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Simulation Study of H1N1 Transmission through Immigrants and Vaccination

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Authors' contributions

This work was carried out in collaboration among all authors. All authors have contributed equally in this research paper.

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Abstract

H1N1 influenza, is a disease caused by the Type A strain of negative-sense single stranded RNA virus which has unique characteristics of reassortment making it different and difficult to control. Due to the unique nature of the virus, the constant globalization of the international population, and the lack of effective immunization, this virus is a great threat to public health. There is only limited data available on impacts of immigrants on H1N1 transmission. So, there is real need to study impacts of immigrants on H1N1 transmission. We have studied a mathematical model for simulation study of H1N1 transmission through immigrants and vaccination. The class of susceptible people is divided in two types: vaccinated immigrants and non-vaccinated immigrants. The medications and hospitalizations are the remedial steps to get cured. The rate at which people get medicated or hospitalized is analyzed using SEIR model. The system of non-linear ordinary differential equation is formulated for the given model and the reproduction number is then calculated using next generation matrix method which indicates the recovery rate of an individual stability. Numerical simulations are then carried out. Our study showed that increasing the awareness among the general population, immigrants about clinical features and modes of transmission of H1N1 and improving the vaccination rate helps decrease the transmission of H1N1.

Keywords: H1N1; mathematical model; basic reproduction number; stability; simulation.



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1 Introduction

H1N1 influenza, caused by the Type A strain of influenza virus, and is a disease transmitted from infectious pigs to humans. Similar to many other influenza viruses, H1N1 viruses mutate continuously, a characteristic they share with many other influenza viruses. Reassortment is the unique nature of H1N1 which occurs as influenza viruses from different species infect pigs, and new viruses that are a mix of genes from swine, human and/or avian influenza viruses can emerge. H1N1, H1N2, H3N2, and H3N1 are the four main influenza type A virus subtypes that have been isolated in pigs [1].

The Adjacency of humans to infected pigs at the places like pig barns and livestock exhibits housing pigs at fairs can lead to direct transmission of Influenza A virus from pigs to people [1]. Human-to-human transmission of swine flu occurs through direct contact with an infected person or indirect contact via contaminated surfaces and objects [2]. Other bodily fluids (e.g. diarrheal stool) should also be considered potentially infectious [1] as well as respiratory secretions. Lack of immunity to the virus from previous exposure due to frequent mutations of influenza viruses is one of the reasons for influenza to be able to spread to a pandemic level [3]. According to the CDC, extremes of age and people with chronic diseases or with reduced immunity are at higher risk for H1N1 influenza complications.

Simple measures like proper hand hygiene, avoiding contact with contaminated surfaces, and covering mouth with sleeve or tissues during coughing and sneezing have been shown to reduce the risk of transmission of influenza [4]. Alcohol-based sanitizing gels are better instead of soap and water when hands are not visibly soiled [5]. Studies done by Steelfisher et al. [6], Ferguson et al. [7], Glass et al. [8], Tahir et al. [9], Tahir et al. [10] showed that influenza mitigation efforts should include hygiene (e.g. hand washing) social distancing (e.g. avoiding places where many people gather) and pharmaceutical interventions (e.g. medications and vaccination).

With widespread community transmission on at least two continents [1], WHO has officially declared the 2009 H1N1 influenza to be a pandemic on June 11th, 2009 [4] More than 170 countries and territories reported influenza cases by August 2009 [5]. One of the main reasons for the fast spread of the disease across nations was the lack of knowledge at the individual level. Since 2010, about 1 million people are immigrating lawfully and obtaining permanent residency in the United States per year according to immigration yearbook statistics 2017 [11]. Constant international migration of the population can promote infectious disease spread between different countries. Alfonso Rodriguez et al. [12] has shown vaccine education targeting immigrants from bilateral countries can help mitigate the spread of influenza. Influenza has resulted in around 9.3 million–49.0 million illnesses, around 140,000–960,000 hospitalizations and around 12,000–79,000 deaths annually since 2010 [13].

Annual influenza vaccination is recommended for those aged 6 months and older in the United States and is the most effective way to prevent influenza and its complications. Inactivated influenza vaccine (IIV), live attenuated influenza vaccine (LAIV), and recombinant influenza vaccine (RIV) are approved in the United States. In fact, it is recommended that travelers to the tropics, who have not received influenza vaccine during the preceding fall or winter should consider influenza vaccination 2 or more weeks before the departure (CDC). A study done by Conway et al. [14] showed that a mere increase in vaccination rate of the population is not enough but the timing of vaccination is very important. Vaccination started 2 weeks before the anticipated epidemic helps decrease morbidity and mortality by about 80-90% compared to non-vaccination individuals [14].

The socio-economic burden and impact of H1N1 pandemic are huge. Compared to developing countries, developed countries are better prepared to alleviate the impact of the influenza pandemic. Barriers faced by developing countries are high prevalence of the infective disease, large population, poor public knowledge about the disease, difficulty in accessing medical care, poorly developed public health infrastructure, and low socio-economic conditions. Previous research by Kowal et al. [15] has demonstrated that one of the reasons for the low vaccination rate among immigrants is due to language barriers leading to ineffective communication between provider and patients as well as lack of massive public health awareness campaign.

Research done by Rodriguez et al. [12] has shown that the low vaccination rate was primarily due to lack of time, poor understanding about the seriousness of the disease, and questions about the safety and effectiveness of vaccines. Studies done by Lu et al. [16], Linn et al. [17], Bish et al. [18], Lindley et al. [19] shows that socioeconomic status of the population, access to health care services, and their attitude towards the vaccine, provider and illness plays a key role in their ultimate decision of vaccination. In addition to the low vaccination rate, host factors, like malnutrition and limited availability of antiviral drugs (oseltamivir and zanamavir) can also influence morbidity and mortality [20].

The influenza virus is a serious risk to global public health due to the limited availability of proper vaccines and antiviral drugs, the constant movement of the world's population, and unique nature of the virus [21]. For the same reasons, many countries are not well prepared for the epidemics of H1N1. Massive public awareness and knowledge about the disease may help reduce the morbidity and mortality during future outbreaks [1]. We found good review of literature of the transmission of H1N1, but we have limited information available on impact of immigrants on H1N1 transmission. With constant migration of the world's population, and H1N1 influenza disease has been raised up to pandemic level in 2009 with serious risk to global public health, there is real need to study effects of immigrants on H1N1 transmission as well as impact of vaccination.

The aim of this study is to help understand H1N1 transmission through immigration and vaccination. Incorporation of monitoring data with mathematical models helps public health planners to maximize the benefits to the general population from offered therapeutic options before the epidemic reaches its worst. We believe that increased awareness about vaccination and their availability, improved general population awareness about H1N1 transmission, steps that individual and government can take to prevent the rapid spread of the viruses by different preventive techniques can decrease the socio-economic burden on society and decrease morbidity and mortality from H1N1.

