A Model Describing the Evolution of West Nile-Like Encephalitis in New York City

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Abstract—Encephalitis is a virus that is carried by mosquitoes and transmitted to humans and birds. Mosquitoes and birds do not show any signs of the virus and the main life cycle is carried between the two. Humans contract the virus from infected mosquitoes and the results can be deadly. In the Summer of 1999, New York City and the surrounding area were struck with a West Nile strain of encephalitis. We develop a difference equation model describing the evolution of the virus. The model incorporates a control variable that accounts for pesticide sprayed to influence mosquito populations in New York City. Using the theory of asymptotically autonomous difference equations, we arrive at parameter calculations that will eradicate the disease. © 2001 Elsevier Science Ltd. All rights reserved.

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1. INTRODUCTION

In the late Summer of 1999, New York City and its surrounding area experienced an outbreak of a West Nile-like encephalitis virus (WNLV). The last known case for the year occurred on September 17, 1999. There were a total of 50 cases, 27 confirmed, and 23 probable, which included seven deaths [1].

The virus is transmitted to humans by mosquitoes, but, cannot be transmitted back to mosquitoes. The life cycle of the virus circulates between mosquitoes and birds. Although most birds do not show signs of illness, 50–80% of crows die from the disease. Researchers used the location of dead crows that had tested positive for WNLV to define the geographic distribution of the outbreak. The affected regions included Nassau, Suffolk, Rockland, Orange, and Westchester counties in New York, Fairfield County in Connecticut and Bergen, Middlesex, Essex, and Union counties in New Jersey.

The last identified case of WNLV in humans around the New York City area coincided with the first cold snap of the year. Infected birds migrated to South America for the winter and mosquito larvae carrying the virus became dormant. New York City health officials expect to see a reoccurrence of the disease in early Spring 2000.

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Unfortunately, there is no available cure for WNLV and as a result the main focus has been to break the virus life cycle. The Giuliani administration began such attempts in September of 1999 by spraying pesticide over infected locations. Figure 1 depicts the reduction in WNLV cases prior to and after spraying began. This article develops a difference equation model that uses discontinuous periodic spraying as a control. Asymptotic results leading to the eradication of the virus are determined using the theory of asymptotically autonomous difference equations developed by Hirsch, Smith and Zhao [2].

In Section 2, we discuss details of the disease that lead to the difference equation model. Theoretical properties, such as nonnegativity of solutions, are determined in this section. Section 3 defines asymptotically autonomous systems and provides a general result. The result is used to show that under certain parameter criteria, the infection dies out. Section 4 summarizes the results and discusses how the model can be improved. Problems for future work on the subject are outlined.

2. THE MODEL AND PRELIMINARIES

The West Nile encephalitis virus has a complex life cycle that mainly circulates between mosquitoes and birds. Both, mosquitoes and most birds rarely show any adverse effects of the disease. In the New York City area, only crows showed signs of illness and approximately 50–80% of infected crows died [1]. Humans can contract the virus from mosquito bites, however, do not transfer the virus back to mosquitoes. Therefore, humans are a dead end host in the life cycle of the encephalitis virus (Figure 2). Unfortunately, humans exhibit adverse effects from the encephalitis virus. The virus leads to inflammation of the brain and usually results in flu-like symptoms such as fever, vomiting, and stiffness of the neck [3]. More severe symptoms, such as alteration of consciousness, tremor of extremities, paralysis, and death increase with age [4]. There were a total of seven deaths due to the disease in New York City and its vicinity from the dates of August 4, 1999 to October 5, 1999 [1].

Mosquitoes contract the disease by biting birds that carry the virus. The virus goes through a one week incubation period where the mosquito carries the virus but cannot transmit the infection to birds or humans. The incubation period lengthens as temperature drops. A percentage of the mosquito larvae will still carry the virus.

Birds sustain the virus for approximately one week. After a week, antibodies are generated and the bird becomes immune to the disease. Humans that do not show severe effects of the virus also become immune in a week. Birds and humans do not transfer immunity to their offspring.
There is no specific treatment available for encephalitis, and therefore, control of the disease depends on breaking the life cycle of the virus. Moreover, experts are unsure what triggers an outbreak of the virus. Researchers believe a combination of climate, birds, and mosquito dynamics and other variable factors can lead to initial outbreak of the virus. However, the exact combination of these factors remains a mystery. Since it is not known what conditions cause an occurrence of West Nile and how many summers West Nile will reappear, health officials feel that the best method of controlling West Nile encephalitis is elimination of the virus in one season by targeting mosquito populations. The Giuliani administration attempted to prevent transmission by spraying pesticide periodically over infected locations. We hope that through a mathematical model, we will be able to obtain exact parameter ranges that lead to eradication of the disease using spraying as a control.

