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Computational Modelling of Two Strain Meningitis Disease Outbreak

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Abstract: Meningococcal meningitis is a significant contributor to increased deaths globally, particularly the vulnerable children aged between 0-5 years. This paper formulates a robust two-strain epidemic model for the transmission dynamics of bacterial meningitis by incorporating interventions such as treatment and vaccination. The aim of the article is to formulate a meningitis epidemic model and study the time dependent dynamics of meningitis in the presence of antibiotic resistance to treatment threats while assessing the impact of vaccination proportion. The study uses the 4^{th} order Runge Kutta numerical approach to solve the problem and Maple mathematical tool to undertake simulations. The meningitis model qualitative study reveals existence of disease-free state when infection dies out and endemic state when disease persist in the community. The disease-free case is found to be stable only if effective reproduction number $R_e < 1$ and the community enjoys disease free scenario. Meningitis disease-free state reveals a locally asymptotically stable (LAS) transmission dynamics. The endemic equilibrium state i.e., $R_e > 1$ exists and persistence occurs in the community. The impact of parameter control measures on the spread of meningitis disease through sensitivity study of the key parameter, i.e., R_e, which revealed the key target parameters that can wipe out meningitis disease. We perform numerical solution of the considered model equations to display the qualitative findings and describe the asymptotical transmission dynamics of the disease. The effects of meningitis disease prevention and control approaches are analyzed. Key findings are shown using graphs and tables. We obtain a threshold vaccination proportion value beyond which the meningitis disease will be perfectly wiped out of the community and below which the disease acquires endemic state.

Keywords: Meningococcal Meningitis, Effective Reproductive Number, Vaccination Coverage, Endemic Equilibrium, Sensitivity Indices, Numerical Simulation

1. Introduction

Meningitis disease whose causative agent is the bacterium Neisseria meningitidis (n.m) is known to be a bacterial disease that leads to acute, life threatening and severe illness. Meningococcal disease is responsible for high cases of child morbidity and mortality in the sub-Saharan Africa region being approximately 400 million annually [8]. The incubation of meningococcal bacterium takes approximately 3-4 days in the mucosal cells to be infectious, with a range of 1 to 10 days. Meningococci is mainly spread from one individual to another via excretions from respiratory system of an infectious persons with asymptomatic and symptomatic stages of meningococcal disease [7]. The symptoms visible in bacterial meningitis infected individual include: sudden onset of fever, severe headache accompanied by a stiff neck, nausea, vomiting, eye sensitivity to light (photophobia) etc. see [7, 8]. Meningococcal meningitis grows rapidly, even in healthy looking individuals, and consequently may lead to increased morbidity and mortality (it leads to deaths of an estimated 50%-80% of untreated cases) [6]. In as much as, early diagnosis and a prompt treatment may be undertaken, still 5% to 10% patients succumb to the disease within 24 to 48 hours after the symptoms are seen, while survivors approximated at 10%-20% of population may suffer longterm effects from meningitis [6, 9].

Meningococcal disease is a common form of infectious disease in young children under five years. Due to the threat posed by meningococcal disease, the discovery of effective vaccines for meningococcal diseases is a life-saving

achievement to humanity and necessitates that health agencies to get guided on decision making in case of an outbreak of the disease [7]. Over the years, vaccination has proved to be the efficient and effective approach to wipe out infectious diseases. Thus, the need to formulate of a predictive model capable of recommending an accurate vaccine proportion that is enough to eliminate the transmission of meningitis disease in the community is crucial. In this regard, mathematical computational methods employed on epidemic models play a crucial role while making numerical solution and predicting the unforeseen dynamics of the disease in the community [6].

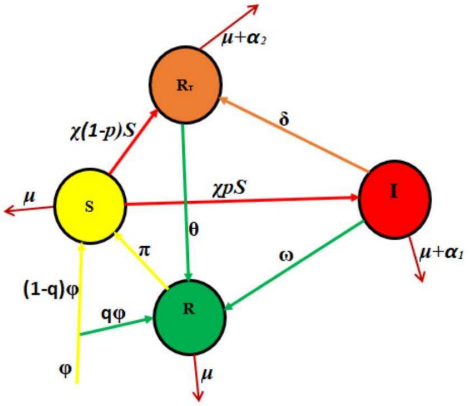

Figure 1. *Meningitis model flow chart.*

The articles main target is to formulate and analyze a meningitis epidemic predictive model that investigates the dynamics of meningococcal diseases with antibiotic resistance threats in the presence of vaccines. We organized as follows: Section 2 was devoted to formulation of a meningitis model and indicate the parameters and assumed values. In Section 3, we study the model properties qualitatively and identify key determinants that shape the dynamics. In section 4, we apply the numerical method to get the approximate solutions. Section 5 analyzes the computational findings of the study using tables and graphs. In Sections 6, was solely used to drawing conclusions and areas for further studies.

