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Summer 2014

Epistasis in Predator-Prey Relationships

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EPISTASIS IN PREDATOR-PREY RELATIONSHIPS

by

IULIIA INOZEMTSEVA

(Under the Direction of James Braselton)

ABSTRACT

Epistasis is the interaction between two or more genes to control a single phenotype. We model epistasis of the prey in a two-locus two-allele problem in a basic predator-prey relationship. The resulting model allows us to examine both population sizes as well as genotypic and phenotypic frequencies. In the context of several numerical examples, we show that if epistasis results in an undesirable or desirable phenotype in the prey by making the particular genotype more or less susceptible to the predator or dangerous to the predator, elimination of undesirable phenotypes and then genotypes occurs.

EPISTASIS IN PREDATOR-PREY RELATIONSHIPS

by

IULIIA INOZEMTSEVA

B.S. in Mathematics

A Thesis Submitted to the Graduate Faculty of Georgia Southern University in Partial

Fulfillment

of the Requirement for the Degree

MASTER OF SCIENCE

STATESBORO, GEORGIA

2014

EPISTASIS IN PREDATOR-PREY RELATIONSHIPS

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Electronic Version Approved:

June 10, 2014

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CHAPTER 1

INTRODUCTION

The usual starting point for models in population genetics is a model of random mating for a one-locus, two-allele problem with Hardy-Weinberg proportions for the alleles. Refer to Karlin, [5], for an overview of mathematical models of population genetics. In genetic problems, the **genotype** is defined to be the genetic makeup of an organism. For example, in a one-locus, two-allele model, one can label the alleles as A (dominant) and a (recessive). The possible genotypes are then AA, Aa, and aa. On the other hand, the **phenotype** is defined to be the set of observable characteristics of an individual resulting from the interaction of its genotype with the environment. In the absence of **epistasis**, defined in the next paragraph, with the above genotypes, the phenotypes would be A for the genotypes of type AA and Aa and a for the genotype of type aa.

Epistasis occurs when the genotype results in a phenotype different from that expected. For example, in the context of the example above, epistasis would occur if either the AA or Aa genotype did not result in the phenotype expected, A.

One of the more interesting examples of epistasis in humans occurs in human blood in the form of the *Bombay Phenotype*, which has been popularized in the daytime soap opera, *General Hospital*, the 2012 film "Get the Gringo," and the Canadian television series "My Babysitter's a Vampire." The *Bombay Phenotype* is a *very rare* blood phenotype in humans that is scientifically referred to as the hh (the H) antigen that results in a phenotype of type O, even though the genotype of these individuals is of type ABO, which typically would be expressed as of type AB. People with the *Bombay Phenotype* can donate to any member of the ABO blood group system, but they can only receive blood from others with the *Bombay Phenotype*. The *Bombay Phenotype* is *very* rare and only occurs in approximately 1 in 250,000 humans.

In the study of epistasis discussed here the modifications introduced are that epistasis is incorporated into a standard predator-prey model, so that one genotype of the prey may be more or less susceptible to the predator than other prey with a different genotype, but the same phenotype in the absence of epistasis. Second, we introduce epistasis in the standard predator-prey model so that one genotype of the prey is dangerous (lethal or poisonous) to the predator than other prey with different genotypes but the same expected phenotype in the absence of epistasis.

This is accomplished by a perturbation of the random mating model within the context of established ecological models for the evolution of a predator-prey population.

Of course, general mating preferences almost certainly depend on numerous external factors that might include but are not limited to available mates, parasites, season, climate, and such that are beyond the scope of the model discussed here, but could lead to interesting extensions of the situations discussed here.

