)S \\ -\xi_a U_a - \gamma_a T_a + \mu Q_a \end{bmatrix}$$

The alcohol model has a disease-free equilibrium $X_a^0 = (\Lambda/\mu, 0, 0, 0, 0)$. The matrices F and V at the disease-free equilibrium, following the next generation matrix method in [31], are given by:

$$F = \begin{bmatrix} \beta_1 & \beta_1 \theta_1 & \beta_1 \theta_2 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \sigma_a + \mu & 0 & 0 \\ -\sigma_a & \alpha_a + \xi_a + \delta_a + \mu & -\psi_a \\ 0 & -\alpha_a & \psi_a + \gamma_a + \mu \end{bmatrix}.$$

or

$$V = \begin{bmatrix} \sigma_a + \mu & 0 & 0 \\ -\sigma_a & b_1 & -\psi_a \\ 0 & -\alpha_a & b_2 \end{bmatrix},$$

where $b_1 = \alpha_a + \xi_a + \delta_a + \mu$ and $b_2 = \psi_a + \gamma_a + \mu$. The basic reproduction number \mathscr{R}_{a0} corresponds to the spectral value of the matrix FV^{-1} , so

$$\mathscr{R}_{a0} = \frac{\beta_1}{\sigma_a + \mu} + \frac{\beta_1 \theta_1 \sigma_a}{b_1 (\sigma_a + \mu)(1 - \Phi_a)} + \frac{\beta_1 \theta_2 \sigma_a \alpha_a}{b_1 b_2 (\sigma_a + \mu)(1 - \Phi_a)},$$

where $\Phi_a = \alpha_a \psi_a / b_1 b_2$.

3.3. Marijuana abuse model. Taking S together with the right column of Figure 1, one can see that the Marijuana abuse model is given by

$$\frac{dS}{dt} = \Lambda - (\mu + \tilde{\lambda}_2)S \tag{19}$$

$$\frac{dE_m}{dt} = \tilde{\lambda}_2 S - (\sigma_m + \mu) E_m \tag{20}$$

$$\frac{dU_m}{dt} = \sigma_m E_m + \psi_m T_m - (\alpha_m + \delta_m + \mu)U_m$$
(21)

$$\frac{dT_m}{dt} = \alpha_m U_m - (\psi_m + \gamma_m + \mu)T_m \tag{22}$$

$$\frac{dQ_m}{dt} = \xi_m U_m + \gamma_m T_m - \mu Q_m \tag{23}$$

where

$$\tilde{\lambda}_2 = \beta_2 \bigg(\frac{E_m + \epsilon_1 U_m + \epsilon_2 T_m}{N} \bigg).$$

The corresponding matrices to apply the next generation method to are

$$\mathscr{F} = \begin{bmatrix} \tilde{\lambda}_2 S \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad \text{and} \quad \mathscr{V} = \begin{bmatrix} (\sigma_m + \mu)E_m \\ -\sigma_m E_m - \psi_m Tw + (\alpha_m + \xi_m + \delta_m + \mu)U_m \\ -\alpha_m U_m + (\psi_m + \gamma_m + \mu)T_m \\ -\Lambda + (\mu + \tilde{\lambda}_2)S \\ -\xi_a U_a - \gamma_a T_a + \mu Q_a \end{bmatrix}.$$

Similarly to the alcohol model, the marijuana model has a disease-free equilibrium $X_m^0 = (\Lambda/\mu, 0, 0, 0, 0)$, and the matrices F and V at the marijuana-free equilibrium, following the next generation matrix method, are given by

$$F = \begin{bmatrix} \beta_2 & \beta_2 \epsilon_1 & \beta_2 \epsilon_2 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \sigma_m + \mu & 0 & 0 \\ -\sigma_m & \alpha_m + \xi_m + \mu & -\psi_m \\ 0 & -\alpha_m & \psi_m + \gamma_m + \mu \end{bmatrix}$$

or

$$V = \begin{bmatrix} \sigma_a + \mu & 0 & 0 \\ -\sigma_m & c_1 & -\psi_m \\ 0 & -\alpha_m & c_2 \end{bmatrix},$$

where $c_1 = \alpha_m + \xi_m + \mu$ and $c_2 = \psi_m + \gamma_m + \mu$. The basic reproduction number \mathscr{R}_{m0} is

$$\mathscr{R}_{m0} = \frac{\beta_2}{\sigma_m + \mu} + \frac{\beta_2 \epsilon_1 \sigma_m}{c_1 (\sigma_m + \mu) (1 - \Phi_m)} + \frac{\beta_2 \epsilon_2 \sigma_m \alpha_m}{c_1 c_2 (\sigma_m + \mu) (1 - \Phi_m)}$$

where $\Phi_m = \alpha_m \psi_m / c_1 c_2$.

