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Analysis of SEIR Model for Effectiveness of Religious Leaders on Non-Pharmaceutical and Pharmaceutical Interventions against Covid -19 Spread In Kenya

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Abstract: **This research analyses the effectiveness of religious leaders' on adherence to pharmaceutical and non-pharmaceutical intervention measures to reduce COVID-19 spread in Kenya. Disease-free and endemic equilibrium was determined, and subsequently, the local and global stability was carried out. A SEIR compartmental model that considers the religious leaders was developed. The basic reproduction number (R_0) was determined using the next-generation matrix method. Sensitivity analysis was done to determine which parameters have the most significant impact on the model. Religious leaders were found to have significant impact on curbing the Covid-19 pandemic.**

Keywords: **Pharmaceutical; Non-pharmaceutical; COVID-19; Reproduction number; Mathematical modeling; Stability analysis; Disease-free equilibrium.**

I. INTRODUCTION

COVID-19 which is highly contagious was reported in Wuhan City, Hubei Province of China Wang et al. (2020). China has been focused worldwide due to (COVID-19) outbreak, which is brought about by (SARS-CoV-2). The disease incubation period ranges from 2 to 14 days Hethcote (2020). During this time, the individuals who are infected may be asymptomatic though, they may be unaware of the infection, but they have the ability of infecting other people Li et al (2020). As of 28th September, 2021, over 249,434 cases and 5,116 deaths in 188 countries had been recorded Bazzani et al. (2020). The common COVID-19 symptoms include; increase in temperatures, dry cough, fatigue and loss of sense of smell and sense of taste. Mild symptoms include sores in the throat, severe headache, aches, joint pains, loose stool, and pimples on the skin or decreased melanin on the figures or toes and itchy watery eyes.

Severe symptoms include straining when breathing, loss of speech or balance, confusion, and pain in the respiratory surface. Several mathematical models have been put forward to know on how to alleviate the pandemic. Osei-Tutu et al. (2021), studied the analysis of COVID-19 and religious leaders' restrictions on the health of Ghana Christians. Ullah and Khan (2020), studied on dynamics of COVID-19 with quarantine and isolation with real statistics cases reported in Mainland China. They used the SEIAQHR compartmental model and found that the fractional model was more suitable than the classical one. Okyere et al. (2020), researched on the non-linear dynamics of COVID 19 with optimal control analysis. They used the SEIR compartmental model for the 2019 corona infections in Ghana. Benahmadi et al. (2021), carried out research on the control measures of COVID-19 in Morocco. They used the SEAIR model with seven compartments; sensitivity analysis was done to know which parameters had much effect on R_0. Kim et al. (2020), developed a model to predict transmission dynamics of coronavirus using a model in mathematics considering changes in behavior in Korea. They came up with a model in mathematics on transmission of coronavirus based on SEIR model with a compartment of hospital quarantined people.

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Mbogo and Odhiambo (2021), studied the COVID-19 outbreak and control in Kenya. They employed the SEIHCRD mathematical transmission model and proposed NPIs intervention are needed for some time so as to stop COVID-19 epidemic effectively; otherwise, this pandemic will continue increasing despite the increased degree of recovering.

Mostly religious leaders can play an essential role in managing the COVID-19 pandemic by informing the congregants on the importance of using the pharmaceutical and non-pharmaceutical intervention measures of COVID-19, which contribute towards disseminating the health information around, hence religion can make sense of this dangerous COVID-19 pandemic. In the recent past, different models have been developed to describe the dynamics of COVID-19 in Kenya but no model has been developed to analyze the effectiveness of religious leaders in Kenya. This study is geared on analyzing the effectiveness of religious leaders on pharmaceutical and non-pharmaceutical intervention to curb COVID-19 spread in Kenya.

II. MATHEMATICAL FORMULATION

The total population $\mathrm{N}(t)$ at time t, is divided mainly into eight subpopulations; Susceptible, S (t), Stay-at-home susceptible, $S_h(t)$, Exposed, $E(t)$,Infected and symptomatic, $I_S(t)$,infected and Asymptomatic, $I_A(t)$,Home-based Treatment, $T(t)$ Hospitalized, $H(t)$ and Recovered, $R(t)$. Susceptible individuals are recruited into the population at a constant rate, $\prod$. β_1 is the contact rate of susceptible individuals with asymptomatic and β_2 is the contact rate of susceptible individuals with infected individuals and they move to the exposed compartment. Again, it is presumed that the rate at which susceptible individuals stay at home is $\upsilon+\theta_1$ and $\tau-\theta_1$ is the rate at which individuals move from stay-at-home due to different reasons, hence become susceptible to the pandemic. It is also assumed that θ_1 is the rate of adherence to covid-19 protocols due to religious leader's influence to the non-pharmaceutical intervention and θ_2 is the rate of adherence to covid-19 protocols due to religious leader's influence to pharmaceutical intervention (vaccination). After completing the period of incubation, the individuals now become infected at a rate of γ. Out of this, a proportion αγ do not show the COVID-19 symptoms while the others (1 − α) γ exhibit the symptoms . After carrying out tests $\sigma\delta$ proportion of asymptomatic individuals require treatment and join the home-based treatment compartment. The other $(1-\sigma)\delta$ proportion of asymptomatic individuals naturally recover from the disease. Now, from individuals who are infected, a fraction $C\varepsilon\theta_2$ of individuals go to the hospitalized compartment. The rest receive treatment at their home at a constant rate $(1-C)\varepsilon\theta_2$. However, individuals who get treatment can fail to recover hence, $\phi\rho$ fraction move to the hospitalized compartment. The remaining proportion now recovers. Individuals who recover can get into contact with the disease again at a rate of ω and individuals who are hospitalized recover from the pandemic a rate κ. The asymptomatic, symptomatic, treated and hospitalized individuals die due to the disease at a rate ρ_1 , ρ_2, ρ_3, ρ_4 respectively. The whole population have an average non-disease related death rate of μ.

