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Department of Mathematics

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EXPERIENTIAL LEARNING

A Behavioural Vaccination Model with application to Meningitis spread in Nigeria

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CONTENTS

ABSTRACT

This study explores the impact of vaccine hesitancy on the transmission of meningococcal meningitis in Nigeria using a behavioral epidemic model. The model incorporates an information index, representing the influence of information and rumors on individuals' decisions to vaccinate. The model was applied to data from the 2016-2017 meningitis outbreak in Nigeria, the largest ever global epidemic of serogroup C meningitis. The findings reveal that voluntary vaccination alone is insufficient to eradicate the disease, as the control reproduction number remains unaffected by the information parameters. The analysis estimates that the public was aware of approximately 69% of official data, with a delay of 4.27 days for information dissemination. The study demonstrates the utility of behavioral epidemic models with information indices in understanding the dynamics of disease spread and the challenges posed by vaccine hesitancy in controlling epidemics.

We analyze the equations and graphs obtained from the paper, incorporate in 3 new parameters, viz. A viz., **Social Influence, Differential Access to Healthcare, and Misinformation Index**, and conduct a comparative study through the medium of a website.

INTRODUCTION

The meninges, membranes surrounding the brain and spinal cord, can be affected by a devastating disease, the meningitis. Although the disease affects all ages, young children are most at risk. Bacteria, fungi, viruses and parasites are the most common causes of meningitis, and the highest global burden of disease is seen with meningitis caused by bacteria. There are four main causes of acute bacterial meningitis: Neisseria meningitidis (meningococcus), Streptococcus pneumoniae (pneumococcus), Haemophilus influenzae, Streptococcus agalactiae (group B streptococcus). These bacteria were responsible for over 50% of the 250,000 deaths from all–cause meningitis in 2019.

Neisseria meningitidis (Nm) is the only bacterium capable of generating large epidemics of meningococcal disease. Explosive epidemics with incidence rates reaching 1,000 cases per 100,000 inhabitants have been reported, particularly in the African meningitis belt, an area of sub–Saharan Africa which stretches from Senegal in the west to Ethiopia in the east, involving 26 countries.Nm bacteria typically live in the pharyngeal passages of healthy humans without causing symptoms. Therefore, carriers play an important role for this disease and it is believed that they represent the main source of transmission. The bacteria, which only infect humans, are transmitted from person–to–person through respiratory droplets or throat secretions. At least 12 serotypes of meningococcus have been identified, of which groups A, B, C are responsible for about 90% of meningococcal disease cases.

Public Health Challenge: Meningococcal meningitis poses a significant health threat, particularly in regions like Nigeria, where outbreaks can lead to high mortality and morbidity.

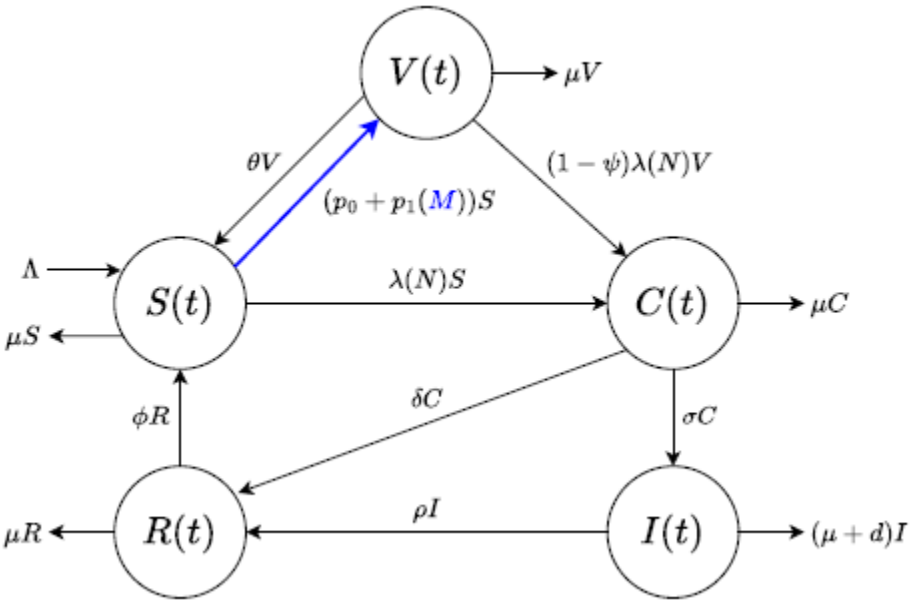
Behavioral Factors in Vaccination: The paper explores the influence of public awareness and information on vaccine uptake, highlighting the importance of socio-behavioral elements in disease control.

Compartmental Model: The study introduces a detailed epidemiological model that categorizes the population into different compartments (Susceptible, Vaccinated, Carriers, Ill, Recovered) to better understand disease dynamics.
Information Index: An innovative aspect of the model is the inclusion of an information index, which quantifies the public's awareness of the disease and its impact on vaccination decisions.
Application to Nigerian Outbreak: The model is applied to the 2016-2017 meningitis outbreak in Nigeria, providing valuable insights into how different factors influence the spread of the disease and the effectiveness of control measures.

The paper highlights the issue of vaccine hesitancy and how it affects the effectiveness of vaccination campaigns. It seeks to understand how behavioral factors, such as the spread of information and rumors, influence individuals' decisions to vaccinate and how these factors impact the overall dynamics of disease transmission. The study aims to develop and apply a behavioral vaccination model that incorporates these elements to better understand and address the challenges in controlling meningitis spread in Nigeria. 3 additional parameters are later added into the model and further analyzed.

MODEL AND ITS PROPERTIES

Compartmental epidemic model to describe the spread of meningococcal meningitis within a population, assuming that a vaccine is available but vaccination can be not mandatory. We assume that the total population is divided into **five** disjoint compartments: **susceptible individuals** (healthy subjects who can contract the disease); **vaccinated** (healthy individuals that received partial and temporal protection by immunization); **carriers** (infected and infectious individuals that carry the Nm, but do not show signs of the invasive disease); **ill individuals** (infected and infectious subjects showing signs of the invasive disease); **recovered** (individuals who recovered after the infectious period). The time–dependent function representing the size of the aforementioned five compartments is denoted, respectively, by S, V , C, I, R. The size of the total population is denoted by N (therefore $N = S + V + C + I + R$).



