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Case Study

Avian Influenza Type A-H5N1 Epidemiological Model: Puerto Rico as a Case Study

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Keywords SIR; Avian Influenza; Epidemiology; Metapopulation; Vaccination; Basic Reproductive Number; Simulations Spread of Diseases

Abstract

Our research focused on Avian Influenza Type A-H5N1, specifically on an epidemiological model centered in Puerto Rico. Our main goal is to address the following: first, to determine the potential outbreaks of this disease in Puerto Rico using as a base the location of the poultry industry as a hub, we are interested in the repercussions of the infection among the human-to-human potential interaction. The second goal centers on the possibility of vaccination to mitigate an epidemic among humans. In order to address these goals and future ones, we will construct a mathematical model and use parameters according to two cases; the first is a single population model and the second one is a metapopulation model involving 5 cities in Puerto Rico. Our research will specifically target the spread of this particular disease, to investigate possible alternatives to mitigate the spread using measures of immunization. Our results show that a 30% vaccination regime will eradicate the disease in cities that are immunized.

Introduction

There are three types of influenza viruses: A, B and C. They are divided into subtypes on the basis of two proteins on the surface of the virus: Hemagglutinin (HA) that has 17 subtypes known and Neuraminidase (NA) that has 10 known subtypes [1]. Epidemiologists need to make an educated guess for the inclusion of such variants in annual vaccines in order to assure proper immunization of the population and mitigate a possible epidemic among the population. However, due to the lack of immunity in humans against new mutations of the virus, epidemics or even pandemics can emerge resulting in high morbidity and mortality [2]. In this article we focus on Avian Influenza Type A-H5N1 that is a zoonotic disease (i.e. spread from animal to humans) it is an acute and recurring respiratory disease, occurring in particular during winter months and straining the public health system worldwide. It can only spread from infected poultry to humans that had been in contact with poultry or infected soil. Avian influenza has high mortality rate, as high as 60% [2-6] hence it is a mayor concern if it mutates and can spread from human to human. As any type of influenza it is fatal for immune compromised individuals; children, elderly, patients with chronic illnesses and pregnant women. The initial symptoms are: high temperature 38°C or 100.4°F, upper respiratory tract symptoms, diarrhea, vomiting, abdominal pain, inflammation of the lungs (pleuritic pain) and nose bleeding [3-7].

We implemented modification of a simpler SIR model (Susceptible-Infected-Recovered) developed by *Kermack and McKendrick* [8], then implemented an SEIR (adding Exposed individuals) for a single population to study the effect of different levels of vaccination in the population that can potentially contain or eradicate the disease in a hypothetical scenario of an epidemic in Puerto Rico. We will base our models in past epidemics for different countries that have suffered from epidemics or outbreaks of Avian Influenza Type A-H5N1 [2]. In 1959 chickens spread the disease in Scotland, in 1992 the disease was spread in England by turkeys, in 1997 chickens also spread the disease in Hong Kong [3-6]. In different countries in Asia and the Middle East, between 2005 and 2006 the avian species that spread the disease predominantly were domestic and wild poultry [3-6].

This article will specifically target the spread of infectious diseases, which will be held in perspective with a mathematical-epidemiological model to determine possible alternatives to mitigate the potential spread of Avian Influenza Type A-H5N1 using measures such as immunization. Particularly, we address the spread of influenza type A-H5N1 in the country of Puerto Rico in the town of Cayey, using as a possible site of infection the city of Aibonito. The hub of infection will be set in Cayey where there is a poultry factory located near. We would also work on constructing a metapopulation model involving 5 cities of Puerto Rico connected by the main highways. A meta population model consists of a group of interacting spatially separated populations of the same species, it is defined as a set of differential equation coupled together. After our first simple model we implemented a SIR-type mobility model for five cities in Puerto Rico

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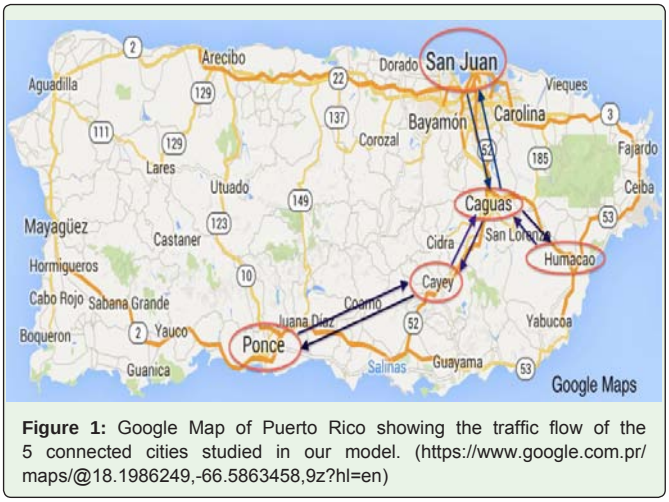