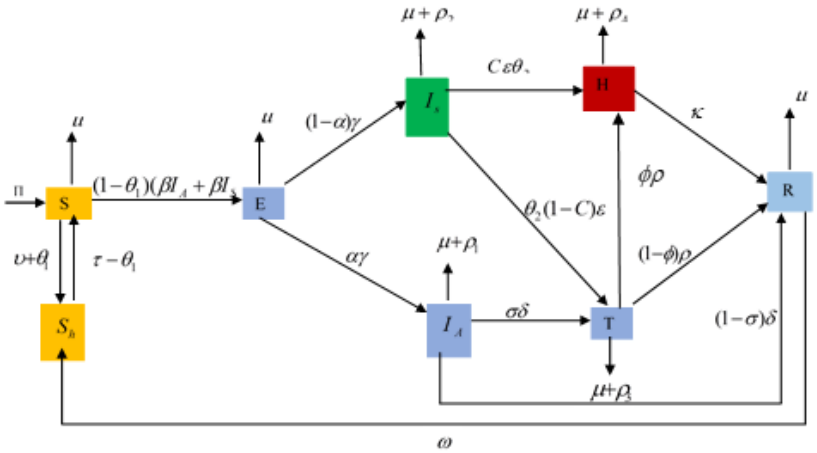

Fig. 1. SEIR Model

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MODEL EQUATIONS

Based on the above assumptions, a set of differential equation are established to analyze the stability of the disease free equilibrium and endemic equilibrium as shown below;

$$\left.\begin{aligned}
\frac{dS}{dt} &= \prod + (\tau - \theta_1)S_h - (1-\theta_2)(\beta_1 I_A + \beta_2 I_S)S - (\mu + \upsilon + \theta_1)S \\
\frac{dS_h}{dt} &= (\upsilon + \theta_1)S + \omega R - \mu S_h - (\tau - \theta_1)S_h \\
\frac{dE}{dt} &= (1-\theta_2)(\beta_1 I_A + \beta_2 I_S)S - (\mu + \gamma)E \\
\frac{dI_A}{dt} &= \alpha\gamma E - (\rho_1 + \delta + \mu)I_A \\
\frac{dI_S}{dt} &= (1-\alpha)\gamma E - (\varepsilon\theta_2 + \mu + \rho_2)I_S \\
\frac{dT}{dt} &= \theta_2(1-C)\varepsilon I_S + \sigma\delta - (\mu + \rho + \rho_3)T \\
\frac{dH}{dt} &= C\varepsilon\theta_2 I_S + \phi\rho T - (\kappa + \mu + \rho_4)H \\
\frac{dR}{dt} &= (1-\sigma)\delta I_A + \kappa H + (1-\phi)\rho T - (\omega + \mu)R
\end{aligned}\right\} \quad (1)$$

Given the initial conditions,

$$\begin{aligned}
&S(0) = S_0 \geq 0, S_h(0) = S_h \geq 0, E(0) = E_0 \geq 0, I_A(0) = I_A \geq 0, I_S(0) = I_S \geq 0, \\
&T(0) = T_0 \geq 0, H(0) = H_0 \geq 0, R(0) = R_0 \geq 0
\end{aligned} \quad (2)$$

MODEL ANALYSIS

i. Invariant region.

The analysis of the model equations (1) is performed in a region Ω of biological interest. The following theorem is on the region that model (1)-(2) is restricted to.

Theorem 2.1. *The feasible region* Ω*for model (1)-(2) is defined by;*

$$\Omega = \left\{ S(t), S_h(t), E(t), I_A(t), I_S(t), T(t), H(t), R(t) \in N \leq N \leq \max\{N(0), \frac{\prod}{\mu}\} \right\}$$

The initial conditions in (2) are positively invariant and attracting with respect to model system (1) for all $t \geq 0$.

Proof. Summing up the differential equations in model system (1), we obtain that the total population satisfies the differential equation;

$$\frac{dN(t)}{dt} = \prod - \mu N - \rho_1 I_A - \rho_2 I_S - \rho_3 T - \rho_4 H,$$

(3)

In the absence of covid-19 infection, it follows that,

$$\frac{dN(t)}{dt} + \mu N \leq \prod \quad (4)$$

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Upon integrating (4) we get;

$$N(t) \le \frac{\prod}{\mu} + \left(N(0) - \frac{\prod}{\mu} \right) e^{-\mu t} \tag{5}$$

Where $N(0)$ is the initial population size. From (5), we observe that at $t = 0, N = N(0)$ as $t \to \infty$, $N(t) \to \frac{\prod}{\mu}$. -

This means that $N(t) \le \max\left\{ N(0), \frac{\prod}{\mu} \right\}$. Therefore $N(t)$ is bounded above. Subsequently,

$\{S(t), S_h(t), E(t), I_A(t), I_S(t), T(t), H(t) \text{ and } R(t)\}$ are bounded above.

ii. Positivity of the model solutions

The model (1) – (2) describes human populations and hence it is important to prove that the solutions to model (1) with non-negative initial conditions will remain non-negative for all $t \ge 0$. Thus, we have the following theorem:

Theorem 2.2: *If the initial conditions of the model (1)-(2) are positive, then the solution set is positive for future time* $t \ge 0$.

