ABSTRACT

JOYNER, SARAH LYNN. Dynamic Models for Insect Mortality Due to Exposure to Insecticides. (Under the direction of Professor H. T. Banks).

Chemical pesticides have long been used to control insect populations on field crops, and their widespread availability and ease of manipulation present an ideal system for the exploration of the relationship between vital rates and population dynamics. Research has shown that models of population effects of insecticides are more accurate when time-varying parameters are used, but the scientific community has been slow to accept such models, which may involve complicated mathematics such as partial differential equations. Additionally, many of the pesticides now in use have both acute and sublethal effects, and these differing modes of action, along with concerns for the scientific community’s understanding of the models, motivate a new approach to mathematical modeling of the effects of insecticides on population dynamics. We first consider an ordinary differential equation model, which, while simple, provides highly accurate fits to population data, though it accounts for only one generation at a time without explicitly considering reproductive effects. An evaluation of the effects of insecticides on insect fecundity is considered separately. We then develop, implement, and analyze a coupled time-delay differential equation model, which incorporates information for multiple generations and is an intermediate model in terms of ease of understanding for non-mathematicians. Sensitivity results for the time delay kernel are also presented.
Dynamic Models for Insect Mortality Due to Exposure to Insecticides

by

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A thesis submitted to the Graduate Faculty of
North Carolina State University
in partial fulfillment of the
requirements for the Degree of
Master of Science

Computational Mathematics

Raleigh, North Carolina

2008

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Dr. Robert H. Martin

Dr. H. T. Banks
Chair of Advisory Committee
DEDICATION

To my family and to Quinn, who have faithfully supported me in every decision and encouraged me from day to day.
BIOGRAPHY

Sarah Lynn Joyner was raised in Raleigh, North Carolina and graduated from Sanderson High School in 2002, whereupon she attended Meredith College, graduating summa cum laude in May 2006 with a bachelor of science in mathematics and a bachelor of arts in chemistry. Her graduate school experience at North Carolina State University began in May 2006 with a summer research assistantship through the Center for Research in Scientific Computation, under the direction of Dr. H. Thomas Banks. In May 2008, she graduated with a master of science in computational mathematics, and in August 2008, she will begin working in Charlotte for Accenture as a consulting analyst.
ACKNOWLEDGMENTS

First of all, I would like to extend my heartfelt thanks and appreciation to my advisor, Dr. H.T. Banks, who has served as a great mentor and given me invaluable guidance and encouragement throughout my time at N.C. State. His willingness to teach me and his patience as I learned, along with his overflowing love of doing mathematics, have been instrumental in my mathematical development. I would also like to thank Dr. Bob Martin and Dr. Pierre Gremaud for their support on my committee as well as all my professors and fellow graduate students for their roles in my education.

Thank you to John Banks, John Stark, and Lara Dick for their excellent work on this problem prior to mine, and whose input has been a great help in my understanding of the data and ecology. I also greatly appreciate the help of Hoan Nguyen and Sava Dediu, who mentored me as post-doctoral students in the Center for Research in Scientific Computation. I must also thank the CRSC, the Lord Corporation, and the North Carolina State Alumni Association, who have supported me with fellowships.

I am particularly grateful to my family, who have lovingly supported each decision I made. Most of all, I thank my fiance and best friend, Quinn, who has patiently borne with me through this entire process, encouraging and advising me, and demonstrating his love in innumerable ways. I am eagerly anticipating the life we will share together.
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Chapter 1

Introduction

Mathematical models of population dynamics play an important role in understanding anthropogenic disturbances such as the use of chemical pesticides. Because pesticides are widely available and easy to manipulate in both laboratory and field environments, they provide an ideal system for the exploration of how altering vital rates affects population dynamics. The effects of the pesticides are characterized by population-specific parameters that change with time and thus present a convenient system for examining the link between vital rates and population processes. Reproductive and mortality rates, which often vary with time, are two such parameters that are easily manipulated by pesticide exposure. In these cases, constant-parameter models often fail to adequately capture the dynamics of the insect populations; however, time-varying parameter models may provide a much better fit to population data. Differential equations models, which can represent vital rates in continuous time, are especially well-suited to modeling population dynamics with time-varying parameters [2]. We present here an analysis of insect-pesticide population dynamics that explores the utility of employing differential equation models with time-varying vital rates as a means of predicting population outcomes.

1.1 Insect Population

The pea aphid, *Acyrthosiphon pisum*, is a long-legged, deep-green color insect with red eyes, measuring approximately one-sixth of an inch long [3, 4]. The species is found throughout the United States and Canada and infests various types of peas, clover, alfalfa, and some leguminous weeds [3]. The aphids feed on host plants by extracting sap from the
leaves and stems, and in larger infestations, “honey dew,” a shiny, sticky material produced by the aphids, may be visible on the plants along with white-colored skins cast on the ground during molting [3, 4]. Pea aphids transmit certain viral diseases, including the pea enation mosaic and the yellow bean mosaic viruses [3].

Male aphids appear only in the fall, while females are present throughout the year [4]. Adult female pea aphids produce from six to eight offspring per day, totaling around 100 per individual in a lifetime [3]. The neonate aphids require four molts to reach the adult stage, and maturation to adulthood occurs between five and seven days of age [3, 1]. While the majority of aphids are wingless, overcrowding or climate changes can result in the birth of more winged young, who migrate to nearby fields to form new colonies [4].

1.2 Experimental Procedure and Data Description

In 1995, Stark and Wennergren [1] conducted a study on the sublethal effects of the neem insecticide Margosan-O (MO) on pea aphids (A. pisum). Broad bean plants which had grown to a height of 25 cm were thinned to five per pot, and the upper and under surfaces of the leaves of the plants in each pot were sprayed with water (control) or one of six concentrations of MO. Effects of MO on both neonate and adult pea aphids were studied in two separate procedures.

Both procedures began with two clip cages, each containing one young apterous adult female aphid, being attached to the underside of the leaves of each of the five plants in twenty-one pots for a total of ten aphids per pot. In the study of neonate exposure, three pots had been treated per concentration of MO or water, for a total of 30 aphids per concentration. Twenty-four hours after introduction of the adult aphids, all aphids except one first instar were removed from each clip cage in order to ensure that only newborn aphids, exposed at birth to the treatments, would be observed. Mortality and reproduction data were taken daily throughout the lifespan of these newborn aphids. Time measurements were recorded in terms of age classes whose duration was one day, beginning with age class zero. Mortality data was recorded as the number of the original 30 aphids alive each day, and reproduction data was recorded as the number of new aphids born per day. No data was recorded for these newborn aphids other than the number born; after being counted, these first instars were removed from the clip cages, leaving only the original population of
aphids that had been exposed to the treatments.

In the study of adult exposure, all of the plants in each of the twenty-one pots were treated with water, and twenty-four hours after introduction of the adult aphids, all aphids except one first instar were removed from the cages. One day after these aphids had molted to the adult stage (i.e., aphids were now 7 days old), a separate set of broad bean plants was treated with either water or one of the six concentrations of MO as described above. After the leaves had dried, the aphids were transferred to the treated plants and contained in new clip cages, with a total of 30 aphids per concentration. Mortality and reproduction were recorded daily throughout the lifespan of these aphids in the same manner as for the neonates.

The neonate data sets for both mortality and reproduction at each insecticide exposure level are plotted together in Figure 1.1, and the same plots for the adult data sets are shown in Figure 1.2. For a more detailed explanation of the experimental procedure, see [1].

1.3 Necessity of Time-Varying Parameters

The use of time-dependent parameters with this data was first considered in the context of a widely used partial differential equation model [5, 6]. The Sinko-Streifer [7] or McKendrick-von Forester [8] model for age- or size-structured population dynamics was used
Figure 1.2: Adult mortality and reproduction data for experiment described in [1].

as a continuous-time approximation to the mortality data. This model can be expressed as

$$\frac{\partial u}{\partial t} + \frac{\partial}{\partial x} gu = -\mu u$$  \hspace{1cm} (1.1)$$

where $x \in (x_0, x_{\text{max}})$ and $t \in (0, T)$ with the boundary condition

$$R(t) = g(x_0, t)u(x_0, t) = \int_{x_0}^{x_{\text{max}}} k(x, t)u(x, t)dx$$

and initial condition

$$u(x, 0) = \phi(x).$$

Here, $u(x, t)$ represents the population density at age $x$ and time $t$, $g(x, t)$ is the individual growth rate, $\phi(x)$ is the initial age distribution, $k$ represents the fecundity rate, and $\mu$ is the mortality rate.