Figure 1: Google Map of Puerto Rico showing the traffic flow of the 5 connected cities studied in our model. (https://www.google.com.pr/maps/@18.1986249,-66.5863458,9z?hl=en)

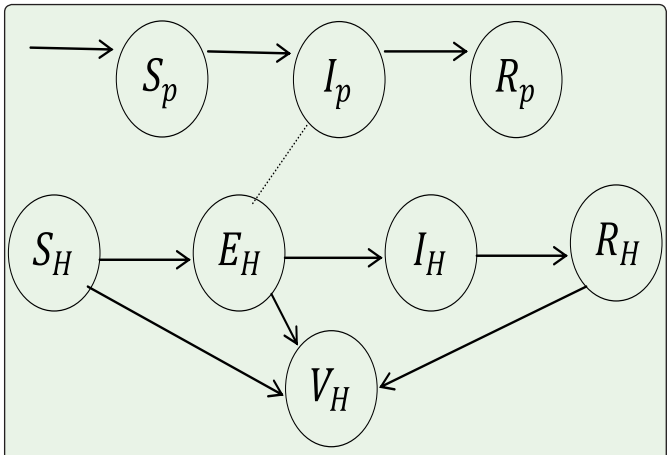

Figure 2: Schematic of the mathematical model for Avian Influenza A-H5N1.

Table 1: Epidemiological status of humans and poultry

Class	Definitions
N_p	Total poultry population
S_p	Susceptible poultry
I_p	Infected poultry
R_p	Recovered poultry
N_h	Total human population
S_h	Susceptible humans
I_h	Infected humans
R_h	Recovered humans
E_h	Exposed humans-Incubation virus
V_h	Vaccination humans

to investigate the potential outbreak of this disease in the linked areas of: San Juan, Caguas, Cayey, Ponce and Humacao as seen in Figure 1 in the methodology section.

Our focus is on the behavior of the epidemic of human interaction and the effect on the entire population. However, we implemented the mathematical model to address these specific aims: first, to determine the outbreaks of this disease in Puerto Rico using as a base the location of the poultry industry as a hub. The second goal centers on the possibility of vaccination to mitigate an epidemic among humans. Third, for the meta population networking approach we want to determine the potential outbreaks of this disease in Puerto Rico and how different vaccination implementations mitigate or reshape the epidemic outbreak as time goes by.

The research questions we want to address are: What mechanisms are effective to contain a potential Avian A-H5N1 epidemic? Which dynamics of disease spreading affect an outbreak among cities? What mechanisms are effective to contain a potential avian flu epidemic among cities? In order to address this question and further ones we will construct a mathematical model and use a different set of parameters, for now we are focusing first

Table 2: Parameter values of the mathematical model.

Parameter	Definitions	Range	References
β_p	Infectious rate for chickens	2.5	[11]
β_h	Infectious rate for humans – humans interaction	0.5	[11]
βh_p	Infectious rate for humans – chickens interaction	0.2	[11]
γ_h	Lower infectivity for the interaction between exposed and susceptible humans	0.95	[11]
α_h	Recovery rate for humans	0.1	[11]
α_p	Recovery rate for chickens	0.002	[11]
δ_p	Death rate due to infection in chickens	0.05	[11]
δ_h	Death rate due to infection in humans	0.005	[11]
$1/\sigma_h$	Incubation period for humans	2-17 days	[2]
π_h	Vaccination rate for humans	Effectiveness of 60 to 90%	[11]
μ_p	Demographics - birth rate and mortality	0.005	[11]
b_p	Demographics - birth rate massive chicks rate	0.1	[11]

on the mitigation of the epidemic using vaccination strategies. Important to mention, we are interested in the repercussion of the infection among human-to-human interactions for the networking approach.