2 Mathematical Model

Here we formulate a mathematical model for simulation study of H1N1 transmission through immigrants and vaccination. The notations along with its parametric values are shown in Table 1.

Notation		Parametric values
В	New recruitment rate	0.10
v	Rate of immigrant individuals	0.01
S(t)	Number of individuals susceptible to H1N1 at any instant of time t	20
$V_{C}(t)$	Number of individuals who get vaccinated at any instant of time t	12
$N_{v}(t)$	Number of individuals who get non-vaccinated at any instant of time t	4
I(t)	Number of individuals who get infected at any instant of time t	14
M(t)	Number of individuals who get medicated at any instant of time t	8
R(t)	Number of recovered individuals	6
eta_1	Rate at which susceptible individuals gets infected	0.05
β_2	Rate at which susceptible individuals gets vaccinated	0.6
β_{3}	Rate at which susceptible individuals gets non-vaccinated	0.2
eta_4	Rate at which no vaccinated gets vaccinated	0.3
β_5	Rate at which vaccinated gets infected	0.1
$m{eta}_6$	Rate at which non-vaccinated get infected	0.5

Table 1. Notation and its parametric values

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Notation		Parametric values
β_7	Rate at which infected get medicated	0.2
${m eta}_8$	Rate at which medicated get recovered	0.7
eta_9	Rate at which recovered get again susceptible	0.5
μ	Natural death rate	0.4
N(t)	Sample size at any instant time of t	100

The transmission diagram of H1N1 through immigrants and vaccination is shown in Fig. 1.



Fig. 1. Flowchart of H1N1 transmission trough immigrants and vaccination

In this model, Susceptible(S) enters with the new recruitment rate B and v. v is the rate of immigrants. Susceptible people are split in two ways. Firstly, susceptible people who get vaccinated enter with the rate of β_2 and secondly susceptible people who get non-vaccinated enter with the rate of β_3 .

Susceptible people get directly infected for H1N1 at the rate of β_1 . Non-Vaccinated people enter in the compartment of vaccinated at the rate of β_4 . Vaccinated and non-vaccinated people get infected with the rate of β_5 and β_6 respectively. Infected people get medicated/hospitalized at the rate of β_7 and medicated/hospitalized people recovered at the rate of β_8 . It may happen that recovered people again go back to the susceptible at the rate of β_9 . Here, B and μ describes new recruitment rate and natural escape rate respectively.

Fig. 1 is described by the following set of non-linear ordinary differential equations.

$$\frac{dS}{dt} = B + v - \beta_1 SI - \beta_2 SV_c - \beta_3 SN_v + \beta_9 R - \mu S$$

$$\frac{dV_c}{dt} = \beta_2 SV_c + \beta_4 N_v - \beta_5 V_c - \mu V_c$$

$$\frac{dN_v}{dt} = \beta_3 SN_v - \beta_4 N_v - \beta_6 N_v - \mu N_v$$
(1)

$$\frac{dI}{dt} = \beta_5 V_c + \beta_6 N_v + \beta_1 SI - \beta_7 I - \mu I$$
$$\frac{dM}{dt} = \beta_7 I - \beta_8 H - \mu M$$
$$\frac{dR}{dt} = \beta_8 H - \beta_9 R - \mu R$$

where $S + V_c + N_v + I + M + R = N$. Also $S > 0, V_c \ge 0, N_v \ge 0, I \ge 0, M \ge 0, R \ge 0$

Adding the above set of equations of system (1), we have

$$\frac{d}{dt}(S + V_c + N_v + I + M + R) = B - \mu(S + V_c + N_v + I + M + R) \ge 0$$
(2)

which implies that

$$\lim_{t \to \infty} Sup(S + V_c + N_v + I + M + R) \le \frac{B}{\mu}$$
(3)

Thus, the feasible region of the model is,

$$\Lambda = \left\{ (S, V_c, N_v, I, M, R) / S + L + H + A + C + R \le \frac{B}{\mu}, S > 0, V_c \ge 0, N_v \ge 0, I \ge 0, M \ge 0, R \ge 0 \right\}$$
(4)

On solving these set of equation (1) by putting equal to zero, we get the equilibrium points. Therefore, the equilibrium points of the model are

$$E_{0} = \left(\frac{B}{\mu}, 0, 0, 0, 0, 0\right),$$

$$E_{1} = (S_{1}, 0, 0, I_{1}, M_{1}, R_{1}),$$

$$E_{2} = (S_{2}, V_{c_{2}}, 0, I_{2}, M_{2}, R_{2}),$$

$$E_{3} = (S_{3}, 0, N_{v_{3}}, I_{3}, M_{3}, R_{3})$$

Where,

$$S_{1} = \frac{\varphi_{4}}{\beta_{1}} , \qquad I_{1} = -\frac{\varphi_{4}\varphi_{5}\varphi_{6}(B - \mu + \nu)}{\beta_{1}(\beta_{7}\beta_{8}\beta_{9} - \varphi_{4}\varphi_{5}\varphi_{6})} , \qquad M_{1} = -\frac{\varphi_{4}\beta_{7}\varphi_{6}(B - \mu + \nu)}{\beta_{1}(\beta_{7}\beta_{8}\beta_{9} - \varphi_{4}\varphi_{5}\varphi_{6})}$$
$$R_{1} = -\frac{\beta_{7}\varphi_{4}\beta_{8}(B - \mu + \nu)}{\beta_{1}(\beta_{7}\beta_{8}\beta_{9} - \varphi_{4}\varphi_{5}\varphi_{6})}$$