The mortality rate $\mu$ was first considered as a constant and was estimated using an ordinary least squares technique to find the parameter value that optimized the fit between the data and the model. Next, the mortality rate was taken to be time-dependent and was estimated as a piecewise constant function $\mu = \mu(t) \approx \mu^M(t) = \sum_{j=1}^{M} a_j \chi_j(t)$. The ordinary least squares (OLS) technique was applied in this case to calculate a time-varying mortality rate across a given number of nodal points, and a $\chi^2$ statistical test was performed to determine whether increasing the number of nodes significantly improved the fit of the model to the data [9]. It was determined that using any more than $M = 5$ nodal points did not result in a statistically significant improvement in the model fit when using model (1.1) with piecewise constant time varying $\mu$. 
Finally, a comparison was made between the fit to the data of the Sinko-Streifer models with constant mortality and time-varying mortality, respectively. The time-varying mortality model generated a good fit to the data, capturing the aphid decline over the entire duration of the experiment, whereas the constant mortality model overestimated the population decline across the entire range of pesticide concentrations, particularly within the first 15 days. This comparison reveals the potential merits of using time-varying parameters in modeling population trajectories of organisms subjected to pesticides: the constant-parameter version of the model requires that the mortality rate be fixed at the outset, while the time-varying parameter model allows for changing mortality rates due to the cumulative effects of the toxicant.
Chapter 2

Ordinary Differential Equation Model

Though the Sinko-Streifer model with time-varying parameters provided a highly accurate fit to the data, it is a partial differential equation model and thus may prove difficult for those without a strong theoretical mathematical background to understand. This desire for a wider audience to understand and use mathematical models for insect population dynamics, along with information about the chemical properties and physical effects of the insecticide, motivates another approach to modeling the action of the insecticide. In this section, we summarize the results previously presented in [10], where we propose an ordinary differential equation model to describe population processes and analyze its fit to the same experimental data recorded in [1].

2.1 Model Development

The application of \( MO \) has both acute and sublethal toxic effects on aphids. First, Neem oil combined with the surfactant that facilitates adherence of the insecticide to the plants’ leaves can coat an aphid’s exoskeleton upon contact, causing the aphid to suffocate immediately. Second, \( MO \) inhibits insect growth by acting as a molt hormone regulator. Juvenile insects exposed to the insecticide may fail to molt or may molt improperly, resulting in delayed death. Finally, exposure of adult insects to \( MO \) can reduce egg production, a sublethal effect on population growth over time.

The Sinko-Streifer model with time-varying mortality provided a highly accurate
fit to the data due to its ability to capture changing mortality rates over time. However, the aphid-pesticide system can be modeled with a simple ordinary differential equation if the effects of the pesticide are attributed to accumulation through time rather than as specific to each age class.

To reflect the modes of action of the pesticide, we refine the time-varying mortality used in the Sinko-Streifer model by partitioning it into three separate mortality rates: mortality due to the direct or immediate action of the pesticide, denoted $p_{\text{dir}}$, and the delayed, cumulative effect of the insecticide, $p_{\text{delay}}$. Finally, the function $\mu(t)$ models the background or natural mortality experienced by all aphids. The ordinary differential equation that models the population dynamics is a function of these three types of mortality and can be written

$$\dot{x}(t) = -\left[p_{\text{dir}}(t) + p_{\text{delay}}(t) + \mu(t)\right]x(t),$$

$$x(0) = x_0. \quad (2.1)$$

The reproductive effects of the insecticide are not explicitly considered in this model due to the nature of the data available. Since all new first instars were removed from the cages daily without any record of their population dynamics being kept, the effects of $MO$ on population growth over several generations cannot be seen and hence are not modeled directly in this presentation.

### 2.2 Numerical Techniques

To numerically solve (2.1), each mortality rate is taken as a function of time measured in days, and these three functions are approximated using piecewise linear splines. Each rate function has the form

$$q(t) = \sum_{j=0}^{M} \alpha_j \ell_j(t), \quad j = 0, 1, \ldots, M, \quad (2.2)$$

where $n$ is the number of intervals into which the time span is partitioned, the $\alpha_j$ are constants (nodal values), and $\ell_j$ is the linear spline function defined as

$$\ell_j(t) = \begin{cases} 
\frac{t - t_{j-1}}{t_j - t_{j-1}} & \text{if } t \in [t_{j-1}, t_j], \\
\frac{t_{j+1} - t}{t_{j+1} - t_j} & \text{if } t \in [t_j, t_{j+1}], \\
0 & \text{otherwise.}
\end{cases} \quad (2.3)$$
The support for $\mu(t)$ is taken as the entire time span $[0, T]$, while the supports for $p_{\text{dir}}(t)$ and $p_{\text{delay}}(t)$ are restricted to smaller intervals. These intervals differ between the neonates and the adults due to the differing exposure times and the molt regulatory action of the Neem insecticide. We take uniform 1-day discretizations $\{t_j\}$ on each support region for the insecticide-induced mortalities so that $[t_{j-1}, t_j]$ corresponds to one day for all piecewise linear time dependent parameters. The discretization for $\mu(t)$ is taken at 16 uniform intervals rather than daily to reduce the risk of over-parameterization.

Neonate aphids become adults between ages 5 and 7 days, and they undergo several molts until they have reached this stage. Molting no longer occurs for adult aphids, so the only direct effect is suffocation, which takes place over a shorter time period than does the molting. Additionally, the neonates are exposed to the insecticide at time $t = 0$, while the adults are exposed at time $t = 7$. Thus we take the support for $p_{\text{dir}}^N(t)$, the immediate mortality for neonates, to be the interval $[0, 6]$, while the support of $p_{\text{dir}}^A(t)$ for immediate adult mortality is taken to be the interval $[7, 9]$. In each case the rate function is constrained so that the value at the final endpoint is zero. Thus there are a total of 7 nodes and hence 6 nontrivial coefficients $\alpha_j^N$ for the neonates and 3 nodes with 2 corresponding nontrivial coefficients $\alpha_j^A$ for the adults, with $\alpha_7^N = \alpha_3^A = 0$.

Support for $p_{\text{delay}}(t)$ for both neonates and adults begins at the last node of the support for $p_{\text{dir}}(t)$ and continues through the end of the time span for the data, again with the intervals partitioned such that nodes are taken daily. Here no constraints are imposed upon the spline coefficient values at the ends of the interval.

### 2.3 Parameter Estimation

Due to the nature of the data, we must estimate a separate set of parameters $\theta(\alpha, \gamma)$ for each insecticide exposure level for both neonate and adult aphids, where $\alpha$ is the age class and $\gamma$ is the exposure level. The parameter estimation is performed using the ordinary least squares (OLS) inverse problem technique, where values of $\theta$ are sought to provide the most accurate fit of the population dynamics to the data. To find these parameters, we minimize the least squares cost functional

$$J(\theta) = \sum_{j=0}^{n} |x(t_j; \theta) - d_j|^2,$$  \hspace{1cm} (2.4)
where \( x(t_j; \theta) \) is the solution of the model ordinary differential equation (2.1) at time \( t_j \) for a given set of parameters \( \theta = (\mu, p_{\text{dir}}, p_{\text{delay}}) \), and \( d_j \) is the experimental data at time \( t_j \). We choose the MATLAB optimization routine \textit{fmincon} to find the optimal parameters \( \hat{\theta} \) which minimize (2.4) and use the medium-scale option, which implements a constrained optimization method using a line search. The constraints on the nodal values for each function are taken to be zero as a lower bound and five as an upper bound.

### 2.3.1 Estimation of Natural Mortality Parameters

The natural or background mortality \( \mu(t) \) for aphids in the absence of insecticide is estimated using the OLS technique for (2.1) with \( p_{\text{dir}}(t) = p_{\text{delay}}(t) \equiv 0 \), which simplifies the model to

\[
\dot{x}(t) = -\mu(t)x(t)
\]

(2.5)
in the control situation. Since separate control data is given for each of the two age groups, a set of nodal values \( \{\beta_i\}_{i=0}^{16} \) is estimated for each natural mortality function \( \mu^N(t) \) and \( \mu^A(t) \) for neonates and adults, respectively. The results of these calculations (model solutions and parameters functions) using the optimized nodal values shown in Table 2.1 are plotted in Figure 2.1. The small residuals seen in Table 2.2 confirm the qualitative observation that the model provides a highly accurate fit to the data. It can also be observed that the plot of the estimated natural mortality \( \mu(t) \) behaves as expected for both age groups: the death rate is low throughout the time span until the last three to five days, when the rate increases sharply, indicating the natural limit of the aphids’ lifespan is approaching.

<table>
<thead>
<tr>
<th>Parameter Function</th>
<th>Interval of Support</th>
<th># of Equally Spaced Nodes</th>
<th>Nodal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \mu^N(t) )</td>
<td>[0, 31]</td>
<td>17</td>
<td>(0, 0, 0, 0, 0, 0, 0, 0.0263, 0.0091, \ldots 0.0, 0, 0.0273, 0, 0, 0.3577, 0.4491)</td>
</tr>
<tr>
<td>( \mu^A(t) )</td>
<td>[0, 31]</td>
<td>17</td>
<td>(0, 0, 0, 0, 0, 0.0017, 0.0034, 0.0339, \ldots 0.0724, 0.0143, 0, 0.0173, 0.0189, \ldots 0.0517, 0.1269, 0.6165, 0.4598)</td>
</tr>
</tbody>
</table>
2.3.2 Estimation of Parameters for Insecticide-Induced Mortality

Using the estimated parameters for $\mu(t)$ in (2.1), we again employ the OLS technique to find parameters for $p_{\text{dir}}(t)$ and $p_{\text{delay}}(t)$ which provide the most accurate fit to the population dynamics for populations subjected to insecticide exposure. We estimate parameters for each level of exposure for both neonates and adults, and these results are displayed in Tables 2.3 and 2.4 and in Figures 2.2-2.5, with the minimized residuals summarized in Table 2.2.

The different modes of action of the insecticide are shown clearly in the results of the parameter estimation. For example, Figures 2.2(b) and (d) and 2.3(b),(d), and (f) depict $p_{\text{dir}}^N(t)$ increasing with insecticide concentration, while Figures 2.4 and 2.5 reveal that $p_{\text{dir}}^A(t)$ is nearly negligible. These results are consistent with the fact that the growth hormone regulatory effects play an important role in the action of MO: neonates are affected strongly immediately after exposure, while adults have ceased molting and thus do not experience the
Table 2.2: Summary of least squares residuals for ODE model fit to pea aphid data for exposure to $MO$.