Methodology

We implemented the modification of a simpler SIR epidemiological model evolving to a SEIRV and use parameter values of outbreaks that occurred in different countries in the past to modify them to the case of Puerto Rico [9,10]. We want to explore what would happen in Puerto Rico if the disease of Avian Influenza Type A-H5N1 emerged, taking into consideration what happened in different countries such as: Europe, Latin America and Middle East [11]. In order to present our simple SEIRV mathematical model, let's define our epidemiological classes as shown in Table 1 and parameters as shown in Table 2. We show in Figure 1 a schematic of the transition of individuals into the different epidemiological classes. Then we present our system of ordinary differential equations and our simulations. Afterwards we implemented a meta population model modification of a simpler mobility model for five cities in Puerto Rico on a SIR-type model, for future work we will consider an SIRV network model [12-14]. In this meta population approach we want to address the potential outbreak of this disease in the areas of: San Juan, Caguas, Cayey, Ponce and Humacao that are linked as shown in Figure 1.

SEIRV single city epidemiological model

Let's describe our modeling approach, starting with the description of the model shown in Figure 2 and the equations that follow. The model classifies the subjects into the different compartments according to species and disease stages. The model classifies the population into a unique epidemiological class or compartment in general as described in Table 1: susceptible subjects (S_i), exposed individuals (E_i), infected (I_i), recovered (R_i) and vaccinated (V_i) individuals. $\mathbf{N_i}$ is the total population; where $i \in \{p,h\}$ the subscripts p and h indicate poultry or human subjects respectively. Here we assume that the total population of poultry is 60,000 and the population of the city of Cayey (that we are using for a one city model) is 48,119 according to the 2010 census data.

The parameters used in the mathematical model for Avian Influenza Type A-H5N1 are shown in Table 2 in general (recall that subscripts of p are h are imposed to identify poultry or humans respectively) they are defined as:μ_i that represent the demographics which is defined as the rate of birth and death for chickens and b is the "birth rate" when the chickens are massively produced and distributed to factories, the β_i parameter is the ratio of effective contact between the interaction of a susceptible and an infected subject, the recovery rate is α_i where $(1/\alpha_i)$ is the number of days the person was ill, $1/\sigma_i$ is the incubation period and π_h is the vaccination rate for humans.

We present the schematic flow of the model on Figure 2 and below we present the ordinary differential equations that define them. Each box represents a compartment where subjects are categorized into the epidemiological classes described in Table 1 at the rates described and shown in Table 2. The differential equation system is as follows:

$$dS_p/dt = -\beta_p S_p I_p/N_p + bN_p + \mu N_p + \mu S_p$$

$$dI_p/dt = \beta_p S_p I_p/N_p + \alpha_p I_p - \delta_p I_p - \mu I_p$$

$$dR_p/dt = \alpha_p I_p - \mu R_p$$

$$dS_h/dt = -(\gamma_h \beta_h S_h E_h + \beta_h S_h I_h + \beta_{hp} S_h I_p)/N_h - \pi_h S_h$$

$$dE_h/dt = (\gamma_h \beta_h S_h E_h + \beta_h S_h I_h + \beta_{hp} S_h I_p)/N_h - \sigma_h E_h - \pi_h E_h$$

$$dI_h/dt = \sigma_h E_h - \alpha_h I_h - \delta_h I_h$$

$$dR_h/dt = \alpha_h I_h - \pi_h R_h$$

$$dV_h/dt = \pi_h (S_h + E_h + R_h)$$

An arrow entering a compartment indicates inflow of individuals (or poultry), to address this in the equations this quality has a plus sign (+). Similarly an arrow leaving a compartment indicates that the sign is negative (-).

SIR epidemiological mathematical model with respect to the poultry: Entering S_p are susceptible chickens, the parameter b or μ, indicates (with a + sign) the demographics (birth rate $\mu N_p$and death rate μS_p) of chickens on a free range environment or import of massive amounts of chickens to a factory indicated with the factor bN_p. The factors $-\mu S_p$, $-\mu I_p$, $-\mu R_p$are the natural death of the poultry. The susceptible chickens that get into the I_p class are indicated with the factor $-\beta_p S_p I_p/N_p$with a - sign is going out of S_p into the I_p class it represents the contact between a susceptible and an infected subject, hence it has a + sign getting into the infected poultry I_p. The parameter $\alpha_p I_p$indicates the recovery ratio of the poultry with a - sign getting out of the infected class and a + sign getting into the recovery class. Chickens that died from the disease are indicated with the factor $-\delta_p I_p$.