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$$\begin{split} S_{2} &= \frac{\varphi_{2}}{\beta_{2}}, \ V_{c_{2}} = -\frac{\varphi_{2}\varphi_{6}\varphi_{2}(B - \mu + \nu)(\beta_{1}\varphi_{2} - \beta_{2}\varphi_{4})}{(\beta_{1}\beta_{5}\varphi_{5}\varphi_{6}\varphi_{6}\varphi_{2} - \beta_{1}\varphi_{2}^{2}\varphi_{5}\varphi_{6} - \beta_{2}\beta_{5}\beta_{7}\beta_{8}\beta_{9} + \beta_{2}\varphi_{5}\varphi_{6}\varphi_{4}\varphi_{2})\beta_{2}}, \\ I_{2} &= \frac{\beta_{5}\varphi_{5}\varphi_{6}\varphi_{6}(2 - \mu + \nu)}{\beta_{1}\beta_{5}\varphi_{5}\varphi_{6}\varphi_{6}\varphi_{2} - \beta_{1}\varphi_{2}^{2}\varphi_{5}\varphi_{6} - \beta_{2}\beta_{5}\beta_{7}\beta_{8}\beta_{9} + \beta_{2}\varphi_{5}\varphi_{6}\varphi_{4}\varphi_{2}}, \\ R_{2} &= -\frac{\beta_{7}\beta_{5}x_{2}\beta_{8}(B - \mu + \nu)}{\beta_{1}\beta_{5}\varphi_{5}\varphi_{6}\varphi_{6}\varphi_{2} - \beta_{1}\varphi_{2}^{2}\varphi_{5}\varphi_{6} - \beta_{2}\beta_{5}\beta_{7}\beta_{8}\beta_{9} + \beta_{2}\varphi_{5}\varphi_{6}\varphi_{4}\varphi_{2}}, \\ S_{3} &= \frac{\varphi_{3}}{\beta_{3}}, \\ N_{\nu_{3}} &= \frac{\varphi_{5}\varphi_{6}\varphi_{3}(B - \mu + \nu)(\beta_{1}\varphi_{3} - \beta_{3}\varphi_{4})(-\beta_{2}\varphi_{3} + \beta_{3}\varphi_{2})}{(\beta_{3}(-\beta_{1}\beta_{2}\varphi_{2}^{2}\varphi_{5}\varphi_{6} + \varphi_{6}((\beta_{1}\varphi_{2} + \beta_{2}\varphi_{4}))\beta_{3} + \beta_{1}\beta_{2}(\beta_{4} + \beta_{6}))\varphi_{5}\varphi_{3}^{2} - \beta_{3}(\varphi_{5}\varphi_{6}\varphi_{4}\varphi_{2}\beta_{3} + (\varphi_{4}\beta_{4}\beta_{2} + \beta_{1}(\beta_{4}\beta_{5} + \beta_{6}x_{2}))\varphi_{9}\varphi_{6} + \beta_{7}\beta_{8}\beta_{5}\beta_{2}\beta_{6})\varphi_{3} + \beta_{7}\beta_{8}\beta_{5}\beta_{3}^{2}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2}))), \\ I_{3} &= -\frac{\varphi_{6}(-\beta_{2}\beta_{6}\varphi_{3} + \beta_{3}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2}))(B - \mu + \nu)\varphi_{5}\varphi_{3}}{-\beta_{1}(\beta_{2}\varphi_{2}^{2}\varphi_{5}\varphi_{6} + \varphi_{6}((\beta_{1}\varphi_{2} + \beta_{2}\varphi_{4})\beta_{3} + \beta_{1}\beta_{2}(\beta_{4} + \beta_{6}))\varphi_{5}\varphi_{3}^{2} - \beta_{3}(\varphi_{5}\varphi_{6}\varphi_{4}\varphi_{2}\beta_{3} + (\varphi_{4}\beta_{4}\beta_{2} + \beta_{1}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2})))\varphi_{9}\varphi_{6} + \beta_{7}\beta_{8}\beta_{5}\beta_{2}\beta_{6})\varphi_{3} + \beta_{7}\beta_{8}\beta_{5}\beta_{3}^{2}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2})) \\ M_{3} &= -\frac{\varphi_{6}(-\beta_{2}\beta_{6}\varphi_{3} + \beta_{3}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2}))(B - \mu + \nu)\beta_{7}\varphi_{3}}{-\beta_{1}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2}))\varphi_{9}\varphi_{6} + \beta_{7}\beta_{8}\beta_{5}\beta_{2}\beta_{6})\varphi_{3} + \beta_{7}\beta_{8}\beta_{5}\beta_{3}^{2}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2}))} \\ M_{3} &= -\frac{\varphi_{6}(-\beta_{2}\beta_{6}\varphi_{3} + \beta_{3}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2}))(B - \mu + \nu)\beta_{7}\varphi_{3}}{-\beta_{1}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2}))\varphi_{7}\varphi_{6} + \beta_{7}\beta_{8}\beta_{5}\beta_{2}\beta_{6})\varphi_{3} + \beta_{7}\beta_{8}\beta_{5}\beta_{3}^{2}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2})} \\ R_{3} &= -\frac{\beta_{6}(-\beta_{2}\beta_{6}\varphi_{3} + \beta_{5}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2}))(B - \mu + \nu)\beta_{7}\varphi_{3}}{-\beta_{1}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2})})\varphi_{5}\varphi_{6} + \beta_{7}\beta_{8}\beta_{5}\beta_{2}\beta_{6})\varphi_{3} + \beta_{7}\beta_{8}\beta_{5}\beta_{3}^{2}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2})} \\ R_$$

Where,

$$\varphi_2 = \beta_5 + \mu \quad \varphi_3 = \beta_4 + \beta_6 + \mu, \varphi_4 = \beta_7 + \mu, \varphi_5 = \beta_8 + \mu, \text{ and } \varphi_6 = \beta_9 + \mu$$

Now we need to calculate the threshold called basic reproduction number for each equilibrium points. The basic Reproduction Number R_0 :

 $R_{\scriptscriptstyle 0}\,$ is one of the most powerful tools for analyzing and interpreting endemic model.

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The basic reproduction number is typically defined as the mean number of secondary infections generated by a single infectious individual in a whole susceptible population. We required R_0 to access the stability of the disease-free equilibrium point and endemic equilibrium point. The next generation matrix method gives spectral radius of matrix fv^{-1} where f and v are the Jacobian matrices of F and V evaluated with respect to each compartment at an equilibrium state.

Let
$$X = (S, V_c, N_v, I, M, R)$$

Therefore

$$\frac{dX}{dt} = F(X) - V(X) \tag{5}$$

Where F(X) denotes the rate of production of new people who get infected and V(x) represents the rate of transition between states, which gives us:

$$F(X) = \begin{bmatrix} \beta_2 S V_c \\ \beta_3 S N_v \\ \beta_1 S I \\ 0 \\ 0 \\ 0 \end{bmatrix} \text{ and } V(X) = \begin{bmatrix} -\beta_4 N_v + \beta_5 V_c + \mu V_c \\ \beta_4 N_v + \beta_6 N_v + \mu N_v \\ -\beta_5 V_c - \beta_6 N_v + \beta_7 I + \mu I \\ -\beta_7 I + \beta_8 H + \mu M \\ -\beta_8 H + \beta_9 R + \mu R \\ -(B+v) + \beta_1 S I + \beta_2 S V_c + \beta_3 S N_v - \beta_9 R + \mu S \end{bmatrix}$$

Now, the derivative of F and V calculated at an equilibrium point E_0 gives matrices F and v of order 6 X 6 defined as:

$$f = \left[\frac{\partial F_i(E_0)}{\partial X_j}\right] \text{ and } v = \left[\frac{\partial V_i(E_0)}{\partial X_j}\right] \text{ for } i, j = 1, 2, 3, 4, 5, 6$$

So,

$$v = \begin{bmatrix} \varphi_2 & -\beta 4 & 0 & 0 & 0 & 0 \\ 0 & \varphi_3 & 0 & 0 & 0 & 0 \\ -\beta_5 & -\beta_6 & \varphi_4 & 0 & 0 & 0 \\ 0 & 0 & -\beta_7 & \varphi_5 & 0 & 0 \\ 0 & 0 & 0 & -\beta_8 & \varphi_6 & 0 \\ \frac{\beta_2 B}{\mu} & \frac{\beta_3 B}{\mu} & \frac{\beta_1 B}{\mu} & 0 & -\beta_9 & \mu \end{bmatrix}$$

Here v is non-singular matrix.