In order to develop a sufficiently accurate yet tractable mathematical model, we need to determine a set of assumptions based on the properties of the virus and simplifications for mathematical analysis. The following list summarizes the assumptions used to establish the model:

1. The virus does not have any adverse effect on mosquitoes.
2. Most birds will recover from the virus and become immune to further infection.
3. The virus causes illness and in some cases death in human beings. Human beings carrying the virus will either recover with full immunity or die.
4. The virus is transmitted to humans by a mosquito bite.
5. The probability that an individual bird gets bit by a mosquito is equal for all birds.
6. The probability that an individual human is bit by a mosquito is equal for all humans.
7. Mosquitoes, birds, and humans grow exponentially. Although bird and human populations are typically modelled by logistic growth equations, we are choosing exponential models due to the short season of West Nile encephalitis. Over the course of a few months, birds and humans can be modelled accurately by exponential equations.
8. A percentage of the virus in mosquitoes can be transferred through reproduction. The amount of virus that is transferred by reproduction in humans and birds is negligible.
9. The pesticide kills a fixed percent of the mosquito population every week that pesticide is sprayed. The percentage of mosquitoes that are killed due to spraying is called the spray kill rate.
10. There is a delay or incubation period where mosquitoes have incurred the disease but are unable to transmit the disease to birds or humans. The delay is assumed to be one week.
11. The mosquito population does not recover from the virus.

Figure 2. Lifecycle of WNLV (reprinted from [5] with permission).
Under these assumptions we have the following state variables:

\[ TM_n = \text{total number of mosquitoes at week } n = SM_n + IM_n + IN_n, \]
\[ SM_n = \text{total number of susceptible mosquitoes at week } n, \]
\[ IM_n = \text{total number of effectively infected mosquitoes at week } n, \]
\[ IN_n = \text{total number of mosquitoes carrying the virus in incubation at week } n, \]
\[ TB_n = \text{total number of birds at week } n = SB_n + IB_n + RC_n, \]
\[ SB_n = \text{total number of susceptible birds at week } n, \]
\[ IB_n = \text{total number of infected birds at week } n, \]
\[ RC_n = \text{total number of recovered birds at week } n, \]
\[ S_n = \text{total number of susceptible humans at week } n, \]
\[ I_n = \text{total number of infected humans at week } n, \]
\[ RCH_n = \text{total number of recovered humans at week } n. \]

A table of parameters with restrictions, domain, and meaning are listed in Table 1.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Meaning</th>
<th>Domain</th>
<th>Restrictions</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \tau_M )</td>
<td>percent gain of mosquitoes due to births</td>
<td>( 0 \leq \tau_M \leq 1 )</td>
<td>none</td>
</tr>
<tr>
<td>( d_M )</td>
<td>percent loss due to natural mosquito deaths</td>
<td>( 0 \leq d_M \leq 1 )</td>
<td>none</td>
</tr>
<tr>
<td>( s )</td>
<td>percent mosquito loss due to pesticide spraying</td>
<td>( 0 \leq s \leq 1 )</td>
<td>( 1 - s - d_M \geq 0 )</td>
</tr>
<tr>
<td>( \mu )</td>
<td>percent of susceptibles gained from incubant/inf ective birth</td>
<td>( 0 \leq \mu \leq 1 )</td>
<td>( \mu + \beta = \tau_M )</td>
</tr>
<tr>
<td>( \alpha_b )</td>
<td>( \alpha_b = -\ln(1 - p_b) ) where ( p_b ) is the probability that one mosquito bites one bird</td>
<td>( \alpha_b &gt; 0 )</td>
<td>None</td>
</tr>
<tr>
<td>( \alpha_{pi} )</td>
<td>( \alpha_{pi} = -\ln(1 - p_{pi}) ) where ( p_{pi} ) is the probability that one incubant becomes effectively infected</td>
<td>( \alpha_{pi} \geq 0 )</td>
<td>None</td>
</tr>
<tr>
<td>( \beta )</td>
<td>percent of births from incubants/inf ectives that carry the virus</td>
<td>( 0 \leq \beta \leq 1 )</td>
<td>( \mu + \beta = \tau_M )</td>
</tr>
<tr>
<td>( \alpha_b )</td>
<td>percent gain of birds due to births</td>
<td>( 0 \leq \alpha_b \leq 1 )</td>
<td>none</td>
</tr>
<tr>
<td>( \delta_b )</td>
<td>percent loss of birds due to deaths</td>
<td>( 0 \leq \delta_b \leq 1 )</td>
<td>none</td>
</tr>
<tr>
<td>( \alpha_{ib} )</td>
<td>( \alpha_{ib} = -\ln(1 - p_{ib}) ) where ( p_{ib} ) is the probability that one infected bird recovered</td>
<td>( \alpha_{ib} \geq 0 )</td>
<td>none</td>
</tr>
<tr>
<td>( \alpha_h )</td>
<td>( \alpha_h = -\ln(1 - p_h) ) where ( p_h ) is the probability that one human gets bit by one mosquito</td>
<td>( \alpha_h \geq 0 )</td>
<td>none</td>
</tr>
<tr>
<td>( \tau_H )</td>
<td>growth rate in humans</td>
<td>( 0 \leq \tau_H \leq 2 )</td>
<td>none</td>
</tr>
<tr>
<td>( \alpha_{ih} )</td>
<td>( \alpha_{ih} = -\ln(1 - p_{ih}) ) where ( p_{ih} ) is the probability that one human recovers from the disease</td>
<td>( \alpha_{ih} \geq 0 )</td>
<td>none</td>
</tr>
</tbody>
</table>

