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Review

Epidemiological model of influenza a (H1N1) transmission in Ashanti Region of Ghana, 2012

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The pandemic potential of influenza A (H1N1) has required decision makers to act in the face of uncertainties. A deterministic susceptible-exposed-infectious-recovered model was developed to study the spread of H1N1 using population data from the Ashanti region of Ghana. We assumed the population to be constant with birth rate equals death rate and they interact freely (homogeneous mixing). We determined the equilibria and stability of the equilibria with the aim of finding threshold conditions under which the disease spread or die out and illustrate the outcome with numerical solutions. Our results suggest that vaccinating 0.64% of the susceptible population can significantly control the spread of the disease.

Key words: Vaccination, stability, basic reproduction number, homogeneous mixing.

INTRODUCTION

Influenza is a viral infection that affects mainly the nose, throat, bronchi and occasionally, lungs. Infection usually lasts for about a week, and is characterized by sudden onset of high fever, aching muscles, headache and severe malaise, non-productive cough, sore throat and rhinitis" (World Health Organization (WHO)/Influenza). Influenza is caused by Ribonucleic acid (RNA) virus in the family of Orthomyxoviridae. The virus is divided into three main types (A, B, and C) which are distinguished by differences in two major proteins; hemagglutinin (HA) and neuraminidase (NA). Influenza Type B infects humans, producing a milder disease that can cause epidemics. Type C apparently infects only humans and typically produces either a very mild illness indistinguishable from a common cold or no symptoms at all. Type C does not cause epidemics. Influenza type A is the most dangerous; it infects a wide variety of mammals and birds. It causes the most cases of the disease in humans and is the type most likely to become pandemic. Influenza A is further divided into subtypes based on differences in the membrane protein HA and NA, which are the most important targets for the immune system. Influenza type A has 16 hemagglutinin subtypes (H1 to H16) and 9 neuraminidase subtypes (N1 to N9) known in birds. Only H1, 2, and 3 and N1 and 2, are commonly found in humans (Stephenson and Democratis, 2006). There are currently two subtypes circulating in humans: H1N1 and H3N2 (Stephenson and Democratis, 2006). An antigenic shift in the influenza A virus can produce a pandemic affecting most of the world within a matter of months.

Influenza A (H1N1) is transmitted from person to person through large respiratory droplets expelled directly through coughing or sneezing, indirectly through contact with respiratory droplets or secretions, followed by touching the nose or the mouth, and one needs not to be more than one meter to be infected (Racaniello, 2009). Preventing transmission requires removing one or more of the conditions necessary for transmission for example, blocking and or minimizing the ways by which the virus can get to a susceptible host, inhibiting or killing the virus

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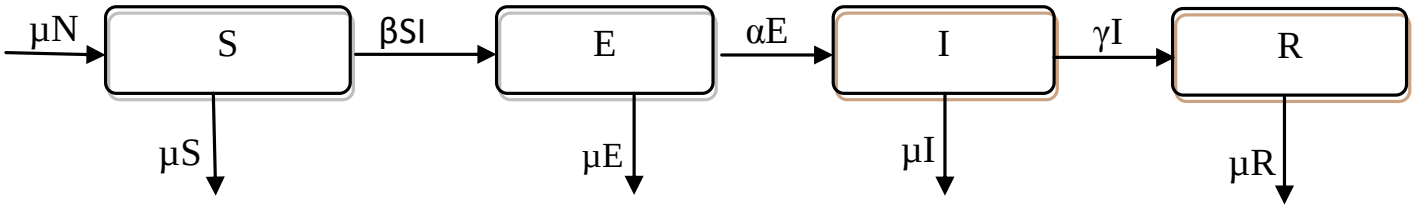

Figure 1. Flowchart for SEIR model.

(Tietjen et al., 2003). People infected with H1N1 first pass through latent and incubation period where they are not infectious and do not have the symptoms. The period of incubation for H1N1 is 1 to 4 days and the infectious period for a confirmed case is defined as 1 day prior to the onset of symptoms to 7 days after onset (Gu et al., 2009). The symptoms of influenza A (H1N1) are: cough, nausea, diarrhea, fever and chills, headache, sore throat, muscle aches, runny nose, shortness of breath, joint pains etc. (Eccles, 2005).