Proof*:* To prove the existence of positivity of solution to model (1)-(2) given that all the model parameters are positive we proceed as below;

The first equation in model (1) can be written as;

$$\frac{dS}{dt} = \prod + (\tau - \theta_1) S_h - \{(1 - \theta_2)(\beta_1 I_A + \beta_2 I_S) + (\mu + \upsilon + \theta_1)\} S \tag{6}$$

From (6) we obtain the inequality;

$$\frac{dS}{dt} \ge -\{(1 - \theta_2)(\beta_1 I_A + \beta_2 I_S) + (\mu + \upsilon + \theta_1)\} S \tag{7}$$

Which is variable separable, i.e;

$$\frac{dS}{S} \ge -\{(1 - \theta_2)(\beta_1 I_A + \beta_2 I_S) + \mu + \upsilon + \theta_1\} d\bar{t} \tag{8}$$

Upon integrating (8) w.r.t $\bar{t}$ from 0 to t yields;

$$\int_0^t \frac{dS}{S} \ge -\int_0^t \{(1 - \theta_2)(\beta_1 I_A + \beta_2 I_S) + \mu + \upsilon + \theta_1\} d\bar{t} \tag{9}$$

$$\Rightarrow \ln S(t) - \ln S(0) \ge -\{(1 - \theta_2)(\beta_1 I_A + \beta_2 I_S) + \mu + \upsilon + \theta_1\} t$$

Therefore,

$$\ln\left[S(t) \big/ S(0) \right] \ge -\{(1 - \theta_2)(\beta_1 I_A + \beta_2 I_S) + \mu + \upsilon + \theta_1\} t \tag{10}$$

Introducing the exponent on both sides of (10) we get;

$$S(t) \ge S(0) \exp\{-(1 - \theta_2)(\beta_1 I_A + \beta_2 I_S) + \mu + \upsilon + \theta_2\} t$$

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Which shows that;

$S(t) \geq 0$ for $t \geq 0$ since $S(0) \geq 0$ and $\exp\{-(1-\theta_2)(\beta_1 I_A + \beta_2 I_S) + \mu + \upsilon + \theta_1\}t \geq 0$ for $t \geq 0.$

Similarly, we have the following;

$$S_h(t) \geq S_h(0)\exp\{-[\mu + \tau - \theta_1)S_h]t\} > 0,$$
$$E(t) \geq E(0)\exp\{-(\gamma + \mu)t\} > 0,$$
$$I_A(t) \geq I_A(0)\exp\{-(\rho_1 + \delta + \mu)t\} > 0,$$
$$I_S(t) \geq I_S(0)\exp\{-(\mu + \varepsilon\theta_2 + \rho_2)t\} > 0,$$
$$T(t) \geq T(0)\exp\{-(\rho_3 + \mu + \rho)t\} > 0,$$
$$H(t) \geq H(0)\exp\{-(\kappa + \mu + \rho_4)t\} > 0,$$
$$R(t) \geq R(0)\exp\{-(\mu + \omega)t\} > 0.$$

iii. Disease free equilibrium (DFE)

Disease-free equilibrium is a state in which the number of infected individuals in a population remains constant over time, and no new cases of the disease are introduced. To obtain this equilibrium point of model (1), the derivatives with respect to time in model (1) are set to zero, while all the infected and recovered classes are also set to zero.

That is,

$$E = 0, I_A = 0, I_S = 0, T = 0, H = 0, R = 0,$$

(11)

$$\prod + (\tau - \theta_1)S_h - (\mu + \upsilon + \theta_1)S = 0$$

(12)

$$(\upsilon + \theta_1)S - \mu S_h - (\tau - \theta_1)S_h = 0 \quad (13)$$

Solving (12) and (13) simultaneously yields;

$$S = \frac{\prod(\upsilon + \tau - \theta_1)}{\mu(\upsilon + \tau - \theta_1) + \upsilon(\upsilon + \theta_1)}$$

$$S_h = \frac{\prod(\upsilon + \theta_1)}{u(\upsilon + \tau - \theta_1) + \upsilon(\upsilon + \theta_1)}$$

Therefore, the equilibrium point is given as;

$$(E_0 = S^0, S_h{}^0, E^0, I_A{}^0, .I_S{}^0, T^0, H^0, R^0)$$

$$\Rightarrow E_0 = \left(\frac{\prod\{\upsilon + \tau - \theta_1)\}}{\mu\{\upsilon + \tau - \theta_1) + \upsilon(\upsilon + \theta_1\}}, \frac{\prod(\upsilon + \theta_1)}{\mu\{\upsilon + \tau - \theta_1) + \upsilon(\upsilon + \theta_1\}}, 0, 0, 0, 0, 0, 0\right)$$