SEIRV epidemiological mathematical model with respect to the human population: In this model since we are running the simulations and considering the time of the epidemic for less than a year we are not including demographics for the human population. The most important factor in this model is the force of infection:

$$(\gamma_h \beta_h S_h E_h + \beta_h S_h I_h + \beta_{hp} S_h I_p)/N_h$$

that represents how a susceptible human gets infected with A-H5N1 by means of interacting with exposed individuals $(\gamma_h \beta_h S_h E_h)/N_h$ that can transmit the disease at a lower rate than the interaction with infected individuals $\beta_h S_h I_h/N_h$ or by means of interaction with infected poultry $\beta_{hp} S_h I_p/N_h$ in a factory. This factor is negative leaving the susceptible individuals and positive enter into the infected class. The factors $\pi_h S_h$, $\pi_h E_h$ and $\pi_h R_h$ are the individuals that get vaccinated. Notice that we vaccinate not only the susceptible class, but also the recovered and exposed individuals, since they are not aware of their epidemiological status and hospitals or medical personal will vaccine individuals that are not symptomatic, hence infected individuals will not get vaccinated. The importance of these distinctions is that we can get a rough estimate of the wasted vaccines (i.e. vaccines given to individuals that had already immunity in the recovered class) or will get sick independently since they where incubating the virus already. The factor $\sigma_h E_h$ are the individuals that where incubating and now leave the exposed class because they are symptomatic. The factor $\alpha_h I_h$ are the individuals that recover from the disease, leaving the class I_h getting into the recovered class with a positive sign. Humans that died from the disease are indicating with the factor $-\delta_h I_h$.

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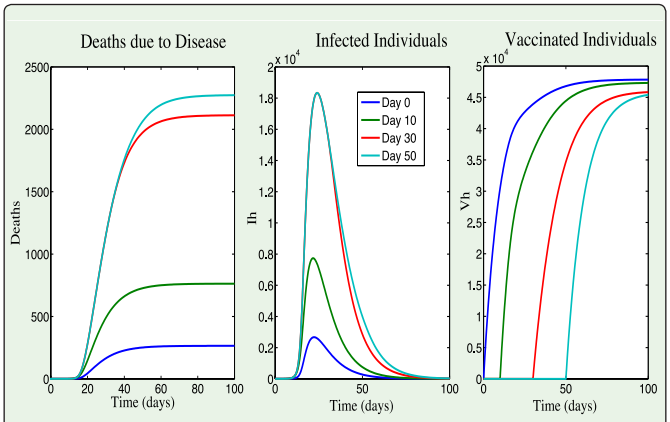

Figure 3: Vaccinating 10% of the population we vary the initial day of vaccination from 0 (blue), 10 (green), 30 (red) to 50 (turquoise) days. As we delay the vaccination, the number of deaths in the human population changes where on day 30 increase dramatically. The infected population also increases as the vaccination start later changing the duration and the peak of the epidemic.

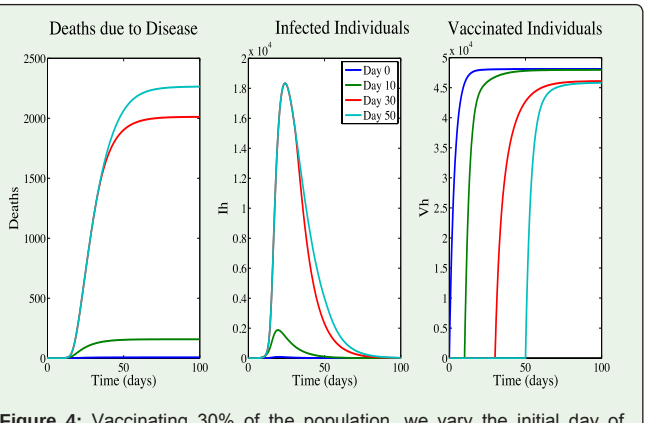

Figure 4: Vaccinating 30% of the population, we vary the initial day of vaccination from 0 (blue), 10 (green), 30 (red) to 50 (turquoise) days. As we delay the vaccination the number of deaths in the human population changes where on day 30 increase dramatically. The infected population also increases as the vaccination start later changing the duration and the peak of the epidemic. We notice a difference in the epidemic size for starting days 0 and 10 in comparison with Figure 3.