The state variables and parameters can be formulated into the following difference equation model:

\[
TM_{n+1} = \underbrace{TM_n + \tau_M TM_n - d_M TM_n + ((-1)^n - 1)}_{\text{births}} \frac{s}{2} TM_n, \quad (1)
\]

\[
SM_{n+1} = e^{-a_b IB_n + RSM_n} \underbrace{SM_n + e^{-a_b IB_n} ((-1)^n - 1)}_{\text{births from infectives and incubants}} \frac{s}{2} SM_n, \quad (2)
\]

\[
+ \underbrace{\mu (IN_n + IM_n)}_{\text{death due to spraying}},
\]
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natural growth rate of newly infected susceptibles

\[ IN_{n+1} = \frac{1}{(1 - e^{-\alpha_h IB_n}) R_M S_M n + \beta(IM_n + IN_n) \text{ gain due to births}} \]

death due to spraying

\[ + (1 - e^{-\alpha_h IB_n}) SM_n + e^{-\alpha_p} IN_n \left( (-1)^n - 1 \right) \frac{s}{2} \]

natural growth of infectives

\[ + e^{-\alpha_p} R_M IN_n \]

natural loss of infectives

\[ IM_{n+1} = \left( 1 - \frac{d_M}{IM_n} \right) IM_n + \left( 1 - e^{-\alpha_p} \right) (1 - d_M + \left( (-1)^n - 1 \right)) \frac{s}{2} IN_n \text{ gain from incubants} \]

death due to spraying

\[ + \left( (-1)^n - 1 \right) \frac{s}{2} IM_n, \]

natural growth of birds

\[ TB_{n+1} = \frac{1}{R_b TB_n} \]

birth from infected/recovered birds

\[ SB_{n+1} = \frac{r_b(IB_n + RC_n)}{R_b TB_n} + e^{-\alpha_h IM_n} R_b SB_n \text{ natural growth of susceptible birds} \]

death of infected birds

\[ IB_{n+1} = e^{-\alpha_h} IB_n (1 - \delta_b) + (1 - e^{-\alpha_h} IM_n) R_b SB_n, \]

past generation minus deaths

\[ RC_{n+1} = \frac{RC_n - \delta_b RC_n}{RC_n} + (1 - e^{-\alpha_h})(1 - \delta_b) IB_n, \]

loss due to infection

\[ S_{n+1} = e^{-\alpha_h} IM_n S_n + r_M (S_n + I_n + RCH_n), \]

natural growth

\[ I_{n+1} = \left( 1 - e^{-\alpha_h} IM_n \right) S_n + e^{-\alpha_h} I_n \text{ gain due to infected susceptibles} \]

loss due to recovery

\[ \text{past generation} \]

\[ RCH_{n+1} = \frac{RHC_n}{RHC_n} + (1 - e^{-\alpha_h}) I_n \text{ gain due to newly recovered} \]

with initial conditions: \( TB_0, SM_0, IN_0, IM_0, TB_0, SB_0, IB_0, RC_0, S_0, I_0, RCH_0 \geq 0 \) and where \( R_M = 1 - d_M + r_M \) and \( R_b = 1 - \delta_b + r_b \). We now show properties of solutions to system (1)-(11).