Then the basic reproduction number for System (1)-(13) is given by

 $\mathscr{R}_{am} = \max\{\mathscr{R}_{a0}, \mathscr{R}_{m0}\}.$

Graphs for \mathscr{R}_{a0} , \mathscr{R}_{m0} , and \mathscr{R}_{am} were obtained using Matlab, the graphs gave us an insight for the behaviour of the basic reproduction number when parameters are varied. Most of the parameters used were found from publicly available data and recent literature.

In Figure 2, notice that $\Re_{a0} > 1$ for values of $\sigma_a < 0.3$. From the data $\sigma_a = 0.056 < 0.3$, meaning that alcoholism is not under control, a similar situation is observed for the marijuana model, since $\Re_{m0} > 1$ for values of $\sigma_m < 0.2$, and from the data $\sigma_m = 0.0011 < 0.2$. So marijuana use is not under control either. For this

range of values, notice that $\mathscr{R}_{am} = \mathscr{R}_{a0}$. Then the alcohol-marijuana co-abuse is an epidemic and can become a pandemic if severe actions are not implemented.



FIGURE 2. \mathscr{R}_{a0} and \mathscr{R}_{m0} with $.1 \leq \sigma_a, \sigma_m \leq .9, \theta_1 = 1.01, \epsilon_1 = 1.05$ and $\theta_2 = .01, \epsilon_2 = .7$. On this interval, $\mathscr{R}_{am} = \mathscr{R}_{a0}$.

Theorem 2. The disease-free equilibrium X_a^0 for the Alcohol abuse model is stable. Proof. The Jacobian for the Alcohol abuse model at X_a^0 , is given by

$$J_a(X_a^0) = \begin{bmatrix} -(\mu + \lambda_1) & 0 & 0 & 0 & 0 \\ \tilde{\lambda}_1 & -(\sigma_a + \mu) & 0 & 0 & 0 \\ 0 & \sigma_a & -(\alpha_a + \xi_a + \delta_a + \mu) & \psi_a & 0 \\ 0 & 0 & \alpha_a & -(\psi_a + \gamma_a + \mu) & 0 \\ 0 & 0 & \xi_a & \gamma_a & -\mu \end{bmatrix}$$

The eigenvalues of $J_a(X_a^0)$ are given by

$$- \mu, \quad -(\mu + \lambda_1), \quad -(\mu + \sigma_a), \\ -\frac{1}{2}\alpha_a - \frac{1}{2}\delta_a - \frac{1}{2}\gamma_a - \mu - \frac{1}{2}\psi_a - \frac{1}{2}\xi_a - \frac{1}{2}(\alpha_a^2 + 2\alpha_a\delta_a - 2\alpha_a\gamma_a + 2\alpha_a\psi_a + 2\alpha_a\xi_a + \delta_a^2 - 2*\delta_a\gamma_a - 2\delta_a\psi_a + 2\delta_a\xi_a + \gamma_a^2 + 2\gamma_a\psi_a - 2\gamma_a\xi_a + \psi_a^2 - 2\psi_a\xi_a + \xi_a^2)^{1/2},$$

and

$$\frac{1}{2} \left(\alpha_a^2 + 2\alpha_a \delta_a - 2\alpha_a \gamma_a + 2\alpha_a \psi_a + 2\alpha_a \xi_a + \delta_a^2 - 2\delta_a \gamma_a - 2\delta_a \psi_a + 2\delta_a \xi_a + \gamma_a^2 + 2\gamma_a \psi_a - 2\gamma_a \xi_a + \psi_a^2 - 2\psi_a \xi_a + \xi_a^2 \right)^{1/2} - \frac{1}{2} \delta_a - \frac{1}{2} \gamma_a - \mu - \frac{1}{2} \psi_a - \frac{1}{2} \xi_a - \frac{1}{2} \alpha_a.$$

notice that the real parts for the five eigenvalues are negative, therefore when tapproaches infinity, the solutions approach X_a^0 .

Similarly, it is possible to show results for the marijuana abuse model, and for the alcohol-marijuana co-abuse model.

