



Modeling the Impact of Public Enlightenment on Measles Transmission Dynamic in Kafin Hausa Metropolitan

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ABSTRACT

Measles is a very contagious illness with symptoms such as fever, rash, cough and redness of eyes. It can cause very dangerous consequences such as brain infection and pneumonia. In this research work, SIR model was formulated in order to assess the effect of public enlightenment on the transmission dynamics of measles infection in Kafin Hausa metropolis of Jigawa state. In the mathematical analysis section, the existence, boundedness, and positivity of the solution have been established. The existence of the disease equilibria indicates that there exist two equilibria for the model. They are disease-free equilibrium (DFE) and disease endemic equilibrium point (DEEP). It was determined that the DFE is locally asymptotically stable when the effective reproduction number is less than one ($R_c < 1$) and the DEEP is globally asymptotically stable when the effective reproduction number is greater than one ($R_c > 1$). The numerical simulation reveals that, the public enlightenment rate on Atypically and Severe exposed individuals is the important parameter to reduce the dynamics transmission of measles disease in Kafin Hausa metropolitan, Jigawa State.

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INTRODUCTION

The measles, which is also called rubeola, is a very contagious and deadly airborne disease caused by the virus called Morbillivirus (WHO). Measles spreads among individuals when sick people release the virus through their coughs and sneezes. Its symptoms include fever, cough, runny nose, inflamed eyes and a generalized, maculopapular. Measles also results in a red rash of characteristic appearance and has been known to cause fatal complications such as pneumonia, diarrhea, and encephalitis. Those infected with the disease may develop blindness, deafness or have poor vision. Children below five years of age are particularly vulnerable to the infection. The period between the infection and the development of measles symptoms averages between 7 to 21 days (usually 10-12 days). It is considered an airborne illness because it is transmitted by breathing, coughing, sneezing, or talking in an infected individual. Kafin Hausa Local Government is a high-burden area with generally low vaccination coverage, which contributes to recurrent outbreaks and associated mortality. While not specific to Kafin Hausa, a 2016 report confirmed six child deaths in a measles and whooping cough outbreak in the nearby Sule-Tankarkar LGA, highlighting the localized mortality risk during outbreaks in rural Jigawa communities with low immunization rates.

Measles remains a leading cause of death in young children globally. In 2024, an estimated 95,000 people died from measles, mostly children under the age of five (CDC 2024). The risk of death is higher in developing countries, in malnourished children (especially those with vitamin A deficiency), and in individuals with weakened immune systems (WHO 2023).

Mathematical modeling of infectious diseases can be used to comprehend the dynamics of measles disease transmission and to inform public health policy makers on how to implement effective intervention programs to fight infections.

Numerous models have been developed to understand the transmission dynamics of measles. For example, (Adama and Somma, 2025) developed a mathematical model to examine the transmission dynamics of measles and explore effective control measures. And the results indicate that the equilibrium is locally asymptotically stable when the basic reproduction number R_0 is less than or equal to unity. Numerical simulations were conducted to validate the analytical findings, demonstrating that measles can be eradicated if a sufficiently high level of treatment is applied to the infected population. Also, Hicham *et.al.* (2025) presented a dynamic mathematical model constructed using comprehensive mortality data from the World Health Organization and actual data on measles outbreak propagation. Both local and global stability for scenarios with and without the presence of the disease was demonstrated, sensitivity analysis on the model's parameters to assess their impact on the basic reproduction number R_0 was conducted, and the result was sustained numerically using MATLAB. The mathematical model of measles disease with double dose vaccination control strategy was provided by Agbata *et.al* (2025). In this model, which was formulated using the system of differential equations, the basic reproduction number (R_0) is calculated using the next generation operator approach. From their work, one can see the efficiency of vaccination interventions to control the disease. In the paper "Numerical analysis on Novel mathematical model of measles pneumonia co-infection with treated vaccinated compartment", global sensitivity analysis is conducted in order to investigate the effect of vital parameters on co-infection disease transmission and their effect on the reproduction number of disease. The result demonstrates the effectiveness of the implemented techniques in enhancing the accuracy of the outbreak of predictions (Adil *et.al* 2025). In this research we intend to mathematically model and analyzed the effect of

measles diseases in Kafin Hausa metropolitan to curb its menace.

Model Formulation

The total population $N(t)$ is composed of six epidemiological groups: Susceptible Kafin Hausa individuals (S), Exposed unaware Kafin Hausa individuals (E_u), Exposed aware Kafin Hausa individuals (E_a), Atypical Measles Kafin Hausa individuals (I_t), Severe infectious Kafin Hausa individuals (I_s) and recovered Kafin Hausa individuals (R). Let π be the constant recruitment rate and λ be the force of infection. Suppose μ denotes the natural death

rate, σ_1, σ_2 , is the progression rates of unaware and aware exposed Kafin Hausa individual to I_t . where $\gamma_1, \gamma_2, \gamma_3$ are the treatments rates of E_a, I_t and I_s to R respectively, and ε is the progression rate of atypical measles to I_s . δ denotes the measles induced death rate and β is the effective contact rate while θ represents public enlightenment. The modification parameter η is to reduced infectiousness of I_t . The total population is defined as,