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CHAPTER 2

FORMULATION OF THE MODEL

2.1   The Standard Predator-Prey Equations

</div>

Let $x = x(t)$ denote the size or density of the prey population and $y = y(t)$ denote the size or density of the predator population. Then the standard predator-prey equations take the form

$$x' = x\,(a - ky) = xa - kxy$$
$$y' = y(-c + dx) = dxy - cy \qquad (2.1)$$
$$x(0) = x_0,\; y(0) = y_0,$$

where a, k, c, and d are all positive constants. Refer to Karlin, [5], for basic details regarding the standard Predator-Prey model. In the absence of the predator, the prey has linear growth, given by the $x' = ax$ term. The death rate of the prey is governed by predator-prey interactions given by the quadratic $-k\,xy$ term, which contributes to the growth rate of the predator by the $d\,xy$ term. We interpret k to be the *catchability* of the prey x by the predator y. Details regarding the standard predator-prey equations and different interpretations of the coefficients are discussed in most introductory differential equations texts like Abell and Braselton, [1], or introductory mathematical modeling and/or mathematical biology texts such as Beltrami, [2], or Murray, [7].

The most important result for system (2.1) is that the equilibrium (rest) point $(x_0, y_0) = (c/d, a/k)$ is classified as a *center* in the corresponding linearized system because the eigenvalues of the Jacobian of system (2.1),

$$\mathbf{J}(x,y) = \begin{pmatrix} \dfrac{\partial}{\partial x}(xa - kxy) & \dfrac{\partial}{\partial y}(xa - kxy) \\ \dfrac{\partial}{\partial x}(dxy - cy) & \dfrac{\partial}{\partial y}(dxy - cy) \end{pmatrix} = \begin{pmatrix} a - ky & -kx \\ dy & -c + dx \end{pmatrix}$$
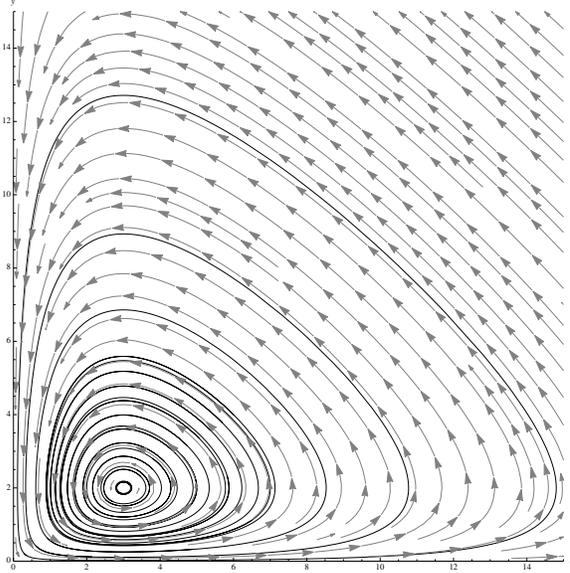
Figure 2.1: We choose $a = 2$, $k = -1$, $c = -3$, and $d = 1$. Observe how the solution curves revolve about the center, $(x_0, y_0) = (3, 2)$.

evaluated at the equilibrium (rest) point $(x_0, y_0) = (c/d, a/k)$ are

$$
\lambda_{1,2} = \begin{vmatrix} a - ky & -kx \\ dy & -c + dx \end{vmatrix}_{(x_0, y_0) = (c/d, a/k)} = \begin{vmatrix} 0 & -ck/d \\ ad/k & 0 \end{vmatrix} = \pm i \sqrt{ac}.
$$

While the stability of the equilibrium point in the nonlinear system is generally inconclusive in this case, other solution methods can verify that the equilibrium point is, in fact, a center. A typical example is shown in Figure 2.1, where we have used the values $a = 2$, $k = -1$, $c = -3$, and $d = 1$. In Figure 2.1, observe how the limit cycles revolve about the center, $(x_0, y_0) = (3, 2)$.