- The model takes into account the demographic turnover given by immigration and newborns (represented by a constant net **inflow Λ** into the susceptible compartment), and by the **natural mortality of individuals (represented by a rate** μ). Disease–induced mortality of ill individuals occurs at a **rate** d.
- The susceptible population S decreases following infection and vaccination, but vaccinated and recovered individuals become susceptible again because of the waning of vaccine–induced immunity and post–recovery immunity, *respectively*. The transmission is characterized by a force of infection, i.e. the per capita rate at which susceptible individuals become infected.

- **Force of infection** is given by: $\mathrm{FoI} = \beta \frac{\varepsilon C + I}{N}$, ($\beta$ is the disease transmission rate and ε is a transmission modification factor for carriers, $\varepsilon \geq 1$.) This last parameter is motivated by the asymptomatic nature of carriers, who can move freely within the population, while ill individuals are subject to identification and hospitalization.
- The vaccination rate is modeled according to some important aspects of vaccine acceptance. Firstly, it is reasonable to assume that part of the population is strongly pro–vaccine or the vaccine is strongly recommended (or even mandatory) for them like in the case of high risk categories. Such individuals are inclined to be vaccinated independently of the information. This propensity is represented by a positive constant vaccination rate $p\mathbf{0}$. Secondly, we assume that another fraction of susceptibles choose to get vaccinated because of the social alarm caused by the disease. To describe this phenomenon, we follow an approach of the behavioural epidemiology of infectious diseases based on the introduction of the so–called **information index** M, which summarizes the information about the current and past values of the disease. Precisely, the vaccination rate includes also continuous, differentiable and increasing function $p1(M)$, such that $p1(0) = 0$.

$$p_1(M) = (p_{\max} - p_0)\frac{DM}{1 + DM},$$

where $D > 0$ and $p\max > p0$. This formulation implies that $p\max$ is the asymptotic overall rate when the level of circulating information is very high (say, $M \to \infty$), i.e. when there is a very high perceived risk of contagion. Moreover, the parameter D may be interpreted as a measure of how quickly the individuals react to information and voluntarily get vaccinated.

- Individuals move from the susceptible compartment S to the vaccinated compartment V after vaccination. The **size of the compartment** V **decreases** due to infection, although at a reduced transmission rate $(1 - \psi)\beta$, where $\psi \in (0, 1]$ represents the **vaccine effectiveness**, and decreases also because of the **waning of the vaccine–induced immunity, described by a rate** θ.
- Carrier individuals arise as the results of new infections of susceptible and vaccinated individuals. The carrier population C **decreases at a rate** σ due to *transfer to the ill stage at the onset of signs of the invasive disease*, and also **decreases at a rate** δ due to *recovery*.
- Individuals move from the **carrier compartment** C **to the ill compartment** I after *disease progression*. The size of the compartment I decreases at a rate ρ because of recovery. After the infectious period, individuals from the compartments C and I move to the recovered compartment R, but as mentioned above their immunity is not permanent and become susceptible again at a **rate** ϕ.
- Information Index **M** is given by the distributed delay:

$$M(t) = \int_{-\infty}^{t} k\frac{I(\tau)}{\bar{N}} H(\tau) a e^{-a(t-\tau)} d\tau,$$

where H denotes the Heaviside step function. The main assumptions behind this formulation are that the individuals react in response to the current and the past perceived risks associated with the disease, and that the memory of the population is exponentially fading.

Here, as past we mean the period from a given initial time, say $t = 0$, to the current time.

$k\frac{I(t)}{\bar{N}}$, describes the perceived risks associated to the disease, which are assumed to depend on the size of the compartment I. The quantity $\mathcal{N} = \Lambda/\mu$ **is a reference value** ($\mathcal{N}$ is the steady state value of the total population in the disease–free case or when d = **0**).

k is the information coverage, which summarizes two opposite phenomena in the dissemination of information: the under– and over–reporting. The **under–reporting** is due to technical difficulties (e.g. limited resources for case finding) or errors in data recording, and leads to *underestimate* the number of ill cases in the community. The **over–reporting** is due to media and rumors that tend to amplify the public alarm about the disease. We assume here that the former phenomenon prevails on the latter, that is $k \in$ **(0, 1]** (where **1** denotes the appropriate amount of reporting.)

a is the rate parameter of the exponentially fading memory

- By applying the linear chain trick, we obtain the differential equation ruling the dynamics of M:

$$\dot{M} = a\left(k\frac{I}{\bar{N}} - M\right), \qquad \text{(upper dot on M denotes dM/dt)}$$

- According to the above descriptions, the following non-linear ODE's are obtained

$$\dot{S} = \Lambda - \beta S \frac{\varepsilon C + I}{N} - \left(p_0 + (p_{\max} - p_0)\frac{DM}{1+DM}\right) S + \theta V + \phi R - \mu S,$$

$$\dot{V} = \left(p_0 + (p_{\max} - p_0)\frac{DM}{1+DM}\right) S - (1-\psi)\beta V \frac{\varepsilon C + I}{N} - (\theta + \mu) V,$$

$$\dot{C} = \beta (S + (1-\psi)V)\frac{\varepsilon C + I}{N} - (\sigma + \delta + \mu) C,$$

(dot denotes 1st order derivative)

$$\dot{I} = \sigma C - (\rho + \mu + d) I,$$

$$\dot{R} = \delta C + \rho I - (\phi + \mu) R,$$

$$\dot{M} = a(hI - M), \quad \text{where } h = k/\bar{N} = k\mu/\Lambda.$$

$$S(0) = S_0 > 0,\ V(0) = V_0 \geq 0,\ C(0) = C_0 \geq 0,$$

$$I(0) = I_0 \geq 0,\ R(0) = R_0 \geq 0,\ M(0) = M_0 \geq 0.$$

Temporal horizon, initial conditions and parameters values for model (6).