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iv. Reproduction number

The basic reproduction number is a measure of average number of secondary infections caused by a single infected individual in a population where everyone is susceptible to the disease. The next generation matrix method Hethcote (2000), is used to compute the basic reproduction number of the model (1). Using the notation F for the new infections and V for the transition terms, the following is obtained,

$$F=\begin{pmatrix}(1-\theta_2)(\beta_1 I_A+\beta_2 I_S)S(t)\\0\\0\\0\\0\end{pmatrix} \text{ and } V=\begin{pmatrix}-(\mu+\gamma)E\\ \alpha\gamma E-(\rho_1+\delta+\mu)A\\ (1-\alpha)\gamma E-(\varepsilon\theta_2+\mu+\rho_2)T\\ \theta_2(1-C)\varepsilon I_S+\sigma\delta I_A-(\mu+\rho+\rho_3)T\\ \phi\rho I_A+C\theta_2\varepsilon I_S-(\kappa+\mu+\rho_4)H\end{pmatrix} \quad (14)$$

The partial derivatives of F and V with respect to the infected classes evaluated at the disease free equilibrium E_0 are denoted by F and V respectively and obtained as follows:

$$\mathrm{F}=\begin{pmatrix}0 & \beta_1(1-\theta_2)S^0 & \beta_2(1-\theta_2)S^0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0\end{pmatrix} \quad (15)$$

And,

$$\mathrm{V}=\begin{pmatrix}-(\gamma+\mu) & 0 & 0 & 0 & 0\\ -\alpha\gamma & -\psi_1 & 0 & 0 & 0\\ -(1-\alpha)\gamma & 0 & -\psi_2 & 0 & 0\\ 0 & 0 & -(1-C)\varepsilon\theta_2 & -\psi_3 & 0\\ 0 & 0 & -C\varepsilon\theta_2 & -\phi\rho & -\psi_4\end{pmatrix} \quad (16)$$

Where;

$$\psi_1=\rho_1+\delta+\mu,\ \psi_2=\rho_2+\theta_2\varepsilon+\mu,\ \ \psi_3=\rho_3+\rho+\mu,\ \psi_4=\rho_4+\kappa+\mu,$$

The basic reproduction number is given as the dominant eigenvalue of the spectral radius of the matrix FV^{-1};

That is;

$$R_0=\frac{\left((1-\alpha)(\delta+\rho_1+\mu)\beta_2+(\varepsilon\theta_2+\rho_2+\mu)\alpha\beta_1\right)\gamma\prod(1-\theta_2)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)(\delta+\rho_1+\mu)(\varepsilon\theta_2+\rho_1+\mu)}$$

v. Local stability of DFE

Local stability is analyzed by examining the eigenvalues of the Jacobian matrix associated with the system of differential equations that describe the dynamics of the disease spread. If all eigenvalues have negative real parts, the disease-free equilibrium is considered locally stable. If one or more eigenvalues have positive real parts, the disease-free equilibrium is considered unstable. The eigenvalues are obtained by getting the PDE of the function Badshah and Akbar (2021). Routh-Hurwitz criterion Yang et al. (2021) was utilized so as to get the local stability.

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Theorem 3.3. *The disease free equilibrium of model (1)-(2) is locally asymptotically stable if;* $R_0 < 1$ *and unstable otherwise.*

Proof: The Jacobian matrix of model (1) evaluated at disease free equilibrium (E_0) is first obtained as;

$$JE_0 = \begin{pmatrix} -(\mu+\upsilon+\theta_1) & (\tau-\theta_1) & 0 & \beta_1(1-\theta_2)S^0 & \beta_2(1-\theta_2)S^0 & 0 & 0 & 0 \\ \upsilon+\omega+\theta_1 & -(\mu+\tau-\theta_1)) & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\mu+\gamma) & \beta_1(1-\theta_2)S^0 & \beta_2(1-\theta_2)S^0 & 0 & 0 & 0 \\ 0 & 0 & \alpha\gamma & -\psi_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & (1-\alpha)\gamma & 0 & -\psi_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \theta_2(1-C)\varepsilon & -\psi_3 & 0 & 0 \\ 0 & 0 & 0 & 0 & C\theta_2\varepsilon & \phi\rho & -\psi_4 & 0 \\ 0 & 0 & 0 & (1-\sigma)\delta & 0 & (1-\phi)\rho & 0 & -(\omega+\mu) \end{pmatrix}$$

(17)

Where;

$$\psi_1 = \rho_1 + \delta + \mu,\ \psi_2 = \rho_2 + \theta_2\varepsilon + \mu,\ \ \psi_3 = \rho_3 + \rho + \mu,\ \psi_4 = \rho_4 + \kappa + \mu,$$

$$S^0 = \frac{\prod(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)}$$

The first five Eigen values are listed as;

$$\lambda_1 = -(\mu+\upsilon+\theta_1), \lambda_2 = -(\mu+\tau-\theta_1), \lambda_3 = -\psi_3, \lambda_4 = -\psi_4, \lambda_5 = -(\mu+\omega).$$

The other eigenvalues with negative real parts can be obtained from the characteristic polynomial;

$$P(\lambda) = \lambda^3 + \eta_1\lambda^2 + \eta_2\lambda + \eta_3$$

(18)

Where;

$$\eta_1 = \psi_1 + \psi_2 + K$$

$$\eta_2 = K\psi_2 + K\psi_1 - \lambda\psi_1\psi_2 + \frac{\beta_1\prod\alpha\gamma\lambda(1-\theta_2)(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)} + \frac{\beta_2\prod\psi_1\lambda(1-\theta_2)(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)} + \frac{\beta_2\prod\lambda\gamma(1-\theta_2)+(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau(1-\theta_1)+\upsilon(\upsilon+\theta_1)} - \frac{\beta_2\prod\lambda\alpha\gamma(1-\theta_2)+(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)}$$

$$\eta_3 = -\left\{K\psi_1\psi_2 + \frac{\beta_1\prod\alpha\gamma\psi_1(1-\theta_2)(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)} + \frac{\beta_2\prod\alpha\gamma\psi_1(1-\theta_2)(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)}\right\}$$

$$K = -(\mu+\tau)$$

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To establish the Eigen values positivity, we used the Routh-Hurwitz criteria Yang et al. (2021). If $\eta_1 > 0, \eta_2 > 0$ and $\eta_1\eta_2 - \eta_3 > 0$ holds then all the roots of the characteristic equation have negative real part and hence the equilibrium point (DFE) point is stable.