Thus, the basic reproduction number R_0 at the point E_0 :

$$R_{0} = \frac{1}{\mu \varphi_{4} \varphi_{2} \varphi_{3}} \begin{bmatrix} (\beta_{1} + \beta_{2} + \beta_{3}) \mu^{2} + (\beta_{4} + \beta_{5} + \beta_{6}) \beta_{1} + (\beta_{4} + \beta_{6} + \beta_{7}) \beta_{2} \\ + (\beta_{5} + \beta_{7}) \beta_{3} \end{bmatrix} \mu \\ + \beta_{5} (\beta_{4} + \beta_{6}) \beta_{1} + \beta_{7} ((\beta_{4} + \beta_{6}) \beta_{2} + \beta_{3} \beta_{5}) \beta \end{bmatrix}$$

Using all the values of parameters in table 1 we get $R_0 = 0.36250 < 1$.

The basic reproduction number R_0 at the point E_1 :

$$R_{0} = \frac{1}{\beta_{1}\varphi_{2}\varphi_{3}} \begin{bmatrix} (\beta_{1} + \beta_{2} + \beta_{3})\mu^{2} + (\beta_{4} + \beta_{5} + \beta_{6})\beta_{1} + (\beta_{4} + \beta_{6} + \beta_{7})\beta_{2} \\ + (\beta_{5} + \beta_{7})\beta_{3} \end{bmatrix} \mu \\ + \beta_{5}(\beta_{4} + \beta_{6})\beta_{1} + \beta_{7}((\beta_{4} + \beta_{6})\beta_{2} + \beta_{3}\beta_{5}) \end{bmatrix}$$

The basic reproduction number R_0 at the point E_2 :

$$R_{0} = \frac{1}{\mu\beta_{2}\varphi_{4}\varphi_{2}\varphi_{3}} \begin{bmatrix} (\beta_{1} + \beta_{2} + \beta_{3})\mu^{2} + (\beta_{4} + \beta_{6} + \beta_{7})\beta_{2} + (\beta_{1} + \beta_{3})\beta_{5} \\ + (\beta_{4} + \beta_{6})\beta_{1} + \beta_{3}\beta_{7} \end{bmatrix} \mu \\ + \beta_{7}(\beta_{4} + \beta_{6})\beta_{2} + \beta_{5}((\beta_{4} + \beta_{6})\beta_{1} + \beta_{3}\beta_{7}) + (-\beta_{4} + \beta_{5} + \mu) \end{bmatrix}$$

The basic reproduction number R_0 at the point E_3 :

$$R_{0} = \frac{1}{\beta_{3}\varphi_{4}\varphi_{2}} \begin{bmatrix} (\beta_{1} + \beta_{2} + \beta_{3})\mu^{2} + (\beta_{4} + \beta_{5} + \beta_{6})\beta_{1} + (\beta_{4} + \beta_{6} + \beta_{7})\beta_{2} \\ + (\beta_{5} + \beta_{7})\beta_{3} \\ + \beta_{5}(\beta_{4} + \beta_{6})\beta_{1} + ((\beta_{4} + \beta_{6})\beta_{2} + \beta_{3}\beta_{5})\beta_{7} \end{bmatrix} \mu$$

3 Stability Analysis of Disease-Free Equilibrium

The equilibrium for the local and global stability at the points E_0, E_1, E_2 and E_3 of the H1N1 transmission through immigrant and vaccination model is discussed at here.

3.1 Local stability

3.1.1 Theorem

(Stability at E_0): If $\frac{\beta_3 B}{\mu} < \beta_4 + \beta_6 + \mu$ and $\frac{\beta_1 B}{\mu} < \beta_7 + \mu$ then $E_0 = \left(\frac{B}{\mu}, 0, 0, 0, 0, 0\right)$ is locally asymptotically stable.

Proof: The Jacobian Matrix J at the equilibrium point E_0 is given by:

$$J(E_0) = \begin{bmatrix} -\mu & \frac{-\beta_2 B}{\mu} & \frac{-\beta_3 B}{\mu} & \frac{-\beta_1 B}{\mu} & 0 & \beta_9 \\ 0 & -\beta_5 - \mu & \beta_4 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta_3 B}{\mu} - \beta_4 - \beta_6 - \mu & 0 & 0 & 0 \\ 0 & \beta_5 & \beta_6 & \frac{\beta_1 B}{\mu} - \beta_7 - \mu & 0 & 0 \\ 0 & 0 & 0 & \beta_7 & -\beta_8 - \mu & 0 \\ 0 & 0 & 0 & 0 & \beta_8 & -\beta_9 - \mu \end{bmatrix}$$

Eigenvalues:

$$\begin{bmatrix} -\mu \\ -\beta_9 - \mu \\ -\beta_8 - \mu \\ -\beta_5 - \mu \\ \underline{\beta_3 B - \beta_4 \mu - \beta_6 \mu - \mu^2} \\ \mu \\ \underline{\beta_1 B - \beta_7 \mu - \mu^2} \\ \mu \end{bmatrix}$$

 E_0 is locally stable if all Eigen values are less than zero. Here λ_{11} , λ_{12} , λ_{13} and λ_{14} are less than zero. Where $\lambda_{11} = -\mu$, $\lambda_{12} = -\beta_9 - \mu$, $\lambda_{13} = -\beta_8 - \mu$, $\lambda_{14} = -\beta_5 - \mu$. E_0 is locally stable if,

$$\frac{\beta_3 B}{\mu} < \beta_4 + \beta_6 + \mu \text{ and } \frac{\beta_1 B}{\mu} < \beta_7 + \mu.$$
(6)

3.1.2 Theorem

(Stability at E_1): If $S_1\beta_1x_5x_6 > \beta_8\beta_7\beta_1\beta_9$ then $E_1 = (S_1, 0, 0, I_1, M_1, R_1)$ is locally asymptotically stable.