The H1N1 virus had infected more than one million people worldwide (World Health Organisation 2009). Ashanti Region is no exception to the menace of the influenza virus H1N1. The region was first hit by the influenza A (H1N1) pandemic on August 31, 1918, on a ship arriving from Freetown, Sierra Leone and it spread across Ghana along the main lines of communication, killing at least 100,000 people. This has been followed by so many influenza outbreaks, for instance the 1957 to 1958 Asian Flu (H2N2) and 1968 to 1970 Hong Flu (H3N2). In April, 2005, outbreak of influenza A H5N1 and March, 2010, confirmed first case of pandemic Influenza A (H1N1) (Ghana Health Service, Kumasi, 2009).

The H1N1 poses public health and developmental challenges similar to challenges posed by communicable and chronic diseases. This has required decision makers to act in the face of substantial uncertainties. Even though vaccines are available for many infectious diseases, these diseases still cause suffering and mortality in Ghana and Ashanti region in particular. It is against this backdrop that this research is called for to ascertain the wide spread of the influenza A (H1N1) virus.

MODEL FORMULATION

The model we decided to use in studying the H1N1 virus is the susceptible-exposed-infectious-recovered compartmental model, or more commonly the SEIR model (Anderson and May, 1991). This model is the same as the SIR model, except that before the individual becomes infectious, of course he/she will be exposed to the environment. For the model, we consider four basic classes:

1. Susceptible (S);
2. Exposed (E);
3. Infectious (I);
4. Recovered (R).

Susceptible class are individuals in the population who are at risk of becoming infected with H1N1 virus. The exposed class are individuals who have been infected with the H1N1 virus but not infectious (show no symptoms and cannot pass on the disease). Infectious class are Individuals who have been infected with the H1N1 and can pass it on to susceptible persons. Lastly, the recovered class are individuals who have recovered or been removed from H1N1 infection (Uhavax, 2001). For the model, we assume births and deaths occur at equal rate and that all newborns are susceptible (no inherited immunity). We denote the average birth and death rate by μ. The rate at which individuals are born into the susceptible class with no passive is μS. We also assume the population mix homogeneously, with no restriction of age, mobility or other social factors. Once infected, you become exposed to the environment before becoming infectious. The rate at which susceptible enters the exposed class without been infectious is βSI and the rate at which an exposed person becomes infectious is αE. The rate at which an infected individual may recover and will remain until death is γ. The transmission coefficient is $\beta > 0$, the latency coefficient $\alpha > 0$, the recovery coefficient $\gamma > 0$ and the capital death rate $\mu > 0$. The flow diagram for the SEIR model is given in Figure 1.

The following system of ordinary differential equations (ODEs) is used to represent this model:

$$S' = \mu N - \mu S - \beta IS$$

$$E' = \beta IS - (\mu + \alpha)E,$$

$$I' = \alpha E - (\gamma + \mu)I,$$

$$R' = \gamma I - \mu R,$$

The ODEs satisfy

$$S' + E' + I' + R' = 0$$

And hence;

$$S + E + I + R = N$$

The ODEs above have a disease-free equilibrium (DFE) and an endemic equilibrium (EE), one can show that independently form biologically meaningful initial conditions,

$$\big(S(0),E(0),I(0),R(0)\big) \in \{(S,E,I,R) \in [0,N]^4 : S \geq 0, E \geq 0, I \geq 0, R \geq 0, S+E+I+R=N\}$$

It holds that:

$$R_0 \leq 1 \Longrightarrow \lim_{t\to+\infty}\big(S(t),E(t),I(t),R(t)\big) = DFE$$

$$R_0 > 1, I(0) > 0 \Longrightarrow \lim_{t\to+\infty}\big(S(t),E(t),I(t),R(t)\big) = EE$$

Expressing the ODEs as a proportion of the population we obtain:

$$s(t)=\frac{S(t)}{N}, e(t)=\frac{E(t)}{N}, i(t)=\frac{I(t)}{N}, r(t)=\frac{R(t)}{N}$$