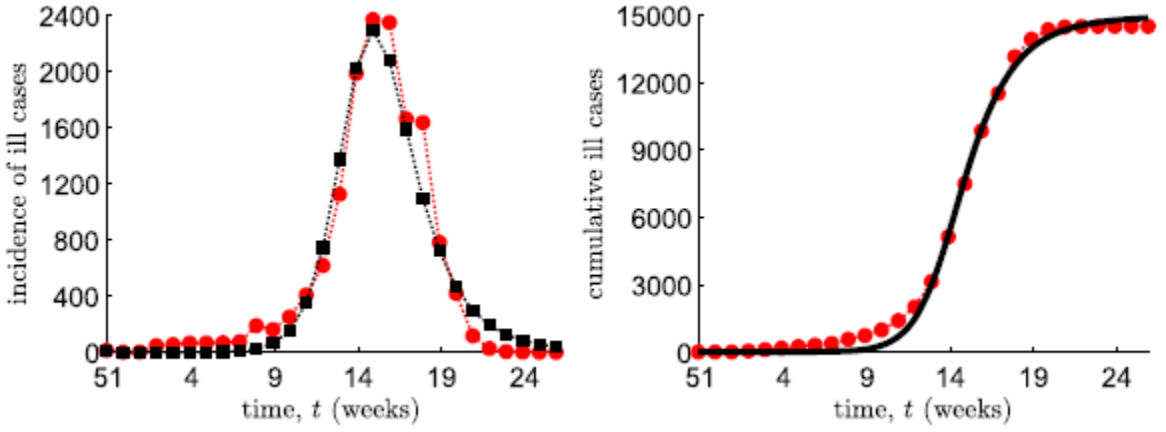
Parameter	Description	Baseline value
t_f	Temporal horizon	27 weeks
$\bar{N}$	Total population at DFE	186,121,000
V_0	Initial number of vaccinated individuals	0
C_0	Initial number of carriers	$10I_0$
I_0	Initial number of ill individuals	14
R_0	Initial number of recovered individuals	0
M_0	Initial value of the information index	$kI_0/\bar{N}$
$\mathcal{R}_0$	Basic reproduction number	1.889
μ	Natural mortality rate	$1/54$ years^{-1}
β	Transmission rate	0.27 days^{-1}
ε	Modification factor w.r.t. transmission from C	1
p_0	Information–independent vaccination rate	$0 - 0.01p_{\max}$
$p_{\max}$	Upper bound of overall vaccination rate	0.01 days^{-1}
D	Reactivity factor to voluntary vaccination	$0 - 6.5 \cdot 10^3$
ψ	Vaccine effectiveness	0.85
θ	Waning rate of vaccine–induced immunity	$1/5$ years^{-1}
σ	Rate at which carriers develop symptoms	0.0051 years^{-1}
δ	Recovery rate for carriers	$1/7$ days^{-1}
ρ	Recovery rate for ill individuals	0.1128 days^{-1}
d	Disease–induced mortality rate	0.01 days^{-1}
ϕ	Waning rate of natural immunity	0.0032 days^{-1}
k	Information coverage	0.6945
a	Inverse of information delay	0.2341 days^{-1}

MODELING THE OUTBREAK (2016/17)

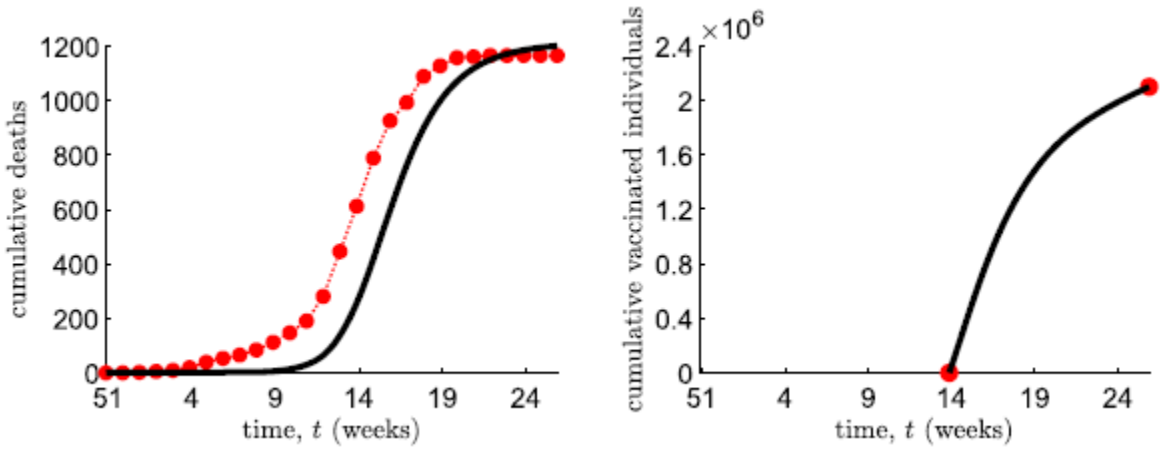
- Reproduce the time evolution of the 2016/2017 Nigerian meningitis outbreak with information–dependent vaccination rate.
- Numerical simulations are performed in MATLAB. We use the *ode45* solver for integrating the system and the platform–integrated functions for getting the plots.
- In Fig. below, a comparison is shown between the official data of the Nigerian outbreak and the solutions of model (6), where we identify the ill cases with the suspected cases reported by Nigerian health authorities. The left panel refers to the incidence of ill cases at each week j, WIj , and the right panel refers to the cumulative number of ill cases at time t, CI. According to model (6), we have

$$\mathrm{WI}_j = \int_{7j-6}^{7j} \sigma C(t)dt, \quad \mathrm{CI} = \int_0^t \sigma C(\tau)d\tau,$$

where j denotes the j–th week and t is measured in days.