For the point $E_1(V_c = N_v = 0)$, Jacobian matrix is:

$$J(E_1) = \begin{bmatrix} -\varphi_1 & -\beta_2 S_1 & -\beta_3 S_1 & -\beta_1 S_1 & 0 & \beta_9 \\ 0 & -\varphi_2 & \beta_4 & 0 & 0 & 0 \\ 0 & 0 & -\varphi_3 & 0 & 0 & 0 \\ \beta_1 I_1 & \beta_5 & \beta_6 & -\varphi_4 & 0 & 0 \\ 0 & 0 & 0 & \beta_7 & -\varphi_5 & 0 \\ 0 & 0 & 0 & 0 & \beta_8 & -\varphi_6 \end{bmatrix}$$

where $\varphi_1 = \beta_1 I_1 + \mu$, $\varphi_2 = \beta_5 + \mu$, $\varphi_3 = -\beta_3 S_1 + \beta_4 + \beta_6 + \mu$, $\varphi_4 = -\beta_1 S_1 + \beta_7 + \mu$, $\varphi_5 = \beta_8 + \mu$ and $\varphi_6 = \beta_9 + \mu$. The Eigen values for the above matrix is $\lambda_1 = -\varphi_2$ and $\lambda_2 = -\varphi_3$. So, the reduced form of the above matrix has the characteristic polynomial equation

$$\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0.$$

where,

$$a_{3} = \varphi_{6} + \varphi_{5} + \varphi_{4} + \varphi_{1} > 0$$

$$a_{2} = I_{1}S_{1}\beta_{1}^{2} + \varphi_{1}(\varphi_{4} + \varphi_{5} + \varphi_{6}) + \varphi_{4}(\varphi_{5} + \varphi_{6}) + \varphi_{5}\varphi_{6} > 0$$

$$a_{1} = (I_{1}S_{1}\beta_{1}^{2}(\varphi_{5} + \varphi_{6})(I_{1}S_{1}\beta_{1}^{2} + \varphi_{1}\varphi_{4}) + \varphi_{5}\varphi_{6}(\varphi_{1} + \varphi_{4}) > 0$$

$$a_{0} = I_{1}S_{1}\beta_{1}^{2}\varphi_{5}\varphi_{6} - \beta_{8}\beta_{7}\beta_{1}I_{1}\beta_{9} + \varphi_{1}\varphi_{4}\varphi_{5}\varphi_{6} > 0$$

So, E_1 is locally asymptotically stable if $S_1\beta_1\varphi_5\varphi_6 > \beta_8\beta_7\beta_1\beta_9$ which is obvious true. (7)

3.1.3 Theorem

(Stability at E_2): If $S_2\beta_1\varphi_5\varphi_6 > \beta_7\beta_8\beta_9$ then $E_2 = (S_2, V_{c_2}, 0, I_2, M_2, R_2)$ is locally asymptotically stable.

For the point $E_2(N_v = 0)$

$$J(E_2) = \begin{bmatrix} -\varphi_1 & -\beta_2 S_2 & -\beta_3 S_2 & -\beta_1 S_2 & 0 & \beta_9 \\ \beta_2 V_{c_2} & -\varphi_2 & \beta_4 & 0 & 0 & 0 \\ 0 & 0 & -\varphi_3 & 0 & 0 & 0 \\ \beta_1 I_2 & \beta_5 & \beta_6 & -\varphi_4 & 0 & 0 \\ 0 & 0 & 0 & \beta_7 & -\varphi_5 & 0 \\ 0 & 0 & 0 & 0 & \beta_8 & -\varphi_6 \end{bmatrix}$$

where,

 $\varphi_1 = \beta_1 I_2 + \beta_2 V_{c_2} + \mu$, $\varphi_2 = \beta_5 + \mu$, $\varphi_3 = -\beta_3 S_2 + \beta_4 + \beta_6 + \mu$, $\varphi_4 = -\beta_1 S_2 + \beta_7 + \mu$, $\varphi_5 = \beta_8 + \mu$ and $\varphi_6 = \beta_9 + \mu$. The Eigen value for the above matrix is $\lambda_1 = -\varphi_3 < 0$. Hence, above matrix has the characteristic polynomial equation.

$$\lambda^{5} + a_{4}\lambda^{4} + a_{3}\lambda^{3} + a_{2}\lambda^{2} + a_{1}\lambda + a_{0} = 0$$

where,

$$\begin{aligned} a_{4} &= (\varphi_{1} + \varphi_{2} + \varphi_{4} + \varphi_{5} + \varphi_{6}) > 0 \\ a_{3} &= (I_{2}S_{2}\beta_{1}^{2} + S_{2}V_{c_{2}}\beta_{2}^{2} + \varphi_{1}(\varphi_{2} + \varphi_{4} + \varphi_{5} + \varphi_{6}) + \varphi_{2}(\varphi_{4} + \varphi_{5} + \varphi_{6}) + \varphi_{4}(\varphi_{5} + \varphi_{6}) + \varphi_{5}\varphi_{6}) > 0 \\ a_{2} &= (I_{2}S_{2}\beta_{1}^{2}(\varphi_{2} + \varphi_{5} + \varphi_{6}) + (\varphi_{4} + \varphi_{5} + \varphi_{6})(S_{2}V_{c_{2}}\beta_{2}^{2} + \varphi_{1}\varphi_{2}) + SV_{c_{2}}\beta_{1}\beta_{2}\beta_{5} + \varphi_{4}(\varphi_{5} + \varphi_{6})(\varphi_{1} + \varphi_{2}) \\ &+ \varphi_{5}\varphi_{6}(\varphi_{1} + \varphi_{2} + \varphi_{4})) > 0 \\ a_{1} &= ((\varphi_{5} + \varphi_{6})(I_{2}S_{2}\beta_{1}^{2}\varphi_{2} + SV_{c_{2}}\beta_{1}\beta_{2}\beta_{5} + \varphi_{1}\varphi_{2}\varphi_{4}) + S_{2}V_{c_{2}}\beta_{2}^{2}(\varphi_{4}\varphi_{5} + \varphi_{4}\varphi_{6} + \varphi_{5}\varphi_{6}) \\ &+ \varphi_{1}\varphi_{5}\varphi_{6}(\varphi_{2} + \varphi_{4}) + \varphi_{1}\varphi_{4}\varphi_{5}\varphi_{6} + I_{2}\beta_{1}(S_{2}\beta_{1}\varphi_{5}\varphi_{6} - \beta_{7}\beta_{8}\beta_{9})) > 0 \\ a_{0} &= (I_{2}\beta_{1}x_{2}(S_{2}\beta_{3}\varphi_{5}\varphi_{6} - \beta_{7}\beta_{6}\beta_{0}) + V_{c}\beta_{2}\beta_{5}(S_{2}\beta_{3}\varphi_{5}\varphi_{6} - \beta_{7}\beta_{6}\beta_{0}) + \varphi_{4}\varphi_{5}\varphi_{6}(S_{2}V_{c}\beta_{2} + \varphi_{4}\varphi_{5})) > 0 \end{aligned}$$

$$a_0 = (I_2\rho_1x_2(S_2\rho_1\varphi_5\varphi_6 - \rho_7\rho_8\rho_9) + v_{c_2}\rho_2\rho_5(S_2\rho_1\varphi_5\varphi_6 - \rho_7\rho_8\rho_9) + \varphi_4\varphi_5\varphi_6(S_2v_{c_2}\rho_2 + \varphi_1\varphi_2)) > 0$$

 E_2 is locally asymptotically stable if $S_2\beta_1\varphi_5\varphi_6 > \beta_7\beta_8\beta_9$ (8)

3.1.4 Theorem

(Stability at E_3) If $S_2\beta_1\varphi_5\varphi_6 > \beta_7\beta_8\beta_9$ then $E_3 = (S_3, 0, N_{\nu_3}, I_3, M_3, R_3)$ is locally asymptotically stable.