And with $r(t) = 1 - s(t) - e(t) - i(t)$, we have the ODEs as a reduced three dimensional system;

$$s' = \mu - (\mu + \beta i)s$$

$$e' = \beta si - (\mu + \alpha)e$$

$$i' = \alpha e - (\gamma + \mu)i$$

The probability to survive the latency and to enter the infectious period equals to $\frac{\alpha}{\alpha+\mu}$ (Bjørnstad, 2005), therefore for this model, the basic reproduction number is;

$$R_O = \frac{\beta\alpha}{(\mu+\gamma)(\mu+\alpha)}$$

$$\mu - (\mu + \beta i)s = 0$$

$$\beta si - (\mu + \alpha)e = 0$$

$$\alpha e - (\gamma + \mu)i = 0$$

Setting the differential equations equal to 0 gives;

At $i = 0$, from the first equation we have $s = 1$, also $e = 0$. Hence the infection-free equilibrium $(s,e,i) = (1,0,0)$

To determine the endemic state we set $e = \frac{\gamma+\mu}{\alpha} i$ in the third equation. We then substitute e into the second equation and we obtain $s = \frac{1}{\beta\alpha}(\mu+\alpha)(\gamma+\mu) = \frac{1}{R_0}$. Putting the value of s into the first equation we have $i = \frac{\mu}{\beta}(R_O - 1)$ and $e = \frac{\gamma+\mu}{\alpha} i = \frac{\mu(R_0-1)}{R_0(\mu+\alpha)}$. Thus the endemic equilibrium point is given by;

$$(s^*, e^*, i^*) = \left(\frac{1}{R_o}, \frac{\mu(R_o-1)}{R_o(\mu+\alpha)}, \frac{\mu(R_o-1)}{\beta}\right)$$

We calculate the local stability of these steady states by linearizing the ODEs. The Jacobian matrix is found to be

$$J = \begin{bmatrix} -\mu - \beta i & 0 & -\beta s \\ \beta i & -(\mu+\gamma) & \beta s \\ 0 & \alpha & -(\gamma+\mu) \end{bmatrix}$$

The Jacobian at the disease-free equilibrium is;

$$J_{DFE} = \begin{bmatrix} -\mu & 0 & -\beta \\ 0 & -(\mu+\alpha) & \beta \\ 0 & \alpha & -(\gamma+\mu) \end{bmatrix}$$

We have; $det(J-\lambda I) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3$

Where;

$$a_1 = (3\mu+\gamma+\alpha)$$

$$a_2 = [(\mu+\gamma)(\mu+\alpha) - \beta\alpha + \mu(2\mu+\gamma+\alpha)]$$

$$a_3 = \mu[(\mu+\alpha)(\mu+\gamma) - \beta\alpha]$$

From Routh-Hurwitz stability criterion if $a_1 > 0,\ a_3 > 0\ and\ a_1a_2 - a_3 > 0$ are true, then all the roots of the characteristic equation have negative real part which means stable equilibrium (Flores, 2013). The disease-free equilibrium is stable when $R_0 < 1$ otherwise unstable. Next we have the Jacobian at the endemic equilibrium point;

$$J_{EE} = \begin{bmatrix} -\mu R_o & 0 & \dfrac{-(\mu+\alpha)(\mu+\gamma)}{\alpha} \\ \mu(R_o-1) & -(\mu+\alpha) & \dfrac{(\mu+\alpha)(\mu+\gamma)}{\alpha} \\ 0 & \alpha & -(\gamma+\mu) \end{bmatrix}$$

We have; $det(J-\lambda I) = \lambda^3 + b_1\lambda^2 + b_2\lambda + b_3$

Where;

$$b_1 = \alpha + \gamma + (2+R_o)\mu$$

$$b_2 = \mu R_o(2\mu + \alpha + \gamma)$$

$$b_3 = \mu(R_o-1)[\mu^2 + \mu(\alpha+\gamma) + \alpha\gamma]$$

From Routh-Hurwitz stability criterion, if the coefficient of the characteristic equation $b_1 > 0,\ b_3 > 0$ and $b_1b_2 - b_3 > 0$ are true, then all the roots of the characteristic equation have negative real parts which means a stable equilibrium (Flores, 2013). The first two conditions are true for $R_0 > 1$ as $b_1\ and\ b_3$ are both positive quantities. The third condition $b_1b_2 - b_3 > 0$ given by $\mu[R_o\{(3\mu+\alpha+\mu R_o)(\alpha+\gamma) + \mu^2(3+2R_o) + \gamma^2\} + \mu^2 + \mu(\alpha+\gamma) + \alpha\gamma]$ is greater than zero (for all parameter values and $R_O > 1$), hence it is also true. Thus the endemic steady state is stable when $R_o > 1$ by the Routh-Hurwitz criteria.