2. Meningitis Model

The model is divided into 5 groups which include: Susceptible group (S), Drug sensitive infectives (I), drug resistant infectives (R_T) and recoveries (R). The model has a population fraction q that is vaccinated and a fraction of $(1-q)$ of the total population that become susceptible to the diseases. The susceptible individual gets infection through contact with drug sensitive infective or through contact with drug resistant infectives with an infection rate of $\chi = \frac{\xi I(t)+\xi \Upsilon R_T(t)}{N}$, where $\xi = \kappa\tau$ is the effective infection rate, κ is the rate getting infected, τ is the likelihood of an infective person can transmit an infection and Υ is coefficient for the drug resistant to cause an infection.

$$\begin{cases} \frac{dS}{dt} = (1-q)\varphi N + \pi R - (\chi+\mu)S \\ \frac{dI}{dt} = \chi pS - (\omega+\delta+\mu+\alpha_1)I \\ \frac{dR_T}{dt} = \chi(1-p)S + \delta I - (\theta+\mu+\alpha_2)R_T \\ \frac{dR}{dt} = q\varphi N + \omega I + \theta R_T - (\pi+\mu) \end{cases} \tag{1}$$

subject to the initial conditions $S(0) = S_0, I(0) = I_0, R_T(0) = R_{T0}$ and $R(0) = R_0$ with $N = S + I + R_T + R$. A sum of the governing model equations in (1) yields,

$$\frac{dN}{dt} = (\varphi-\mu)N - \alpha_1 I - \alpha_2 R_T \tag{2}$$

which is a varying population size with deaths due to fatal meningitis disease strains. The classes are scaled by population N using the following variables, $s = \frac{S}{N}, i = \frac{I}{N}, r_T = \frac{R_T}{N}$ and $r = \frac{R}{N}$ which normalizes the population, such that, the new system gives;

$$\begin{cases} \frac{ds}{dt} = (1-q)\varphi + \pi r - \left(\left(\xi\left(i(t)+\Upsilon r_T(t)\right)+\varphi\right)s + \alpha_1 si + \alpha_2 s r_T \right. \\ \frac{di}{dt} = p\xi\left(i(t)+\Upsilon r_T(t)\right)s - (\omega+\delta+\varphi+\alpha_1)i + \alpha_1 i^2 + \alpha_2 i r_T \\ \frac{dr_T}{dt} = (1-p)\xi\left(i(t)+\Upsilon r_T(t)\right)s + \delta i - (\theta+\varphi+\alpha_2)r_T + \alpha_1 i r_T + \alpha_2 r_T^2 \\ \frac{dr}{dt} = q\varphi + \omega i + \theta r_T - (\pi+\varphi)r + \alpha_1 i r + \alpha_2 r_T r \end{cases} \tag{3}$$

3. Model Properties

3.1. Feasible Region

In the absence of deaths due to Meningitis disease, model equations in (1) gives,

$$\frac{dN}{N} \leq (\varphi - \mu)dt, \text{ on integration we obtain } N = N_0 \exp(\varphi - \mu)t$$

where N_0 is the initial population computed at $S(0) = S_0, I(0) = I_0, R_T(0) = R_{T0}$ and $R(0) = R_0$, as $t \to \infty$ the total population size $N \to 1$ which means that $0 \leq s + i + r_T + r \leq 1$.

Thus, the feasibility region of equations in (1) is,

$$\Omega = \{(s, i, r_T, r) \in \mathbb{R}_+^4 : 0 \leq s + i + r_T + r \leq 1\} \tag{4}$$

Hence, the meningitis model is epidemiologically and mathematically well stated.

3.2. Non-negative Variables

Here, positive solutions of variables and parameters as time changes to reflect the model variables which are a population of persons.

Theorem 1. Let $\Omega = \{(s, i, r_T, r) \in \mathbb{R}_+^4 : s(0) = s_0, i(0) = i_0, r_T(0) = r_{T0}$ and $r(0) = r_0 \geq 0\}$ with solutions $\{s, i, r_T, r\}$ are non-negative for $t \geq 0$.