<table>
<thead>
<tr>
<th>Data Type</th>
<th>Exposure</th>
<th>Residual</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonate</td>
<td>Control</td>
<td>2.8317</td>
</tr>
<tr>
<td></td>
<td>10 ppm</td>
<td>5.9589</td>
</tr>
<tr>
<td></td>
<td>20 ppm</td>
<td>3.6614</td>
</tr>
<tr>
<td></td>
<td>40 ppm</td>
<td>2.9377</td>
</tr>
<tr>
<td></td>
<td>60 ppm</td>
<td>4.7432</td>
</tr>
<tr>
<td></td>
<td>80 ppm</td>
<td>$4.8990 \times 10^{-8}$</td>
</tr>
<tr>
<td>Adult</td>
<td>Control</td>
<td>6.7592</td>
</tr>
<tr>
<td></td>
<td>10 ppm</td>
<td>17.6949</td>
</tr>
<tr>
<td></td>
<td>20 ppm</td>
<td>7.7444</td>
</tr>
<tr>
<td></td>
<td>40 ppm</td>
<td>13.4492</td>
</tr>
<tr>
<td></td>
<td>60 ppm</td>
<td>2.6536</td>
</tr>
<tr>
<td></td>
<td>80 ppm</td>
<td>8.0715</td>
</tr>
<tr>
<td></td>
<td>100 ppm</td>
<td>1.1594</td>
</tr>
</tbody>
</table>

same effects. For the adult populations, $p_{\text{delay}}^A(t)$ plays a more fundamental role throughout the time span as the cumulative effects of the pesticide build, while $p_{\text{delay}}^N(t)$ is secondary to the direct effect of the insecticide. In both neonates and adults, population density at a given time declines more and more sharply as insecticide concentration increases, with the neonates eliminated almost immediately at the highest concentration.
Table 2.3: Nodal values for parameter functions $p_{dir}(t)$ for each age class and exposure level.

<table>
<thead>
<tr>
<th>Age Class</th>
<th>Exposure Level</th>
<th>Support</th>
<th># of Nodes</th>
<th>Nodal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonate</td>
<td>10 ppm</td>
<td>[0, 6]</td>
<td>7</td>
<td>(0.0005, 0.0002, 0.0182, ..., 0.0959, 0, 0, 0)</td>
</tr>
<tr>
<td></td>
<td>20 ppm</td>
<td>[0, 6]</td>
<td>7</td>
<td>(0.0045, 0.0150, 0.0375, ..., 0.0140, 0.0128, 0.0526, 0)</td>
</tr>
<tr>
<td></td>
<td>40 ppm</td>
<td>[0, 6]</td>
<td>7</td>
<td>(0.0076, 0.0111, 0.0349, ..., 0.1806, 0.2469, 0.2163, 0)</td>
</tr>
<tr>
<td></td>
<td>60 ppm</td>
<td>[0, 6]</td>
<td>7</td>
<td>(0.0078, 0.1447, 0.4615, ..., 0.6717, 0.1142, 0)</td>
</tr>
<tr>
<td></td>
<td>80 ppm</td>
<td>[0, 6]</td>
<td>7</td>
<td>(0.6673, 0.8570, 0.8369, ..., 1.3821, 0.0011, 0, 0)</td>
</tr>
<tr>
<td></td>
<td>100 ppm</td>
<td>[7, 9]</td>
<td>3</td>
<td>(0.0002, 0.0305, 0)</td>
</tr>
<tr>
<td>Adult</td>
<td>10 ppm</td>
<td>[7, 9]</td>
<td>3</td>
<td>(0.0010, 0.0343, 0)</td>
</tr>
<tr>
<td></td>
<td>20 ppm</td>
<td>[7, 9]</td>
<td>3</td>
<td>(0.0095, 4.9528 $\times$ 10^{-6}, 0)</td>
</tr>
<tr>
<td></td>
<td>40 ppm</td>
<td>[7, 9]</td>
<td>3</td>
<td>(0.0137, 0, 0)</td>
</tr>
<tr>
<td></td>
<td>60 ppm</td>
<td>[7, 9]</td>
<td>3</td>
<td>(0.0171, 0.0130, 0)</td>
</tr>
<tr>
<td></td>
<td>80 ppm</td>
<td>[7, 9]</td>
<td>3</td>
<td>(0.0386, 0.0042, 0)</td>
</tr>
</tbody>
</table>
Table 2.4: Nodal values for parameter functions $p_{\text{delay}}(t)$ for each age class and exposure level.

<table>
<thead>
<tr>
<th>Age Class</th>
<th>Exposure Level</th>
<th>Support</th>
<th># of Nodes</th>
<th>Nodal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonate</td>
<td>10 ppm</td>
<td>[6, 29]</td>
<td>24</td>
<td>(0.0016, 0, 0.0249, 0, 0, 0, 0.00149, ..., 0.0512, 0.0902, 0.1133, 0.0873, 0.1021, ..., 0.1737, 0.0969, 0.0514, 0.1617, 0, 0, 0.8573)</td>
</tr>
<tr>
<td></td>
<td>20 ppm</td>
<td>[6, 25]</td>
<td>20</td>
<td>(0.0146, 0.0912, 0.0749, 0, 0.0069, 0.0390, ..., 0, 0, 0, 0, 0.0565, 0.2059, 0.0721, 0.2006, ..., 0.1338, 0, 0.3784, 0.9415, 1.3016)</td>
</tr>
<tr>
<td></td>
<td>40 ppm</td>
<td>[6, 22]</td>
<td>17</td>
<td>(0.2414, 0.2218, 0.1415, 0, 0.0024, 0.0697, ..., 0.0938, 0, 0, 0, 0, 0, 0, 0.0943, 0.7155, 0)</td>
</tr>
<tr>
<td></td>
<td>60 ppm</td>
<td>[6, 20]</td>
<td>15</td>
<td>(0, 0.3987, 0.3787, 0.0498, 0, 0, 0, 0, 0, 0, ..., 0, 0, 0.1238, 1.9055)</td>
</tr>
<tr>
<td></td>
<td>80 ppm</td>
<td>[6, 8]</td>
<td>3</td>
<td>(5.8600 × 10⁻⁹, 3.1535 × 10⁻⁸, 1.0847 × 10⁻⁸)</td>
</tr>
<tr>
<td>Adult</td>
<td>10 ppm</td>
<td>[9, 31]</td>
<td>23</td>
<td>(0.0006, 0.0852, 0, 0, 0, 0.0003, 0.0002, 0, ..., 0.0005, 0, 0, 0.0058, 0, 0.0107, 0.1879, 0.0741, ..., 0.0406, 0.0176, 0.0509, 0, 0.0017, 2.2888, 3.6428)</td>
</tr>
<tr>
<td></td>
<td>20 ppm</td>
<td>[9, 28]</td>
<td>20</td>
<td>(0.0232, 0.0636, 0.0235, 0.0105, 0.0044, ..., 0.0262, 0.0810, 0.1560, 0.0451, 0.0150, 0, ..., 0, 0.0209, 0.0558, 0.1593, 0.3629, 0.0799, ..., 0.0073, 0.2041, 2.3725)</td>
</tr>
<tr>
<td></td>
<td>40 ppm</td>
<td>[9, 29]</td>
<td>21</td>
<td>(0.0509, 0.0437, 0.0196, 0.0013, 0.1280, ..., 0.1258, 0.1834, 0.0102, 0.0199, 0.0446, ..., 0.0649, 0.0713, 0.0867, 0.1574, 0.2042, ..., 0.1364, 0.0665, 0.2531, 0.4369, 0.2185, 0.0098)</td>
</tr>
<tr>
<td></td>
<td>60 ppm</td>
<td>[9, 26]</td>
<td>18</td>
<td>(0.0551, 0.0501, 0.0480, 0.0897, 0.1340, ..., 0.2031, 0.0804, 0.0326, 0.0619, 0.0027, ..., 0, 0.0077, 0.1632, 0.2171, 0.0484, 0.1654, ..., 0.6556, 1.7188)</td>
</tr>
<tr>
<td></td>
<td>80 ppm</td>
<td>[9, 26]</td>
<td>18</td>
<td>(0.2746, 0.1772, 0.0625, 0.0129, 0.0236, ..., 0.0846, 0.2047, 0.0378, 0.0210, 0.0042, ..., 0.0451, 0.1976, 0.1794, 0.2328, 0.1943, ..., 0.7007, 0.7516, 0.3234)</td>
</tr>
<tr>
<td></td>
<td>100 ppm</td>
<td>[9, 22]</td>
<td>14</td>
<td>(0.0751, 0.0313, 0.2060, 0.0939, 0.0461, ..., 0.1892, 0.4723, 0.2884, 0, 0, 0, 0.0001, ..., 0.0003, 2.1104)</td>
</tr>
</tbody>
</table>
Figure 2.2: Model fit to neonate (a) 10 ppm and (c) 20 ppm neem exposure data and corresponding linear spline approximations (b) and (d) to $p_{dir}(t)$ and $p_{delay}(t)$ along with $\mu_N(t)$. 
Figure 2.3: Model fit to neonate (a) 40 ppm, (c) 60 ppm, and (e) 80 ppm neem exposure data and corresponding linear spline approximations (b), (d), and (f) to $p_{dir}(t)$ and $p_{delay}(t)$ along with $\mu^N(t)$. 
Figure 2.4: Model fit to adult (a) 10 ppm, (c) 20 ppm, and (d) 40 ppm neem exposure data and corresponding linear spline approximations (b), (d), and (f) to $p_{\text{dir}}(t)$ and $p_{\text{delay}}(t)$ along with $\mu^A(t)$. 
Figure 2.5: Model fit to adult (a) 60 ppm, (c) 80 ppm, and (e) 100 ppm neem exposure data and corresponding linear spline approximations (b), (d), and (f) to $p_{\text{dir}}(t)$ and $p_{\text{delay}}(t)$ along with $\mu_A(t)$. 
2.4 Application to Other Insect/Pesticide Systems

To examine the usefulness of the ODE model, another set of data for the same species was considered. Walthall and Stark examined the effects of the pesticide imidacloprid on pea aphids [11]. In this experiment, the aphids were exposed to the insecticide imidacloprid in a similar manner as in the Neem experiment. The mortality and reproduction data for all exposure levels are shown in Figure 2.6. Walthall and Stark observed that the aphids experienced an acute lethal response only: no sublethal effects were noted. For a more detailed explanation of the experimental procedure, see [11].