We proposed the herd immunity threshold (H_1) as the sole immunization strategy. The herd immunity threshold is the percentage of the population that needs to be immune to control transmission of the disease. It protects directly the immune individuals from reinfection but also provides an indirect protection to susceptible population. The equation given by Diekmann and Heesterbeek (2000) for estimating the herd immunity threshold is; $H_1 = 1 - \dfrac{1}{R_O}$

Model analysis

We used Ashanti regional data, and had $N = 4725042$, $\beta = 0.3016$, $\alpha = 0.5$, $\gamma = 0.2857$ and $\mu = 0.0088$. The basic reproduction number was found to be $R_0 = 1.0064$. The disease-free equilibrium $(s,e,i) = (1,0,0)$ was unstable for $R_0 \prec 1$ and the endemic equilibrium $(s^*,e^*,i^*) = (0.994, 0.000109, 0.000187)$ was stable for $R_0 > 1$. The herd immunity threshold was found to be $H_1 = 0.0064$.

Table 1. Sensitivity analysis of the disease-free equilibrium state.

Nature of steady state	μ	β	α	γ	R_O
Unstable	0.0088	0.4	0.5	0.2857	1.3347
Stable	0.0088	0.175	0.5	0.2857	0.5839
Stable	0.0088	0.3016	0.5	0.5	0.5825
Unstable	0.0088	0.3016	0.5	0.105	2.6044

Table 2. Sensitivity analysis of the endemic equilibrium point.

Nature of steady state	μ	β	α	γ	R_O
Stable	0.0088	0.4000	0.5	0.2857	1.3347
Unstable	0.0088	0.1750	0.5	0.2857	0.5839
Unstable	0.0088	0.3016	0.5	0.5000	0.5825
Stable	0.0088	0.3016	0.5	0.1050	2.6044

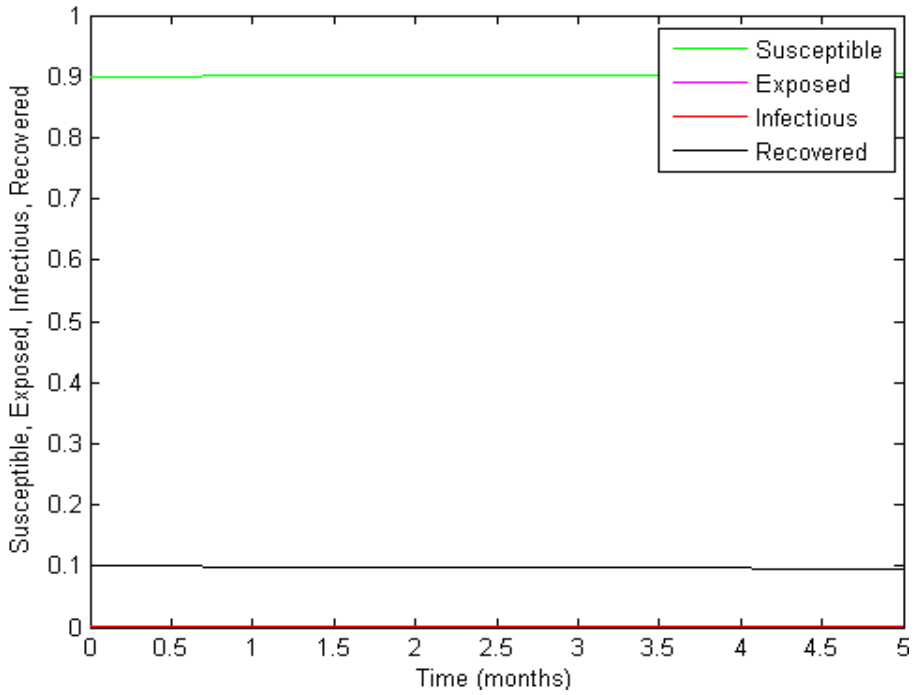

Figure 2. Dynamics of the various compartments at the initial outbreak of H1N1.