4. CHARACTERIZATION OF THE ENDEMIC EQUILIBRIUM

Analytic expressions for the endemic equilibrium are presented in this section. Setting equations from System (1)-(13) to zero and performing several calculations, the endemic equilibrium is obtained depending on the force of infectious λ_1^* and λ_2^* , and the parameters for the model. System (1)-(13) was sub-divided to accomplish this task, the first set of equations correspond to the variables S, E_a, E_{am}, E_m , as follows:

$$S^* = \frac{\Lambda}{\mu + \lambda_1^* + \lambda_2^*} \tag{24}$$

$$E_{am}^* = \frac{\lambda_1^* \lambda_2^* [b_5 \eta_a + b_4 \eta_m]}{b_3 b_4 b_5 [1 - \Phi_3]} S^*$$
(25)

$$E_a^* = \frac{\rho_1}{b_4} E_{am}^* + \frac{\lambda_1}{b_4} S^*$$
(26)

$$E_m^* = \frac{\rho_2}{b_5} E_{am}^* + \frac{\lambda_2}{b_5} S^*$$
(27)

where $b_3 = \rho_1 + \rho_2 + \sigma_{am} + \mu$, $b_4 = \eta_a \lambda_2^* + \sigma_a + \mu$, $b_5 = \eta_m \lambda_1^* + \sigma_m + \mu$ and $\Phi_3 = \frac{\eta_a \lambda_2^* \rho_1}{b_3 b_4} + \frac{\eta_w \lambda_1^* \rho_2}{b_3 b_5}$. The second set of equations correspond to the variables U_a, U_m, U_{am} , as follows:

$$U_{am}^* = \frac{\sigma_a \eta_a \lambda_2^*}{\Gamma_{am} \Phi_4} E_a^* + \frac{\sigma_m \eta_m \lambda_1^*}{\Gamma_{am} \Phi_5} E_m^* + \frac{\sigma_{am}}{\Gamma_{am}} E_{am}^*$$
(28)

$$U_{a}^{*} = \frac{\rho_{3}}{\Phi_{4}} U_{am}^{*} + \frac{\sigma_{a}}{\Phi_{4}} E_{a}^{*}$$
(29)

$$U_m^* = \frac{\rho_4}{\Phi_5} U_{am}^* + \frac{\sigma_m}{\Phi_5} E_m^*, \tag{30}$$

where $c_3 = \psi_{am} + \gamma_{am} + \mu$, $c_4 = \rho_3 + \rho_4 + \alpha_{am} + \xi_m + \delta_{am} + \mu$, $\Phi_4 = \eta_a \lambda_2^* + b_1 - \frac{\alpha_a \psi_a}{b_2}$, $\Phi_5 = \eta_m \lambda_1^* + c_1 - \frac{\alpha_m \psi_m}{c_2}$, and $\Gamma_{am} = c_4 - \frac{\alpha_{am} \psi_{am}}{c_3} - \frac{\rho_3 \eta_a \lambda_2^*}{\Phi_4} - \frac{\rho_4 \eta_m \lambda_1^*}{\Phi_5}$. The last set of equations correspond to the variables T_{am}^* , T_a^* , T_m^* , Q_{am}^* , Q_a^* ,

 Q_m^* , as follows:

$$T_{am}^* = \frac{\alpha_{am}}{c_3} U_{am}^* \tag{31}$$

$$T_a^* = \frac{\alpha_a}{b_2} U_a^* \tag{32}$$

$$T_m^* = \frac{\alpha_m}{c_2} U_m^*,\tag{33}$$

and

$$Q_{am}^* = \left(\frac{\xi_{am}}{\mu} + \frac{\gamma_{am}\alpha_{am}}{\mu c_3}\right) U_{am}^* \tag{34}$$

$$Q_a^* = \left(\frac{\xi_a}{\mu} + \frac{\gamma_a \alpha_a}{\mu b_2}\right) U_a^* \tag{35}$$

$$Q_m^* = \left(\frac{\xi_m}{\mu} + \frac{\gamma_m \alpha_m}{\mu c_2}\right) U_m^* \tag{36}$$

Reaching an analytic expression for the endemic equilibrium is considered of great value in epidemiological models because the disease free-equilibrium and the endemic equilibrium are the two stages that the population approaches, in the long term. But, in general, for the majority of contagious diseases, the disease remains in the populations, and therefore approaches to the endemic equilibrium.

5. NUMERICAL SIMULATIONS

In this section, numerical simulations are presented.