$$N(t) = S + E_u + E_a + I_t + I_s + R \quad (1)$$

Model Follow Chart

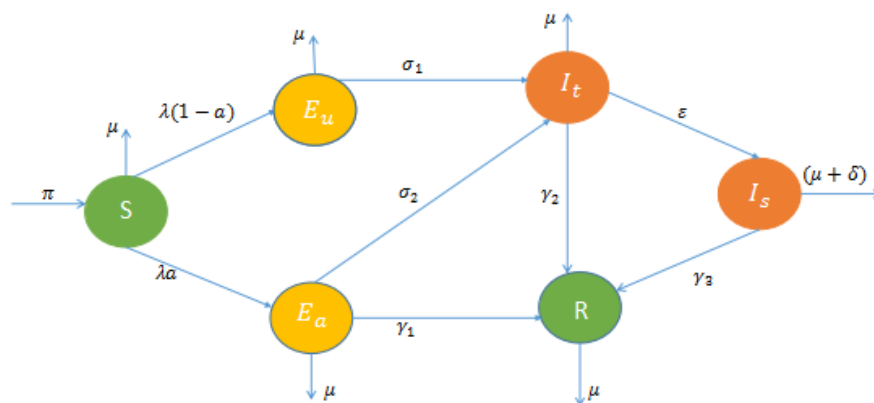


Figure 1: Sketched Diagram of Measles Model

Table 1: The Model Parameters

Compartment	Definition
S	Susceptible K/Hausa individuals
E_u	Exposed Un-aware K/Hausa individuals
E_a	Exposed aware K/Hausa individuals
I_t	Atypical measles K/Hausa individuals
I_s	Severe infectious K/Hausa individuals
R	Recovered K/Hausa individuals
Parameters	Descriptions
μ	Natural death rate
π	Recruitment rate
λ	Force of infection
σ_1, σ_2	Progression rate of unaware and aware exposed K/Hausa individuals to I_t
$\gamma_1, \gamma_2, \gamma_3$	Treatment rate of E_a, I_t and I_s to R respectively.
ε	Progression rate of Atypical measles to I_s
δ	Measles induced death rate
β	Contact rate
θ	Public enlightenment
η	Parameter to reduced infectiousness of I_t .

Model equation

$$\frac{dS}{dt} = \pi - \lambda S - \lambda a S - \mu S$$

$$\frac{dE_u}{dt} = \lambda S - \lambda a S - (\sigma_1 + \mu) E_u$$

$$\frac{dE_a}{dt} = \lambda a S - (\sigma_2 + \gamma_1 + \mu) E_a$$

$$\frac{dI_t}{dt} = \sigma_1 E_u + \sigma_2 E_a - (\gamma_2 + \varepsilon + \mu) I_t$$

(2)

$$\frac{dI_s}{dt} = \varepsilon I_t - (\gamma_3 + \mu + \delta) I_s$$

$$\frac{dR}{dt} = \gamma_1 E_a + \gamma_2 I_t + \gamma_3 I_s - \mu R$$

$$\text{Where, } \lambda = \frac{\beta(1-\theta)(I_s + \eta I_t)}{N}$$

Properties of the model

Model Positivity

We want to show that system (2) is positive means the variable are non-negative since the human population cannot be negative. Let the suitable feasible region for the model be $\mathfrak{D}$ defined by,

$$\mathfrak{D} = \left\{ (S, E_u, E_a, I_t, I_s, R) \in \mathfrak{R}_+^6 : N \leq \frac{\pi}{\mu} \right\} \quad (3)$$

Theorem 1.1: Let the initial condition of the model be

$$\{(S(0), E_u(0), E_a(0), I_t(0), I_s(0), R(0)) \geq 0, \} \in \mathfrak{D}. \quad (4)$$

The set of solution $\{S(t), E_u(t), E_a(t), I_t(t), I_s(t), R(t)\}$ of system (2) is positive for all $t \geq 0$.

Proof: We want to prove that the solution variables $\{S(t), E_u(t), E_a(t), I_t(t), I_s(t), R(t)\}$ of the model correspond to initial conditions $\{(S(0) > 0, E_u(0) > 0, E_a(0) > 0, I_t(0) > 0, I_s(0) > 0, R(0) > 0)\}$ are non-negative. Considering the first equation of system (2),

$$\frac{dS}{dt} = \pi - \lambda S - (2\lambda a + \mu)S \quad (5)$$

For all $t \in [0, 1], \pi \geq 0$, it follows that $\frac{dS}{dt} + (\lambda + 2\lambda a + \mu)S \geq 0$,

By using variable separation method, we can integrate and get,

$$s(t) \geq \frac{\pi}{(\lambda + 2\lambda a + \mu)} + C \exp^{(\lambda + 2\lambda a + \mu)t} \quad (6)$$

as $t \rightarrow \infty, s(t) \geq 0$,

Similarly, apply to all other cases, $E_u(t) = 0, E_a(t) = 0, I_t(t) = 0, I_s(t) = 0, R(t) = 0$ lead to the same contradiction. Thus $E_u(t) \geq 0, E_a(t) \geq 0, I_t(t) \geq 0, I_s(t) \geq 0, R(t) \geq 0$ as $t \rightarrow \infty$.

Where C is an arbitrary constant for all parameters. The model is positive since it shows that is non negative. *Proved*

3.2 Boundedness Property

Theorem 1.2: The region $\mathfrak{D}$ in (3) above, is positively invariant and the system restricted to non-negative condition.

Proof: we need to show that for the total number of human population at time t, $N(t)$ the bounded property holds that $0 \leq N(t) \leq \pi/\mu$ if and only if $0 \leq N(0) \leq \pi/\mu$. From (1), the derivative of the total population is,

$$\frac{dN}{dt} = \pi - \mu N - \sigma_1 I_t - \gamma_2 I_s \leq \pi - \mu N \quad (7)$$

$$\Rightarrow \frac{dN}{dt} + \mu N + \sigma_1 I_t + \gamma_2 I_s = \pi \leq \pi - \mu N \quad (8)$$

as $t \rightarrow 0, N(t) \rightarrow N(0) \Rightarrow N(t) \leq \frac{\pi}{\mu}$ and as $t \rightarrow$

$\infty, N(t) \rightarrow \frac{\pi}{\mu}$. Therefore, the feasible region $\mathfrak{D}$ is positively

invariant for all positive initials condition. *Proved*

3.3 Existence and uniqueness of solutions**Endemic Equilibrium Point**

It is essential to find a positive disease equilibrium point. To achieve this, the RHS of system (2) made to be zero and solve simultaneously. Then, we have

$$\begin{aligned} S^* &= \frac{\pi}{(\lambda + \mu)} \\ E_u^* &= \frac{\lambda(1-a)\pi}{k_1(\lambda + \mu)} \\ E_a^* &= \frac{\lambda a \pi}{k_2(\lambda + \mu)} \\ I_t^* &= \frac{\sigma_1 \lambda(1-a)\pi}{k_1 k_3(\lambda + \mu)} + \frac{\sigma_2 \lambda a \pi}{k_2 k_3(\lambda + \mu)} \\ I_s^* &= \frac{\varepsilon \sigma_1(1-a)\pi \lambda}{k_1 k_3 k_4(\lambda + \mu)} + \frac{\varepsilon \sigma_2 a \pi \lambda}{k_2 k_3 k_4(\lambda + \mu)} \\ R^* &= \frac{\gamma_1 a \pi \lambda}{k_2 \mu(\lambda + \mu)} + \frac{\gamma_2 \sigma_1(1-a)\pi \lambda}{\mu k_1 k_3(\lambda + \mu)} + \frac{\gamma_2 \sigma_2 a \pi \lambda}{\mu k_2 k_3(\lambda + \mu)} + \frac{\varepsilon \gamma_3 \sigma_1(1-a)\pi \lambda}{\mu k_1 k_3 k_4(\lambda + \mu)} + \frac{\varepsilon \gamma_3 \sigma_2 a \pi \lambda}{\mu k_2 k_3 k_4(\lambda + \mu)} \end{aligned} \quad (10)$$

where $k_1 = (\sigma_1 + \mu), k_2 = (\sigma_2 + \gamma_1 + \mu), k_3 = (\gamma_2 + \varepsilon + \mu), k_4 = (\gamma_3 + \mu + \delta)$