2.2 A Two-Locus, Two-Allele Model

In the two-locus, two-allele problem, the number of genotypes is nine but in the absence of epistasis, the number of phenotypes is four. If the A and B alleles are dominant, the expected result is four phenotypes AB (x_1, x_2, x_4, and x_5), Ab (x_3

and x_6), aB (x_7 and x_8), and ab (x_9), as described next. For the two-locus, two-allele problem, we consider a population $x = x(t)$ with size (or density) $x = x_1 + x_2 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7 + x_8 + x_9$, where

- x_1 is the size of the population of type $AABB$ (expected phenotype AB),

- x_2 is the size of the population of type $AABb$ (expected phenotype AB),

- x_3 is the size of the population of type $AAbb$ (expected phenotype Ab),

- x_4 is the size of the population of type $AaBB$ (expected phenotype AB),

- x_5 is the size of the population of type $AaBb$ (expected phenotype AB),

- x_6 is the size of the population of type $Aabb$ (expected phenotype Ab),

- x_7 is the size of the population of type $aaBB$ (expected phenotype aB),

- x_8 is the size of the population of type $aaBb$ (expected phenotype aB) and

- x_9 is the size of the population of type $aabb$ (expected phenotype ab).

The proportion of gametes of type AB, Ab, aB, and ab are given by

$$p_x = \frac{1}{x}\left(x_1 + \frac{1}{2}(x_2 + x_4) + \frac{1}{4}x_5\right),$$

$$q_x = \frac{1}{x}\left(x_3 + \frac{1}{2}(x_2 + x_6) + \frac{1}{4}x_5\right),$$

$$r_x = \frac{1}{x}\left(x_7 + \frac{1}{2}(x_4 + x_8) + \frac{1}{4}x_5\right), \tag{2.2}$$

and

$$s_x = \frac{1}{x}\left(x_9 + \frac{1}{2}(x_6 + x_8) + \frac{1}{4}x_5\right),$$

respectively. Observe that $p_x + q_x + r_x + s_x = 1$. Table A.1 in the **Appendix** shows the expected ratio of offspring produced by each x_i-x_j combination. Refer to articles like Szathmáry, [8], for details regarding these and similar calculations.

Assuming random mating between the genotypes of the prey, the predator-prey equations (2.1) become

$$x_1' = axp^2 - k_1 x_1 y$$

$$x_2' = 2axpq - k_2 x_2 y$$

$$x_3' = axq^2 - m_3 x_3 y$$

$$x_4' = 2axpr - k_4 x_4 y$$

$$x_5' = 2ax\,(ps + rq) - k_5 x_5 y$$

$$x_6' = 2axqs - m_6 x_6 y \tag{2.3}$$

$$x_7' = axr^2 - f_7 x_7 y$$

$$x_8' = 2axrs - f_8 x_8 y$$

$$x_9' = axs^2 - l_9 x_9 y$$

$$y' = y(-c + dx)$$

with initial conditions

$$x_1(0) = x_{10},\ x_2(0) = x_{20},\ x_3(0) = x_{30},\ x_4(0) = x_{40},\ x_5(0) = x_{50},$$
$$x_6(0) = x_{60},\ x_7(0) = x_{70},\ x_8(0) = x_{80},\ x_9(0) = x_{90},\ y(0) = y_0, \tag{2.4}$$

where we have used Table A.1 in the Appendix to compute the coefficients and simplified the results using equation (2.2) as well as omitted the subscripts for the $p = p_x$, $q = q_x$, $r = r_x$, and $s = s_x$ terms.

If $k = k_{1,2,4,5} = m_{3,6} = f_{7,8} = l_9$, adding system (2.3), substituting the proportion of gametes given in equation (2.2), and adding $x = x_1 + x_2 + x_2 + x_4 + x_5 + x_6 + x_7 + x_8 + x_9$ results in the predator-prey equations, system (2.1). Also, using equations (2.2), the allele frequencies of A, a, B, and b are given by

$$\frac{1}{x}\left(x_1 + x_2 + x_3 + \frac{1}{2}\left(x_4 + x_5 + x_6\right)\right) = p + q,$$

$$\frac{1}{x}\left(x_7 + x_8 + x_9 + \frac{1}{2}\left(x_4 + x_5 + x_6\right)\right) = r + s,$$

$$\frac{1}{x}\left(x_1 + x_4 + x_7 + \frac{1}{2}\left(x_2 + x_5 + x_8\right)\right) = p + r, \tag{2.5}$$

and

$$\frac{1}{x}\left(x_3 + x_6 + x_9 + \frac{1}{2}\left(x_2 + x_5 + x_8\right)\right) = q + s,$$

respectively.