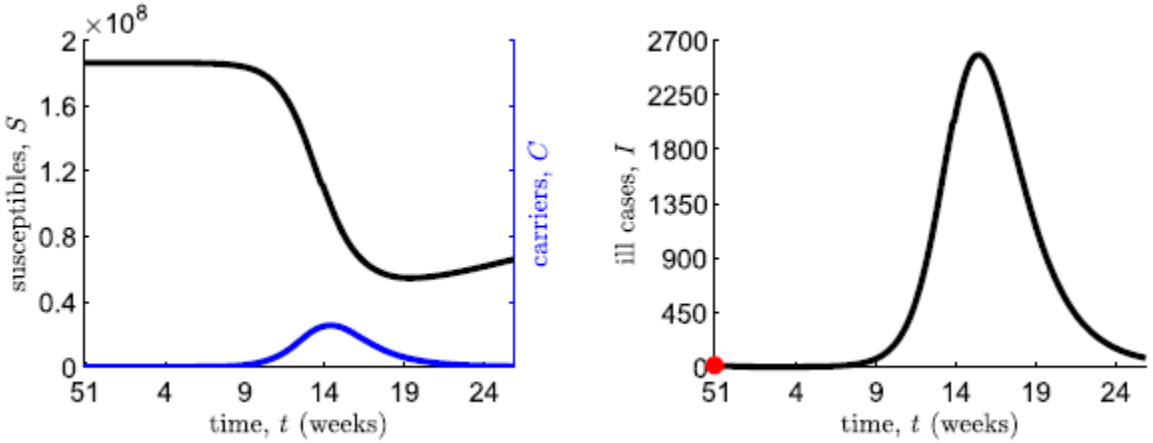
Comparison between official data of 2016/17 outbreak (in **red dots**) and the solutions of model equations **(black lines)**. Left fig.: weekly incidence of ill cases, Right fig.: cumulative ill cases



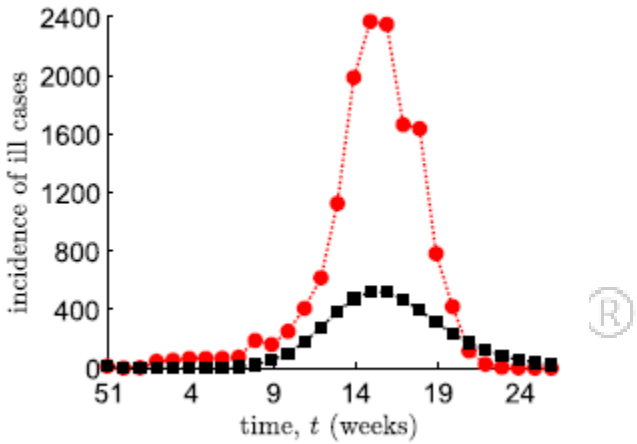
Comparison between official data of 2016/17 outbreak (**red dots**) and model solutions (**black lines**). Left panel: cumulative fig.. Right fig.:cumulated vaccinated individuals

In the above figure, comparison between the official data and the numerical simulations refers to the cumulative number of deaths, CD (left fig.) and the cumulative number of vaccinated individuals, CV (right fig.). These quantities at time t are given by:

$$\mathrm{CD} = \int_0^t dI(\tau)d\tau, \quad \mathrm{CV} = \int_0^t \left(p_0 + (p_{\max} - p_0)\frac{DM(\tau)}{1 + DM(\tau)}\right) S(\tau)d\tau.$$



Epidemic evolution predicted by the model. Left fig.: Susceptibles (**black)** and Carriers (**blue**). Right fig.: Ill cases: the **red dot** indicates the *no. of suspected cases registered at the beginning of the epidemic (14 cases).*



Weekly incidence of ill cases. Comparison between official 2016/17 data (**red dots**) and model solutions (**black lines**) in the case of Highest level of Intervention

MODIFICATIONS AND TASKS PERFORMED

1. Parameters introduced:

   a. Differential Healthcare Access (Γ)

      ➢ The differential healthcare access evolves based on the susceptible and infected populations. It improves with a higher susceptible population and deteriorates with a higher infected population.

      $$\frac{d\Gamma}{dt} = \alpha_\gamma \cdot \frac{S}{N} - \beta_\gamma \cdot \frac{I}{N}$$

      ➢ Alpha and Beta are parameters affecting healthcare access (susceptible population And infected population respectively)

      ➢ If the susceptible population S is large compared to the total population N, healthcare access improves, leading to an increase in γ.

      ➢ Conversely, if the infected population I increases, healthcare access is strained, leading to a decrease in γ.

   b. Social Influence (η)

      ➢ Social influence increases with vaccination rates and decreases due to misinformation. The equation reflects how the spread of vaccination positively influences social behavior, while misinformation has a counteracting effect.

      $$\frac{d\eta}{dt} = \alpha_\eta \cdot \frac{V}{N} - \beta_\eta \cdot \frac{M}{N}$$

➢ Social influence increases as more people get vaccinated (V), but it decreases if misinformation (ξ) spreads.

➢ If the vaccination rate V is initially high, η might rise. However, if misinformation spreads rapidly, it could counteract this effect, leading to a decline or stabilization in η.

c. Misinformation (ξ)

➢ Misinformation spreads within the population and is countered by increasing vaccination. The evolution of misinformation is modeled as a balance between its natural spread and efforts to mitigate it through vaccination.

$$\frac{d\xi}{dt} = \alpha_\xi \cdot M - \beta_\xi \cdot \frac{V}{N}$$

➢ Alpha and Beta are parameters affecting misinformation spread and vaccination counteraction respectively.

➢ Misinformation (ξ) spreads within the population at a rate proportional to its current level, but it can be counteracted by the spread of vaccination.

➢ If misinformation spreads quickly (αξ is large) and vaccination is not widespread enough to counteract it, ξ might increase rapidly.

➢ If vaccination is effective, ξ might eventually stabilize or decrease.

2. Modified Equations:

Addition of the 3 parameters alter the original equations to give:

$$\frac{dS}{dt} = \Lambda - \beta \cdot \frac{S \cdot (\epsilon \cdot C + I)}{N} - \gamma \cdot \left(p_0 + \frac{(p_{max} - p_0) \cdot D \cdot M}{1 + D \cdot M}\right) \cdot S + \theta \cdot V + \phi \cdot R - \mu \cdot S$$

$$\frac{dV}{dt} = \gamma \cdot \left(p_0 + \frac{(p_{max} - p_0) \cdot D \cdot M}{1 + D \cdot M}\right) \cdot S - (1 - \psi) \cdot \beta \cdot \frac{V \cdot (\epsilon \cdot C + I)}{N} - (\theta + \mu) \cdot V$$

$$\frac{dC}{dt} = \beta \cdot \frac{(S + (1 - \psi) \cdot V) \cdot (\epsilon \cdot C + I)}{N} - (\sigma + \delta + \mu) \cdot C$$