The model was adapted to correspond to these physical observations by combining the two mortality rates \( p_{\text{dir}}(t) \) and \( p_{\text{delay}}(t) \), which account for different sublethal modes of action of the insecticide, into the one rate \( p(t) \). Thus the model is

\[
\dot{x}(t) = - [p(t) + \mu(t)] x(t),
\]

\[
x(0) = x_0.
\]  

(2.6)

As before, values for the background mortality \( \mu(t) \) were found by fitting the model to the control data, and the optimal \( \mu \) was used in the model to estimate \( p \) for each exposure level.

As seen in Figures 2.7-2.9, this version of the model provides a good fit to the data. Since there are no sublethal effects, \( p \) takes nonzero values that increase with the exposure level near the beginning of the time interval and values closer to zero with increasing time. These mortality rates are graphed together in Figure 2.10.
Figure 2.6: (a) Mortality for pea aphids exposed to imidacloprid; (b) Daily reproduction of pea aphids exposed to imidacloprid.

Figure 2.7: (a) Model fit to control data; (b) Natural (background) mortality \( \mu(t) \).
Figure 2.8: Model fit to exposure data and mortality rates $p(t)$ and $\mu(t)$: (a,b) 0.07 mg/L, (c,d) 0.10 mg/L, (e,f) 0.15 mg/L, (g,h) 0.25 mg/L.
Figure 2.9: Model fit to exposure data and mortality rates $p(t)$ and $\mu(t)$: (a,b) 0.40 mg/L, (c,d) 0.60 mg/L, (e,f) 1.00 mg/L, (g,h) 1.25 mg/L.
Figure 2.10: Mortality functions $p(t)$ for all exposure levels.
Chapter 3

Effect of Insecticide on Aphid Fecundity

The neem insecticide also affects the fecundity of the aphids. A decrease in fecundity is expected as the concentration of the insecticide increases, and this is indeed the case, as shown in the data for the daily births, plotted in Figures 1.1(b) and 1.2(b) of Chapter 1.

The total number of births per day, $R(t)$, can be modeled as

$$R(t) = r(t)x(t),$$  \hspace{1cm} (3.1)

where $x(t)$ is the number of the original 30 aphids alive at time $t$, and $r(t)$ is the individual birth rate (number of young per aphid per day).

We are given reproduction data for each age class at each exposure level $\gamma$, and if we let this data $R^d_j(\gamma) \approx R(t_j; \gamma)$, we can compute the birth rate $r^d_j(\gamma)$ at each time point $t_j$ by

$$r^d_j(\gamma) = \frac{R^d_j(\gamma)}{d_j(\gamma)};$$  \hspace{1cm} (3.2)

where $d_j(\gamma)$ is population data at time $t_j$ for exposure level $\gamma$. To determine the birth rate corresponding to the model solution $x(t; \hat{\theta}_\gamma)$, we again use the reproduction data $R^d_j(\gamma)$ to compute

$$r(t_j; \hat{\theta}_\gamma) = \frac{R^d_j}{x(t_j; \hat{\theta}_\gamma)};$$  \hspace{1cm} (3.3)

where $\hat{\theta}_\gamma$ is the optimal set of parameters determined for the insecticide exposure level $\gamma$ using (2.4). That is, $\hat{\theta}_\gamma$ is the set of parameters obtained when fitting the population model.
to the data, which does not involve fecundity. The resulting individual birth rate models $r(t_j; \hat{\theta}_\gamma)$ and data $r_{jd}(\gamma)$ are plotted in Figures 3.1 (control), 3.2 (neonate exposures), and 3.3 (adult exposures).

The corresponding fecundity residual using these optimal parameters estimated with (2.4) for the population data is

$$RSS(\hat{\theta}_\gamma) = \sum_{j=1}^{n} \left| r(t_j; \hat{\theta}_\gamma) - r_{jd}(\gamma) \right|^2,$$

(3.4)

provides a quantitative comparison of the two rates. These values are given in Table 3.1. Both qualitatively and quantitatively, the model output for population accurately predicts the birth rate.
Figure 3.2: Birth rate comparisons for neonate exposure: (a) 10 ppm, (b) 20 ppm, (c) 40 ppm, (d) 60 ppm, and (e) 80 ppm.
Figure 3.3: Birth rate comparisons for adult exposure: (a) 10 ppm, (b) 20 ppm, (c) 40 ppm, (d) 60 ppm, (e) 80 ppm, and (f) 100 ppm.
Table 3.1: Fecundity residuals for individual birth rate computed using data and model output for pea aphids exposed to MO.

<table>
<thead>
<tr>
<th>Data Type</th>
<th>Exposure</th>
<th>Residual $RSS(\theta_\gamma)$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonate</td>
<td>Control</td>
<td>$1.3440 \times 10^{-2}$</td>
</tr>
<tr>
<td></td>
<td>10 ppm</td>
<td>$1.8828 \times 10^{-2}$</td>
</tr>
<tr>
<td></td>
<td>20 ppm</td>
<td>$1.1114 \times 10^{-2}$</td>
</tr>
<tr>
<td></td>
<td>40 ppm</td>
<td>$1.0508 \times 10^{-2}$</td>
</tr>
<tr>
<td></td>
<td>60 ppm</td>
<td>$5.4160 \times 10^{-1}$</td>
</tr>
<tr>
<td></td>
<td>80 ppm</td>
<td>$1.2598 \times 10^{-10}$</td>
</tr>
<tr>
<td></td>
<td>100 ppm</td>
<td>$1.7495 \times 10^{-2}$</td>
</tr>
<tr>
<td>Adult</td>
<td>Control</td>
<td>$1.7018 \times 10^{-1}$</td>
</tr>
<tr>
<td></td>
<td>10 ppm</td>
<td>$2.7895 \times 10^{-1}$</td>
</tr>
<tr>
<td></td>
<td>20 ppm</td>
<td>$1.6969 \times 10^{-1}$</td>
</tr>
<tr>
<td></td>
<td>40 ppm</td>
<td>$4.7171 \times 10^{-1}$</td>
</tr>
<tr>
<td></td>
<td>60 ppm</td>
<td>$7.7703 \times 10^{-2}$</td>
</tr>
<tr>
<td></td>
<td>80 ppm</td>
<td>$1.2038 \times 10^{-1}$</td>
</tr>
<tr>
<td></td>
<td>100 ppm</td>
<td>$1.7495 \times 10^{-2}$</td>
</tr>
</tbody>
</table>
Chapter 4

Delay Differential Equation Model

Though the ordinary differential equation model successfully captures the changing population dynamics of aphids exposed to insecticide, it is severely limited due to the fact that it does not incorporate reproductive effects. Ideally, a mathematical model would consider these effects, and we would need to account for multiple generations and their interconnectedness. This consideration indicates the need for a coupled system of equations describing the two separate age classes. Additionally, due to individual differences within the insect population, it is biologically unrealistic to assume that all neonate aphids born on the same day complete molting and reach the adult age class at the same time. In fact, the age at which the insects reach adulthood varies from as few as five to as many as seven days. Hence we must include a term in our model to account for this variability, leading us to develop a coupled delay differential equation model for the insect population dynamics.

4.1 Model Development

In this section we examine the delay between birth and adulthood for neonate pea aphids and derive a first mathematical model that treats this delay as a random variable. Let $A(t)$ and $N(t)$ denote the number of adults and neonates, respectively, in the population at time $t$. We suppose that the time delay varies across the insect population according to a probability distribution $P(\tau)$ for $\tau \in [0, \infty)$ with corresponding density $m(\tau) = \frac{dP(\tau)}{d\tau}$. Next, we lump the mortality due to insecticide into one time-varying parameter $p_A(t)$ for the adults, $p_N(t)$ for the neonates, and denote by $d_A(t)$ and $d_N(t)$ the time-varying background or natural mortalities for adults and neonates respectively. We let $b(t)$ be the time-varying
rate at which neonates are born into the population.