By this principal equation (18) has strictly negative real root since $\eta_1 > 0, \eta_2 > 0$ and $\eta_1\eta_2 - \eta_3 > 0$ .We can clearly see that $\eta_1 > 0, and\ \eta_2 > 0$ since they are sum of positive parameters.

Then considering the equation,

$$\eta_3 = \psi_1\psi_2[1 - R_0] > 0,$$

We conclude that the DFE is locally asymptotically stable when $R_0 < 1$ and thus Covid-19 cannot become a pandemic to invade the population.

vi. Global stability of DFEP

This gives the long-term behavior of a population in the absence of a disease. In this section we investigate the global stability of the disease-free equilibrium by the use of the theorem by Castillo (2001) , we write the model (1) as;

$$\left.\begin{aligned} \frac{dM}{dt} &= F(M,N) \\ \frac{dN}{dt} &= G(M,N),\ G(M,0) = 0 \end{aligned}\right\} \tag{19}$$

Where $M = (S, S_h,\ R,\ \in \Re^3)$ represents the non-disease compartments, $N = (E, I_A, I_S, T, H) \in \Re^5)$ represents the disease compartments of model (1)

The following conditions H_1 and H_2 are required for the global asymptotic stability of the DFE of the model (1).

$(H_1)\ For\ \frac{dM}{dt} = F(M,0)$, M^* is globally asymptotically stable, where $F(M^*,0) = 0$,

$(H_2)\ For\ \frac{dN}{dt} = D_N G(M^*,0) - \hat{G}(M,N), \hat{G}(M,N) > 0\ for\ all\ (M,N) \in \Omega$,

$D_N G(M,N)$ is the region where the system is biologically feasible in Ω and M -matrix whose off-diagonal elements are nonnegative. If the system (19) satisfies the above two conditions, then the following theorem holds.

Theorem 3.4. *The disease-free equilibrium points* $E_0 = (M^*,0)$ *of the system (19) is globally asymptotically stable if* $R_0 < 1$ *and the conditions* H_1 *and* H_2 *are satisfied.*

Proof: We first start by defining new variable and dividing the system (19) into sub-systems. $M = (S, S_h, R)\ and\ N = (E, I_A, I_S, T, H)$. From equation (19) we have two functions

$F(M, N)\ and\ G(M,N)$ given by;

$$F(M,N) = \begin{pmatrix} \prod + (\tau - \theta_1)S_h - (1-\theta_2)(\beta_1 I_A + \beta_2 I_S)S - (\mu + \upsilon + \theta_1)S \\ (\upsilon + \theta_1)S + \omega R - (\mu + \tau - \theta_1)S_h \\ (1-\sigma)\delta I_A + \kappa H + (1-\phi)\rho T - (\mu + \omega)R \end{pmatrix} \tag{20}$$

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And,

$$G(M,N)=\begin{pmatrix}(1-\theta_2)(\beta_1 I_A+\beta_2 I_S)S-(\mu+\gamma)E\\ \alpha\gamma E-(\rho_1+\delta+\mu)I_A\\ (1-\alpha)\gamma E-(\varepsilon\theta_2+\mu+\rho_2)I_S\\ \theta_2(1-C)\varepsilon I_S-(\mu+\rho+\rho_3)T\\ C\varepsilon\theta_2 I_S+\phi\rho T-(\kappa+\mu+\rho_4)Q\end{pmatrix} \tag{21}$$

Now considering the reduced system $\frac{dM}{dt}=F(M,0)$ equation (20) becomes,

$$\left.\frac{dM}{dt}\right|_{N=0}=\begin{pmatrix}\prod+(\tau-\theta_1)S_h-(\mu+\upsilon+\theta_1)S\\ (\upsilon+\theta_1)S+\omega R-(\mu+\tau-\theta_1)S_h\\ -(\mu+\omega)R\end{pmatrix} \tag{22}$$

From conditions H_1, equation (22) becomes,

$$\left.\frac{dM}{dt}\right|=\begin{pmatrix}\prod-\mu S\\ 0\\ 0\end{pmatrix}$$

Hence,

$$M(t)\le\frac{\prod}{\mu}+\left(M(0)-\frac{\prod}{\mu}\right)e^{-\mu t} \tag{23}$$

This implies the global convergence of (23) in Ω since the solution $M(t)$ approaches $\frac{\prod}{\mu}$ as $t\to\infty$. Clearly $M^*=\left(\frac{\prod}{\mu},\ 0\right)$ is the global asymptotic equilibrium point of the system $\frac{dM}{dt}=F(M,0)$. Hence, the convergence of the solution of the reduced system equation (22) is global in Ω.