$$\frac{dI}{dt} = \sigma \cdot C - (\rho + \mu + d) \cdot I$$

$$\frac{dR}{dt} = \delta \cdot C + \rho \cdot I - (\phi + \mu) \cdot R$$

$$\frac{dM}{dt} = a \cdot \left(\frac{k \cdot I}{\Lambda} - M\right) + \eta \cdot M - \xi \cdot M$$

where, the parameters are:

$$\frac{d\Gamma}{dt} = \alpha_\gamma \cdot \frac{S}{N} - \beta_\gamma \cdot \frac{I}{N}$$

$$p_{\text{eff}} = \gamma \left(p_0 + \frac{(p_{\max} - p_0) \cdot D \cdot M}{1 + D \cdot M}\right)$$

$$\frac{d\eta}{dt} = \alpha_\eta \cdot \frac{V}{N} - \beta_\eta \cdot \frac{M}{N}$$

$$\frac{d\xi}{dt} = \alpha_\xi \cdot M - \beta_\xi \cdot \frac{V}{N}$$

MODIFIED	ORIGINAL
$\frac{dS}{dt} = \Lambda - \beta \cdot \frac{S \cdot (\epsilon \cdot C + I)}{N} - \gamma \cdot \left(p_0 + \frac{(p_{max} - p_0) \cdot D \cdot M}{1 + D \cdot M}\right) \cdot S + \theta \cdot V + \phi \cdot R - \mu \cdot S$	$\dot{S} = \Lambda - \beta S \frac{\epsilon C + I}{N} - \left(p_0 + (p_{\max} - p_0) \frac{DM}{1 + DM}\right) S + \theta V + \phi R - \mu S$
$\frac{dV}{dt} = \gamma \cdot \left(p_0 + \frac{(p_{max} - p_0) \cdot D \cdot M}{1 + D \cdot M}\right) \cdot S - (1 - \psi) \cdot \beta \cdot \frac{V \cdot (\epsilon \cdot C + I)}{N} - (\theta + \mu) \cdot V$	$\dot{V} = \left(p_0 + (p_{\max} - p_0) \frac{DM}{1 + DM}\right) S - (1 - \psi) \beta V \frac{\epsilon C + I}{N} - (\theta + \mu) V$
$\frac{dC}{dt} = \beta \cdot \frac{(S + (1 - \psi) \cdot V) \cdot (\epsilon \cdot C + I)}{N} - (\sigma + \delta + \mu) \cdot C$	$\dot{C} = \beta \left(S + (1 - \psi) V\right) \frac{\epsilon C + I}{N} - (\sigma + \delta + \mu) C$
$\frac{dI}{dt} = \sigma \cdot C - (\rho + \mu + d) \cdot I$	$\dot{I} = \sigma C - (\rho + \mu + d) I,$
$\frac{dR}{dt} = \delta \cdot C + \rho \cdot I - (\phi + \mu) \cdot R$	$\dot{R} = \delta C + \rho I - (\phi + \mu) R,$
$\frac{dM}{dt} = a \cdot \left(\frac{k \cdot I}{\Lambda} - M\right) + \eta \cdot M - \xi \cdot M$	$\dot{M} = a(hI - M),$ where $h = k/\bar{N} = k\mu/\Lambda.$

These equations were taken and analysis was done via Python and Matlab and an illustrative depiction has been done on Streamlit, Python.