We have that $m(\tau), \tau < 0$, is the probability per unit time that a neonate who has been in the population $-\tau$ time units becomes an adult. Then the rate at which such neonates become adults is $N(t+\tau)m(\tau)$. Summing over all such $\tau$’s, we obtain that the rate at which neonates become adults is $\int_{0}^{\infty} N(t+\tau)m(\tau)d\tau$. Applying the biological knowledge that the delay occurs between five and seven days, we obtain the system

$$\dot{A}(t) = \int_{-7}^{-5} N(t+\tau)m(\tau)d\tau - (d_A(t) + p_A(t))A(t)$$

$$\dot{N}(t) = b(t)A(t) - (d_N(t) + p_N(t))N(t) - \int_{-7}^{-5} N(t+\tau)m(\tau)d\tau$$

$$A(\theta) = \Phi(\theta), \quad N(\theta) = \Psi(\theta), \quad \theta \in [-7, 0)$$

$$A(0) = A^0, \quad N(0) = N^0$$

where $m$ is a probability density kernel which we have assumed has the property $m(\tau) > 0$ for $\tau \in (-7, -5)$ and $m(\tau) = 0$ for $\tau \in (-\infty, -7] \cup [-5, 0]$.

### 4.2 Functional Differential Equation Formulation

The system of functional differential equations described in (4.1) can be simulated using an algorithm first developed by Banks and Kappel for the linear time-invariant case [12] and later extended by Banks and Rosen to nonlinear and time-dependent systems [13]. To use this algorithm, we first convert the system to an abstract evolution equation (AEE) and then approximate the solutions in a space spanned by piecewise linear splines. Thus we can numerically calculate the generalized Fourier coefficients of the approximate solution in the spline basis representation and recover an approximation to the solution of (4.1).

#### 4.2.1 Abstract Evolution Equation Theory and Implementation

Let

$$x(t) = (A(t), N(t))^T$$

and

$$x_{\tau}(\tau) = x(t + \tau), -7 \leq \tau \leq 0.$$
We define the Hilbert space \( Z \equiv \mathbb{R}^2 \times L^2(-7,0;\mathbb{R}^2) \) with inner product
\[
||| (\eta, \varphi) |||_Z = \left( |\eta|^2 + \int_{-7}^0 |\varphi(\theta)|^2 d\theta \right)^{1/2}, \quad (\eta, \varphi) \in Z,
\]
and let \( z(t) = (x(t), x_t) \in Z \). Then our system (4.1) can be written as
\[
\dot{x}(t) = L(t, x(t), x_t) \quad \text{for} \quad 0 \leq t \leq T,
\]
\[
(x(0), x_0) = (\Phi(0), \Phi) \in Z, \quad \Phi \in C(-7,0;\mathbb{R}^2),
\]
where \( T < \infty \) and for \( \eta = (\psi^0, \zeta^0)^T \in \mathbb{R}^2 \) and \( \varphi = (\psi, \zeta)^T \in C(-7,0;\mathbb{R}^2) \)
\[
L(t, \eta, \varphi) = \begin{bmatrix}
-d_A(t) - p_A(t) & 0 \\
0 & -d_N(t) - p_N(t)
\end{bmatrix} \eta + \begin{bmatrix} 0 & 1 \\
0 & -1 \end{bmatrix} \int_{-7}^{-5} \varphi(\tau) m(\tau) d\tau.
\]
We now define a linear operator \( \mathcal{A} : \mathcal{D}(\mathcal{A}) \subset Z \to Z \) with domain
\[
\mathcal{D}(\mathcal{A}) = \{ (\eta, \varphi) \in Z | \varphi \in H^1(-7,0;\mathbb{R}^2) \text{ and } \eta = \varphi(0) \}
\]
by
\[
\mathcal{A}(t, \eta, \varphi) = (L(t, \eta, \varphi), \dot{\varphi}).
\]
Note that while \( \mathcal{A} \) depends on \( t \), \( \mathcal{D}(\mathcal{A}) \) does not. Then the delay system (4.1) can be formulated as
\[
\dot{z}(t) = \mathcal{A}(t) z(t)
\]
\[
z(0) = z_0,
\]
where \( z_0 = ((A^0, N^0)^T, (\Phi, \Psi)^T) \).

Define \( Z^N \) to be an approximating piecewise linear spline subspace of \( Z \), \( \Pi^N \) as the orthogonal projection of \( Z \) onto \( Z^N \), and \( \mathcal{A}^N(t) \) as the approximating operator for \( \mathcal{A}(t) \) given by \( \mathcal{A}^N(t) = \Pi^N \mathcal{A}(t) \Pi^N \). Then the problem given in (4.7) is approximated by the finite dimensional problem
\[
\dot{z}^N(t) = \mathcal{A}^N(t) z^N(t), \quad t \geq 0,
\]
\[
z^N(0) = \Pi^N z_0.
\]
We fix the basis \( Z^N_1 \) for a special case of \( Z^N \) corresponding to the partition \( t^N_j = -j(7/N) \) for \( j = 0, \ldots, N \). Then the basis is defined by
\[
\hat{\beta}^N = (\hat{\beta}^N(0), \hat{\beta}^N) \text{ where } \hat{\beta}^N = (e^N_0, e^N_1, \ldots, e^N_N) \otimes I_2,
\]
where \( e^N_0, e^N_1, \ldots, e^N_N \) are piecewise linear splines.
and the $e_j^N$'s are piecewise linear splines defined by

$$e_j^N(t_i^N) = \delta_{ij} \text{ for } i, j = 0, 1, \ldots, N.$$  

When $\mathcal{A}(t)$ is restricted to $Z_1^N$, we have a matrix representation $A_1^N(t)$ of $\mathcal{A}(t)$. Define $w^N(t)$ so that $z^N(t) = \hat{\beta}^N w^N(t)$. Then solving for $z^N(t)$ in the finite dimensional system (4.8) is equivalent to solving for $w^N(t)$ in the linear system

$$\begin{align*}
\dot{w}^N(t) &= A_1^N(t)w^N(t) \\
w^N(0) &= w_0^N,
\end{align*}$$

where $\hat{\beta}^N w_0^N = \Pi^N z_0$. We note that having obtained $w^N$, the product $\hat{\beta}^N w^N$ converges uniformly in $t$ to the solution of (4.7), $z(t) = (x(t), x_t)$.

### 4.2.2 Numerical Approximation

To solve (4.10) numerically, we follow the method outlined by Banks and Rosen [13]. We compute the inner products

$$Q_1^N = \langle \hat{\beta}^N, \hat{\beta}^N \rangle$$

and

$$H_1^N(t) = h^N(L(t, \beta^N), D\beta^N)$$

where

$$h^N(\eta, \varphi) = \langle \hat{\beta}^N, (\eta, \varphi) \rangle$$

to obtain the matrices

$$Q_1^N = \frac{7}{N} \begin{bmatrix}
\frac{N}{7} + \frac{1}{3} & \frac{1}{6} & 0 & \cdots & 0 \\
\frac{1}{6} & \frac{2}{3} & \frac{1}{6} & \cdots & \vdots \\
0 & \ddots & \ddots & \ddots & \vdots \\
\vdots & \ddots & \ddots & \ddots & 0 \\
0 & \cdots & 0 & \frac{1}{6} & \frac{2}{3} & \frac{1}{6}
\end{bmatrix} \otimes I_2$$

and

$$H_1^N(t) = H_{11}^N(t) + H_{12}^N.$$  

(4.12)
where

$$H_{11}^N(t) = \begin{bmatrix} A_0(t) + D_0^N & D_1^N & \cdots & D_{N-1}^N & D_N^N \\ \vdots & \ddots & \ddots & \vdots \\ 0 & \cdots & \cdots & 0 \end{bmatrix}$$

(4.13)

with

$$A_0(t) = \begin{bmatrix} -d_A(t) - p_A(t) \\ b(t) \end{bmatrix}, \quad D_0^N = \begin{bmatrix} 0 \\ -d_N(t) - p_N(t) \end{bmatrix}$$

(4.14)

and

$$D_j^N = \begin{bmatrix} 0 & 1 \\ 0 & -1 \end{bmatrix} \int_{-7}^0 e_j^N(\tau)m(\tau)d\tau$$

(4.15)

and

$$H_{12}^N = \begin{bmatrix} \frac{1}{2} & -\frac{1}{2} & 0 & \cdots & \cdots & 0 \\ \frac{1}{2} & 0 & -\frac{1}{2} & \cdots & \vdots \\ \vdots & \ddots & \ddots & \ddots & \vdots \\ 0 & \cdots & \cdots & \cdots & \frac{1}{2} \end{bmatrix} \otimes I_2.$$  

(4.16)

We would normally find the matrix $A_1^N$ to use in solving the linear system (4.10) by solving

$$Q_1^N A_1^N(t) = H_1^N(t),$$

(4.17)

but the time-dependence of $A_0(t)$ requires that $H_1^N(t)$ be recomputed with each time step of the integrator. However, only the first $2 \times 2$ submatrix of $H_1^N(t)$ changes with time, so we can compute the majority of the entries of $A_1^N(t)$ outside of the integrator and simply update the time-varying submatrix at each step. Thus rather than solving (4.17), we will determine $A_1^N(t)$ as the sum of a constant matrix and an update matrix.