**Theorem 2.1.** Solutions of system (1)-(11) under the parameter restrictions in Table 1 are nonnegative.
Mosquito Dynamics

Figure 3. Mosquito dynamics given by difference equation model.

**Proof.** We will show that \( \{S_{M_k}\}, \{I_{M_k}\}, \) and \( \{I_{N_k}\} \) are nonnegative. All other solutions are shown to be nonnegative in a similar manner.

Since \( S_{M0}, I_{N0}, I_{M0} \geq 0 \) we have that,

\[
S_{M1} = e^{-a_1B_0} R_M S_{M0} + e^{-a_1B_0} ((-1)^n - 1) \frac{s}{2} S_{M0} + \mu(I_{N0} + I_{M0}) \geq 0,
\]

\[
I_{M1} = (1 - d_M)I_{M0} + (1 - e^{-a_1B_0}) (1 - d_M + ((-1)^n - 1)) \frac{s}{2} I_{N0} + ((-1)^n - 1)) \frac{s}{2} I_{M0} \geq 0,
\]

\[
I_{N1} = (1 - e^{-a_1B_0}) R_M S_{M0} + \beta(I_{M0} + I_{N0})
+ ((1 - e^{-a_1B_0}) S_{M0} + e^{-a_1B_0} I_{N0} ((-1)^n - 1)) \frac{s}{2} + e^{-a_1B_0} R_M I_{N0} \geq 0.
\]

Assume that \( S_{M_k}, I_{M_k}, \) and \( I_{N_k} \) are all nonnegative. Using equations (2)–(4) we can show similar to the above calculation that \( S_{M_{k+1}}, I_{M_{k+1}}, I_{N_{k+1}} \) will all be nonnegative. Therefore, by induction, \( \{S_{M_k}\}, \{I_{M_k}\}, \) and \( \{I_{N_k}\} \) are nonnegative.

**Corollary 2.2.** For each \( n \geq 0 \), \( T_{M_n} \geq S_{M_n}, I_{N_n}, I_{M_n} \). Likewise, for each \( n \geq 0 \), \( T_{R_n} \geq S_{B_n}, I_{B_n}, R_{C_n} \).

**Proof.** The total number of mosquitoes at week \( n \) should be the sum of the total number of susceptible, incubative, and infective mosquitoes at week \( n \). Summing equations (2)–(4) yields
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Figure 4. Bird dynamics given by difference equation model.

Figure 5. Human population dynamics given by difference equation model.

equation (1). By Theorem 2.1, $S_{M_n}$, $I_{N_n}$, and $I_{M_n}$ are all nonnegative. This leads to the conclusion of the theorem.

The proof for the bird equations are similar.

Solutions to system (1)–(10) that display properties verified in Theorem 2.1 and Corollary 2.2 are depicted in Figures 3–5.

THEOREM 2.3. The solution to equation (1) is given by

$$TM_k = \begin{cases} R_M(R_M(R_M-s))^{(k-1)/2}TM_0, & \text{if } k \text{ is odd}, \\ (R_M(R_M-s))^{k/2}TM_0, & \text{if } k \text{ is even}, \end{cases}$$

where $R_M = 1 + r_M - d_M$. Similarly, the solution to equation (5) is given by

$$TB_k = (1 + r_b - \delta_b)^kTB_0.$$
Figure 6. Mosquito dynamics when $s > R_M - (1/R_M)$.

**PROOF.** Let $R_M = 1 + r_M - d_M$. Iterating equation (1) yields

$$TM_1 = R_MT_M0,$$
$$TM_2 = R_MT_M1 = R_M(R_M - s)TM_0,$$
$$TM_3 = R_MT_M2 = R^2_M(R_M - s)TM_0,$$

$$TM_k = \begin{cases} R_M(R_M(R_M - s))^{(k-1)/2}TM_0, & \text{if } k \text{ is odd,} \\ (R_M(R_M - s))^{k/2}TM_0, & \text{if } k \text{ is even.} \end{cases}$$

The general form of (12) can be verified using induction.