Also, using system (2.3) and equations (2.2), we have

$$\begin{aligned}
\frac{x_1' + x_2' + x_3' + \frac{1}{2}\left(x_4' + x_5' + x_6'\right)}{x_1 + x_2 + x_3 + \frac{1}{2}\left(x_4 + x_5 + x_6\right)} - \frac{x'}{x} &= 0, \\[4pt]
\frac{x_7' + x_8' + x_9' + \frac{1}{2}\left(x_4' + x_5' + x_6'\right)}{x_7 + x_8 + x_9 + \frac{1}{2}\left(x_4 + x_5 + x_6\right)} - \frac{x'}{x} &= 0, \\[4pt]
\frac{x_1' + x_4' + x_7' + \frac{1}{2}\left(x_2' + x_5' + x_8'\right)}{x_1 + x_4 + x_7 + \frac{1}{2}\left(x_2 + x_5 + x_8\right)} - \frac{x'}{x} &= 0, \\[4pt]
\frac{x_3' + x_6' + x_9' + \frac{1}{2}\left(x_2' + x_5' + x_8'\right)}{x_3 + x_6 + x_9 + \frac{1}{2}\left(x_2 + x_5 + x_8\right)} - \frac{x'}{x} &= 0.
\end{aligned} \tag{2.6}$$

Integrating and exponentiating each equation in (2.6) results in the following

$$p + q = p(0) + q(0), \quad p + r = p(0) + r(0), \quad q + s = q(0) + s(0), \quad \text{and} \quad r + s = r(0) + s(0). \tag{2.7}$$

This proves the theorem

Theorem 2.1. *For random mating, the relative frequencies of the alleles A, a, B, and b are constant, agreeing with the Hardy-Weinberg equation.*

Theorem 2.2. *If $k = k_{1,2,4,5} = m_{3,6} = f_{7,8} = l_9$, there are up to 14 equilibrium (rest) points, provided that the appropriate quantities are nonnegative.*

Proof. In the following, $x_1 = x_1(0)$, $x_2 = x_2(0)$, $x_3 = x_3(0)$, $x_4 = x_4(0)$, $x_5 = x_5(0)$, $x_6 = x_6(0)$, $x_7 = x_7(0)$, $x_8 = x_8(0)$, and $x_9 = x_9(0))$.

$E_{1,2}$ are given by

$$x_1{}^* = \pm \frac{x_5 \left(2\sqrt{2}\sqrt{cx_5^2 x_8 \left(2cx_8 - d(x_5 + 2x_8)^2\right)} \pm 4cx_5 x_8 + dx_5(x_5 + 2x_8)^2\right)}{8dx_8(x_5 + 2x_8)^2}$$

$$x_2{}^* = \frac{x_5^2}{4x_8},$$

$$x_3{}^* = -\frac{dx_5^3(x_5 + 2x_8)^2}{8x_8\left(\mp 2\sqrt{2}\sqrt{cx_5^2 x_8 \left(2cx_8 - d(x_5 + 2x_8)^2\right)} - 4cx_5 x_8 + dx_5(x_5 + 2x_8)^2\right)},$$

$$x_4{}^* = \pm \frac{2\sqrt{2}\sqrt{cx_5^2 x_8 \left(2cx_8 - d(x_5 + 2x_8)^2\right)} \pm 4cx_5 x_8 + dx_5(x_5 + 2x_8)^2}{2d(x_5 + 2x_8)^2},$$

$$x_5{}^* = x_5,$$

$$x_6{}^* = \pm \frac{dx_5^2(x_5 + 2x_8)^2}{4\sqrt{2}\sqrt{cx_5^2 x_8 \left(2cx_8 - d(x_5 + 2x_8)^2\right)} \pm 8cx_5 x_8 \mp 2dx_5(x_5 + 2x_8)^2},$$