Considering (10), all the state variables are positives provided $(\lambda + \mu) > 0$, hence we deduced that the DEEP of the model exist.

Basic Reproduction Number

Local stability of $\mathfrak{D}_0$ of system (2) was determined using next generation operator approach with the notation as

System (2) will be tested to ascertain whether its solution exist, and if the solution exists then it is necessary to show its uniqueness. The existence of the solution is given as, Let,

$$m_1 = \pi - \left[\frac{\beta(1-\theta)(I_s + \eta I_t)}{N} + 2a \left(\frac{\beta(1-\theta)(I_s + \eta I_t)}{N} \right) \right] S - \mu S$$

$$m_2 = \left[(1-a) \left(\frac{\beta(1-\theta)(I_s + \eta I_t)}{N} \right) \right] S - (\sigma_1 + \mu) E_u$$

$$m_3 = \left[\frac{\beta(1-\theta)(I_s + \eta I_t)}{N} \right] a S - (\sigma_2 + \gamma_1 + \mu) E_a$$

$$m_4 = \sigma_1 E_u + \sigma_2 E_a - (\gamma_2 + \varepsilon + \mu) I_t$$

$$m_5 = \varepsilon I_t - (\gamma_3 + \mu + \delta) I_s$$

$$m_6 = \gamma_1 E_a + \gamma_2 I_t + \gamma_3 I_s - \mu R$$

Theorem 1.3: System (2) has a unique solution in the region $\mathfrak{D}$, since that,

$\frac{dm_i}{dt}, i = 1, 2, 3, 4, 5, 6$, in $\mathfrak{D}$ is bounded and continuous.

Proof: Finding the derivatives of m_1 partially with respect to all state variables in (2) we got,

$$\left| \frac{\partial m_1}{\partial S} \right| = \left| - \left[\frac{\beta(1-\theta)(I_s + \eta I_t)}{N} + 2a \left(\frac{\beta(1-\theta)(I_s + \eta I_t)}{N} \right) \right] + \mu \right|$$

$$\left| \frac{\partial m_1}{\partial E_u} \right| = \left| \frac{\partial m_1}{\partial E_a} \right| = \left| \frac{\partial m_1}{\partial R} \right| = 0 < \infty$$

$$\left| \frac{\partial m_1}{\partial I_t} \right| = \left| \frac{\eta \beta(1-\theta)}{N} + 2a \left(\frac{\eta \beta(1-\theta)}{N} \right) \right| < \infty$$

$$\left| \frac{\partial m_1}{\partial I_s} \right| = \left| \frac{\beta(1-\theta)}{N} + 2a \left(\frac{\beta(1-\theta)}{N} \right) \right| < \infty$$

In the same way, for m_2, m_3, m_4, m_5 , and m_6 we can use the same approach, Because all the partial derivatives are smaller than infinity, and therefore the model has a unique solution in the region $\mathfrak{D}$. *Proved*

The Equilibrium Points

System (2) has two equilibrium points (Disease Free Equilibrium and Endemic Equilibrium Point) which are critical for understanding the behavior of the system under different conditions. And these equilibrium points will help to analyzed the dynamics and stability of the system.

Disease Free Equilibrium (DFE)

Next generation operator method was used on system (2) to establish local stability of $\mathfrak{D}^0$. The Disease-free equilibrium was determined as,

$$\mathfrak{D}^0 = (S^0, E_u^0, E_s^0, I_t^0, I_s^0, R^0) = \left(\frac{\pi}{\mu}, 0, 0, 0, 0, 0 \right) \quad (9)$$

defined in (9), the matrices F and V corresponding to the newly introduced infection terms and transfer terms

respectively, so that the basic reproduction number is given by,

$$F = \begin{bmatrix} 0 & 0 & \eta(1-a)\beta(1-\theta) & (1-a)\beta(1-\theta) \\ 0 & 0 & \eta a\beta(1-\theta) & a\beta(1-\theta) \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

And

$$V = \begin{bmatrix} k_1 & 0 & 0 & 0 \\ 0 & k_2 & 0 & 0 \\ -\sigma_1 & -\sigma_2 & k_3 & 0 \\ 0 & 0 & -\varepsilon & k_4 \end{bmatrix}$$

Then V^{-1} is given by,

$$V^{-1} = \begin{bmatrix} \frac{1}{k_1} & 0 & 0 & 0 \\ 0 & \frac{1}{k_2} & 0 & 0 \\ \frac{\sigma_1}{k_1 k_3} & \frac{\sigma_2}{k_2 k_3} & \frac{1}{k_3} & 0 \\ \frac{\varepsilon \sigma_1}{k_1 k_3 k_4} & \frac{\varepsilon \sigma_2}{k_2 k_3 k_4} & \frac{\varepsilon}{k_3 k_4} & \frac{1}{k_4} \end{bmatrix}$$

Also, FV^{-1} is given by,

$$FV^{-1} = \begin{bmatrix} \psi_1 & \psi_2 & \psi_3 & \psi_4 \\ \psi_5 & \psi_6 & \psi_7 & \psi_8 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}$$