Figure 3 shows the behavior for compartments S, E, U, T, Q, for the sub-model of alcohol abuse. Observe that with a small initial amount of latent population (E), after interactions with susceptible individuals, the number of alcohol users (U)increased, peaking in the fifth year. With treatment programs in effect, the number of alcoholics decreases after the fifth year. Notice that population Q increases, indicating treatment is effective.

The most relevant compartment to observe in this work is the compartment U_a of alcohol-dependent individuals. In Figure 3, it is noticeable that if the alcoholism effective rate increases, the number of alcoholic individuals will increase, having a peak around the fifth year. As treatment plans and programs are implemented, the number of alcohol-dependent users starts decreasing, even though the treatment rate is very low.

For the marijuana abuse model, Figure 4 shows that the population of marijuana users U_m steadily increases during the first five years after 2017. Between five to thirty years after 2017, the user population appears to transition from a slow increase to a slow decrease. After the thirty year mark, the user population decreases more quickly, perhaps due to treatment for marijuana-abuse not being as ubiquitous as treatment for alcohol-abuse.

When simulating System (1)–(13), in Figure 5 notice that the compartment classes E_{am} , U_{am} , T_{am} , and Q_{am} for co-abuse reach their maximum close to the first year, meaning that the use of both alcohol and marijuana lead faster to addiction that the independent use of the substances. Again, if entering treatment happen as soon as the individual detect an addiction, then the probability of recovery is higher than without treatment.



FIGURE 3. Simulation of alcohol-abuse system with $\theta_1 = 1.05$ and $\theta_2 = 0.7$.



FIGURE 4. Simulation of marijuana-abuse system with $\epsilon_1 = 1.05$ and $\epsilon_2 = 0.7$.

For the alcohol abuse model, in Figure 6, alcohol users U_a increases significantly during the first five years after the year 2017. Notice that the dominant graph of U_a corresponds to $\sigma_a = 0.076$, which is the maximum value for σ_a .



Co-Abuse ODE System: all compartments

FIGURE 5. Simulation of co-abuse system with $\epsilon_1 = 1.05$ and $\epsilon_2 = 0.7$.



FIGURE 6. U_a with $.25 \le \sigma_a \le .9$, $\theta_1 = 0.01$ and $\theta_2 = 1.01$.

For the marijuana abuse model, in Figure 7, data corresponding to marijuana treatment is not available yet because legalization is very recent in many states.



FIGURE 7. T_m with $.1 \le \xi_m \le .3$, $\epsilon_1 = 1.01$ and $\epsilon_2 = 1.01$.



FIGURE 8. Q_m with $.1 \le \xi_m \le .3$, $\theta_1 = 0.01$ and $\theta_2 = 1.01$.

The parameters used for this simulations were estimated based on parameters for treatment for other types of drugs.

For the marijuana abuse model, in Figure 8, the parameter in consideration ξ corresponds to the quitting rate of marijuana users without treatment. The reason Q_m increases slowly is because of a lack of treatment programs for marijuana addicts.

6. Conclusions

Even though alcohol-marijuana co-abuse will always be present in modern society, the question of how to prevent co-abuse from becoming a pandemic can be asked. A mathematical model that describes the dynamics of the alcohol-marijuana co-abuse allowed us to identify the most relevant parameters to help control this epidemic. Alcohol and marijuana are two addictive substances that when combined can cause severe damage to young generations. This co-abuse is a social problem that is growing out of control, and if public health entities do not implement prevention programs, susceptible individuals are at high risk of becoming addicted to both substances. Analytical evaluations and numerical simulations show that for some parameters the alcohol-marijuana co-abuse can be controlled under certain constraints. The basic reproduction number for the independent models of alcohol and marijuana, and for the co-abuse model, in terms of the parameters, is a very standard way of defining public policies with the purpose of avoiding pandemics. This work can be implemented for any region by changing the parameters for the model using data values that correspond to each region. The model is well defined since positiveness and boundedness of solutions were shown. Stability for the disease-free equilibrium was attained by evaluation of eigenvalues for the sub-matrix for the newly infectious (latent and alcoholic individuals). Additionally, we were able to find an analytic expression for the endemic equilibrium which will help to identify where the individuals from different compartments will approach in the long term. Simulations for the most relevant parameters were included, showing that it is possible to control the co-abuse by implementing different approaches. These approaches include voluntarily quitting the use of the substances or, for a faster recovery, by entering a treatment program. Unfortunately, if quitting excessive alcohol consumption is not accomplished on time, the consequences of alcoholism can cause irreparable damage in the individual, for example, cardio vascular diseases, diabetes, cirrhosis, and some type of cancers, among others. Similarly, recent research shows that uncontrolled use of marijuana can cause neural damage, mental health disorders such as anxiety, depression, and in some cases paranoia. Also, some studies show that the constant use of marijuana causes digestive problems. When modeling co-abuse, it is expected that health consequences will be worse. Therefore, conduction of more research in this area, and collection of data are very important in order to create awareness and prevention programs for a real problem in society.