$$x_7{}^* = -\frac{x_8\left(\mp 2\sqrt{2}\sqrt{cx_5^2 x_8 \left(2cx_8 - d(x_5 + 2x_8)^2\right)} - 4cx_5 x_8 + dx_5(x_5 + 2x_8)^2\right)}{2dx_5(x_5 + 2x_8)^2},$$

$$x_8{}^* = x_8,$$

$$x_9{}^* = \pm \frac{dx_5 x_8(x_5 + 2x_8)^2}{4\sqrt{2}\sqrt{cx_5^2 x_8 \left(2cx_8 - d(x_5 + 2x_8)^2\right)} \pm 8cx_5 x_8 \mp 2dx_5(x_5 + 2x_8)^2}, and$$

$$y^* = \frac{a}{k}.$$

Next, $E_{3,4}$ are given by

$$x_1{}^* = 0, \ x_2{}^* = 0, \ x_3{}^* = 0, \ x_4{}^* = 0, \ x_5{}^* = 0, \ x_6{}^* = 0,$$

$$x_7{}^* = \frac{c - dx_8 \pm \sqrt{c(c - 2dx_8)}}{2d},$$

$$x_8{}^* = x_8, \quad x_9{}^* = \frac{c - dx_8 \mp \sqrt{c(c - 2dx_8)}}{2d} \quad \text{and} \quad y^* = \frac{a}{k}.$$

$E_{5,6}$ are given by

$$x_1{}^* = 0, \; x_2{}^* = 0, \; x_4{}^* = 0, \; x_5{}^* = 0, x_6{}^* = x_6,$$

$$x_7{}^* = 0, x_8{}^* = 0, \; y^* = \frac{a}{k}, \; x_3{}^* = \frac{c - dx_6 \pm \sqrt{c(c - 2dx_6)}}{2d}, \; \text{and}$$

$$x_9{}^* = \frac{c - dx_6 \mp \sqrt{c(c - 2dx_6)}}{2d}.$$

$E_{7,8}$ are given by

$$x_2{}^* = 0, \; x_3{}^* = 0, \; x_4{}^* = x_4, \; x_5{}^* = 0, x_6{}^* = 0,$$

$$x_8{}^* = 0, x_9{}^* = 0, \; y^* = \frac{a}{k}, \; x_1{}^* = \frac{c - dx_4 \pm \sqrt{c(c - 2dx_4)}}{2d}, \; \text{and}$$

$$x_7{}^* = \frac{c - dx_4 \mp \sqrt{c(c - 2dx_4)}}{2d}.$$

$E_{9,10}$ are given by

$$x_2{}^* = x_2, \; x_4{}^* = 0, \; x_5{}^* = 0, \; x_6{}^* = 0, x_7{}^* = 0,$$

$$x_8{}^* = 0, x_9{}^* = 0, \; y^* = \frac{a}{k}, \; x_1{}^* = \frac{c - dx_2 \pm \sqrt{c(c - 2dx_2)}}{2d}, \; \text{and}$$

$$x_3{}^* = \frac{c - dx_2 \mp \sqrt{c(c - 2dx_2)}}{2d}.$$

E_{11} is given by $x_1{}^* = 0$, $x_2{}^* = 0$, $x_3{}^* = 0$, $x_4{}^* = 0$, $x_5{}^* = 0$, $x_6{}^* = 0$, $x_7{}^* = 0$, $x_8{}^* = 0$, $x_9{}^* = c/d$, and $y^* = a/k$.

E_{12} is given by $x_1{}^* = 0$, $x_2{}^* = 0$, $x_3{}^* = c/d$, $x_4{}^* = 0$, $x_5{}^* = 0$, $x_6{}^* = 0$, $x_7{}^* = 0$, $x_8{}^* = 0$, $x_9{}^* = 0$, and $y^* = a/k$.

E_{13} is given by $x_1{}^* = 0$, $x_2{}^* = 0$, $x_3{}^* = 0$, $x_4{}^* = 0$, $x_5{}^* = 0$, $x_6{}^* = 0$, $x_7{}^* = c/d$, $x_8{}^* = 0$, $x_9{}^* = 0$, and $y^* = a/k$.