Where, $\psi_1 = \frac{\eta(1-a)\beta(1-\theta)\sigma_1}{k_1 k_3} + \frac{(1-a)\beta(1-\theta)\varepsilon\sigma_1}{k_1 k_3 k_4}$

$$\psi_2 = \frac{\eta(1-a)\beta(1-\theta)\sigma_2}{k_2 k_3} + \frac{(1-a)\beta(1-\theta)\varepsilon\sigma_2}{k_2 k_3 k_4}$$

$$\psi_3 = \frac{\eta(1-a)\beta(1-\theta)}{k_3} + \frac{(1-a)\beta(1-\theta)\varepsilon}{k_3 k_4}$$

$$\psi_4 = \frac{(1-a)\beta(1-\theta)}{k_4}$$

$$\psi_5 = \frac{\eta a\beta(1-\theta)\sigma_1}{k_1 k_3} + \frac{a\beta(1-\theta)\varepsilon\sigma_1}{k_1 k_3 k_4}$$

$$\psi_6 = \frac{\eta a\beta(1-\theta)\sigma_2}{k_2 k_3} + \frac{a\beta(1-\theta)\varepsilon\sigma_2}{k_2 k_3 k_4}$$

$$\psi_7 = \frac{\eta a\beta(1-\theta)}{k_3} + \frac{a\beta(1-\theta)\varepsilon}{k_3 k_4}$$

$$\psi_8 = \frac{a\beta(1-\theta)}{k_4}$$

And the eigen values is given as, $\rho(FV^{-1}) = R_c$

Global Asymptotic Stability of Measles Endemic Equilibrium point

Let $D^* = \{(S^*, E_u^*, E_a^*, I_t^*, I_s^*, R^*) \in \mathfrak{R}_+^6\}$ (15)

Theorem 1.5: Equation (10) of the model (Disease Endemic Equilibrium Point) is globally asymptotically stable in $\mathfrak{D}$ whenever $(\lambda + \mu) > 0$.

Proof: We begin in the proof by setting $\sigma_2 = \gamma_1 = \gamma_2 = 0$ then system (2) becomes,

$$\frac{dS}{dt} = \pi - \lambda S - \mu S$$

$$\frac{dE_u}{dt} = \lambda(1-a)S - k'_1 E_u$$

$$\frac{dE_a}{dt} = \lambda a S - k'_2 E_a$$

$$\frac{dI_t}{dt} = \sigma_1 E_u - k'_3 I_t$$

$$\frac{dI_s}{dt} = \varepsilon I_t - k_4 I_s$$

$$\frac{dR}{dt} = \gamma_3 I_s - \mu R$$

Where $k'_1 = (\sigma_1 + \mu)$, $k'_2 = (\sigma_2 + \gamma_1 + \mu)$, $k'_3 = (\gamma_2 + \varepsilon + \mu)$, and $k_4 = (\gamma_3 + \mu + \delta)$.

Let $\mathfrak{U}$ be a Goh-Volterra non-linear Lyapunov function.

$$\mathfrak{U} = \left(S - S^{**} - S^{**} \ln \frac{S}{S^{**}} \right) + \left(E_u - E_u^{**} - E_u^{**} \ln \frac{E_u}{E_u^{**}} \right) + \left(E_a - E_a^{**} - E_a^{**} \ln \frac{E_a}{E_a^{**}} \right) + A_1 \left(I_t - I_t^{**} - I_t^{**} \ln \frac{I_t}{I_t^{**}} \right) + A_2 \left(I_s - I_s^{**} - I_s^{**} \ln \frac{I_s}{I_s^{**}} \right) + A_3 \left(R - R^{**} - R^{**} \ln \frac{R}{R^{**}} \right) \quad (17)$$

Differentiating (17) with respect to time gives,

$$\dot{\mathfrak{U}} = \dot{S} - \frac{S^{**}}{S} \dot{S} + \dot{E}_u - \frac{E_u^{**}}{E_u} \dot{E}_u + \dot{E}_a - \frac{E_a^{**}}{E_a} \dot{E}_a + A_1 \left(\dot{I}_t - \frac{I_t^{**}}{I_t} \dot{I}_t \right) + A_2 \left(\dot{I}_s - \frac{I_s^{**}}{I_s} \dot{I}_s \right) + A_3 \left(\dot{R} - \frac{R^{**}}{R} \dot{R} \right) \quad (18)$$

Putting (2) in to (18) we now have,

$$R_c = \frac{\beta(1-\theta)(\eta k_4 + \varepsilon)(a\sigma_2 k_1 + (1-a)\sigma_1 k_2)}{k_1 k_2 k_3 k_4} \quad (11)$$

Where R_0 is the basic reproductive number, when there is no measures to control the diseases, I.e. setting all control parameters to zero ($\gamma_1 = \gamma_2 = \gamma_3 = 0$), (11) turn to the basic reproduction number (R_0).

$$R_0 = \frac{\beta(1-\theta)(\eta k_4^* + \varepsilon)(a\sigma_2 k_1 + (1-a)\sigma_1 k_2^*)}{k_1 k_2^* k_3^* k_4^*} \quad (12)$$

Where $k_2^* = (\sigma_2 + \mu)$, $k_3^* = (\varepsilon + \mu)$, $k_4^* = (\mu + \delta)$

Local Stability of the Disease-Free Equilibrium

Theorem 1.4: The Disease-free equilibrium is locally asymptotically stable if the basic reproduction number is less than zero ($R_0 < 0$), otherwise unstable if the reproduction number is greater than zero ($R_0 > 0$).

Proof: The Jacobian matrix for the system (2) evaluated at disease free equilibrium is,

$$J(D^0) = \begin{bmatrix} -\mu & 0 & 0 & -\beta(1-\theta)\eta & -\beta(1-\theta) & 0 \\ 0 & -k_1 & 0 & (1-a)\beta(1-\theta)\eta & (1-a)\beta(1-\theta) & 0 \\ 0 & 0 & -k_2 & a\beta(1-\theta)\eta & a\beta(1-\theta) & 0 \\ 0 & \sigma_1 & \sigma_2 & -k_3 & 0 & 0 \\ 0 & 0 & 0 & \varepsilon & -k_4 & 0 \\ 0 & 0 & \gamma_1 & \gamma_2 & \gamma_3 & -\mu \end{bmatrix}$$

From the Jacobian matrix the eigenvalues are $-k_2, -k_1, -\mu$.