Sensitivity analysis was performed for both the disease-free and the endemic equilibrium point. The results were shown in Tables 1 and 2.

From Table 1, as the transmission rate or the recovery rate was increased or decreased, respectively the $R_O > 1$ and the disease-free equilibrium was found to be unstable. This means in the cause of an outbreak, the disease will spread. On the other hand, as the transmission rate or the recovery rate was reduced or increased, respectively $R_O < 1$ and the disease-free equilibrium was found to be stable, meaning the disease failed to spread. From Table 2, as the transmission rate or the recovery rate was increased or decreased, respectively the $R_O > 1$ and the endemic equilibrium point was stable. On the other hand, as the transmission rate or the recovery rate was decreased or increased, respectively the $R_O < 1$ and the endemic equilibrium was unstable. We ran a simulation for a period of 5 months for interaction between susceptible, infectious and recovered patients using the parameter values given. The Ashanti regional data showed that at the month of March $S(0) = 4725042, E(0) = 2, I(0) = 2 \ and \ R_0 = 0$. Dividing through by the total population of Ashanti region which was 4725046 (Ghana statistical service, 2010), we had; $s(0) = 0.999999915, e(0) = 4.232763025 \times 10^{-7}$ $i(0) = 4.232763025 \times 10^{-7}$ and $r(0) = 0.0$. From the

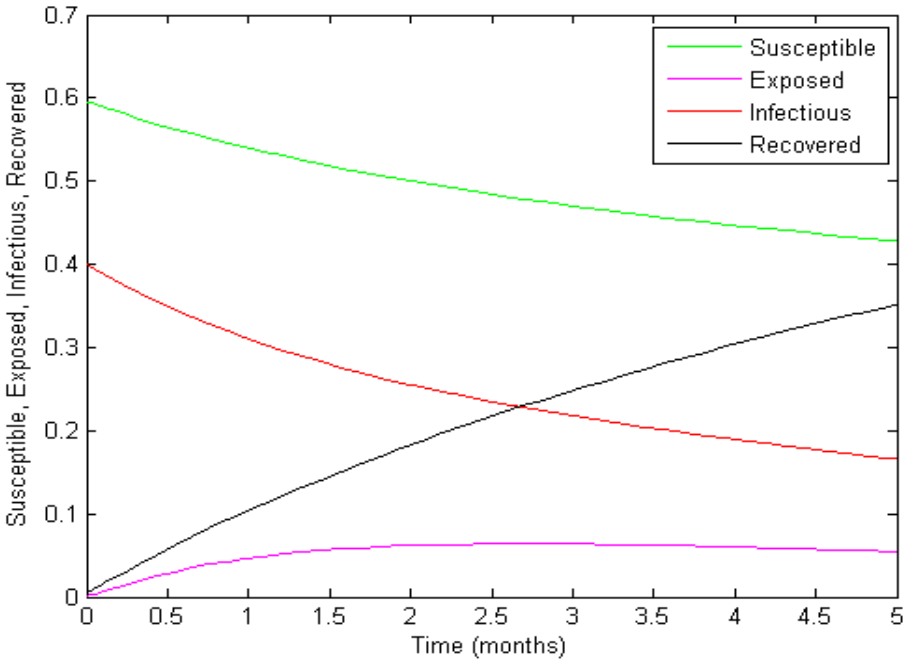

Figure 3. Graph of an increased in the proportion of infectives (5 months period) on various compartments**.**