Next we obtain;

$$G(M,N)=D_N\hat{G}(M^*,0)N-\hat{G}(M,N),\hat{G}(M,N)\ge 0$$

By taking;

$$B=\begin{pmatrix}-\mu-\gamma & \frac{\beta_1 I_A\prod(1-\theta_2)(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)} & \frac{\beta_2 I_S\prod(1-\theta_2)(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)} & 0 & 0\\ \alpha\gamma & -\psi_1 & 0 & 0 & 0\\ (1-\alpha)\gamma & 0 & -\psi_2 & 0 & 0\\ 0 & 0 & \theta_2(1-C)\varepsilon I & -\psi & 0\\ 0 & 0 & C\varepsilon\theta_2 & \phi\rho & -\psi_4\end{pmatrix} \tag{24}$$

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Where;

$$B = D_N G(M,N)N$$

And;

$$\hat{G}(M,N) = \begin{pmatrix} -\mu-\gamma & \beta_1 I_A(1-\theta_2)\prod S^0 & \beta_2 I_S(1-\theta_2)\prod S^0 & 0 & 0 \\ \alpha\gamma & -\psi_1 & 0 & 0 & 0 \\ (1-\alpha)\gamma & 0 & -\psi_2 & 0 & 0 \\ 0 & 0 & \theta_2(1-C)\varepsilon I & -\psi & 0 \\ 0 & \sigma\delta & C\varepsilon\theta_2 & \phi\rho & -\psi_4 \end{pmatrix} \quad (25)$$

Hence, by subtracting (25) from (24) we obtain;

$$G(M,N) = \begin{pmatrix} \left|\frac{\prod(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)}\right|(1-\theta_2)(\beta_1 I_A+\beta_2 I_S) \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} - S^0$$

Now since; $\frac{\prod(\upsilon+\tau-\theta_1)}{\mu(\upsilon+\tau-\theta_1)+\upsilon(\upsilon+\theta_1)} = M_0 \geq M$, hence the proof that;

$G(M,N) = D_N\hat{G}(M^*,0)N - \hat{G}(M,N), \hat{G}(M,N) \geq 0$ *for all* $(M,N) \in \Omega$. Thus, the two H_1 *and* H_2 conditions are satisfied and the conclusion that the Covid-19 free equilibrium of the model (1) is globally asymptotically stable

vii. Endemic equilibrium point

Equilibrium occurs when at least one of the infected compartments of model (1) are non-zero, Yousefpour et al. (2020) . In order to obtain this endemic equilibrium from our model (1), the right-hand side of equations in model (1) are set to zero to obtain;

$$\left.\begin{aligned} &\prod+(\tau-\theta_1)S_h^* - (1-\theta_2)(\beta_1 I_A^* + \beta_2 I_S^*)S^* - (\mu+\upsilon+\theta_1)S^* = 0 \\ &(\upsilon+\theta_1)S^* + \omega R^* - \mu S_h^* - (\tau-\theta_1)S_h^* = 0 \\ &(1-\theta_2)(\beta_1 I_A^* + \beta_2 I_S^*)S^* - (\mu+\gamma)E^* = 0 \\ &\alpha\gamma E^* - (\rho_1+\delta+\mu)I_A^* = 0 \\ &(1-\alpha)\gamma E^* - (\varepsilon\theta_2+\mu+\rho_2)I_S^* = 0 \\ &\theta_2(1-C)\varepsilon I_S^* - (\mu+\rho+\rho_3)T^* = 0 \\ &C\theta_2\varepsilon I_S^* + \phi\rho T^* + (\kappa+\mu+\rho_4)H^* = 0 \\ &(1-\sigma)\delta I_A^* + \kappa H^* + (1-\phi)\rho T^* - (\omega+\mu)R^* = 0 \end{aligned}\right\} \quad (26)$$

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Now we solve $S^*, S_h^*, E^*, I_A^*, I_S^*, T^*, H^*, R^*$ from equation (26) to obtain the following;

$$S^* = \frac{(\prod + \tau - \theta_1)\zeta_1(\upsilon + \theta_1 + \omega)\{(\mu + \tau(1-\theta_2))(\gamma + \mu)(\rho_1 + \delta + \mu)(\varepsilon\theta_2 + \mu + \rho_2)\}}{\gamma\zeta_2\zeta_1(\rho_1 + \delta + \mu)(\varepsilon\theta_2 + \mu + \rho_2)} \Big/ \frac{\gamma\beta_1\zeta_1\alpha\{(1-\theta_1)(1-\theta_2)(\varepsilon\theta_2 + \mu + \rho_2)\} + \zeta_1\gamma(1-\theta_2)(1-\alpha)(\rho_1 + \delta + \mu)\beta_2 + \gamma\zeta_1\zeta_2(\mu + \upsilon + \theta_1)(\rho_1 + \delta + \mu)(\varepsilon\theta_2 + \mu + \rho_2)}{} \quad (27)$$

$$S_h^* = \frac{(\upsilon + \theta_1 + \omega)\zeta_1\{(\mu + \tau - \theta_2))(\gamma + \mu)(\rho_1 + \delta + \mu)(\varepsilon\theta_2 + \mu + \rho_2)\}}{\gamma(\mu + \tau - \theta_2)\{\zeta_1\alpha(1-\theta_2)(\varepsilon\theta_2 + \mu + \rho_2)\beta_1 + (\zeta_1(1-\theta_2)(1-\alpha)(\rho_1 + \delta + \mu)\beta_2\}} \quad (28)$$