When $\chi_1 = -k_2$, $\chi_2 = -k_1$ then the remaining sub matrix by $J(D^0)$ are,

$$\chi_3 = \frac{\eta\sigma_1(1-\theta)(1-a)\beta k_2 + \eta a\beta\sigma_2 k_1(1-\theta) - k_1 k_2 k_3}{k_1 k_2} \quad (13)$$

$\chi_3 < 0$, iff $\eta\sigma_1(1-\theta)(1-a)\beta k_2 + \eta a\beta\sigma_2 k_1(1-\theta) < k_1 k_2 k_3$ and $k_1 k_2 > 0$

$$\chi_4 = \beta(1-\theta)(\eta k_4 + \varepsilon)a\sigma_2 k_1 + \beta(1-\theta)(\eta k_4 + \varepsilon)(1-a)\sigma_1 k_2 - k_1 k_2 k_3 k_4$$

$$\Rightarrow \chi_4 = \frac{k_1 k_2 k_3 k_4 [\beta(1-\theta)(\eta k_4 + \varepsilon)a\sigma_2 k_1 + \beta(1-\theta)(\eta k_4 + \varepsilon)(1-a)\sigma_1 k_2 - 1]}{k_1 k_2 k_3 k_4}$$

$$\chi_4 = \frac{k_1 k_2 k_3 k_4 (R_c - 1)}{k_1 k_2 k_3 k_4} \quad (14)$$

if $R_c < 0$ implies $\chi_4 < 0$

0, it is obvious to show that χ_5, χ_6 are less than zero.

As seen from the above, all the eigenvalues of the Jacobian matrix $J(D^0)$ will be negative when $R_c < 0$; hence, the disease-free equilibrium is locally asymptotically stable.

$$\begin{aligned}\ddot{U} = & \pi - \lambda S - \lambda a S - \lambda a S - \mu S - \frac{S^{**}}{S}(\pi - \lambda S - \lambda a S - \lambda a S - \mu S) + \lambda S - \lambda a S - k'_1 E_u - \frac{E_u^{**}}{E_u}(\lambda S - \lambda a S - k'_1 E_u) + \\ & \lambda a S - k'_2 E_a - \frac{E_a^{**}}{E_a}(\lambda a S - k'_2 E_a) + A_1 \sigma_1 E_u - A_1 k'_3 I_t - \frac{I_t^{**}}{I_t}(A_1 \sigma_1 E_u - A_1 k'_3 I_t) = A_2 \varepsilon I_t - A_2 k_4 I_s - \\ & \frac{I_s^{**}}{I_s}(A_2 \varepsilon I_t - A_2 k_4 I_s) + A_3 \gamma_3 I_s - A_3 \mu R - \frac{R^{**}}{R}(A_3 \gamma_3 I_s - A_3 \mu R)\end{aligned}\quad (19)$$

At EEP $\pi = \lambda(1-a)S^{**} + \lambda a S^{**} + \mu S^{**}$ substituting the value of π in (19) and simplify we got,

$$\begin{aligned}\ddot{U} = & \lambda S^{**} - \lambda a S^{**} + \lambda a S^{**} + \mu S^{**} - \mu S - \lambda \frac{S^{**2}}{S} - \lambda a \frac{S^{**2}}{S} - \lambda a \frac{S^{**2}}{S} - \frac{S^{**2}}{S} \mu + \lambda a S^{**} + \lambda S^{**} - \lambda a S^{**} + \mu S^{**} - \lambda S \frac{E_u^{**}}{E_u} - \lambda a S \frac{E_u^{**}}{E_u} + k_1 E_u^{**} - \\ & k'_2 E_a - \lambda a S \frac{E_a^{**}}{E_a} + k'_2 E_a^{**} - A_1 \sigma_1 E_u - A_1 k'_3 I_t + \frac{I_t^{**}}{I_t} + A_1 k'_3 I_t^{**} - A_2 \varepsilon I_t \frac{I_s^{**}}{I_s} + A_2 k_4 I_s^{**} - A_3 \mu R - A_3 \gamma_3 I_s \frac{R^{**}}{R} + A_3 \mu R^{**}\end{aligned}\quad (20)$$

$$-k'_1 E_u + A_1 \sigma_1 E_u = 0, \Rightarrow A_1 = \frac{k'_1}{\sigma_1} \quad (21)$$

$$-A_1 k'_3 I_t + A_2 \varepsilon I_t = 0, \Rightarrow A_2 = \frac{k'_1 k'_3}{\varepsilon \sigma_1} \quad (22)$$

$$-A_2 k_4 + A_3 \gamma_3 = 0, \Rightarrow A_3 = \frac{k'_1 k'_3 k_4}{\gamma_3 \varepsilon \sigma_1} \quad (23)$$

Substituting (21), (22) and (23) in to (20) we have,

$$\begin{aligned}\ddot{U} = & \lambda S^{**} - \lambda a S^{**} + \lambda a S^{**} + \mu S^{**} - \mu S - \lambda \frac{S^{**2}}{S} - \lambda a \frac{S^{**2}}{S} - \lambda a \frac{S^{**2}}{S} - \mu \frac{S^{**2}}{S} + \lambda a S^{**} + \lambda S^{**} - \lambda a S^{**} + \mu S^{**} - \lambda S \frac{E_u^{**}}{E_u} - \\ & \lambda a S \frac{E_u^{**}}{E_u} + k_1 E_u^{**} - k'_2 E_a - \lambda a S \frac{E_a^{**}}{E_a} + k'_2 E_a^{**} - k'_1 E_u \frac{I_t^{**}}{I_t} + \frac{k'_1 k'_3}{\sigma_1} I_t^{**} - \frac{k'_1 k'_3}{\sigma_1} I_t \frac{I_s^{**}}{I_s} + \frac{k'_1 k'_3 k_4}{\varepsilon \sigma_1} I_s^{**} - \frac{k'_1 k'_3 k_4}{\gamma_3 \varepsilon \sigma_1} \mu R - \frac{k'_1 k'_3 k_4}{\varepsilon \sigma_1} I_s \frac{R^{**}}{R} + \\ & \frac{k'_1 k'_3 k_4}{\gamma_3 \varepsilon \sigma_1} \mu R^{**}\end{aligned}\quad (24)$$

At Endemic Equilibrium Point

$$k'_1 = \left(\lambda \frac{S^{**}}{E_u^{**}} - \lambda a \frac{S^{**}}{E_u^{**}} \right), \quad k'_2 = \lambda a \frac{S^{**}}{E_a^{**}}, \quad k'_3 = \frac{\sigma_1 E_u^{**}}{I_t^{**}}, \quad k_4 = \frac{\varepsilon I_t^{**}}{I_s^{**}}, \quad \mu = \frac{\gamma_3 I_s^{**}}{R^{**}} \quad (25)$$