Since the right-hand side of equation (5) is linear and continuous, the general solution is well known to be

$$TB_k = (1 + r_0\delta_0)^kTB_0.$$  

**COROLLARY 2.4.** Suppose $s > R_M - (1/R_M)$. Then

$$\lim_{n \to \infty} TM_n = \lim_{n \to \infty} SM_n = \lim_{n \to \infty} IN_n = \lim_{n \to \infty} IM_n = 0.$$  

**PROOF.** We arrive at the result, $\lim_{n \to \infty} TM_n = 0$, by simply taking the limit of the solution, (12) provided in Theorem 2.3. To obtain the remaining limits, we apply Corollary 2.2.

Corollary 2.4 provides parameter ranges on how much pesticide should be sprayed in order to force the mosquitoes to die out, and hence, break the life cycle of the virus. Figure 6 portrays the mosquito asymptotics when $s > R_M - (1/R_M)$. The dynamics of the remaining state variables can be determined by the theory of asymptotically autonomous difference equations developed by Hirsch, Sinitui and Zhao [2].

### 3. AN APPLICATION OF ASYMPTOTICALLY AUTONOMOUS SYSTEMS

The theory of asymptotically autonomous difference equations can be used to analyze a nonautonomous system where the time varying terms converge to a limit. Specifically, a nonautonomous
difference equation in $\mathbb{R}^m$,

$$x_{n+1} = g(n, x_n)$$  \hspace{1cm} (13)

is *asymptotically autonomous* with limiting system,

$$y_{n+1} = f(y_n)$$  \hspace{1cm} (14)

if

$$\lim_{n \to \infty} g(n, x) = f(x),$$

and the convergence is uniform over compact sets in $\mathbb{R}^m$. The functions $f$ and $g$ are usually assumed to be continuous.

It may be initially unclear that equations (13) and (14) do not necessarily share asymptotic behavior. The following two counter-examples show that the systems do not always share the same dynamics.

**Example 3.1.** For $n \geq 1$ consider the discrete system,

$$x_{n+1} = x_n + \frac{1}{n}.$$  \hspace{1cm} (15)

As $n \to \infty$, we obtain the autonomous limiting equation,

$$y_{n+1} = y_n.$$  \hspace{1cm} (16)

The general solution for equation (16) is $y_n = y_1$. That is, every initial condition in $\mathbb{R}$ is a fixed point. However, the nonautonomous equation, (15), has the general solution $x_n = x_1 + 1 + 1/2 + \cdots + 1/n$, which is the initial condition plus the partial sum of the harmonic series. Therefore, every solution of equation (15) goes to infinity.

This particular example seems to indicate that the limiting system should have isolated equilibria in order to share asymptotic results with the original nonautonomous system.

The second example, which was obtained by discretizing a differential equation in [6], shows that a semi-stable fixed point can become a global repeller. This example shows that dynamics can change even if the convergence is exponential.

**Example 3.2.** Let $\alpha > 0$ and for $n \geq 0$ consider the nonautonomous discrete system,

$$x_{n+1} = x_n + |x_n| + e^{-\alpha n}.$$  \hspace{1cm} (17)

The limiting system for equation (17) is,

$$y_{n+1} = y_n + |y_n|.$$  \hspace{1cm} (18)

The general solution for equation (18) if $y_0 > 0$ is given by $y_n = 2^n y_0$. If $y_0 \leq 0$, then $y_n = 0$ for $n \geq 1$. Therefore, the solution to (18) converges to infinity if the initial condition is positive and 0 if the initial condition is nonpositive, and so the zero equilibrium is semistable.

Now for equation (17), if $x_0 > 0$, the solution $x_n \geq c 2^n$ where $c > 0$, and so $x_n$ converges to infinity. But if $x_0 \leq 0$, $x_1 = 1 > 0$. So in one iteration, $x_n$ becomes positive, and hence, also converges to infinity. Thus, zero is now a global repeller.

This particular example indicates that the forward orbits of (13) probably should be bounded for a general result on asymptotics.

Thieme has written a series of articles on the theory of asymptotically autonomous differential equations. One of the main results, which depends on the Poincaré Bendixon Theorem for differential equations, derives conditions when forward bounded orbits of the nonautonomous system converge to equilibria of the autonomous system. Hirsch, Smith and Zhao prove general results for asymptotically autonomous difference equations. They show that when $g(n, x)$ is continuous in $x$ and $g(n, x)$ converges locally uniformly to $f(x)$ as $n$ approaches infinity, then the $\omega$-limit set of a forward bounded orbit of (13) must be contained in a compact invariant set of (14). The implication of this theorem is that when the compact invariant sets of (14) are equilibria, then the forward bounded orbits of (13) must converge to an equilibrium of (14). The following theorem is not as general as the work in [2], since $f(x)$ is restricted to be a contraction. However, we lift the condition that $g(n, x)$ be continuous in $x$. 