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References

- [1] Virginia, va, us, data, retrieved from https://www.samhsa.gov/data/report/virginia-va, 2014.
- [2] USA, data, https://www.census.gov/, 2020.
- [3] Alcohol-related death in virginia, 2016–2020, Rep., Virginia Department of Health, Richmond, VA, 2022.
- [4] NIDA: National institute of drug abuse, data, 2022.
- [5] CDC: Excessive alcohol use, https://www.cdc.gov/chronicdisease/resources/publications/, 2023.
- [6] Impact on alcohol use in human health and well-being, 2023.
- [7] Abiodun, O. E., Adebimpe, O., Ndako, J. A., Oludoun, O., Aladeitan, B., Adeniyi, M., Mathematical modeling of hiv-hcv co-infection model: Impact of parameters on reproduction number, *F1000Research*, 11 (2022), 1153, https://dx.doi.org/10.12688/f1000research.124555.2.
- [8] Akanni, J. O., Adediipo, D. A., Kehinde, O. O., Ayanrinola, O. W., Adeyemo, O. A., Mathematical modelling of the co-dynamics of illicit drug use and terrorism, *Inform. Sci. Lett, 11* (2022), 559–572, https://dx.doi.org/10.18576/isl/110224.
- Badurally Adam, N.-R., Dauhoo, M. Z., Kavian, O., An analysis of the dynamical evolution of experimental, recreative and abusive marijuana consumption in the states of Colorado and Washington beyond the implementation of i-502, *The Journal of Mathematical Sociology*, 39 (4) (2015), 257–279, https://dx.doi.org/10.1080/0022250X.2015.1077240.
- [10] Beckmeyer, J. J., Weybright, E. H., Perceptions of alcohol use by friends compared to peers: Associations with middle adolescents' own use, *Substance Abuse*, 37 (3) (2016), 435–440, https://dx.doi.org/10.1080/08897077.2015.1134754.
- [11] Burggren, A. C., Shirazi, A., Ginder, N., London, E. D., Cannabis effects on brain structure, function, and cognition: considerations for medical uses of cannabis and its derivatives, *The American Journal of Drug and Alcohol Abuse*, 45 (6) (2019), 563–579, https://dx.doi.org/10.1080/00952990.2019.1634086.
- [12] Carvalho, A. F., Heilig, M., Perez, A., Probst, C., Rehm, J., Alcohol use disorders, *The Lancet*, 394 (10200) (2019), 781–792, https://dx.doi.org/10.1016/S0140-6736(19)31775-1.
- [13] Chapwanya, M., Lubuma, J. M.-S., Lutermann, H., Matusse, A., Nyabadza, F., Terefe, Y., A mathematical model for the cannabis epidemic in a South African province with a non-linear incidence rate, *Journal of Statistics and Management Systems*, 24 (8) (2021), 1627–1647, https://dx.doi.org/10.1080/09720510.2020.1843274.
- [14] Chen, B. T., Yau, H.-J., Hatch, C., Kusumoto-Yoshida, I., Cho, S. L., Hopf, F. W., Bonci, A., Rescuing cocaine-induced prefrontal cortex hypoactivity prevents compulsive cocaine seeking, *Nature*, 496 (7445) (2013), 359–362, https://dx.doi.org/10.1038/nature12024.
- [15] Christakis, N. A., Fowler, J. H., Social contagion theory: examining dynamic social networks and human behavior, *Statistics in Medicine*, 32 (4) (2013), 556–577, https://dx.doi.org/10.1111/add.15751.