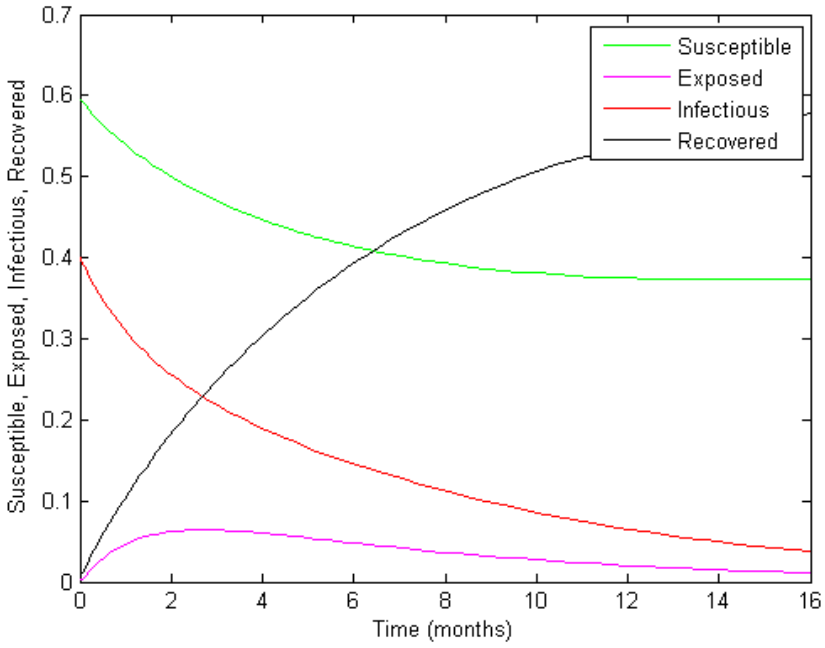

Figure 4. Graph of an increased in proportion of infectives (16 months period) on various compartment.

simulation we obtain Figure 2.

From Figure 2, the initial proportion of infectious has minimal or no effect on the susceptible population, hence we had disease-free state. We varied the proportion of infectives (taken $i(0) = 0.4$) around the neighbourhood of the endemic equilibrium point for a period of 5 and 16 months. This is illustrated in the Figures 3 and 4, respectively. From Figure 3, when the proportion of infectives was increased to 0.4 around the neighborhood of the endemic equilibrium, the proportion of exposed individuals initially increased from 0, reaches a peak of 0.06 in the second month then declines gradually to a minimum value of 0.05 by the fifth month. The

proportion of susceptible on the other hand declines from a value of 0.6 during the first month to a minimum value of 0.44 by the fifth month. The proportion of recovered on the other hand increases exponentially with time and reaches a maximum value of 0.35 by the fifth month. Also, the recovered population equals the infective around the third month of the outbreak.

From Figure 4, when the proportion of infectives was increased to 0.4 around the neighborhood of the endemic equilibrium point, the proportion of exposed individuals initially increased from zero, then reached a peak of 0.06 in the second month and declined gradually to a minimum value of 0.01 by the sixteenth month. The proportion of susceptible on the other hand declined from a value of 0.6 during the first month to a minimum value of 0.38 by the sixteenth month. The proportion of recovered on the other hand increased exponentially with time and reached a maximum value of 0.58 by the sixteenth month. Also, the recovered population was equal the infective around the third month of the outbreak at a value of 0.23 and the susceptible around the seventh month at a value of 0.41.

DISCUSSION

From the results, the reproduction number for the SEIR epidemiological model estimated indicated that $R_0 > 1$. This means the disease will spread in the cause of an outbreak. The sensitivity analysis revealed that whenever the transmission rate is increased or the recovery rate is reduced, the disease spread, but whenever the transmission rate is reduced or the recovery rate is increased, the disease dies out. From the simulation (Figure 2) we found out that the initial proportion of infectives had no effect on the various compartments. As the proportion of infective was increased to 0.4 as shown in Figures 3 and 4 around the neighborhood of the endemic equilibrium state, the SEIR model exhibited a decline in the proportion of susceptible. This means that as more and more people are infected with the H1N1 virus, the disease will become endemic in the region. Furthermore, the recovered proportion of the population increases exponentially with time. This is as a result of a relatively high recovery rate such that even though the susceptible population was infected, a high amount of them recovered quickly, providing herd immunity. The herd immunity threshold was estimated to be 0.0064, meaning about 0.64% of the Ashanti region population has to be vaccinated in order to bring the disease under control in case of an outbreak.

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