The proof. Given the equations in (3), we consider the first equation;

$$\frac{ds}{dt} = (1 - q)\varphi + \pi r - \Big(\big(\xi\big(i(t) + \Upsilon r_T(t)\big) + \varphi\big)s + \alpha_1 si + \alpha_2 s r_t$$

$$\frac{ds}{s} \geq -\Big(\big(\xi\big(i(t) + r r_T(t)\big) + \varphi\big)dt + \alpha_1 i dt + \alpha_2 r_t dt,$$

On integrating and solving for s (t) we have,

$$s(t) = s_0 \exp - \left(\left(\xi \int_0^t \left(i(t)dt + \Upsilon \int_0^t r_T(t)dt\right) + \varphi t\right) + \alpha_1 \int_0^t i dt + \alpha_2 \int_0^t r_t dt \geq 0.\right.$$

Similarly, it can be shown that, $i(t), r_T(t), r(t) \geq 0$. clearly, the proof of the Theorem is complete.

3.3. Model Analysis of Disease-Free State

The model was analyzed qualitatively in the set Ω. In the case of zero Meningitis disease presence the study reveals that there exists a disease-free state, $E_0 = (1 - q, 0,0,0)$. We compute the Jacobian (J_{E_0}) of the equations in (3) at E_0as follows;

$$J_{E_0} = \begin{bmatrix} -\varphi & (-\xi + \alpha_1)(1 - q) & (-\xi\Upsilon + \alpha_2)(1 - q) & \pi \\ 0 & p\xi(1 - q) - \mathrm{a} & p\xi\Upsilon(1 - q) & 0 \\ 0 & \mathrm{x} + \delta & \mathrm{x}\Upsilon - \mathrm{b} & 0 \\ 0 & \omega & \theta & -\mathrm{c} \end{bmatrix} \tag{5}$$

with, $x = (1 - p)\xi(1 - q), a = (\omega + \delta + \varphi + \alpha_1), b = (\theta + \varphi + \alpha_2), c = (\pi + \varphi)$

We compute $\left|J_{E_0} - \lambda I\right| = 0$, by employing the Jacobian method [16] to get the Threshold value, R_e known as the effective reproduction number that shapes the nature of equilibria.

$$R_e = \frac{p\xi(1-q)}{(\omega+\delta+\varphi+\alpha_1)} + \frac{\xi\Upsilon(1-p)(1-q)}{(\theta+\varphi+\alpha_2)} + \frac{\delta p\xi\Upsilon(1-q)}{(\omega+\delta+\varphi+\alpha_1)(\theta+\varphi+\alpha_2)} \tag{6}$$

By applying Routh-Hurwitz approach [3, 16] which stipulates the criteria to be used to establish and identify whether the eigenvalues of a polynomial have negative values.

We have $P(\lambda) = (-\lambda - \varphi)(-\lambda - (\pi + \varphi))(\lambda^2 + c_1\lambda + c_2) = 0, c_1 = (\theta + \varphi + \alpha_2) - p\xi(1 - q) - (1 - p)\xi\Upsilon(1 - q)$ and $c_2 = (\omega + \delta + \varphi + \alpha_1)(\theta + \varphi + \alpha_2) - (\theta + \varphi + \alpha_2)p\xi(1 - q) - (\omega + \delta + \varphi + \alpha_1)(1 - p)\xi\Upsilon(1 - q) - \delta\rho\xi\Upsilon(1 - q)$ Clearly, $\lambda_1 = -\varphi, \lambda_2 = -(\pi + \varphi)$. Note that, $c_2 > 0$ if $R_e < 1$ and $c_1 > 0$ only if $(\theta + \varphi + \alpha_2) > p(1 - q)\xi + \xi(1 - p)\Upsilon(1 - q)$, Satisfying the conditions for Routh-Hurwitz approach [3, 16]. This confirms that the disease-free state is asymptotically stable locally since that all the negative eigenvalues i.e., $R_e < 1$ and it becomes unstable only when $R_e > 1$. Global analysis study of its stability reveals the set $\left\{(s, i, r_T, r) \in \Omega : \frac{dV}{dt} = 0\right\}$, with the Lyapunov function $V = (\theta + \varphi + \alpha_2)i + (\omega + \delta + \varphi + \alpha_1)\Upsilon r_T$, is the disease-free equilibrium. Global asymptotic stability for E_0 was determined by using Lasalle-Lyapunov theorem [3] $R_e < 1$.