To study the epidemic further, we compute the basic reproductive number R_0, that is the number of people a sick individual infects when inserted into a fully susceptible population. In this case, foran epidemic to occur, we need an $R_0 > 1$. In order to compute R_0, we will be implementing the second-generation operator developed by Van Driessche and Watmough, in 2002 [15]. The R_0 for this model is defined as: $R_{0p} = [\beta_p / (\alpha_p+\delta_p)]$, $R_{0h} = [\beta_h / \sigma_h] + [\beta_h / (\alpha_h+\delta_h)]$ that represent respectively the R_0 for the poultry epidemic by itself and the R_0 for the human epidemic by itself (see Appendix 1 for the computation process).

Results: ***Simulations of the disease Avian Influenza Type A-H5N1***

The parameter values used in the simulations are as described in Table 2, where we use an incubation period for humans of 9 days and change the percentage of vaccinated individuals for the simulations shown and the starting day of the vaccination campaign for Figures 3 and 4.

We focus on the behavior of the epidemic entirely by human interaction and the effect of either a single individual or a population. On the results of the simulations when $R_o>1$, exists the possibility of an epidemic, we focused on the total population of Cayey that is 48,119 people in 2010 according to the census. The mortality rate in infected individuals is greater than 60% according to the World Health Organization (WHO) [16].

Table 3: Parameters values of the meta population SIR model for the five cities.

Parameters	Definitions	Values	References
β	Infectious rate for humans – humans interaction	0.5	[11]
μ	Demography – birth rate and mortality	0.005	[11]
α	Recovery rate for humans	0.1	[11]
δ	Death rate due to infection in humans	0.005	[11]
p_{12}	Instant Transportation for the city San Juan-Caguas	0.0002	Estimated
p_{23}	Instant Transportation for the city of Caguas-Cayey	0.0002	Estimated
p_{25}	Instant Transportation for the city of Caguas-Humacao	0.0002	Estimated
p_{34}	Instant Transportation for the city of Cayey-Ponce	0.0002	Estimated
π	Vaccination rate variations for each city	0, 10, 30%	Estimated

The parameter values used in the simulations from Figures 3 and 5 are as described in the Table 2 where we used an incubation period of 9 days for humans and changed the percentage of individuals vaccinated for the simulations shown and the starting day of the vaccination campaign for Figures 3 and 4. In Figure 3, we delay the vaccination from 0 to 30 days after the start of the epidemic and observed that the number of deaths in the human population increases dramatically if vaccination started 30 days after the epidemic. The infected population also increases as vaccines are delayed. In Figure 4, as we delay the vaccination the number of deaths in the human population on day 30 changes where it increased dramatically. We notice a difference in the epidemic size for starting days 0 and 10 in comparison with Figure 3. In Figure 5, as we increased the percentage of people vaccinated the epidemic morbidity and mortality changes as well, it has been shown computationally that when we vaccinate at least 30% of the population we can eradicate the epidemic.

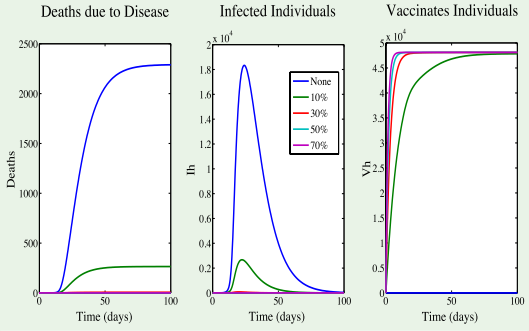

Figure 5: We start the vaccination at the same time the epidemic starts. We change the vaccinated percentage from 10% to 70%. As we increase the vaccination percentage, the epidemic decreases as well as the number of deaths but when we vaccinate at least 30% of the population we can eradicate the epidemic.

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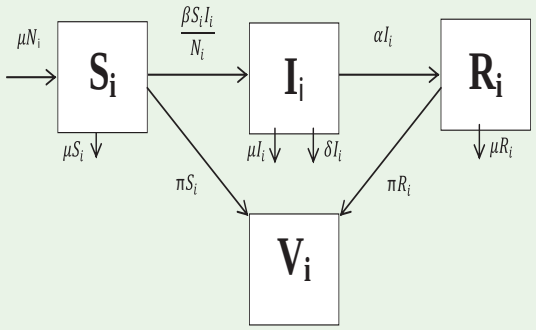

Figure 6: Schematic of the mathematical model for avian flu Type A-H5N1.