To solve the system efficiently, we take advantage of the sparsity that results from our assumption for the density kernel that $m(\tau) = 0$ for $\tau \in [-5, 0]$. To compute the constant entries of $H_1^N(t)$, we determine the index $j_{\text{min}}$ for the linear spline basis functions $e_j$ such that for all $j < j_{\text{min}}$, we have $D_j^N = 0$. Then we calculate $D_j^N$ for $j = j_{\text{min}}, \ldots, N$ to determine the matrix $H_{11}^N(t)$ and thereby matrix $H_1^N(t)$. Next, we solve

$$Q_1^N A_{11}^N = H_1^N(t)$$
and

\[
Q_N^{12}A_N^{12} = \begin{bmatrix}
1 & 0 \\
0 & 1 \\
0 & 0 \\
\vdots & \vdots \\
0 & 0 
\end{bmatrix},
\]

where \(A_N^{11}\) is constant and \(A_N^{12}\) is used to update \(A_N^1(t)\). With each time step of the integrator, we compute the matrix \(A_0(t)\) as defined in (4.14) and update \(A_N^1(t)\) by

\[
A_N^1(t) = A_N^{11} + A_N^{12}A_0(t). \tag{4.18}
\]

This method of updating the matrix greatly increases computational speed, which plays an important role as the number of elements \(N\) increases.

### 4.3 Convergence of Solutions

According to mathematical theory (see [13] for proof), the solutions of (4.8) converge to the solution of (4.7) as the number of elements \(N\) goes to infinity. When we perform an inverse problem using the delay differential equation model, we need to know how many elements to take in \([-7, 0]\) so that we obtain accurate estimates of our parameters. To find the \(N\) at which solutions are converged, we carry out the forward problem for increasing values of \(N\) and plot the solutions.

For the forward problem, we need to know the parameter functions \(d_A(t), p_A(t), d_N(t), p_N(t),\) and \(b(t)\) as well as the probability density \(m(\tau)\). For realistic solutions, such as a non-negative population for all time, we can use the ODE model to estimate mortality parameters for a given level of insecticide exposure and adapt these parameters for use in the DDE model. It is important to note that each of these six time-varying parameter functions will be defined using piecewise linear splines, so as the number of nodes taken for each function increases, we are greatly enlarging our parameter space. Hence we take five equally spaced nodes for each of the functions except for \(m(\tau)\), which we define on \([-7, -5]\)

by

\[
m(\tau) = \sum_{i=0}^{2} a_i \ell_i(t) \tag{4.19}
\]

where the \(\ell_i(t)\)'s are hat functions defined on the partition of \([-7, -5]\) with \(t_i = -7 + i, i = 0, 1, 2\) and \(a_0 = a_2 = 0, a_1 = 1\). That is, the density kernel is the single hat function \(\ell_1(t)\).
Table 4.1: Nodal values of parameter functions for (4.1).

<table>
<thead>
<tr>
<th>Parameter Function</th>
<th>Region of Support</th>
<th># of Equally Spaced Nodes</th>
<th>Nodal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>$d_A(t)$</td>
<td>[0, 15]</td>
<td>5</td>
<td>(0, 0, 0.041437, 0.022689, 0)</td>
</tr>
<tr>
<td>$p_A(t)$</td>
<td>[0, 15]</td>
<td>5</td>
<td>(0.010763, 0.012490, 0.11679, 0.0098291)</td>
</tr>
<tr>
<td>$d_N(t)$</td>
<td>[0, 15]</td>
<td>5</td>
<td>(0, 0, 0, 0, 0.034625)</td>
</tr>
<tr>
<td>$p_N(t)$</td>
<td>[0, 15]</td>
<td>5</td>
<td>(0.19273, 0.29144, 0, 0.073119)</td>
</tr>
<tr>
<td>$b(t)$</td>
<td>[0, 15]</td>
<td>5</td>
<td>(0.64828, 4.1277, 1.0357, 0)</td>
</tr>
<tr>
<td>$m(\tau)$</td>
<td>[−7, −5]</td>
<td>3</td>
<td>(0, 1, 0)</td>
</tr>
</tbody>
</table>

The analogs to $d_A$ and $d_N$ for the ODE model are the background mortalities $\mu$ for adults and neonates respectively, so we use the Neem control data and perform the inverse problem on (2.5) to estimate $\mu(t)$ using five nodes for each age class. The insecticide-induced mortality rates $p_A$ and $p_N$ do not have direct analogs in the ODE model, so we instead combine the two mortality rates $p_{dir}$ and $p_{delay}$. To obtain five nodes, we take one nontrivial node for $p_{dir}$ and four nodes for $p_{delay}$ and estimate these parameters for the 40 parts per million exposure data sets according to the procedure described in Sections 2.2 and 2.3. Then the vector of spline coefficients for $p_A$ is $\left(p_{A_{dir}}, p_{A_{delay}}\right)$, and likewise, we set $p_N = \left(p_{N_{dir}}, p_{N_{delay}}\right)$. Since the minimum length of time the aphids survive after introduction of the insecticide is 15 days for this exposure level, we use only the first 15 days of data to estimate all four mortality parameters, and we likewise define the birth rate on the time interval 0 to 15 days. This rate $b(t)$ is estimated by interpolating the reproduction data for the adult age class at 40 parts per million Neem exposure at five equally spaced nodes. The nodal values for all of the parameter functions are summarized in Table 4.1.

Having set these parameters, we use the Matlab integrator $ode15s$ to solve the system with $N = 16, 32, 64, 128, 256, 512, 1024$. The solutions are plotted together in Figure 4.1. At $N = 16$ and $N = 32$ elements, the solutions are not converged, but they begin to approach the limit at $N = 64$, and by $N = 256$, the curves are nearly indistinguishable. However, one important consideration is the computational time required for one iteration of the forward problem. The times required for the Matlab integrator to solve the system for each $N$ are shown in Table 4.2. Time is not a factor through $N = 128$, but the computational time increases by a factor of 6 when we increase the number of elements to 256. We can thus be safe in saying that solutions are converged at $N = 256$ elements, but
Figure 4.1: Solutions to delay system for increasing $N$.

Table 4.2: Computational time required to solve the delay system for increasing $N$.

<table>
<thead>
<tr>
<th>$N$</th>
<th>Time (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>0.787528</td>
</tr>
<tr>
<td>32</td>
<td>0.985615</td>
</tr>
<tr>
<td>64</td>
<td>1.317057</td>
</tr>
<tr>
<td>128</td>
<td>4.219158</td>
</tr>
<tr>
<td>256</td>
<td>24.521670</td>
</tr>
<tr>
<td>512</td>
<td>200.121309</td>
</tr>
<tr>
<td>1024</td>
<td>11973.085994</td>
</tr>
</tbody>
</table>

in practice, we are more likely to use $N = 128$.

4.4 Inverse Problem and Algorithm Analysis

As we wish to eventually estimate the time-varying parameters $d_A(t)$, $p_A(t)$, $d_N(t)$, $p_N(t)$, and $b(t)$ as well as the probability density $m(\tau)$, we must first verify the numerical accuracy of our model. To do so, we simulate a solution at the converged level with the known parameters used in the convergence analysis and recorded in Table 4.1. We then
Table 4.3: Constraints on delay system parameters.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Lower Bound</th>
<th>Upper Bound</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>$d_A$</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>$p_A$</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>$d_N$</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>$p_N$</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>$b$</td>
<td>0</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>$m(\tau)$</td>
<td>0</td>
<td>—</td>
<td>$\int_{-7}^{-3} m(\tau) , d\tau = 1$</td>
</tr>
</tbody>
</table>

add noise to this solution and finally carry out an inverse problem using the noise-added solution as data to attempt to recover the original parameters.

With the six time-varying parameter functions defined as in Section 4.3, we set $\Theta = (d_A, p_A, d_N, p_N, b, m)$, solve the delay system on the time interval 0 to 15 days, and add noise to the solution to obtain the statistical model

$$y_i = x^N(t_i, \Theta_0) + \epsilon_i$$ (4.20)

where each $\epsilon_i$ is taken from a normal distribution with mean zero and variance one, and $\Theta_0$ is a set of “true” parameters, tacitly assumed to exist in standard statistical treatments.

As with the ODE model, we optimize over a constrained parameter space using the Matlab routine *fmincon* to minimize the least squares cost functional

$$J^N(\Theta) = \frac{1}{2n} \sum_{i=0}^{n} |y_i - x^N(t_i, \Theta)|^2$$ (4.21)

where

$$y_i = (x^N_A(t_i, \Theta_0), x^N_N(t_i, \Theta_0)) + (\epsilon_i^A, \epsilon_i^N)$$

is taken as data and $x^N(t_i, \Theta)$ is the model solution using parameters $\Theta$ with $N$ finite elements in the delay interval $[-7, 0]$. For the optimization, we take $N = 128$, while the data was generated using $N = 256$. The constraints imposed upon the parameters are summarized in Table 4.3. Note that in the case of upper or lower bounds, the bound extends to all spline coefficients that define the given function. Additionally, the constraint on the integral value of $m(\tau)$ is implemented as a linear constraint using the trapezoidal rule.

Since our parameter space consists of a total of 28 parameters, we begin by setting $\Theta = m$ and holding the other five parameters fixed at their true values $d_{A0}$, $p_{A0}$, $d_{N0}$, $p_{N0}$, $b_0$
Table 4.4: Minimal values of (4.21) for increasing size parameter space.