The critical vaccination proportion (q_c) is obtained by setting $R_e = 1$.

$$q_c = 1 - \frac{(\omega + \delta + \varphi + \alpha_1)(\theta + \varphi + \alpha_2)}{(\theta + \varphi + \alpha_2)p\xi + (\omega + \delta + \varphi + \alpha_1)\xi\Upsilon(1 - p) + \delta p\xi\Upsilon}$$

Clearly, $R_e < 1 \Leftrightarrow q > q_c$ i.e., high vaccination coverage that leads to die out of the disease from the community.

3.4. Endemic Equilibrium

The persistence of an infection in a community leads to endemic state, the model attains the endemic equilibrium, $E_* = (s_*, i_*, r_{T*}, r_*)$ computed by equating the nonlinear system of equations in (3) to zero with $s_* > 0, i_* > 0, r_{T*} > 0$ and $r_* > 0$. Solving gives, $s_* = \frac{(\omega+\delta+\varphi+\alpha_1)i_* - \alpha_1 i_*^2 - \alpha_2 i_* r_{T*}}{p\xi(i_* + \Upsilon r_{T*})}$ and $r_* = \frac{q\varphi + \omega i_* + \theta r_{T*}}{\pi + \varphi - \alpha_1 i_* - \alpha_2 r_{T*}}$

Using the second and third equations of system (3) and substituting s_* and r_* gives,

$$s_* = \frac{-\delta i_* + (\theta + \varphi + \alpha_2) r_{T*} - \alpha_1 i_* r_{T*} - \alpha_2 r_{T*}^2}{(1-p)\xi(i_*(t) + \Upsilon r_{T*}(t))} = \frac{(\omega + \delta + \varphi + \alpha_1) i_* - \alpha_1 i_*^2 - \alpha_2 i_* r_{T*}}{p\xi(i_*(t) + r_{T*}}$$

or

$$-\delta p i_* + (\theta + \varphi + \alpha_2) p r_{T*} - \alpha_1 p i_* r_{T*} - \alpha_2 p r_{T*}^2 = (\omega + \delta + \varphi + \alpha_1)(1-p) i_* - \alpha_1 (1-p) i_*^2 - \alpha_2 (1-p) i_* r_{T*}$$

or

$$G i_*^2 + H i_* + K - \{L r_{T*}^2 + M r_{T*} + N\} = 0 \tag{7}$$

were

$$G = \alpha_1(1-p), H = -[(\omega + \delta + \varphi + \alpha_1)(1-p) + \delta p + \alpha_1 p r_{T*}], K = 0, L = \alpha_2 p, M = -[(\theta + \varphi + \alpha_2)p + \alpha_2(1-p) i_*]$$

and $N = 0$.

From equation (7) we have that $f(i_*) + f(r_{T*}) = 0$ which describes a case when the meningitis disease is persistent in the community.

Lemma 1. The non-linear model which has both sensitive and resistant infectious strains will have:

1) Exactly one endemic state if and only if K is less than zero and N is less than zero $\Leftrightarrow R_e > 1$.
2) Exactly one endemic state if H is less than zero, M is less than zero and $K = 0, N = 0$ or $H^2 - 4GK = 0, M^2 - 4LN = 0$.
3) Exactly 2 endemic states if $K > 0, H < 0$ with $H^2 - 4GK > 0$ and $N > 0, M < 0$ with $M^2 - 4LN > 0$.
4) None otherwise.

Now, by Descartes Rule of Signs [15], it follows that there exists one endemic state as proven from equation (7).

Theorem 2. There exists only one endemic state, E_* if and only if R_e is greater than unity.

4. Application of 4th Order Runge-Kutta

The non-linear model solution was done using the numerical 4th order Runge-Kutta criteria [12].