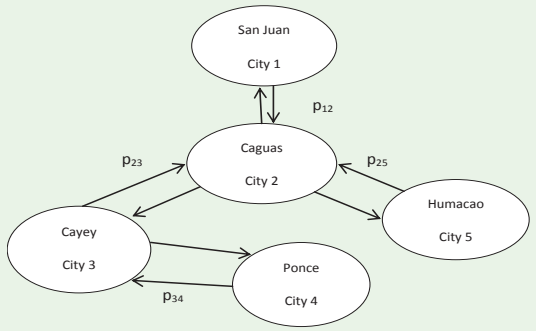

Figure 7: Schematic of the mobility network.

SIR five city meta population model

We implemented the modification of a simpler mobility model for five cities in Puerto Rico on a SIR-type model, to investigate the potential outbreak of this disease in the areas of: San Juan, Caguas, Cayey, Ponce and Humacao. The model classifies the population into a unique epidemiological class or compartment per city as: susceptible subjects (S_i), infected subjects (I_i), recovered subjects (R_i) and vaccinated individuals (V_i). N_i is the total population; where $i \in \{1,2,3,4,5\}$ indicate the city where the human subjects are located.

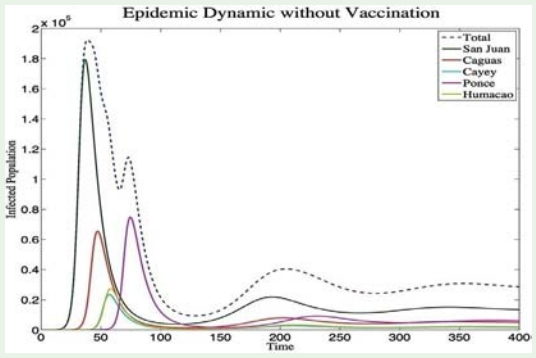

Figure 8: The epidemic starts in the city of San Juan and spreads to Caguas, Humacao, Cayey and finally to Ponce. Notice that the shape of the total epidemic changes depending on the initial conditions, giving the notion of having multiple waves of the epidemic as a whole. As time progresses there is a secondary wave with a pick around day 200 and the epidemic remains endemic.

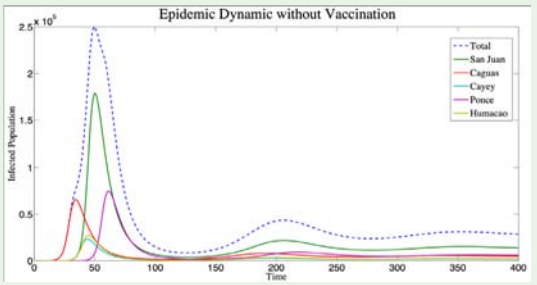

Figure 9: The epidemic starts in the city of Caguas and spreads to San Juan, Humacao, Cayey and finally to Ponce as time progresses there is a secondary wave with a pick around day 200 and we can see that the epidemic remains endemic.

In order to present our mathematical model let's define the schematic of the model as shown in Figure 6, the network or connectivity of the cities in Figure 7 and the parameters as shown in Table 3. For the networking approach we assume that the disease mutates such that it is transmitted from human to human and we only focus on the human interaction and the population traveling from one city to another.

Following the flow of the schematic and descriptions above, we are ready to write down our SIRV system of ordinary differential equations (where the summations $\Sigma_j p_{ij}$ run for j = 1…5) as follows:

$$dS_i/dt = \mu N_i - \mu S_i - \beta S_i I_i / N_i - \pi S_i + \Sigma_j p_{ij} S_j - \Sigma_j p_{ij} S_i$$

$$dI_i/dt = \beta S_i I_i / N_i - (\mu + \alpha +) I_i + \Sigma_j p_{ij} I_j - \Sigma_j p_{ij} I_i$$

$$dR_i/dt = - (\mu + \pi) R_h + \alpha I_i + \Sigma_j p_{ij} R_j - \Sigma_j p_{ij} R_i$$

$$dV_i/dt = \pi (S_i + R_i) + \Sigma_j p_{ij} V_j - \Sigma_j p_{ij} V_i$$

Similar to the SEIRV model we presented earlier in our meta population model schematic (Figure 6) each box represents a compartment where subjects are categorized into the epidemiological classes using the parameter rates described in Table 3. As before, an arrow entering a compartment indicates inflow of individuals represented in the ordinary differential equations coupled system with a plus sign. Similarly an arrow leaving a compartment indicates that the sign is negative. Important to mention is that the **i** subscript represents each city.