For the point $E_3(V_c = 0)$, Jacobian matrix is

$$J(E_3) = \begin{bmatrix} -\varphi_1 & -\beta_2 S_3 & -\beta_3 S_3 & -\beta_1 S_3 & 0 & \beta_9 \\ 0 & -\varphi_2 & \beta_4 & 0 & 0 & 0 \\ \beta_3 N_{\nu_3} & 0 & -\varphi_3 & 0 & 0 & 0 \\ \beta_1 I_3 & \beta_5 & \beta_6 & -\varphi_4 & 0 & 0 \\ 0 & 0 & 0 & \beta_7 & -\varphi_5 & 0 \\ 0 & 0 & 0 & 0 & \beta_8 & -\varphi_6 \end{bmatrix}$$

where,

$$\varphi_1 = \beta_1 I_3 + \mu + \beta_3 N_{\nu_3} , \ \varphi_2 = \beta_5 + \mu , \ \varphi_3 = -\beta_3 S_3 + \beta_4 + \beta_6 + \mu , \ \varphi_4 = -\beta_1 S_3 + \beta_7 + \mu ,$$

$$\varphi_5 = \beta_8 + \mu$$

 $\varphi_6 = \beta_9 + \mu$. The reduced form of the above matrix has the characteristic polynomial equation

$$\lambda^{6} + a_{5}\lambda^{5} + a_{4}\lambda^{4} + a_{3}\lambda^{3} + a_{2}\lambda^{2} + a_{1}\lambda + a_{0} = 0$$

where,

$$a_5 = (\varphi_1 + \varphi_2 + \varphi_3 + \varphi_4 + \varphi_5 + \varphi_6) > 0$$

 $a_{4} = (S_{3}(I_{3}\beta_{1}^{2} + N_{\nu_{3}}\beta_{3}^{2}) + \varphi_{1}(\varphi_{2} + \varphi_{3} + \varphi_{5} + \varphi_{6}) + \varphi_{2}(\varphi_{3} + \varphi_{4} + \varphi_{5} + \varphi_{6}) + \varphi_{3}(\varphi_{4} + \varphi_{5} + \varphi_{6}) + \varphi_{4}(\varphi_{5} + \varphi_{6}) + \varphi_{5}\varphi_{6}) > 0$

$$a_{3} = (I_{3}S_{3}\beta_{1}^{2}(\varphi_{2} + \varphi_{4} + \varphi_{5} + \varphi_{6}) + N_{\nu_{3}}S_{3}\beta_{3}(\beta_{1}\beta_{6} + \beta_{2}\beta_{4}) + N_{\nu_{3}}S_{3}\beta_{3}^{2}(\varphi_{1} + \varphi_{4} + \varphi_{5} + \varphi_{6}) + \varphi_{1}\varphi_{2}(\varphi_{3} + \varphi_{4} + \varphi_{5} + \varphi_{6}) + (\varphi_{4} + \varphi_{5} + \varphi_{6})(\varphi_{1}\varphi_{3} + \varphi_{2}\varphi_{3}) + \varphi_{1}\varphi_{4}(\varphi_{5} + \varphi_{6})) > 0$$

$$a_{2} = (I_{3}S_{3}\beta_{1}^{2}\varphi_{2}(\varphi_{3} + \varphi_{5} + \varphi_{6}) + (\varphi_{5} + \varphi_{6})(I_{3}S_{3}\beta_{1}^{2}\varphi_{3} + N_{v_{3}}S_{3}\beta_{3}^{2} + \varphi_{1}\varphi_{2}\varphi_{4} + \varphi_{1}\varphi_{3}\varphi_{4} + \varphi_{2}\varphi_{3}\varphi_{4}) + N_{v_{3}}S_{3}\beta_{1}\beta_{3}(\beta_{4}\beta_{5} + \beta_{6}\varphi_{2} + \beta_{6}\varphi_{5} + \beta_{6}\varphi_{6} + \beta_{4}\varphi_{4} + \beta_{4}\varphi_{5} + \beta_{4}\varphi_{6}) + (\varphi_{4} + \varphi_{5} + \varphi_{6})(N_{v_{3}}S_{3}\beta_{3}^{2}\varphi_{2} + \varphi_{1}\varphi_{2}\varphi_{3}) + N_{v_{3}}S_{3}\beta_{3}^{2}\varphi_{5}\varphi_{6} + \varphi_{1}\varphi_{5}\varphi_{6}(\varphi_{2} + \varphi_{3} + \varphi_{4}) + \varphi_{2}\varphi_{5}\varphi_{6}(\varphi_{3} + \varphi_{4}) + \varphi_{3}\varphi_{4}\varphi_{5}\varphi_{6}) + I_{3}\beta_{1}(S_{3}\beta_{1}\varphi_{5}\varphi_{6} - \beta_{7}\beta_{8}\beta_{9})) > 0$$

$$\begin{aligned} a_{1} &= ((\varphi_{5} + \varphi_{6})(I_{2}S_{2}\beta_{1}^{2}\varphi_{2}\varphi_{3} + N_{v_{3}}S_{3}\beta_{1}\beta_{3}\beta_{4}\beta_{5} + N_{v_{3}}S_{3}\beta_{1}\beta_{3}\beta_{6}\varphi_{2} + N_{v_{3}}S_{3}\beta_{2}\beta_{3}\beta_{4}\varphi_{4} \\ &+ N_{v_{3}}S_{3}\beta_{3}^{2}\varphi_{2}\varphi_{4} + \varphi_{1}\varphi_{2}\varphi_{3}\varphi_{4}) + N_{v_{3}}S_{3}\beta_{2}\beta_{3}\beta_{4}\varphi_{5}\varphi_{6} + N_{v_{3}}S_{3}\beta_{3}^{2}\varphi_{5}\varphi_{6}(\varphi_{2} + \varphi_{4}) \\ &+ \varphi_{1}\varphi_{2}\varphi_{5}\varphi_{6}(\varphi_{3} + \varphi_{4}) + \varphi_{3}\varphi_{4}\varphi_{5}\varphi_{6}(\varphi_{1} + \varphi_{2}) + I_{3}\beta_{1}\varphi_{2}(S_{3}\beta_{1}\varphi_{5}\varphi_{6} - \beta_{7}\beta_{8}\beta_{9}) \\ &+ I_{3}\beta_{1}\phi_{3}(S_{3}\beta_{1}\phi_{5}\varphi_{6} - \beta_{7}\beta_{8}\beta_{9}) + N_{v_{3}}\beta_{3}\beta_{6}(S_{3}\beta_{1}x_{5}x_{6} - \beta_{7}\beta_{8}\beta_{9})) > 0 \end{aligned}$$