And $E_u \leq E_u^{**}$, $E_a \leq E_a^{**}$ and $S \leq S^{**}$, then substituting the relation in (25) in to (24) and simplify we got;

$$\begin{aligned}\ddot{U} \leq & 2\mu S^{**} - \mu S - \mu \frac{S^{**2}}{S} + 3\lambda a S^{**} - \lambda a \frac{S^{**2}}{S} - \lambda a S^{**} \frac{E_a}{E_a^{**}} - \lambda a S \frac{E_a^{**}}{E_a} + 6\lambda S^{**} - \lambda a S^{**} - \lambda \frac{S^{**2}}{S} - \lambda a \frac{S^{**2}}{S} - \lambda S \frac{E_u^{**}}{E_u} - \\ & \lambda a S \frac{E_u^{**}}{E_u} - \lambda S^{**} \frac{E_u I_t^{**}}{E_u^{**} I_t} - \lambda a S^{**} \frac{E_u I_t^{**}}{E_u^{**} I_t} - \lambda S^{**} \frac{I_t I_s^{**}}{I_t^{**} I_s} - \lambda a S^{**} \frac{I_t I_s^{**}}{I_t^{**} I_s} - \lambda S^{**} \frac{R}{R^{**}} - \lambda a S^{**} \frac{R}{R^{**}} - \lambda S^{**} \frac{I_s R^{**}}{I_s^{**} R} - \lambda a S^{**} \frac{I_s R^{**}}{I_s^{**} R}\end{aligned}\quad (26)$$

This implies

$$\ddot{U} \leq \mu S^{**} \left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \right) + \lambda a S^{**} \left(3 - \frac{S^{**}}{S} - \frac{E_a}{E_a^{**}} - \frac{S E_a^{**}}{S^{**} E_a} \right) + \lambda (1-a) S^{**} \left(6 - \frac{S^{**}}{S} - \frac{S E_u^{**}}{S^{**} E_u} - \frac{E_u I_t^{**}}{E_u^{**} I_t} - \frac{I_t I_s^{**}}{I_t^{**} I_s} - \frac{R}{R^{**}} - \frac{I_s R^{**}}{I_s^{**} R} \right) \quad (27)$$

However, the arithmetic mean is greater than the geometric mean, so we have,

$$\begin{aligned}\left(2 - \frac{S}{S^{**}} - \frac{S^{**}}{S} \right) & \leq 0; \\ \left(3 - \frac{S^{**}}{S} - \frac{E_a}{E_a^{**}} - \frac{S E_a^{**}}{S^{**} E_a} \right) & \leq 0; \\ \left(6 - \frac{S^{**}}{S} - \frac{S E_u^{**}}{S^{**} E_u} - \frac{E_u I_t^{**}}{E_u^{**} I_t} - \frac{I_t I_s^{**}}{I_t^{**} I_s} - \frac{R}{R^{**}} - \frac{I_s R^{**}}{I_s^{**} R} \right) & \leq 0;\end{aligned}$$

Thus, we get $\ddot{U} \leq 0$ under the assumption that $R_c > 1$, because all the variables (S, E_u, E_a, I_t, I_s, R) are at Endemic steady state, this can be substituted in to relevant variables of equation (2), giving us

$$\lim_{t \rightarrow \infty} (S(t), E_u(t), E_a(t), I_t(t), I_s(t), R(t)) = (S^{**}, E_u^{**}, E_a^{**}, I_t^{**}, I_s^{**}, R^{**}) \quad (28)$$

Therefore, the Disease endemic equilibrium point is globally asymptotically stable (GAS) using the invariant principle. $\square$

Table 2: Parameters Estimations and Assumptions

Parameters	Range	Unit	Source
μ	0.2	per year	Momoh (2013)
π	0.32	per year	Momoh (2013)
σ_1, σ_2	0.01	per year	Momoh (2013)
$\gamma_1, \gamma_2, \gamma_3$	[0,1]	per year	Momoh (2013)
ε	0.125	per year	Anes (2012)
δ	0.125	per year	Tilahun (2020)
β	0.01	per year	Momoh (2013)
θ	[0,1]	per year	Assumed
η	0.143	per year	Anes (2012)
λ	0.096	per year	Stephen et al, (2014)

Parameters Estimation and Numerical Simulations

Due to lack of reliable recent data for measles in Kafin Hausa Local Government area we used the parameter values in this work obtained from the relevant literature that can relate to Kafin Hausa Metropolitan.

Theorems 1.5 and 1.4 analytically demonstrated the local stability of the disease-free equilibrium of the model and the global asymptotic stability of the measles endemic equilibrium, respectively. The following numerical figures provide evidence for them:

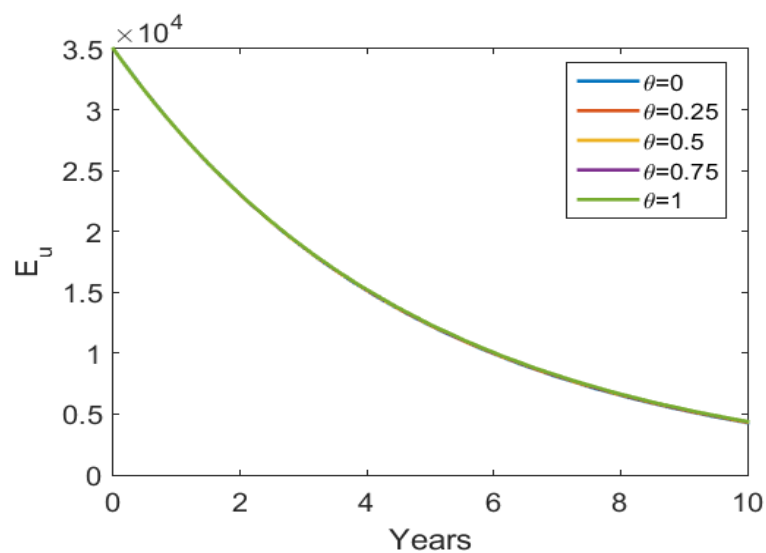


Figure 2: Figure Showing how Public Enlightenment Affects those who are Unaware Exposed Individuals