$$E^* = \frac{(1-\theta_2)\zeta_1}{\gamma\zeta_2} \quad (29)$$

$$I_A^* = \frac{\alpha\gamma\zeta_1(1-\theta_2)}{\gamma\zeta_2(\rho_1 + \delta + \mu} \quad (30)$$

$$I_S^* = \frac{(1-\alpha)(1-\theta_2)\gamma\zeta_1}{\gamma\zeta_2(\rho_1 + \delta + \mu)\gamma\zeta_2(\varepsilon\theta_2 + \mu + \rho_2)} \quad (31)$$

$$T^* = \frac{(1-\alpha)(1-\theta_2)(1-C)\varepsilon\theta_2\gamma\zeta_1}{\gamma\zeta_2(\varepsilon\theta_2 + \mu + \rho_2)(\rho_1 + \delta + \mu)(\mu + \rho + \rho_3)} \quad (32)$$

$$H^* = \frac{\sigma\delta\alpha\zeta_1(1-\theta_1)}{\gamma\zeta_2(\rho_1 + \delta + \mu)(\kappa + \mu + \rho_4)} + \frac{C\varepsilon\theta_2(1-\alpha)(1-\theta_2)\gamma\zeta_1}{\gamma\zeta_2(\rho_1 + \delta + \mu)(\varepsilon\theta_2 + \mu + \rho_2)(\kappa + \mu + \rho_4)} + \frac{\phi\rho(1-\alpha)(1-\theta_2)(1-C)\varepsilon\theta_2\gamma\zeta_1}{\gamma\zeta_2(\varepsilon\theta_2 + \mu + \rho_2)(\rho_1 + \delta + \mu)(\mu + \rho + \rho_3)(\kappa + \mu + \rho_4)} \quad (33)$$

$$R^* = \frac{\delta\alpha\gamma\zeta_1(1-\theta_1)(1-\sigma)}{(\mu + \omega)(\gamma\zeta_2)(\rho_1 + \delta + \mu)} + \frac{\kappa(1-\alpha)(1-\theta_2)C\varepsilon\gamma\zeta_1}{(\omega + \mu^2)\gamma\zeta_2(\varepsilon\theta_2 + \mu + \rho_2)(\kappa + \mu + \rho_4)} + \frac{\sigma\delta\alpha\gamma\zeta_1(1-\theta_1)}{\gamma\zeta_2(\rho_1 + \delta + \mu)(\kappa + \mu + \rho_4)} + \frac{(1-\alpha)(1-\theta_2)(1-\phi)(1-C)\rho\varepsilon\theta_2\gamma\zeta_1}{(\mu + \omega)\gamma\zeta_2(\varepsilon\theta_2 + \mu + \rho_2)(\mu + \rho + \omega_3)} \quad (34)$$

Where;

$$\zeta_1 = (\delta + \rho_1 + \mu)(\alpha - 1)\prod\gamma\beta_2 - (\varepsilon\theta_2 + \rho_2 + \mu)\prod\alpha\gamma\beta_1 + \mu)(\gamma + \mu)$$
$$\{\delta(1-\theta_2)(\varepsilon\theta_2 + \rho_2 + \mu) + \varepsilon\theta_2(\rho_1 + \mu) + \mu(\omega + \rho_1 + \rho_2 + \mu) + \rho_1(\omega + \varphi + \rho_2)$$

$$\zeta_2 = (\gamma + \mu)\{\delta\beta_2(\alpha - 1)(1-\theta_2) - \alpha\varepsilon\theta_2\beta_1\} -$$
$$(\omega + \rho_2 + \mu)(\gamma + \mu)\alpha\beta_1 + (\alpha - 1)(\gamma + \mu)(\rho_1 + \mu)\beta_2$$

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Viii. Sensitivity analysis

Sensitivity analysis is performed to establish parameters that have major influence on the reproduction number (R_0). The normalized forward sensitivity index definition is used to go through sensitivity analysis of basic parameters, Yousefpour et al. (2020). The sensitivity index of R_0 with respect to a given parameter, say h is given by,

$$P = \frac{\partial R_0}{\partial h} \times \frac{h}{R_0}$$

The sign of P is crucial since it determines whether the reproduction number is an increasing or a decreasing function of the corresponding parameter.

$$\nabla_{\beta_1}^{R_0} = \frac{\partial R_0}{\partial \beta_1} \times \frac{\beta_1}{R_0}$$

(35)

Differentiating R_0 with respect to β_1 in (35). We obtain;

$$\frac{\partial R_0}{\partial \beta_1} = \frac{(\theta_2\varepsilon + \omega + \rho_2 + \mu)\alpha\gamma\prod(1-\theta_2)}{(1-\alpha)(\delta+\rho_1+\mu)\beta_2 + (\theta_2\varepsilon+\rho_2+\mu)\alpha\beta_1\prod(1-\theta_2)}$$

(36)

Hence;

Multiplying (36) with $\frac{\beta_1}{R_0}$ and solving we obtain;

$$\frac{\partial R_0}{\partial \beta_1} \times \frac{\beta_1}{R_0} = \frac{(\theta_2\varepsilon + \omega + \rho_2 + \mu)\alpha\gamma\beta_1\prod(1-\theta_2)}{(1-\alpha)(\delta+\rho_1+\mu)\beta_2 + (\theta_1\varepsilon+\rho_2+\mu)\alpha\beta_1\prod(1-\theta_2)} \quad (37)$$