$$a_{0} = (N_{v_{3}}S_{3}\beta_{3}\varphi_{4}\varphi_{5}\varphi_{6}(\beta_{2}\beta_{4} + \beta_{3}\varphi_{2}) + \varphi_{1}\varphi_{2}\varphi_{3}\varphi_{4}\varphi_{5}\varphi_{6} + I_{3}\beta_{1}\varphi_{2}\varphi_{3}(S_{3}\beta_{1}\varphi_{5}\varphi_{6} - \beta_{7}\beta_{8}\beta_{9}) + N_{v_{3}}\beta_{3}\beta_{6}\varphi_{2}(S_{3}\beta_{1}\varphi_{5}\varphi_{6} - \beta_{7}\beta_{8}\beta_{9})) > 0$$

 E_3 is locally asymptotically stable if $S_2\beta_1\varphi_5\varphi_6 > \beta_7\beta_8\beta_9$

(9)

 E_0, E_1, E_2 and E_3 are local stable and satisfy the condition of Routh-criterion [22].

3.2 Global stability

Here, we discuss the global stability behavior of the equilibrium E_0, E_1, E_2 and E_3 by Lyapunov function

3.2.1 Theorem

(Stability at E_0): The disease-free equilibrium point $E_0 = \left(\frac{B}{\mu}, 0, 0, 0, 0, 0\right)$ is globally asymptotically stable.

Proof: Consider the Lyapunov function

$$L(t) = V_{c}(t) + N_{v}(t) + I(t) + H(t) + R(t) \text{ then}$$

$$L(t) = \left(\left(\frac{\beta_{2}B}{\mu} - \mu \right) V_{c} + \left(\frac{\beta_{3}B}{\mu} - \mu \right) N_{v} + \left(\frac{\beta_{1}B}{\mu} - \mu \right) I - \beta_{9}R - \mu H - \mu R \right) \le 0$$
If
$$(i) \frac{\beta_{2}B}{\mu} \le \mu \Longrightarrow \beta_{2}B \le \mu^{2}$$

$$(ii) \frac{\beta_{3}B}{\mu} \le \mu \Longrightarrow \beta_{3}B \le \mu^{2}$$

$$(iii) \frac{\beta_{1}B}{\mu} \le \mu \Longrightarrow \beta_{1}B \le \mu^{2}$$

$$(10)$$

So, if min $\{\beta_1 B, \beta_2 B, \beta_3 B\} \le \mu^2$, then E_0 is globally stable.

3.2.2 Theorem

(Stability at E_1): The endemic equilibrium point $E_1 = (S_1, 0, 0, I_1, M_1, R_1)$ is globally asymptotically stable.

We will discuss the global stability behavior of $E_1(V_c=N_v=0)$ by Lyapunov function

$$L(t) = V_{c}(t) + N_{v}(t) + I(t)$$

$$L(t) = (\beta_{2}SV_{c} - \mu V_{c}) + (\beta_{3}SN_{v} - \mu N_{v}) + (\beta_{1}SI - \mu I) - \beta_{7}I$$

Substituting $\beta_2 SV_c + \beta_3 SN_v + \beta_1 SI$ from the equation (1) $\dot{L}(t) < 0$

By LaSalle's Invariance Principle, E_1 is globally stable.

3.2.3 Theorem

(Stability at E_2): The endemic equilibrium point $E_2 = (S_2, V_{c_2}, 0, I_2, M_2, R_2)$ is globally asymptotically stable

We will discuss the global stability behavior of $E_2(N_v = 0)$ by Lyapunov function

$$L(t) = N_v(t) + I(t) + H(t) + V_c(t)$$

(11)

$$\dot{L}(t) = (\beta_2 SV_c - \mu V_c) + (\beta_3 SN_v - \mu N_v) + (\beta_1 SI - \mu I) - \beta_8 H - \mu H$$

Substituting $\beta_2 SV_c + \beta_3 SN_v + \beta_1 SI$ from the equation (1) $\dot{L}(t) < 0$

By LaSalle's Invariance Principle, E_2 is globally stable.

Same way $E_3(V_c = 0)$ is also globally stable.

4 Sensitivity Analysis

In this section, the sensitivity analysis for all the parameters are shown in Fig. 2.



Fig. 2. Sensitivity analysis of the parameters

Fig. 2 shows the sensitivity of parameters $B, \beta_1, \beta_2, \beta_3, \beta_4, \beta_5, \beta_6$ and β_7 with respect to basic reproduction number. It can be seen from the figure that $B, \beta_1, \beta_2, \beta_3, \beta_4$ are positive which means that infection spreads and $\beta_5, \beta_6, \beta_7$ are negative which means that these parameters are not infected.

5 Numerical Analysis

In this section, we will study the numerical results of all compartments.

Fig. 3 shows within one day; 16 susceptible individuals enter in vaccinated compartment. 14 vaccinated individuals get infected in one day, then after it starts decreasing. During 3 days, 7 susceptible individuals will go in to medicated compartment and after that it starts decreasing. During 3 days, 6 susceptible individuals get recovered. 6 medicated individuals get recovered in 14 days.40 susceptible individuals will go in to non-vaccinated compartment after 4 days.



Fig. 3. Movement of an individual in each compartment

Table 2. Results of	R_0	at the points	E_0 ,	$E_{1},$	E_2 and	E_3
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Equilibrium points								K	2 0	
$E_{0} = \left(\frac{B}{\mu}, 0, 0, 0, 0, 0\right)$								0.	36	
$E_1 = (S_1, 0, 0, I_1, M_1, R_1)$		17.4								
$E_2 = (S_2, V_{c_2}, 0, I_2, M_2, R_2)$		0.48								
$E_3 = (S_3, 0, N_{\nu_3}, I_3, M_3, R_3)$		8.7								
0.9 0.8 0.7 0.7 0.6 0.5 0.4 0.3 0.4 0.3 0.2 0.1 0.2 0 0.1 0 0	0.1									

Fig. 4. Motion of infected individuals towards medication

If community does not opt for vaccination, then on average 18 individuals get infected. Whereas on average vaccinated individual transmits infection to only one. On an average 9 individual get affected from the infected population.