CODE:

```
import streamlit as st
import numpy as np
import matplotlib.pyplot as plt
from scipy.integrate import odeint

# Set up the Streamlit app
st.title('Epidemiological Vaccination Model')
st.markdown("""
Welcome to the *Epidemiological Vaccination Model* app! This app allows you to simulate the spread of a disease within a population and observe the effects of vaccination efforts over time.

You can adjust various parameters to explore different scenarios and understand the dynamics of disease spread, vaccination, and recovery. This model is inspired by real-world epidemiological studies and is tailored to provide insights into managing infectious diseases.

Use the sidebar to input your desired parameters and observe how they affect the susceptible, vaccinated, carrier, infected, recovered populations, and the misinformation index.
""")

# Sidebar for parameters
st.sidebar.header('Model Parameters')
st.sidebar.markdown("""
Adjust the parameters below to simulate different scenarios. These parameters influence the disease dynamics, vaccination effectiveness, and public perception.
""")

Lambda = st.sidebar.number_input('Total Population (Lambda)', value=186121000, min_value=1000, help="Total population size.")
```

```
mu = st.sidebar.number_input('Natural Mortality Rate (mu)', value=1/70, min_value=0.0, max_value=1.0, help="Rate at which individuals naturally die.")
```



```
beta = st.sidebar.number_input('Transmission Rate (beta)', value=0.3, min_value=0.0, max_value=1.0, help="Rate at which the disease spreads.")
epsilon = st.sidebar.number_input('Modification Factor (epsilon)', value=1.0, min_value=0.0, max_value=10.0, help="Factor modifying transmission rate due to carriers.")
p0 = st.sidebar.number_input('Base Vaccination Rate (p0)', value=0.01, min_value=0.0, max_value=1.0, help="Baseline rate of vaccination.")
pmax = st.sidebar.number_input('Max Vaccination Rate (pmax)', value=0.05, min_value=0.0, max_value=1.0, help="Maximum rate of vaccination based on public awareness.")
D = st.sidebar.number_input('Reactivity Factor (D)', value=5000, min_value=0, help="Factor determining public reactivity to the disease.")
psi = st.sidebar.number_input('Vaccine Effectiveness (psi)', value=0.9, min_value=0.0, max_value=1.0, help="Effectiveness of the vaccine.")
theta = st.sidebar.number_input('Waning Rate of Vaccine-Induced Immunity (theta)', value=1/365, min_value=0.0, max_value=1.0, help="Rate at which vaccine-induced immunity wanes.")
sigma = st.sidebar.number_input('Rate of Symptom Development (sigma)', value=0.01, min_value=0.0, max_value=1.0, help="Rate at which carriers develop symptoms.")
delta = st.sidebar.number_input('Recovery Rate for Carriers (delta)', value=1/14, min_value=0.0, max_value=1.0, help="Rate at which carriers recover.")
rho = st.sidebar.number_input('Recovery Rate for Ill (rho)', value=1/10, min_value=0.0, max_value=1.0, help="Rate at which infected individuals recover.")
d = st.sidebar.number_input('Disease-Induced Mortality Rate (d)', value=1/1000, min_value=0.0, max_value=1.0, help="Mortality rate due to the disease.")
phi = st.sidebar.number_input('Waning Rate of Natural Immunity (phi)', value=1/365, min_value=0.0, max_value=1.0, help="Rate at which natural immunity wanes.")
k = st.sidebar.number_input('Information Coverage (k)', value=0.5, min_value=0.0, max_value=1.0, help="Extent of information coverage about the disease.")
a = st.sidebar.number_input('Characteristic Memory Length (a)', value=1/30, min_value=0.0, max_value=1.0, help="Memory length affecting public response to the disease.")
alpha_gamma = st.sidebar.number_input('Growth Rate for Healthcare Access (alpha_gamma)', value=0.01, min_value=0.0, max_value=1.0, help="Rate at which access to healthcare improves.")
beta_gamma = st.sidebar.number_input('Reduction Rate for Healthcare Access (beta_gamma)', value=0.005, min_value=0.0, max_value=1.0, help="Rate at which healthcare access is reduced.")
alpha_eta = st.sidebar.number_input('Growth Rate for Social Influence (alpha_eta)', value=0.02, min_value=0.0, max_value=1.0, help="Rate at which social influence grows.")
```

```
beta_eta = st.sidebar.number_input('Reduction Rate for Social Influence (beta_eta)', value=0.01, min_value=0.0, max_value=1.0, help="Rate at which social influence wanes.")
```



```
alpha_xi = st.sidebar.number_input('Growth Rate of Misinformation (alpha_xi)', value=0.005, min_value=0.0, max_value=1.0, help="Rate at which misinformation spreads.")
beta_xi = st.sidebar.number_input('Reduction Rate of Misinformation (beta_xi)', value=0.002, min_value=0.0, max_value=1.0, help="Rate at which misinformation is countered.")

# Initial conditions
st.sidebar.header('Initial Conditions')
st.sidebar.markdown("Set the initial population distribution among the different groups.")
initial_infected = st.sidebar.number_input('Initial Infected Population', value=1000, min_value=0, max_value=Lambda, help="Initial number of infected individuals.")
initial_vaccinated = st.sidebar.number_input('Initial Vaccinated Population', value=10000000, min_value=0, max_value=Lambda, help="Initial number of vaccinated individuals.")
initial_carriers = st.sidebar.number_input('Initial Carriers Population', value=5000, min_value=0, max_value=Lambda, help="Initial number of carriers.")

# Derived initial conditions
S0 = Lambda - initial_infected - initial_vaccinated - initial_carriers
V0 = initial_vaccinated
C0 = initial_carriers
I0 = initial_infected
R0 = 0
M0 = k * initial_infected / Lambda
gamma0 = 0.8
eta0 = 0.2
xi0 = 0.05

# Time span
tspan = np.linspace(0, 365, 365)

# ODE function
def vaccination_model_dynamic(Y, t, params):
    S, V, C, I, R, M, gamma, eta, xi = Y
     Lambda, mu, beta, epsilon, p0, pmax, D, psi, theta, sigma, delta, rho, d, phi, k, a, alpha_gamma, beta_gamma, alpha_eta, beta_eta, alpha_xi, beta_xi = params
```

N = S + V + C + I + R


```
p_effective = gamma * (p0 + (pmax - p0) * D * M / (1 + D * M))

dSdt = Lambda - beta * S * (epsilon * C + I) / N - p_effective * S + theta * V + phi * R - mu * S
dVdt = p_effective * S - (1 - psi) * beta * V * (epsilon * C + I) / N - (theta + mu) * V
dCdt = beta * (S + (1 - psi) * V) * (epsilon * C + I) / N - (sigma + delta + mu) * C
dIdt = sigma * C - (rho + mu + d) * I
dRdt = delta * C + rho * I - (phi + mu) * R
dMdt = a * (k * I / Lambda - M) + eta * M - xi * M

dGammaDt = alpha_gamma * (S / N) - beta_gamma * (I / N)
dEtaDt = alpha_eta * (V / N) - beta_eta * (M / N)
dXiDt = alpha_xi * M - beta_xi * (V / N)

return [dSdt, dVdt, dCdt, dIdt, dRdt, dMdt, dGammaDt, dEtaDt, dXiDt]

# Parameters tuple
params = (Lambda, mu, beta, epsilon, p0, pmax, D, psi, theta, sigma, delta, rho, d, phi, k, a, alpha_gamma, beta_gamma, alpha_eta, beta_eta, alpha_xi, beta_xi)

# Initial conditions vector
Y0 = [S0, V0, C0, I0, R0, M0, gamma0, eta0, xi0]

# Solving the ODEs
solution = odeint(vaccination_model_dynamic, Y0, tspan, args=(params,))

# Extract solutions
S, V, C, I, R, M, gamma, eta, xi = solution.T

# Plotting the results
st.subheader('Model Results')
st.markdown("""
The graphs below represent the population dynamics over time. Each graph shows the number of individuals in a specific category as the disease spreads and the effects of vaccination take place.
""")
```

```
fig, axs = plt.subplots(3, 2, figsize=(12, 12))
```



```
axs[0, 0].plot(tspan, S, '-b', linewidth=1.5)
axs[0, 0].set_xlabel('Time (days)')
axs[0, 0].set_ylabel('Susceptible Population (S)')
axs[0, 0].set_title('Susceptible Population (S)')
axs[0, 0].grid(True)

axs[0, 1].plot(tspan, V, '-g', linewidth=1.5)
axs[0, 1].set_xlabel('Time (days)')
axs[0, 1].set_ylabel('Vaccinated Population (V)')
axs[0, 1].set_title('Vaccinated Population (V)')
axs[0, 1].grid(True)

axs[1, 0].plot(tspan, C, '-r', linewidth=1.5)
axs[1, 0].set_xlabel('Time (days)')
axs[1, 0].set_ylabel('Carriers Population (C)')
axs[1, 0].set_title('Carriers Population (C)')
axs[1, 0].grid(True)

axs[1, 1].plot(tspan, I, '-m', linewidth=1.5)
axs[1, 1].set_xlabel('Time (days)')
axs[1, 1].set_ylabel('Infected Population (I)')
axs[1, 1].set_title('Infected Population (I)')
axs[1, 1].grid(True)

axs[2, 0].plot(tspan, R, '-c', linewidth=1.5)
axs[2, 0].set_xlabel('Time (days)')
axs[2, 0].set_ylabel('Recovered Population (R)')
axs[2, 0].set_title('Recovered Population (R)')
axs[2, 0].grid(True)


plt.tight_layout()
st.pyplot(fig)
```