<table>
<thead>
<tr>
<th>Θ</th>
<th># of Parameters</th>
<th>( J^{128}(\hat{\Theta}) )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( m )</td>
<td>3</td>
<td>4.0534</td>
</tr>
<tr>
<td>((b, m))</td>
<td>8</td>
<td>3.7009</td>
</tr>
<tr>
<td>((p_N, b, m))</td>
<td>13</td>
<td>3.5224</td>
</tr>
<tr>
<td>((d_N, p_N, b, m))</td>
<td>18</td>
<td>3.5224</td>
</tr>
<tr>
<td>((p_A, d_N, p_N, b, m))</td>
<td>23</td>
<td>3.4628</td>
</tr>
<tr>
<td>((d_A, p_A, d_N, p_N, b, m))</td>
<td>28</td>
<td>3.4628</td>
</tr>
</tbody>
</table>

as given in Table 4.1 for our minimization of (4.21). Once we see that the estimated values for this one parameter provide a better fit to the data than the true parameters, we enlarge our set of parameters to \( \Theta = (b, m) \) and hold the other four parameters fixed at \( d_{A0}, p_{A0}, d_{N0}, p_{N0} \). We carry out the inverse problem to estimate this \( \Theta \), and after obtaining new estimates, we enlarge the parameter space again by one parameter and optimize over the new set of parameters \( \Theta \). We continue in this manner until \( \Theta \) is the set of all six time-varying parameters. This method of gradually increasing the parameter space allows us to see that the inverse problem technique is being carried out correctly. As the number of degrees of freedom increases, we expect that the minimal value of the least squares cost functional should decrease, and we indeed see this phenomenon, as shown in Table 4.4.

Although the value of \( J(\hat{\Theta}) \) decreases as we increase the number of parameters, we do not necessarily obtain accurate estimates of the true parameters. As we see in Figure 4.2, when \( \Theta = (d_A, p_A, d_N, p_N, b, m) \), the estimates for \( m(\tau) \) and \( b \) are very close to the true parameters, but the estimated mortality rates differ drastically from the true parameters. This discrepancy can be attributed to the fact that both the background mortality and insecticide-induced mortality rates have the same region of support. These two mortality rates cannot be estimated independently; it is the result of their sum that is optimized by the routine rather than the individual parameter functions. Thus it is not surprising to obtain inaccurate results for \( d_A, p_A, d_N, \) and \( p_N \), but we expect the estimated sums \( d_A(t) + p_A(t) \) and \( d_N(t) + p_N(t) \) to be accurate approximations to the sums of the true parameter functions \( d_{A0} + p_{A0} \) and \( d_{N0} + p_{N0} \). This accuracy of sums is in fact the case, as we see in Figure 4.3.

However, it is unlikely that we would estimate both the background and insecticide-induced mortality rates at the same time. As we did for the ODE model, we should have
control data which we will use in an inverse problem to estimate $d_A$ and $d_N$. In this case, we would set $p_A = 0$ and $p_N = 0$, reducing the delay system (4.1) to

$$
\dot{A}(t) = \int_{-7}^{-5} N(t + \tau)m(\tau)d\tau - d_A(t)A(t)
$$

$$
\dot{N}(t) = b(t)A(t) - d_N(t)N(t) - \int_{-7}^{-5} N(t + \tau)m(\tau)d\tau.
$$

Having estimated the background mortality rates, we can take these parameter values to be known and optimize over $\Theta = (p_A, p_N, b, m)$ to estimate parameters for cases where we do have exposure to insecticide.

To see that the estimates for the insecticide-induced mortality rates are accurate
when we use this method, we take $d_A = d_{A0}$ and $d_N = d_{N0}$ and find the parameters $\Theta = (p_A, p_N, b, m)$ that minimize (4.21). When we compare the true mortality parameters to the optimal mortality parameters, we see that the results are much more accurate than when we optimize over all four mortality rates. We can examine the results numerically by comparing the infinity norm of the difference between the true and optimal parameters for both cases, and we see that the norm of the error is much smaller when we hold the background mortality rates at the true parameter values. We note that the minimized value of the least squares cost functional in this case is 3.4523, which is the same as when we optimize over all six parameter functions (see Table 4.4). The plots of the true and optimal parameters $p_A$ and $p_N$ are shown in Figure 4.4, and the norm comparisons are detailed in Table 4.5.
Table 4.5: Error comparisons for insecticide-induced mortality rates.

<table>
<thead>
<tr>
<th>Norm</th>
<th>$\Theta = (d_A,p_A,d_N,p_N,b,m)$</th>
<th>$\Theta = (p_A,p_N,b,m)$</th>
<th>Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>$|p_A^5 - p_{A0}^5|_\infty$</td>
<td>$2.3089 \times 10^{-2}$</td>
<td>$1.0726 \times 10^{-2}$</td>
<td>2.1526</td>
</tr>
<tr>
<td>$|p_N^5 - p_{N0}^5|_\infty$</td>
<td>$1.6473 \times 10^{-1}$</td>
<td>$2.0460 \times 10^{-2}$</td>
<td>8.0513</td>
</tr>
<tr>
<td>$J^N(\Theta)$</td>
<td>3.4628</td>
<td>3.4628</td>
<td>1</td>
</tr>
</tbody>
</table>

4.5 Sensitivity Analysis

In this section we follow the theoretical arguments of Banks, Dediu, and Nguyen [14] to derive an equation for the sensitivity of the delay system (4.1) to the density kernel $m(\tau)$. We are particularly concerned with sensitivity to this parameter because it is the one for which we have the least physical information. We know only that neonate aphids become adults at some time between 5 and 7 days of age, but the shape of the distribution is unknown. Thus we desire to investigate how the model output is affected by changes in this parameter.

4.5.1 Sensitivity Equation Derivation

We begin with our delay system (4.1) in the form

$$
F(t,x,x_t,P) = \begin{bmatrix}
-d_A(t) - p_A(t) & 0 \\
 b(t) & -d_N(t) - p_N(t)
\end{bmatrix} x(t) + \begin{bmatrix} 0 & 1 \\
 0 & -1 \end{bmatrix} \int_{-\tau}^{0} x_t(\tau)m(\tau)d\tau,
$$

(4.23)

where $m(\tau) > 0$ for $\tau \in [-7,-5]$ and $m(\tau) = 0$ for $\tau \in [-5,0]$. We now fix another probability distribution $q(\tau)$ defined on $[-7,0]$ with the same properties as $m$ and consider the sensitivity $y = \frac{\partial x}{\partial m}(t,m;q-m)$ of $x$ with respect to $m$, which is defined as the directional derivative of $x$ with respect to $m$ in the direction $q - m$, and satisfies

$$
\dot{y}(t) = \frac{\partial F}{\partial x}(t,x,x_t,m)y(t) + \frac{\partial F}{\partial x_t}(t,x,x_t,m)y_t + \frac{\partial F}{\partial m}(t,x,x_t,m;q-m)
$$

(4.24)

where $F$ is defined in (4.23). The function $y_t$ is the delay sensitivity function and is defined in the same way as the delay function $x_t$ is defined in (4.2). The quantities $\frac{\partial F}{\partial x}$, $\frac{\partial F}{\partial x_t}$, and $\frac{\partial F}{\partial m}$ are the Frechet derivatives of $F$ with respect to $x$ and $x_t$, and finally the directional derivative of $F$ with respect to $m$ in the direction $q - m$. 
For $F$ defined in (abstract form), we have

$$\frac{\partial F}{\partial x}(t,x,x_t,m)y(t) = \begin{bmatrix} -d_A(t) - p_A(t) & 0 \\ b(t) & -d_N(t) - p_N(t) \end{bmatrix} y(t),$$  \hfill (4.25)$$

and

$$\frac{\partial F}{\partial x_t}(t,x,x_t,m)[y_t] = \begin{bmatrix} 0 & 1 \\ 0 & -1 \end{bmatrix} \int_{-7}^{0} y_t(\tau)m(\tau)d\tau,$$  \hfill (4.26)$$

since the integral in (4.23) is linear in $x_t$. Finally, the partial derivative $\partial F/\partial m$ at $m$ in the direction $q - m$ is given by

$$\frac{\partial F}{\partial m}(t,x,x_t,m; q - m) = \lim_{\varepsilon \to \infty} \frac{F(t,x,x_t,m + \varepsilon(q - m)) - F(t,x,x_t,m)}{\varepsilon}$$

$$= \lim_{\varepsilon \to \infty} \begin{bmatrix} 0 & 1 \\ 0 & -1 \end{bmatrix} \left( \int_{-7}^{0} x(t + \tau)[m(\tau) + \varepsilon(q(\tau) - m(\tau))]d\tau - \int_{-7}^{0} x(t + \tau)m(\tau)d\tau \right)$$

$$= \begin{bmatrix} 0 & 1 \\ 0 & -1 \end{bmatrix} \int_{-7}^{0} x(t)(q(\tau) - m(\tau))d\tau.$$  \hfill (4.27)$$

With $F$ defined in (4.23), substitution of (4.25), (4.26), and (4.27) into (4.24) gives the differential equation for the sensitivity

$$\dot{y}(t) = \begin{bmatrix} -d_A(t) - p_A(t) & 0 \\ b(t) & -d_N(t) - p_N(t) \end{bmatrix} y(t) + \begin{bmatrix} 0 & 1 \\ 0 & -1 \end{bmatrix} \int_{-7}^{0} y_t(\tau)m(\tau)d\tau$$

$$+ \begin{bmatrix} 0 & 1 \\ 0 & -1 \end{bmatrix} \int_{-7}^{0} x(t)(q(\tau) - m(\tau))d\tau$$  \hfill (4.28)$$

$$y(0) = (0,0)^T.$$
4.5.2 Numerical Approximation

To numerically approximate (4.28), we could solve the coupled delay differential equation system

\[
\dot{x}(t) = \begin{bmatrix} -d_A(t) - p_A(t) \\ b(t) \\ -d_N(t) - p_N(t) \end{bmatrix} x(t) + \begin{bmatrix} 0 & 1 \\ 0 & -1 \end{bmatrix} \int_{-7}^{0} x_t(\tau)m(\tau)d\tau
\]

\[
\dot{y}(t) = \begin{bmatrix} -d_A(t) - p_A(t) \\ b(t) \\ -d_N(t) - p_N(t) \end{bmatrix} y(t) + \begin{bmatrix} 0 & 1 \\ 0 & -1 \end{bmatrix} \int_{-7}^{0} y_t(\tau)m(\tau)d\tau
\]

\[
+ \begin{bmatrix} 0 & 1 \\ 0 & -1 \end{bmatrix} \int_{-7}^{0} x_t(\tau)[q(\tau) - m(\tau)]d\tau
\]

\[
x(\theta) = (\Phi(\theta), \Psi(\theta))^T, \quad y(\theta) = (0, 0)^T \quad \theta \in [-7, 0),
\]

\[
x(0) = (A^0, N^0)^T, \quad y(0) = (0, 0)^T
\]

taking the delay system algorithm described in [13]. However, coupling the sensitivity equation to the state equation doubles the size of the system, thereby increasing computational time, and also leads to slower convergence in terms of the number of finite elements used to discretize in the delay interval \([-7, 0]\).