As shown in Fig. 4, infected individuals opt for medication after certain time.



Fig. 5. Motion of non-vaccinated individual towards medication

As Fig. 5 indicates, more and more non-vaccinated individuals go for medication. Intensity of the medication in the later stage increases for non-vaccinated individuals.



Fig. 6. The movement of susceptible individuals towards recovery



Fig. 6 shows the movement of susceptible individuals towards recovery. It may happen that these recovered individuals may again become victim of H1N1 after sometime.

Fig. 7. Percentage of vaccinated, Non-vaccinated and Infected out of total population

Fig. 7 shows that out of the total population 39% of individuals opts for vaccination. 34% remains unvaccinated. 27% of the individuals gets infected to H1N1 either due to vaccination or Non-vaccination.

6 Conclusion

Transmission of H1N1 virus occurs rapidly amongst the population from different countries in the world. Simple personal measures may help to avoid seasonal the flu infection: vaccination against flu virus, hand hygiene, well- balanced diet, regular exercise, adequate sleep, and avoiding close contacts. If the general population is aware of the disease, they have a greater opportunity to protect themselves from infection, avoid unnecessary contact, and alleviate the spread. The influenza virus is a serious risk to global public health due to the limited availability of proper vaccines and antiviral drugs, the constant movement of the world's population, and the unique nature of the virus. Many countries are not prepared for the epidemics of H1N1 as they do not have enough resources to tackle this situation. In this context, massive public awareness and knowledge about the disease may help reduce the number of causalities during future outbreaks. Our study showed that if the population is knowledgeable about the modes of transmission, mitigation measures they can adapt, and opt for vaccination, one infected individual transmits the infection to only one on average compare to one non vaccinated infected individual transmits the infection to 18 more people on average. In this study, we have shown that vaccination is a key factor against this disease. The study is valuable to all mankind and shows that to attain valuable protection from this virus, the population needs to adapt to a good and healthy environment, isolation from infected individuals, and vaccination at the proper time. We believe that increased knowledge, timely availability of vaccination, and improved general population awareness about H1N1 transmission are steps that individuals and the government can take to prevent the rapid spread of the viruses by different preventive techniques. This can lead to a decrease in the socio-economic burden on society. The result will be a decrease in morbidity and mortality from H1N1.

Competing Interests

Authors have declared that no competing interests exist.

References

- [1] Centers for disease control and prevention. Questions and answers about 2009 H1N1 Flu (H1N1 influenza); 2009a. Available:https://www.cdc.gov/h1n1flu/qa.htm
- [2] Center for Infectious Disease Research and Policy; 2009. Available:https://www.cidrap.umn.edu/infectious-disease-topics/h1n1-2009-pandemic-influenza
- [3] Mackenzie D. The predictable pandemic. The New Scientist. 2006;6-7.
- [4] World Health Organization. 2009b) Pandemic (H1N1); 2009. Available:http:// www.who.int/csr/disease/swineflu/en/index.html
- [5] Nettleman M. Cases of swine influenza in humans: A review of the literature. eMedicine Health; 2009.
 Available:http:// www.emedicinehealth.com/swine flu/article em.htm
- [6] Steelfisher G, Blendon R, Kang M, Ward J, Kahn E, Maddox K, Lubell K, Tucker M, Ben-Porat E. Adoption of preventive behaviors in response to the 2009 H1N1 influenza pandemic: A multiethnic perspective. Influenza and Other Respiratory Viruses. 2015;9(3):131–142.
- [7] Ferguson NM, Cummings DA, Fraser C, Cajka JC, Cooley PC, Burke DS. Strategies for mitigating an influenza pandemic. Nature. 2006;442:448–452.
- [8] Glass RJ, Glass LM, Beyeler WE, Min HJ. Targeted social distancing design for pandemic influenza. Emerg Infect Dis. 2006;12:1671–1681.
- [9] Tahir M, Shah SA, Zaman G, Khan T. A dynamic compartmental mathematical model describing the transmissibility of MERS-CoV virus in public. Punjab University Journal of Mathematics. 2019;51: 57–71.
- [10] Tahir M, Shah SA, Zaman G. Prevention strategy for superinfection mathematical model tuberculosis and HIV associated with AIDS. Cogent Mathematics & Statistics. 2019;6(1):1637166. Available:https://doi.org/10.1080/25742558.2019.1637166
- [11] DHS: Yearbook of immigration statistics; 2017. Available:https://www.dhs.gov/immigration-statistics/yearbook/2017
- [12] Rodriguez-Lainz A, et al. Influenza vaccination coverage among us-mexico land border crossers: 2009 H1n1 pandemic and 2011-2012 influenza season. Travel Med Infect Dis. 2019;99-103.
- [13] Disease Burden of Influenza | CDC. (n.d.). Available:https://www.cdc.gov/flu/about/burden/index.html
- [14] Conway JM, Tuite AR, Fisman DN, Hupert N, Meza R, Davoudi B, English K, van den Driessche P, Brauer F, Ma J, Meyers LA, Smieja M, Greer A, Skowronski DM, Buckeridge DL, Kwong JC, Wu J, Moghadas SM, Coombs D, Brunham RC, Pourbohloul B. Vaccination against 2009 Pandemic H1n1 in a population dynamical model of vancouver, Canada: Timing Is everything. BMC Public Health. 2011;11:932.
- [15] Kowal SP, Jardine CG, Bubela TM. If they tell me to get it, I'll get it. If They Don't...": Immunization decision-making processes of immigrant mothers. Can J Public Health. 2015;106(4):e230-5.

- [16] Lu P, Bridges CB, Euler GL, Singleton JA. Influenza vaccination of recommended adult populations. US, 1989–2005. Vaccine. 2008;26:1786–1793.
- [17] Linn ST, Guralnik JM, Patel KV. Disparities in influenza vaccine coverage in the United States, 2008. J Am Geriatr Soc. 2010;58:1333–1340.
- [18] Bish A, Yardley L, Nicoll A, Michie S. Factors associated with uptake of vaccine against pandemic influenza: a systematic review. Vaccine. 2011;29:6472–6484.
- [19] Lindley MC, Wortley PM, Winston CA, Bardenheier BH. The role of attitudes in understanding disparities in adult influenza vaccination. Am J Prev Med. 2006;31:281–285.
- [20] Centers for Disease Control and Prevention. Key facts about Swine Influenza (H1N1 influenza); 2009b. Available:http:// www.cdc.gov/flu/swineflu/key facts.htm
- [21] Osterholm MT. Preparing for the next pandemic. New England Journal of Medicine. 2006;352:1839-1842.
- [22] Routh EJ. A treatise on the stability of a given state of motion: Particularly steady motion. Macmillan and company; 1877.

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