From (37) and that $\theta_2, \varepsilon, \alpha, \gamma, \beta_1, \tau, \omega, \rho_2, \upsilon$ are non-negative, we obtain;

$$\frac{\partial R_0}{\partial \beta_1} \times \frac{\beta_1}{R_0} = \frac{(\theta_2\varepsilon + \omega + \rho_2 + \mu)\alpha\gamma\beta_1\prod(1-\theta_2)}{(1-\alpha)(\delta+\rho_1+\mu)\beta_2 + (\theta_1\varepsilon+\rho_2+\mu)\alpha\beta_1\prod(1-\theta_2)} > 0 \quad (38)$$

This indicates there is a direct relationship between β_1 *and* R_0. Meaning increases in β_1 will result to increase in R_0. Since β_1 represents the rate of contact of susceptible with the infected individuals.

Similarly, for the other parameters we obtain,

$$\frac{\partial R_0}{\partial \beta_2} \times \frac{\beta_2}{R_0} = \frac{(1-\alpha)(\delta+\rho_1+\mu)\beta_2\alpha\gamma\prod(1-\theta_2)}{(1-\alpha)(\delta+\rho_1+\mu)\beta_2 + (\theta_1\varepsilon+\rho_2+\mu)\alpha\beta_1\prod(1-\theta_2)} > 0,$$

$$\frac{\partial R_0}{\partial \lambda} \times \frac{\lambda}{R_0} = \frac{(\theta_2\varepsilon + \omega + \rho_2 + \mu)\lambda\alpha\prod(1-\theta_2)}{(1-\alpha)(\delta+\rho_1+\mu)\beta_2 + (\theta_2\varepsilon+\rho_2+\mu)\alpha\beta_1\prod(1-\theta_2)} > 0,$$

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$$\frac{\partial R_0}{\partial \delta}\times\frac{\delta}{R_0}=\frac{-\delta(1-\alpha)\gamma\beta_1\prod(1-\theta_2)}{(1-\alpha)(\delta+\rho_1+\mu)\ \beta_2+(\theta_1\varepsilon+\rho_2+\mu)(\delta+\rho_1+\mu)\alpha\beta_1\prod(1-\theta_2)}<0,$$

$$\frac{\partial R_0}{\partial \varepsilon}\times\frac{\varepsilon}{R_0}=\frac{-\varepsilon\gamma\beta_1\prod(1-\theta_2)}{(1-\alpha)(\delta+\rho_1+\mu)\ \beta_2+(\theta_1\varepsilon+\rho_2+\mu)(\varepsilon\theta_2+\omega+\rho_2+\mu)\alpha\beta_1\prod(1-\theta_2)}<0,$$

$$\frac{\partial R_0}{\partial \rho_1}\times\frac{\rho_1}{R_0}=\frac{-\rho_1(1-\theta_1)\gamma\beta_1\prod(1-\theta_2)}{(1-\alpha)(\delta+\rho_1+\mu)\ \beta_2+(\theta_1\varepsilon+\rho_2+\mu)(\delta+\rho_1+\mu)\alpha\beta_1\prod(1-\theta_2)}<0,$$

$$\frac{\partial R_0}{\partial \rho_2}\times\frac{\rho_2}{R_0}=\frac{-\rho_2\gamma\beta_1\prod(1-\theta_2)}{(1-\alpha)(\delta+\rho_1+\mu)\ \beta_2+(\theta_1\varepsilon+\rho_2+\mu)(\varepsilon\theta_2+\rho_2+\mu)\alpha\beta_1\prod(1-\theta_2)}<0,$$

Table: 1. Sensitivity indices table

Parameter	Sensitivity index
β_1	0.99999
$\prod$	1
β_2	0.258241898
θ_1	-2.064509968
θ_2	-0.6094452335
δ	-0.0305027644
ε	-0.3599326204
ρ_1	-0.7418774828
ρ_2	0.02746556942
γ	0.2582418983

The sensitivity analysis and indices of the basic reproduction number with respect to the main Parameters are found in table (1). We find the positive indices parameters (β_1, β_2, *and* γ) this shows that they have high impact on spreading COVID-19 in the population if their values are rapidly increasing. This occurs because as their values increase in the population the basic reproduction number of the disease also increases. The other parameters like $(\theta_1, \alpha, \theta_2, \delta, \omega, \varepsilon, \rho_1, \rho_2)$ have negative impact and increasing the value of these parameters will minimize the thrust of the disease in the population. Therefore, research advice for interested parties is to work on increasing negative indices parameters so as to curb the pandemic.

III. CONCLUTION

In this paper, we have formulated $SS_hEI_SI_ATHR$ model for analysis of effectiveness of religious leaders on non-pharmaceutical and pharmaceutical intervention measures in COVID -19 spread in Kenya. We analyzed the dynamics of this disease model. Basic reproduction number R_0 was obtained using next generation matrix method and the stability of equilibrium points was investigated. The disease-free equilibrium of model (1) is locally asymptotically stable if; $R_0<1$ and unstable otherwise. Finally, we discussed and analyzed the characteristics of different control strategies according to the basic reproductive number and found that religious leaders had great impact since there was a decline trend of daily infection cases due to the infected people taking medication, many people also got vaccinated and also many people adopted the non-pharmaceutical intervention measures.

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