We note that the differential equation \(\dot{y}(t)\) for the sensitivity takes the form

\[
\dot{y}(t) = L(t, y(t), y_t) + f(t)
\]

where \(L(t, \eta, \varphi)\) is defined in (4.4), and \(f(t)\) is

\[
f(t) = \begin{bmatrix} 0 & 1 \\ 0 & -1 \end{bmatrix} \int_{-7}^{0} x_t(\tau)[q(\tau) - m(\tau)]d\tau.
\]

Thus we can formulate the sensitivity equation as an abstract Cauchy problem

\[
\dot{\bar{z}}(t) = \mathcal{A}(t)\bar{z}(t) + f(t)
\]

\[
\bar{z}(0) = \bar{z}_0,
\]

where \(\bar{z} = (y(t), y_t)\), \(\mathcal{A}(t)\) is defined by (4.5) and (4.6), and \(\bar{z}_0 = (0, 0)^T\).

We use the same approximation scheme \(\{Z^N, \Pi^N, \mathcal{A}^N(t)\}\) as we used earlier to generate an approximation to (4.32), which we express as

\[
\dot{\bar{z}}^N(t) = \mathcal{A}^N(t)\bar{z}^N(t) + \Pi^N f(t)
\]

\[
\bar{z}^N(0) = \Pi^N \bar{z}_0.
\]
As before, we approximate the system in the piecewise linear spline basis \( Z_1^N \), and we have the matrix representation \( A_1^N(t) \) of \( A^N(t) \) when \( A^N(t) \) is restricted to \( Z_1^N \). Using the basis elements \( \hat{\beta}^N \) as defined in (4.9), we define \( F^N(t) \) so that \( \Pi^N f(t) = \hat{\beta}^N F^N(t) \). Then we can solve for the generalized Fourier coefficients \( v^N(t) \) in the linear system

\[
\dot{v}^N(t) = A_1^N(t) v^N(t) + F^N(t)
\]

\( v^N(0) = v_0^N \),

(4.34)

where \( v_0^N = \Pi^N(0, 0) \).

Solving for \( F^N(t) \) numerically amounts to solving

\[
Q_1^N F^N(t) = \beta^N(0)^T f(t) = \begin{bmatrix} f(t) \\ 0 \\ \vdots \\ 0 \end{bmatrix},
\]

where \( Q_1^N \) is the matrix defined by (4.11). The other matrices used in the numerical approximation are computed using the methods described in Section 4.2.2.

### 4.5.3 Convergence

Numerical approximation of (4.33) requires that we apply a quadrature rule to the integral in our forcing function (4.31). To obtain accurate results for the convergence of solutions of (4.34), we must ensure that the value of this integral does not change as we change \( N_y \), the number of finite elements in the delay interval \([-7, 0]\). Note that \( N_y \) need not equal \( N_x \), the number of finite elements in \([-7, 0]\) used in solving (4.8). Using \( M + 1 \) quadrature points, we use the trapezoidal method to obtain

\[
I(t) = \int_{-5}^{-7} g(t, \tau)d\tau \approx I^M(t) = \frac{h}{2} (g(t, -7) + g(t, -5)) + h \sum_{k=1}^{M-1} g(t, \tau_k)
\]

(4.35)

where \( h = 2/M \) and \( g(t, \tau) = x^{N_y}(t + \tau)m(\tau) \) with \( m \) defined as in Sections 4.3 and 4.4.

Given this quadrature, we can write the system that approximates (4.28) as

\[
\dot{\tilde{z}}^{N_y,M}(t) = A^{N_y} \tilde{z}^{N_y,M}(t) + \Pi^{N_y} f(t, m, q)
\]

\( \tilde{z}^{N_y,M}(0) = \Pi^{N_y} \tilde{z}_0 \),

(4.36)

where \( N_y \) is the number of finite elements in \([-7, 0]\). The convergence of the approximations \( \tilde{z}^{N_y,M} \) to the solution \( \tilde{z} \) of (4.32) as \( N_y, M \to \infty \) can be guaranteed using the arguments in [9].
We fix the number of finite elements in $[-7,0]$ for the state at $N_x = 256$ and first solve (4.8) with $m$ defined as in previous sections to obtain the solution $z^{N_x}(t) = (x^{N_x}(t), x_t^{N_x})$. We then compute $I^M$ for different values of $M$ and plot these approximations, as shown in Figure 4.5. We observe that at $M = 256$, it appears that $I^M$ has converged to $I$, but when $M = 2048$, the curve changes. However, this difference can be attributed to over-discretization of the interval $[-7,-5]$; once we have such a large number of elements in this small interval, numerical error grows drastically and leads to this movement away from the converged solutions.

With the value of $M$ fixed at 256, we next solve (4.36), taking $q$ as a uniform distribution on $[-7,-5]$, for different values of $N_y$ to find determine the number of elements in the delay interval $[-7,0]$ required for convergence of solutions $\tilde{z}^{N_y,M}(t)$ to $\tilde{z}(t) = (y(t), y_t)$. As we see in Figure 4.6, the sensitivity of $x$ with respect to $m$ in the direction $q - m$ varies little as we increase $N_y$. Just as was the case for the numerical solution of (4.8), the finite dimensional approximation to (4.1), computational time increases with $N_y$, shown in Figure 4.6.

Figure 4.5: Plots of the integral $I^M(t)$ for different values of $M$. 

![Figure 4.5](image-url)
Table 4.6: Computational time required to solve the finite dimensional approximation (4.36) to the sensitivity of $x$ with respect to $m$ in the direction $q - m$ for increasing $N_y$.

<table>
<thead>
<tr>
<th>$N_y$</th>
<th>Time (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>16</td>
<td>0.967708</td>
</tr>
<tr>
<td>32</td>
<td>1.223329</td>
</tr>
<tr>
<td>64</td>
<td>1.955960</td>
</tr>
<tr>
<td>128</td>
<td>6.970488</td>
</tr>
<tr>
<td>256</td>
<td>58.383277</td>
</tr>
<tr>
<td>512</td>
<td>538.970136</td>
</tr>
</tbody>
</table>

Table 4.6.

Whereas computational time was an important factor in our choice of $N$ for approximations to the state $z(t) = (x(t), x_t)$ because the optimization problem required that we solve (4.8) hundreds of times, we need solve the sensitivity differential equation only once. Thus if we choose $N_y = 128$ or even 256, the time sacrifice is still minimal. However, the improvement in accuracy of the sensitivity does not outweigh the increase in computational time as $N_y$ is increased, so we can take $N_y = 64$ to be the converged level for solutions of (4.36).

4.5.4 Application

Once we have obtained converged solutions to (4.36), we are equipped to study the error in our estimates for the delay kernel $m(\tau)$. To compute confidence intervals for the nodal values of the piecewise linear splines that define $m$, we need the covariance matrix, which uses the sensitivity [14, 15]. These confidence intervals will provide insight into the accuracy of our choice of the form of $m$ and guide our efforts to determine an appropriate shape of this probability density.
Figure 4.6: Convergence of approximations $\tilde{z}^{N_y,M}(t)$ to $\tilde{z}(t)$ for different values of $N_y$ and $M = 256$. 
Chapter 5

Concluding Remarks

As an alternative to age-structured partial differential equation models for population dynamics of aphids under insecticide exposure, we have developed a much simpler ordinary differential equation model as well as a time-delay differential equation model, which can be seen as intermediate in terms of ease of understanding for those without a theoretical mathematical background. While the ODE model provides a highly accurate fit to population data, the DDE model may prove more applicable in future efforts because it accounts for multiple generations.

Future work will depend heavily upon our obtaining new data that will be collected through experiments designed to support the model. Researchers will work in both the laboratory and the field, keeping track of mortality and reproduction for not only the first generation of insects, but also their offspring. We will then use the OLS technique to find parameters for the delay model that provide the best fit to this data, in hopes that our model will effectively capture the population dynamics. Sensitivity analysis on the density kernel \( m \) will be instrumental in determining the appropriate form of the probability density via statistical methods such as confidence intervals. We would also like to determine optimal dosage levels of insecticides using the delay model.
Bibliography