The system of equations in (3) applied to the numerical 4th order Runge-Kutta becomes:

$$\frac{ds}{dt} = f(t, s(t), i(t), r_T(t), r(t)), \frac{di}{dt} = f(t, s(t), i(t), r_T(t), r(t)), \frac{dr_T}{dt} = f(t, s(t), i(t), r_T(t), r(t)) \text{ and } \frac{dr}{dt} = f(t, s(t), i(t), r_T(t), r(t)) \text{ with initial coditions } s(0) = s_0, i(0) = i_0, r_T(0) = r_{T0} \text{ and } r(0) = r_0.$$

where $h = t_{n+1} - t_n, n = 0,1,2,\ldots$ with the Taylor series of $s(t_{n+1}) = s_{n+1}$ about s_n is given by,

$$s(t_{n+1}) = s(t_n) + hf(t_n, s(t_n)) + \tfrac{1}{2!} h^2 f'(t_n, s(t_n)) + \cdots \tag{8}$$

Then, the Runge Kutta method [4] for the equation (8) yields,

$$\begin{aligned} s_{n+1} &= s_n + \tfrac{h}{6}(k_1 s + 2k_2 s + 2k_3 s + k_4 s), \\ i_{n+1} &= i_n + \tfrac{h}{6}(k_1 i + 2k_2 i + 2k_3 i + k_4 i), \\ r_{Tn+1} &= i_n + \tfrac{h}{6}(k_1 r_T + 2k_2 r_T + 2k_3 r_T + k_4 r_T), \\ r_{n+1} &= r_n + \tfrac{h}{6}(k_1 r + 2k_2 r + 2k_3 r + k_4 r), \end{aligned} \tag{9}$$

$$\begin{aligned} k_1 s &= fs(t_n, s_n, i_n, r_{Tn}, r_n), \\ k_1 i &= fi(t_n, s_n, i_n, r_{Tn}, r_n), \\ k_1 r_T &= fr_T(t_n, s_n, i_n, r_{Tn}, r_n), \\ k_1 r &= fr(t_n, s_n, i_n, r_{Tn}, r_n), \end{aligned} \tag{10}$$

$similarly\ k_2 s, k_2 i, k_2 r_T, k_2 r, k_3 s, k_3 i, k_3 r_T, k_3 r$ $and\ k_4 s, k_4 i, k_4 r_T, k_4 r$, can be defined using the RK4 scheme. with $fs = \frac{ds}{dt}, fi = \frac{di}{dt}, fr_T = \frac{dr_T}{dt}$ and $fr = \frac{dr}{dt}$.

The 4th order Runge-Kutta has error of corresponding to

h^5, this means that accurate solutions while using the method can be enhanced through use of small step size of h [3, 12].

5. Computational Results and Discussion

Here, the impact of varying vaccination proportion values on the transmission of a meningitis infections as elaborated by the mathematical equations stated in (3) is investigated. The parameter values obtained from published research articles and estimated parameter values, together with initial conditions ($s_0 = 0.5, i_0 = 0.25, r_{T0} = 0.15$ and $r_0 = 0.1$) of variables used for computations and simulations are shown in Table 1. Computations reveal that the critical vaccination coverage value, $q_c = 0.4502$.

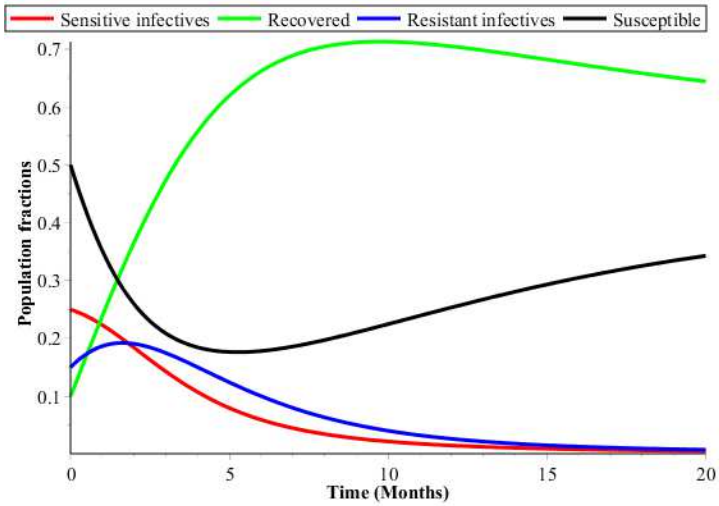

Figure 2. *Population dynamics with time: Case* 1 ($q = 0.8$).