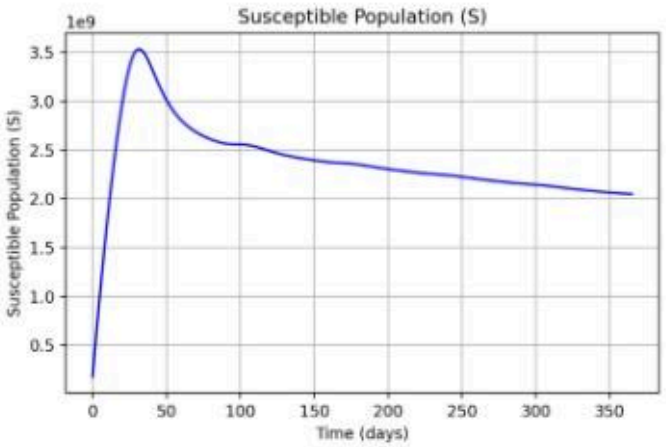
st.subheader('Understanding the Outputs')
st.markdown("""

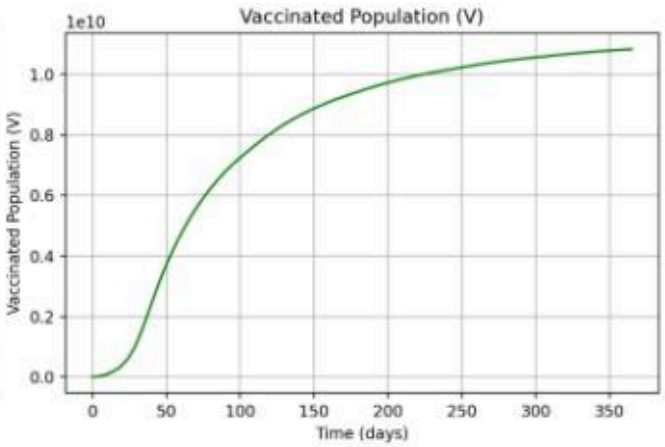
- *Susceptible Population (S)*: This represents individuals who are not infected but are at risk of contracting the disease.
- *Vaccinated Population (V)*: Individuals who have received the vaccine. The effectiveness of the vaccine is reflected in this graph.
- *Carriers Population (C)*: People who carry the disease without showing symptoms, contributing to the disease's spread.
- *Infected Population (I)*: The number of individuals who are currently symptomatic and can spread the disease.
- *Recovered Population (R)*: Individuals who have recovered from the disease and gained natural immunity.
- *Misinformation Index (M)*: Reflects the impact of misinformation on public perception and vaccination rates.

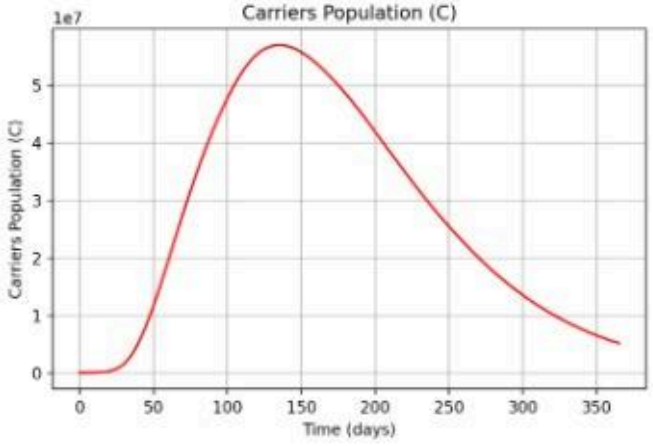
This model allows you to visualize the intricate balance between disease spread, public health interventions, and social factors. Use this tool to gain insights into how different strategies could influence disease outcomes in real-world scenarios.
""")

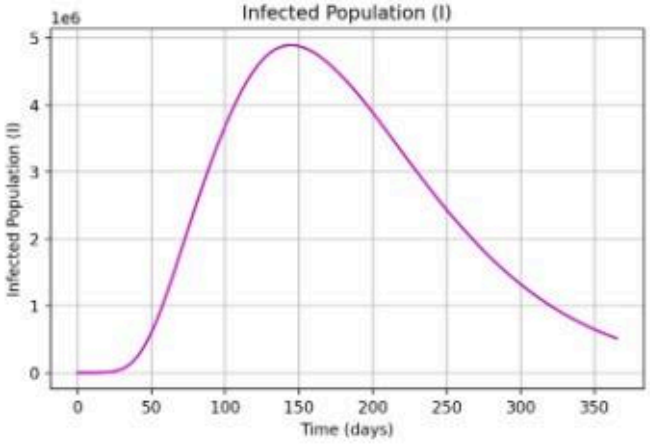
st.subheader('References and Further Reading')
st.markdown("""
- *[Epidemiology: An Introduction](https://example.com/epidemiology-introduction)*: A comprehensive resource to understand the basics of epidemiology.
- *[The Role of Vaccination in Disease Control](https://example.com/vaccination-disease-control)*: An article detailing how vaccination impacts disease dynamics.
- *[Understanding Misinformation and Its Impact](https://example.com/misinformation-impact)*: Learn more about how misinformation affects public health efforts.
""")

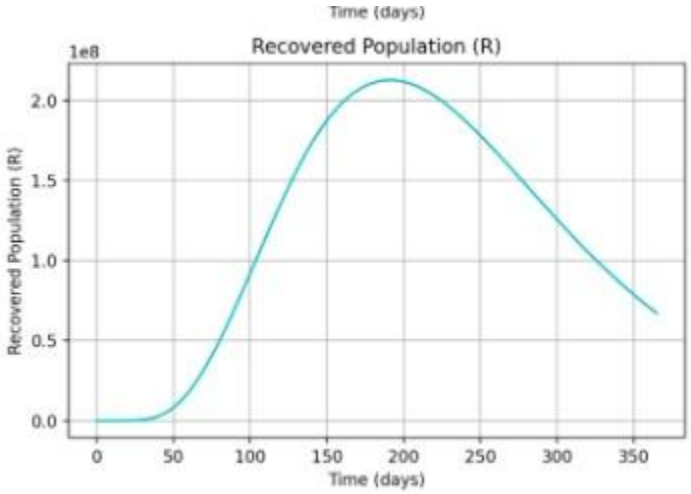
st.markdown("### Thank you for using the Epidemiological Vaccination Model app. Stay informed, stay safe!")