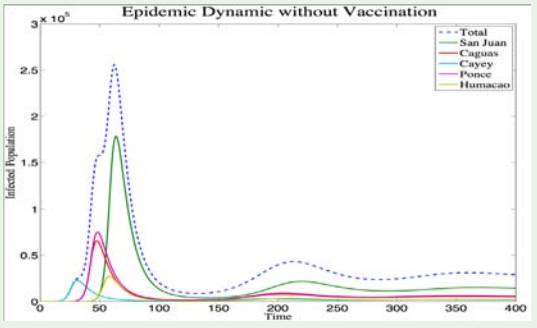

Figure 10 (A): The epidemic starts in the city of Cayey and spreads to Ponce, Caguas, San Juan, and finally to Humacao as time progresses there is a secondary wave with a pick around day 200 and we can see that the epidemic remains endemic.

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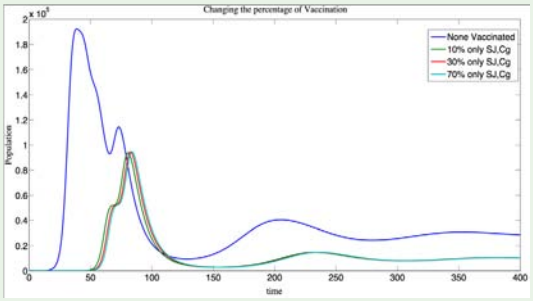

Figure 10 (B): We start the vaccination at the same time the epidemic starts. We change the vaccinated percentage from 10% to 70%. As we increase the percentage the epidemic decreases as well as the number of deaths, but computationally it can be shown that when we vaccinate at least 30% of the population we can eradicate the epidemic.

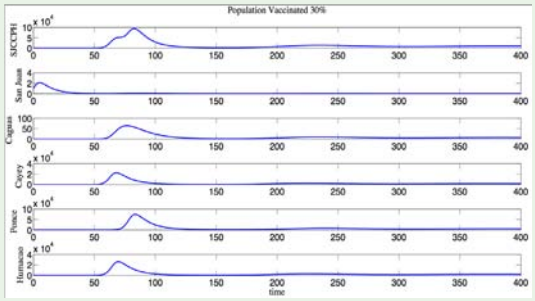

Figure 11 (B): We start the vaccination at the same time the epidemics starts only in San Juan and Caguas. We vaccinate 30% of the population in these two cities. The epidemic is eradicated in the cities that are vaccinated, but there is an outbreak in the other three cities that are the ones that contribute to the total epidemic waves shown in Figure 10.

SIR epidemiological mathematical model networking approach with respect to human population: Entering the susceptible class S_i we have the population birth rate μN_i for city i and leaving the death rate of human population $-\mu S_i$, the infected individuals $\beta S_i I_i / N_i$ that enter into the infected class I_i. The factors μI_i and μR_i are the natural death of the human population for the infected and recovered individuals. The recovery ratio of the human population αI_h has a negative sign getting out of the infected class and a plus sign getting into the recovery class. Humans that die from the disease are indicated with the factor δI_h and vaccinated individuals are indicated by π_i getting out of the class S_i and R_i into the V_i class. We vaccinate all individuals that are not symptomatic, since the individual is not aware of his/her epidemiological class. The CDC recommended in the 2008 influenza pandemic that since people are unaware of what strain of the disease they might have suffered prior they should get vaccinated against the disease [17]. p_{ij}'s are the proportion of the population that travels from city *i* to city *j*. Hence, the summation of the p_{ij} in the differential equations $\Sigma_j p_{ij}$, indicates the individuals traveling from other cities to city *i*. Note that the epidemiological state of the individuals does not change with the mobility because it is an instantaneous change of location.

The mobility component of the model: In this model we introduced traveling to five cities in Puerto Rico as seen in Figure 1 and the schematic of Figure 7 with the intention to extend our model in the future to the 72 municipalities of the island. It is important to mention that the individuals don't change the epidemiological state while they travel. Note that the parameter values of instant transportation are: p_{12} that represents the proportion of the population that travels from San Juan to Caguas, p_{23} is the proportion of the population that travels from Caguas to Cayey, p_{34} is the proportion of the population that travels from Cayey to Ponce, and finally p_{25} is the proportion of the population the travels from Caguas to Humacao. The big cities are San Juan with a population of 395,324 individuals, Caguas with 142,893 individuals and Ponce with 166,327 individuals. The smaller cities are Humacao with 58,466 individuals and Cayey with 48,119 individuals [18].