Table 1. *Computations showing the effects of vaccination coverage on disease transmission.*

Case	ξ	δ	ω	φ	θ	α_1	α_2	Υ	π	p	q	R_e	Comments
1	0.89	0.15	0.3	0.1	0.2	0.2	0.15	1.2	0.04	0.6	0.9	0.2195	LAS (E_0)
2	0.89	0.15	0.3	0.1	0.2	0.2	0.15	1.2	0.04	0.6	0.6	0.8781	LAS (E_0)
3	0.89	0.15	0.3	0.1	0.2	0.2	0.15	1.2	0.04	0.6	0.3	1.5367	Unstable (E_0)
4	0.89	0.15	0.3	0.1	0.2	0.2	0.15	1.2	0.04	0.6	0.1	1.9758	Unstable (E_0)
Ref.	[11]	fitted	[11]	[12, 13]	[1]	[2, 9]	fitted	[10]	[5, 14]	fitted			

Figure 2 illustrates how high vaccination proportion i.e., q=0.8 influences the meningitis disease transmission in the community. The susceptible population decreases exponentially with increasing time to a minimum value then gradually increases to asymptotically to reach a steady level. The decrease occurred because of high vaccination coverage leading to increased recruitment to the recovered group. The rise after minimum point can be as a result of decreasing infective individuals due to treatment. Recovered population displays a sharp increase due to recruitment of vaccinated susceptible individuals. Notably, with an initial low number of resistant infective populations the disease gradually grows until it attains a peak value showing that a resistant strain needs close monitoring and detection to enable health providers to plan for treatment and possible hospital beddings needed to gather for rise in infection. The sensitive and resistant infectives decreases gradually to attain disease-free state asymptotically. The rise of resistant infections initially is due to recruitment of susceptible as a result of the high force of infection associated with the meningitis resistant strain. The sharp decrease from peak to disease free state is due to use of prescribed treatment and high vaccination coverage ($q = 0.8$) . The Population asymptotically acquires zero infection state. This precisely demonstrates that a disease-free state is achievable if and only if $q > q_c$.

Figure 3 investigates a low vaccination proportion case scenario on the transmission of meningitis growth present in the community. The susceptible group reduces gradually to a minimum point then increases gradually with time. Thereafter, we note a higher rise of susceptible population as compared to case 1. The recovered population gradually increased due to recruitment of susceptible individuals to reach its highest point and a minimal decrease to a steady point. The resistant strain of meningitis displayed a gradual rise to attain a peak in less than 5 months showing the high infection rate of the strain then decreased gradually to a steady state. The sensitive strain showed an exponential decrease due to treatment of the infection. However, it is important to observe that the infectives may never be wiped out in the community and the persistent state will be achieved. Hence, low vaccination proportion level, $q < q_c$ results in endemicity of meningitis in the society. Thus, revealing that the number of infectives (sensitive and resistant strains) may never vanish as time increases and an endemic state persists asymptotically.