THEOREM 3.1. Suppose that $g(n, x)$ is a sequence of functions in $n$ and that $f(x)$ is a contraction. Then all forward bounded orbits of (13) converge to the unique fixed point of (14).

PROOF. Since $f(x)$ is a contraction on $\mathbb{R}^m$, $f$ has a unique fixed point $y$. We may assume that $y = 0$ by introducing the change of variables $u_n = x_n - y$ and $v_n = y_n - y$. The new system,

$$
\begin{align*}
    u_{n+1} &= g(n, u_n) = g(n, u_n + y) - y, \\
    v_{n+1} &= f(v_n) = f(v_n + y) - y.
\end{align*}
$$

is asymptotically autonomous with contraction $\tilde{f}$ and $\tilde{f}(0) = 0$.

If we show that a bounded orbit $u_n$ converges to 0 then we have shown that $x_n \to y$ which is the desired result.

Since $f(x)$ is a contraction, we have that

$$
\|f(x)\| \leq C\|x\|,
$$

where $C < 1$. Let $\{x_n\}$ be a bounded orbit of (13) with bound $\|x_n\| \leq B$ for all $n \geq 1$. Let $\epsilon > 0$ be given. Then there is an $N \geq 1$ such that,

$$
\|x_{n+1}\| = \|g(n, x_n)\| \leq \|f(x_n)\| + \epsilon \leq C\|x_n\| + \epsilon,
$$

for all $n \geq N$. Iterating (21) yields,

$$
\begin{align*}
    \|x_{N+1}\| &\leq C\|x_N\| + \epsilon, \\
    \|x_{N+2}\| &\leq C^2\|x_N\| + C\epsilon + \epsilon, \\
    \|x_{N+3}\| &\leq C^3\|x_N\| + C^2\epsilon + C\epsilon + \epsilon, \\
    \vdots \\
    \|x_{N+k}\| &\leq C^k\|x_N\| + \epsilon + \frac{1 - C^k}{1 - C} \leq C^kB + \epsilon + \frac{1 - C^k}{1 - C}.
\end{align*}
$$

When $k \to \infty$ and $\epsilon \to 0$, we see that

$$
\lim_{n \to \infty} \|x_n\| = 0.
$$

This completes the proof.

COROLLARY 3.2. Suppose the convergence of $g(n, x)$ to $f(x)$ is uniform over $\mathbb{R}^m$. Then all forward orbits of (13) are bounded and converge to the unique fixed point of (14).

Theorem 3.1 leads to the following result.

THEOREM 3.3. Suppose that $s > R_M - (1/R_M)$. Then,

$$
\lim_{n \to \infty} I_n = \lim_{n \to \infty} IB_n = 0.
$$

We will apply Corollary 3.2 to the uncoupled linear limiting system

$$
\begin{align*}
    IB_{n+1} &= e^{-a_ih}(1 - \delta_h)IB_n, \\
    I_{n+1} &= e^{-a_ih}I_n.
\end{align*}
$$

The eigenvalues of the linear system are $\lambda_1 = e^{-a_ih}(1 - \delta_h)$ and $\lambda_2 = e^{-a_ih}$ both of which have magnitude strictly less than one. This implies that the limiting system is a contraction. Hence, Theorem 3.3 shows that if $s > R_M - (1/R_M)$, the encephalitis infection would go to zero.
4. CONCLUSION

The model presented captures the basic properties of WNLV targeting its effects on New York City. The virus is controlled through periodic pesticide spraying. Specific amounts of spraying are determined that lead to the elimination of the virus. The resulting dynamics of the mosquito, bird, and human variables are analyzed using the theory of asymptotically autonomous difference equations, thus guaranteeing a robustness of solutions.

This initial model is fairly simple and needs to be modified to encompass more complex behavior. The incubation period of the virus in the mosquito needs to be modeled as a delay term and the delay should be an inversely proportional function of temperature. Future models should include a spatial element so the geographic spread of the virus can be determined. The model should be compartmentalized so that the dynamics of the disease occur for six months in the Northeast United States and then continue for the next six months in South America. Moreover, the birth rate of the bird population should reflect activity only in the spring.

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