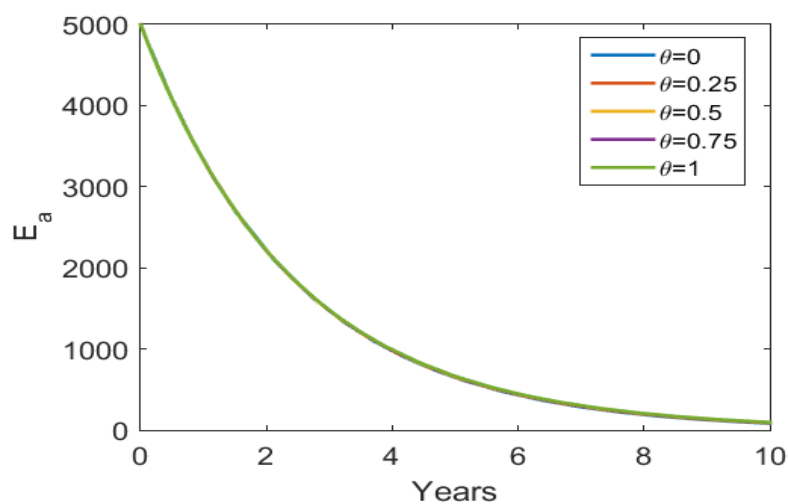


Figure 3: Figure Showing how Public Enlightenment Affects those who are Exposed Aware

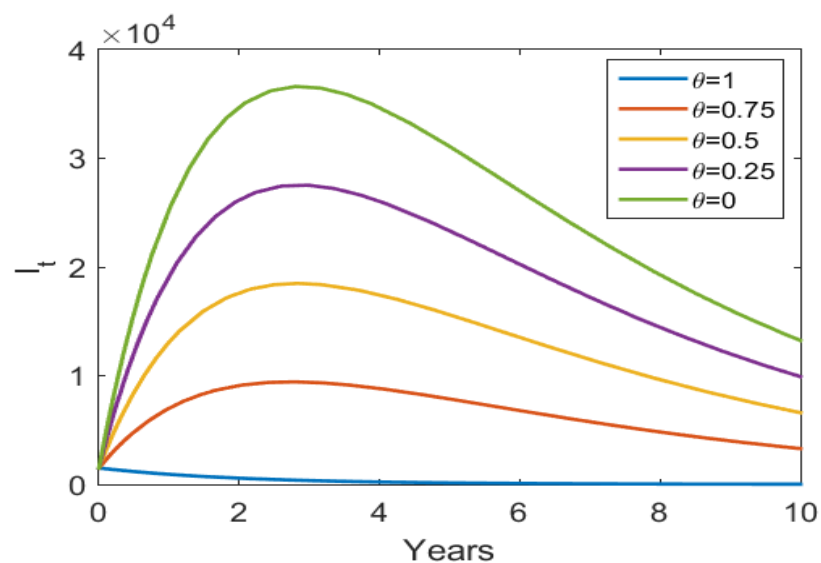


Figure 4: Demonstrating the Effects of Public Enlightenment on Atypical Measles Individuals

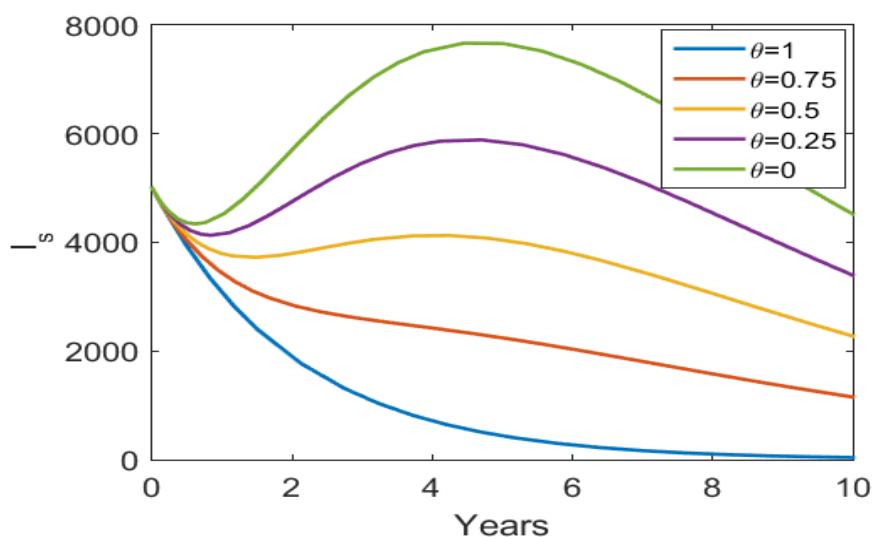


Figure 5: Showing the Effects of Public Enlightenment on Severe Infectious Individuals