Susceptible Population (S)
1e9
Susceptible Population (S)
Time (days)


Vaccinated Population (V)
1e10
Vaccinated Population (V)
Time (days)


Carriers Population (C)
1e7
Carriers Population (C)
Time (days)


Infected Population (I)
1e6
Infected Population (I)
Time (days)


Time (days)
Recovered Population (R)
1e8
Recovered Population (R)
Time (days)

CONCLUSION

Study concerns with a behavioral vaccination model with application to a real case of meningitis outbreak in Nigeria. From the point of view of meningitis disease modeling, there is a large literature where the disease transmission and its control are investigated.
Voluntary vaccination is unable to eliminate the disease, since the control reproduction number Rv is independent of information parameters. A partially or fully mandatory vaccination always leads to Rv<R0, although the vaccine is imperfect.The model may admit multiple endemic equilibria both below and above the critical threshold Rv=1. This is consistent with the dynamics observed for SIS–like behavioral models with imperfect vaccination. Two fundamental information–related quantities can be estimated: k, the information coverage regarding the number of ill cases (normalized with respect to N) and (1/a), the information delay. We find k=0.69, meaning that the public
was aware of about 69% of official data, and 1/a=4.27 days, the average time lag necessary for information to reach the public. The model provides estimates of the unreported data, like the epidemic evolution of susceptibles, carriers and the
prevalence of ill cases. In particular, the model indicates a peak of asymptomatic carriers around 13.7% of the total population. This is in agreement with studies on meningococcal carriage in the African meningitis belt The model estimates that the incidence of ill cases would have been still significant, especially during the weeks
around the peak, even in the hypothetical case of an early and massive vaccination campaign. Cumulatively, both the total number of ill cases and the total deaths would have been about 67% less than the registered ones.
In Phase 2 we successfully executed a Matlab-Python simulation of updated equations using additional parameters and portrayed an illustrative analysis of the same.
Adding the three parameters—Γ (differential healthcare access index), η (social influence index), and ξ (misinformation index)—introduces new dynamics that reflect the influence of healthcare infrastructure, social factors, and misinformation on disease spread and

vaccination rates. Here's an explanation of how these parameters affect the model and the conclusions we can draw:

- ❖ Γ (differential healthcare access index)
  - Impact on the Model:

    Gamma influences the effective vaccination rate by modifying the baseline vaccination rate (p_effective). When healthcare access improves (higher gamma), more people can get vaccinated, leading to a higher vaccination rate. Conversely, poor healthcare access (lower gamma) reduces vaccination coverage.
  - Real-Life Implication:

    In real-world scenarios, improving healthcare infrastructure increases the efficiency and reach of vaccination campaigns, which can significantly slow down the spread of disease. Regions with better healthcare access will see more rapid control of an outbreak.
  - Conclusion:

    Investing in healthcare infrastructure is crucial for effective vaccination campaigns and disease control. Without adequate healthcare access, even highly effective vaccines may fail to reach a significant portion of the population.

- ❖ η (social influence index)
  - Impact on the Model:

    Eta represents the influence of social norms and public perception on vaccination. A higher eta means that social factors strongly encourage vaccination, increasing the vaccination rate (p_effective). If social influence is negative (lower eta), it can lead to vaccine hesitancy or refusal, reducing the vaccination rate.
  - Real-Life Implication:

    Social influence plays a vital role in public health campaigns. Communities where vaccination is seen as a social responsibility will

have higher coverage and better disease control. Conversely, areas with strong anti-vaccination sentiment may struggle to achieve herd immunity, leading to persistent outbreaks.

- Conclusion:

  Public health campaigns should address social factors to maximize vaccination rates. Engaging community leaders and promoting positive social norms around vaccination can help overcome vaccine hesitancy.

❖ ξ (misinformation index)

- Impact on the Model:

  ξ affects the misinformation dynamics in the model. Higher xi indicates stronger misinformation spread, which undermines vaccination efforts by decreasing public trust in vaccines. The misinformation index interacts with the public's response to the disease, making it harder to achieve widespread vaccination if misinformation is rampant.

- Real-Life Implication:

  Misinformation, especially on social media, can have a devastating impact on vaccination rates. False information about vaccine safety or efficacy can cause people to refuse vaccines, prolonging outbreaks and leading to preventable deaths.

- Conclusion:

  Combating misinformation is critical for successful vaccination campaigns. Public health authorities need to actively counter misinformation with accurate, evidence-based information to maintain public trust and ensure high vaccination coverage.

★ Combined Effects:

The inclusion of these three parameters emphasizes the complex interplay between healthcare systems, societal factors, and information dynamics in managing an epidemic:

1. Healthcare Infrastructure: Strengthening healthcare access can directly improve vaccination rates, making it a cornerstone of epidemic control.

2. Social Dynamics: Public perception and social influence can either enhance or undermine vaccination efforts, so public health messaging and community engagement are key.

3. Misinformation: Addressing misinformation is essential for maintaining public trust in vaccines and ensuring the success of vaccination programs.

★ Key Takeaways:

1. Integrated Approaches are Essential: Public health strategies must address not only the biological aspects of disease control but also the social and informational landscape.

2. Healthcare and Social Factors Are Interconnected: Investment in healthcare must go hand in hand with efforts to promote positive social influences and counter misinformation.

3. Vaccine Hesitancy Can be Mitigated: Effective communication strategies that leverage social influence and debunk misinformation are critical for achieving high vaccination rates and controlling the spread of disease.

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