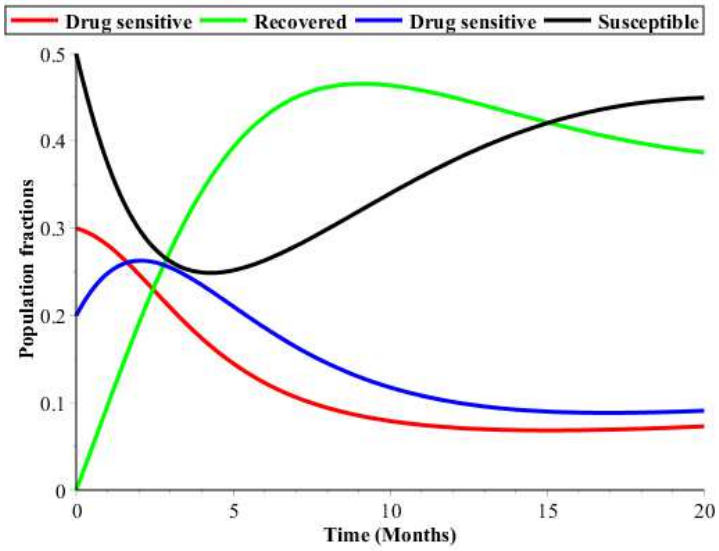

Figure 3. *Population dynamics with time: Case* $1\ (q = 0.2)$*.*

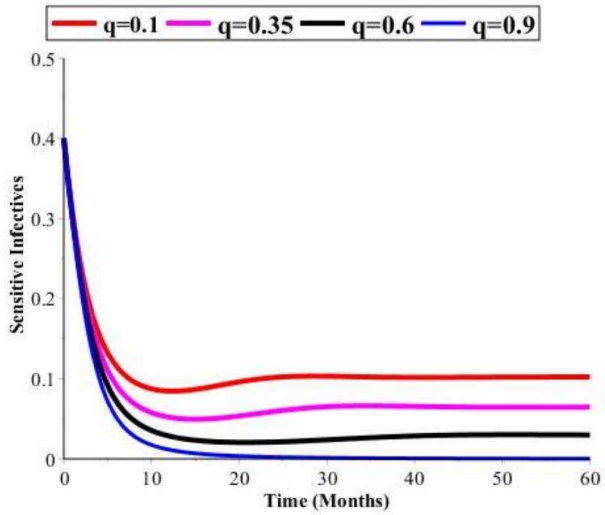

Figure 4. *Investigating the impact of vaccination proportion value on drug sensitive infectives.*

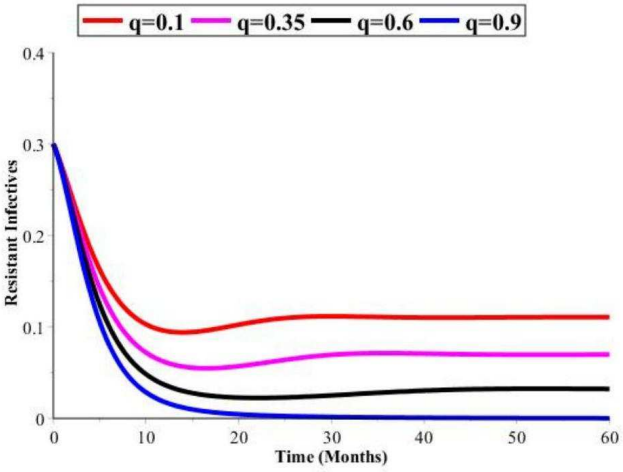

Figure 5. *Investigating the impact of vaccination proportion value on antibiotic resistant infectives.*

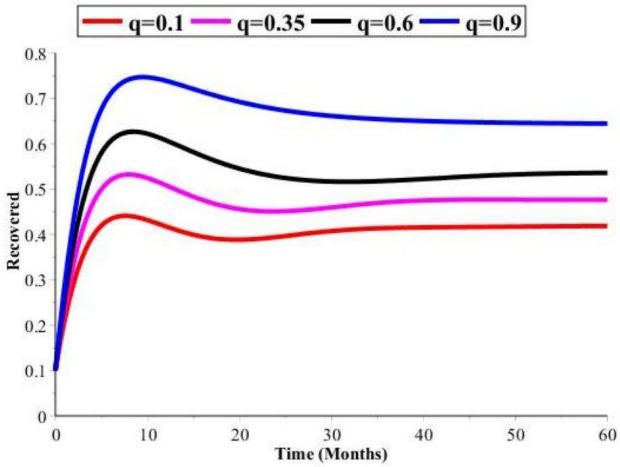

Figure 6. *Investigating the impact of vaccination proportion value on recovered individuals.*

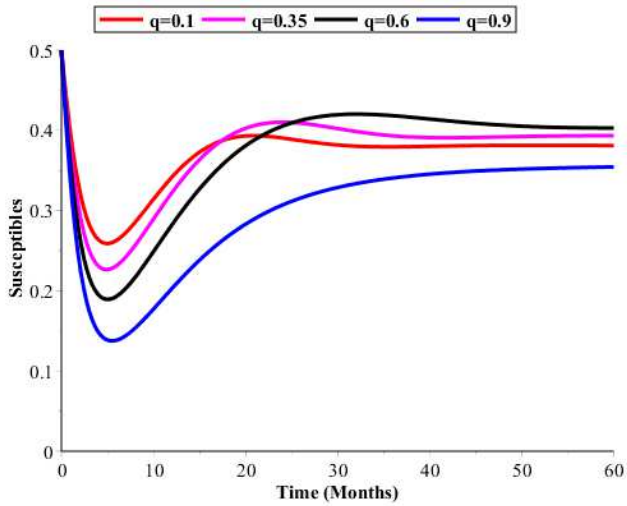

Figure 7. *Investigating the impact of vaccination proportion value on susceptible individuals.*

Figures 4 and 5 shows case 1-4 and reveals that a rise in vaccination value yields a decreasing number of infections (sensitive and resistant strains). Figure 6 shows case 1-4 and reveals that a rise in vaccination coverage (q) yields a corresponding rise in recoveries in the community. Figure 7 shows case 1-4 and reveals that a rise in vaccination coverage yields a reduction to a minimum point then begins to rise.