Results: ***Simulations for the Metapopulation model of Avian flu Type A-H5N1***

The parameter values used in the simulations are as described in Table 3 with variations in the initial conditions or vaccination rate.

Multiple peaks of the same epidemic outbreak can be seen in the total population as shown in Figure 8 and Figure 12, depending on the city of the initial outbreak and the connectivity with the other cities in the network we can observe the different shapes of the epidemic. The important result in our simulations is that with the parameters used from Table 3 avian influenza will remain endemic in the population if there is a mutation that will permit the disease to be transmitted from human to human.

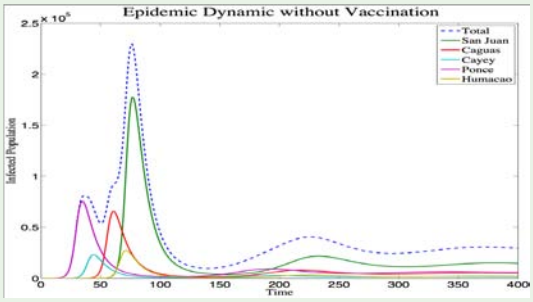

Figure 11 (A): The epidemic starts in the city of Ponce and spreads to Cayey, Caguas, San Juan, and finally to Humacao as time progresses there is a secondary wave with a pick around day 230 and we can see that the epidemic remains endemic.

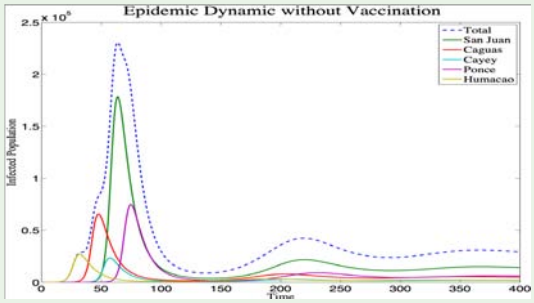

Figure 12: The epidemic starts in the city of Ponce and spreads to Cayey, Caguas, San Juan, and finally to Humacao as time progresses there is a secondary wave with a pick around day 230 and we can see that the epidemic remains endemic.

Changing the percentage of vaccination

The parameter values used in the simulations are as described in the Table 3 where we use an infection period for humans of 7 days and changed the percentage of vaccinated individuals for the simulations shown and the starting day of the vaccination. We used a non-democratic plan of vaccination [19] by vaccinating individuals in the two main cities in Puerto Rico Caguas and San Juan. In Figure 10, we started the vaccination at the same time the epidemics start. We change the vaccinated percentage between 0%, 10%, 30% and 70%. As we increase the percentage the epidemic decreases as well as the number of deaths (not shown). We compared the vaccination percentage from 10% to 30% only in San Juan and Caguas, but when we vaccinate at least 30% of the population we reduced considerably the morbidity of the epidemic. In fact, we eradicated the disease as shown in Figure 11 in the cities vaccinated (San Juan and Caguas). We must take into consideration the people who were not vaccinated in the cities of Cayey, Ponce and Humacao so there is an epidemic in those cities.

Conclusion

To address our research questions: *What mechanisms are effective to contain a potential Avian A-H5N1 epidemic?* We discover that at least a 30% vaccination coverage reduces the morbidity of the epidemic significantly. *Which dynamics of disease spreading affect an outbreak among cities?* The connectivity of the cities, especially where the epidemic starts shape the overall morbidity of a country's epidemic. *What mechanisms are effective to contain a potential avian flu epidemic among cities?* If vaccination is not administered democratically and epidemic cannot be contained in the whole country having a lower morbidity over all but an epidemic on cities that have been neglected.

Future Work

Currently, in our research, we focus on working on avian influenza type A-H5N1 that has evolved over the years, producing more and more serious outbreaks that affect a larger number of birds. The increase in those outbreaks is due to the development of the poultry industry in recent years. The research specifically targets the spread of infectious diseases, which will be held in perspective with mathematical epidemiological models to investigate possible alternatives to mitigate the spread using measures such as treatment, immunization and as for the importance to educate the public or the community about the conduct of the epidemic in humans and how to prevent the spread of the disease. Based on our objectives and specific approaches, we want to study and focus on the behavior of the epidemic entirely by human interaction and the effect on the population. We also want to extend our meta population model on the 72 municipalities of Puerto Rico. Our future plans are to continue this research beyond the preliminary results shown in this article to find scenarios where the disease might be eradicated.

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