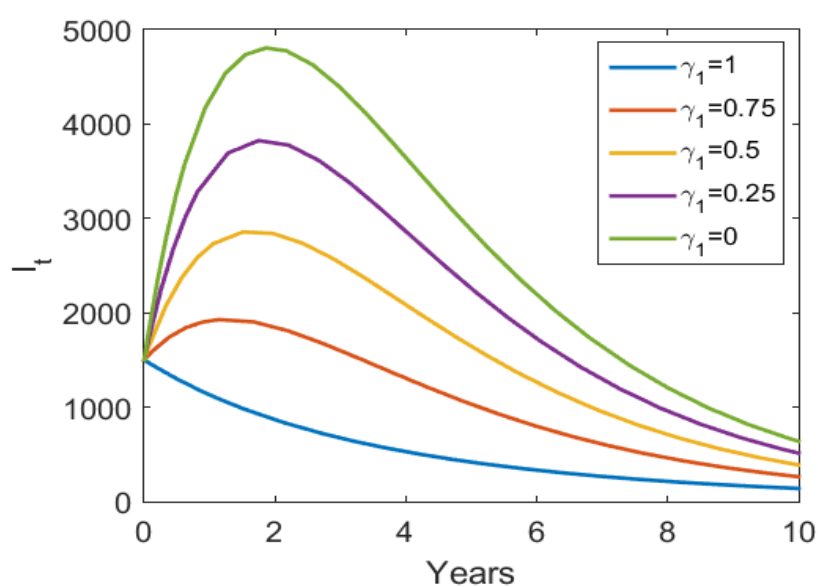


Figure 6: Treatment Control's Effects on People with Atypical Measles Infection

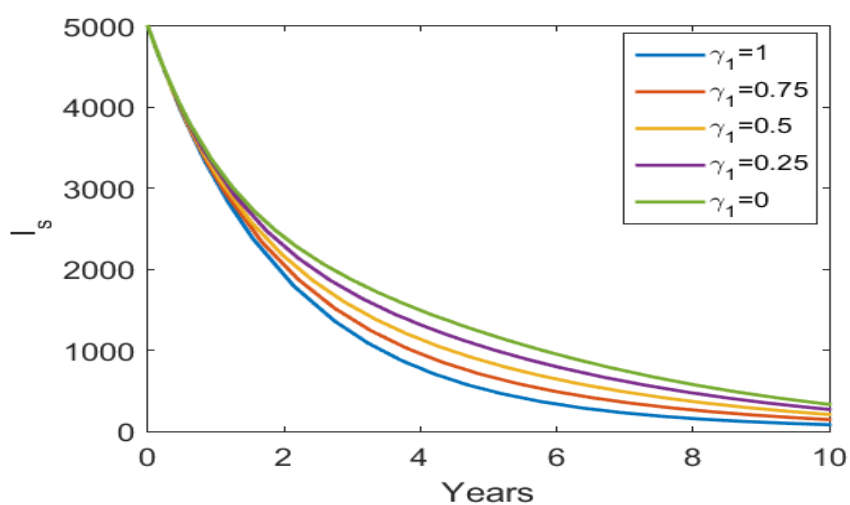


Figure 7: Treatment Control's Effects on Severely Infected People

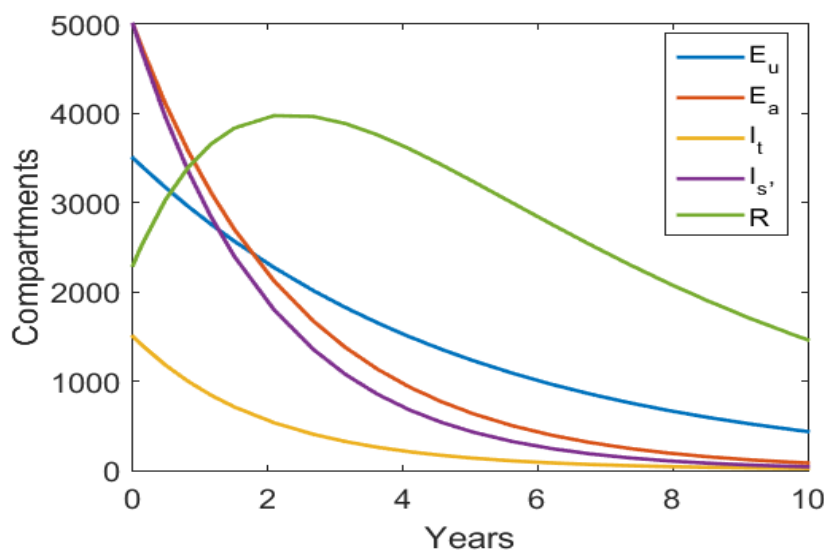


Figure 8: Plotting of Compartmental Behaviours when $R_c > 1$, with all Parameter Values Listed in Table 2

Discussion

The initials conditions of the state variables and the parameter values mentioned in table 2 are used to simulate the dynamical behaviour of the model as it is presented in the paper. We observed the transitions pattern and behavior of the compartments. Figure 2 is simulation associated with effect of public enlightenment on un-aware exposed individuals in which no any changes occur for the values of theta $[0,1]$. Figure 3 is simulated figure on aware exposed individuals for the values of theta indicted in table 2 with no changes for the compartment. Figure 4 is simulated figure on atypically infected individuals with different values of theta $[0,1]$ as the values of theta decreases the number of infected persons increases geometrically, when the value reduced to zero the number of atypically infected person reaches the peak almost near the number of populations. Figure 5 is the effects of public enlightenment on severe infected persons for the values of theta $[0,1]$ when the value reaches 1 the number of infected individuals decreases nearly to zero and when the value of theta is zero the number of infected reaches the peak to the higher number. Figure 6 and 7 shows the impact of treatments controls on atypically and severe infected individuals, the values of gamma (treatments) $[0,1]$ act differently on the number of infected individuals as the values of gamma increases from 0 to 1 the number of infected decreases. In other words, treatment control and public enlightenment have an effect on the measles disease dynamics transmission. **Figure 8** are compartmental behaviors when $R_c > 1$ with other parameters values given in table 2, the values of control parameters (theta and gamma) more especially theta (public enlightenments campaign) make the transitional movement of the compartments from initials state to different states based on the impact of controls parameters on the model.

CONCLUSION

Some data used in this research were derived from related literatures that will coup with Kafin Hausa population while some were estimated to validate the analysis due to lack of accurate record of measles disease in Kafin Hausa local government that will support the research. The impact of public enlightenment on the transmission dynamics of measles disease were modeled using *SIR* model

consisting of six compartments: Susceptible, Un-aware Exposed, Aware exposed, Atypically Infected, Severe Infected and recovered. The model was concentrated on basic Mathematical analysis such as, Stability analysis with numerical simulation. Existence, boundedness, and positivity were examined in the mathematical analysis section. The model's two equilibria, Disease Free Equilibrium (DFE) and Disease Endemic Equilibrium Point (DEEP), were analysed for stability. When $R_c < 0$, the DFE is determined to be locally asymptotically stable. Using a nonlinear Lyapunov function of Go-Volterra type, the global asymptotic stability of the disease endemic equilibrium point (DEEP) has been determined. This indicates that if the effective reproduction number is more than unity and $(\lambda + \mu) > 0$, the DEEP is globally asymptotically stable. The model's numerical simulation shows that the public enlightenment campaign (theta) of atypically and severe infected individuals is the most important parameter to reduce the transmission rate of measles disease in kafin Hausa Metropolitan. Base on that we suggest that priority should be given to public enlightenment to reduce the spread of measles disease in that population.

DECLARATIONS

Conflicts of Interest: This paper contains no conflicts of interest.

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Data Availability: Some information utilized in this study was taken from the pertinent literature and properly cited.

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