6. Conclusions

The article presents a meningitis epidemic model that analyzes and simulates the temporal spread growth of meningitis in a varying population and drug resistance threats. The model incorporates a varying population, resistant and sensitive infectious strains to the community. A qualitative analysis reveals zero disease state which has both local and global asymptotically stability provided $R_e < 1$ otherwise unstable for the case $R_e > 1$. Runge-Kutta 4th Order numerical integration approach was used to determine the solution of the system. The findings reveal a solution which converges to disease-free state as time increases provided $q > q_c$ and is persistent for the case when $q < q_c$. We observe that Runge-Kutta method provided findings that agreed with the analytical findings and the solutions converged as time increases. The findings of this study can be continued by future research studies to include delay differential equations to take care of the duration between getting an infection and showing visible symptoms. We also note that a further study on the optimal control analysis of the model will be helpful in comparing the intervention strategies and choosing the most cost-effective approach that is economical especially in low-income nations.

Declaration of Conflict of Interest

The authors declare no conflict of interest.

Data Availability

The data used in the study of the meningitis model were obtained from published articles and reported studies which have been cited accordingly. Some of the parameter values are estimated.

References

[1] Asamoah, J. K. K., Nyabadza, F., Seidu, B., Chand, M., & Dutta, H. (2018). Mathematical modelling of bacterial meningitis transmission dynamics with control measures. Computational and mathematical methods in medicine, 2018.

[2] Blyuss, K. B. (2016). Mathematical modelling of the dynamics of meningococcal meningitis in Africa. In UK Success Stories in Industrial Mathematics (pp. 221-226). Springer.

[3] Boyce, W. E., DiPrima, R. C., & Haines, C. W. (2001). Elementary differential equations and boundary value problems, volume 9. Wiley New York.

[4] Christodoulou, N. S. (2009). An algorithm using Runge-Kutta methods of orders 4 and 5 for systems of odes. Int. J. Numer. Methods Appl, 2 (1), 47-57.

[5] Karachaliou, A., Conlan, A. J., Preziosi, M.-P., & Trotter, C. L. (2015). Modeling long-term vaccination strategies with menafrivac in the African meningitis belt. Clinical Infectious Diseases, 61 (suppl_5), S594-S600.

[6] Martínez, M. F., Merino, E. G., Sánchez, E. G., Sánchez, J. G., del Rey, A. M., & Sánchez, G. R. (2013). A mathematical model to study the meningococcal meningitis. Procedia Computer Science, 18, 2492-2495.

[7] Mbaeyi, S. A., Bozio, C. H., Duffy, J., Rubin, L. G., Hariri, S., Stephens, D. S., & MacNeil, J. R. (2020). Meningococcal vaccination: recommendations of the advisory committee on immunization practices, United States, 2020. MMWR Recommendations and Reports, 69 (9), 1.

[8] Mbaeyi Sarah, Jonathan Duffy, M. L. A. (2020). Meningococcal disease. CDC Reports.

[9] Organization, W. H. et al. (2007). Standardized treatment of bacterial meningitis in Africa in epidemic and non-epidemic situations. Technical report, World Health Organization.

[10] Tilahun, G. T. (2019). Optimal control analysis of pneumonia and meningitis coinfection. Computational and Mathematical Methods in Medicine, 2019.

[11] Vereen, K. (2008). An scir model of meningococcal meningitis.

[12] Yano, K. T. (2018). SEIR Childhood Disease Model with Constant Vaccination Strategy. PhD thesis, JKUAT-PAUSTI.

[13] Afolabi, M. A., Adewoye, K. S., Folorunso, A. I., & Omoloye, M. A. (2021). A mathematical model on transmission dynamics of meningococcal meningitis. *Iconic Research and Engineering Journal*, (pp. 59–66).

[14] Agusto, F. & Leite, M. (2019). Optimal control and cost-effective analysis of the 2017 meningitis outbreak in Nigeria. *Infectious* Disease Modelling, 4, 161–187.

[15] Pinkert, J. R. (1976). An exact method for finding the roots of a complex polynomial. *ACM Transactions on Mathematical Software* (TOMS), 2 (4), 351–363.

[16] Marsden, J. E., Sirovich, L., Antman, S. S., Iooss, G., Holmes, P., Barkley, D., & Newton, P. Texts in Applied Mathematics 37.