- [16] Costardi, J. V. V., Nampo, R. A. T., A review on alcohol: from the central action mechanism to chemical dependency, NIH Pub Med, 26466222 (2015), https://dx.doi.org/10.1590/1806-9282.61.04.381.
- [17] Galindo, R., Zamudio, P. A., Valenzuela, C. F., Alcohol is a potent stimulant of immature neuronal networks: implications for fetal alcohol spectrum disorder, *Journal of Neurochemistry*, 94 (6) (2005), 1500–1511, https://dx.doi.org/10.1111/j.1471-4159.2005.03294.x.
- [18] Geissler, K. H., Kaizer, K., Johnson, J. K., Doonan, S. M., Whitehill, J. M., Evaluation of availability of survey data about cannabis use, *JAMA Network Open*, 3 (6) (2020), e206039– e206039, https://dx.doi.org/10.1001/jamanetworkopen.2020.6039.
- [19] Hasin, D., Risk factors: Varied vulnerability to alcohol-related harm, NIH: NIAAA Core resource on Alcohol (2023).
- [20] Hingson R, Zha W, S. D., Magnitude and trends in heavy episodic drinking, alcohol-impaired driving, and alcohol-related mortality and overdose hospitalizations among emerging adults of college ages 18–24 in the United States, 1998-2014, *PubMed PMID: 28728636 Stud Alcohol Drugs.*, 78(4) (2017), 540–9, https://dx.doi.org/10.15288/jsad.2017.78.540.
- [21] Kabisa, E., Biracyaza, E., Habagusenga, J. d., Umubyeyi, A., Determinants and prevalence of relapse among patients with substance use disorders: case of icyizere psychotherapeutic centre, *Substance Abuse Treatment, Prevention, and Policy, 16* (1) (2021), 1–12, https://dx.doi.org/10.1186/s13011-021-00347-0.
- [22] Kranzler, H. R., Soyka, M., Diagnosis and pharmacotherapy of alcohol use disorder: a review, Jama, 320 (8) (2018), 815–824, https://dx.doi.org/10.1001/jama.2018.11406.
- [23] Kringelbach, M. L., Berridge, K. C., The functional neuroanatomy of pleasure and happiness, Discovery Medicine, 9 (49) (2010), 579.
- [24] Miller, W. R., Walters, S. T., Bennett, M. E., How effective is alcoholism treatment in the United States?, *Journal of Studies on Alcohol, 62* (2) (2001), 211–220, https://dx.doi.org/10.15288/jsa.2001.62.211.
- [25] Mubayi, A., Greenwood, P. E., Contextual interventions for controlling alcohol drinking, *Mathematical Population Studies*, 20 (1) (2013), 27–53, https://dx.doi.org/10.1080/08898480.2013.748588.
- [26] Orwa, T., Nyabadza, F., Mathematical modelling and analysis of alcohol-methamphetamine co-abuse in the Western Cape Province of South Africa, *Cogent Mathematics & Statistics*, 6 (1) (2019), 1641175, https://dx.doi.org/10.1080/25742558.2019.1641175.
- [27] Sánchez, F., Wang, X., Castillo-Chávez, C., Gorman, D. M., Gruenewald, P. J., Drinking as an epidemic—a simple mathematical model with recovery and relapse, In *Therapist's Guide to Evidence-Based Relapse Prevention*, Elsevier, 2007, pp. 353–368, https://dx.doi.org/10.1016/B978-012369429-4/50046-X.
- [28] Schuler, M. S., Tucker, J. S., Pedersen, E. R., D'Amico, E. J., Relative influence of perceived peer and family substance use on adolescent alcohol, cigarette, and marijuana use across middle and high school, *Addictive Behaviors*, 88 (2019), 99–105, https://dx.doi.org/10.1016/j.addbeh.2018.08.025.
- [29] Sharma, S., Samanta, G., Analysis of a drinking epidemic model, International Journal of Dynamics and Control, 3 (3) (2015), 288–305, https://dx.doi.org/10.1007/s40435-015-0151-8.
- [30] Tulshi D. Saha, B. F. G., Concurrent use of alcohol with other drugs and dsm-5 alcohol use disorder comorbid with other drug use disorders: Sociodemographic characteristics, severity, and psychopathology, *Drug and Alcohol Dependence, Elsevier, 187* (2018), 261–269.
- [31] Van den Driessche, P., Watmough, J., Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Mathematical Biosciences*, 180 (1-2) (2002), 29–48, https://dx.doi.org/10.1016/s0025-5564(02)00108-6.

- [32] Weiss, S. R., Volkow, N. D., Coordinating cannabis data collection globally: Policy implications, Addiction (Abingdon, England), 117 (6) (2022), 1520, https://dx.doi.org/10.1111/add.15751.
- [33] Yu, B., Chen, X., Chen, X., Yan, H., Marijuana legalization and historical trends in marijuana use among us residents aged 12–25: results from the 1979–2016 national survey on drug use and health, BMC Public Health, 20 (2020), 1–10, https://dx.doi.org/10.1186/s12889-020-8253-4.