Finally, E_{14} is given by $x_1{}^* = c/d$, $x_2{}^* = 0$, $x_3{}^* = 0$, $x_4{}^* = 0$, $x_5{}^* = 0$, $x_6{}^* = 0$, $x_7{}^* = 0$, $x_8{}^* = 0$, $x_9{}^* = 0$, and $y^* = a/k$.

For all 14 rest points, $x_1{}^* + x_2{}^* + x_3{}^* + x_4{}^* + x_5{}^* + x_5{}^* + x_7{}^* + x_8{}^* + x_9{}^* = c/9$. $\square$

Observe that the Jacobian, $\mathbf{J}$, for system (2.3) is a 10×10 matrix. We are unable to compute the eigenvalues of $\mathbf{J}$ at E_1 or E_2. However, for the remaining rest points (equilibrium points), E_3, E_4, ..., E_{14}, the eigenvalues of $\mathbf{J}$ evaluated at E_i are $\lambda_{1,2} = 0$, $\lambda_{3,4,5,6,7} = -a$, $\lambda_8 = -a/2$, and $\lambda_{9,10} = \pm i\sqrt{ac}$. Thus, we expect the rest points to *usually* be "center-like," which is illustrated in the computations.

When $k_1 = k_2 = k_4 = k_5 = k$ (phenotype AB), $m_3 = m_6 = m$ (phenotype Ab), or $f_7 = f_8 = f$ (phenotype aB), the interpretation is that the corresponding phenotype of the prey have the same *catchability* to the predator. In this situation, we are not able to find exact formulas for the rest points as in the case when $k = k_{1,2,4,5} = m_{3,6} = f_{7,8} = l_9$. Thus, we conduct numerous numerical studies to explore some of the possibilities.

When all parameter values have similar values as in the standard predator-prey equations (2.1), we typically see a limit cycle that is illustrated in Figure 2.2.

Reviewing the standard predator-prey system of equations (2.1), we expect to see that the higher parameter values of a and c (such as $a = 2$ and $c = 3$) giving the advantage to the prey, because the parameter a is the growth rate of the (prey) species x and the parameter c is the death (or emigration) rate of (predator) species y.

With these parameter values, Figure 2.3 illustrates that the genotype $AaBb$ generally has the highest population sizes/densities. Because $AaBb$-$AaBb$ matings produce all of the other genotypes, all genotypes and phenotypes, AB, Ab, aB and
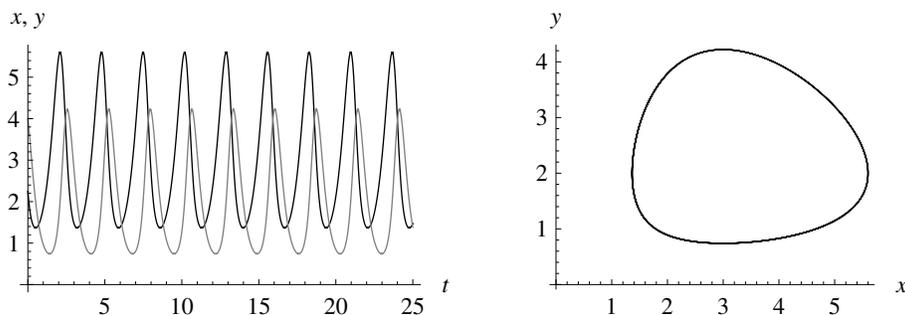
Figure 2.2: We choose $b = k = m = f = l = 1$ and $a = 2, c = 3$. Initial values are $x_{10} = x_{20} = x_{30} = x_{40} = x_{50} = x_{60} = x_{70} = x_{80} = x_{90} = 0.25, y_0 = 4$. All solutions, except for the equilibrium solution $x = 3$, $y = 2$, are periodic so all solution curves are closed curves in the graph on the right.

ab coexist.

Choosing values for k and m to be greater than values for f and l (using the same initial conditions and values for a, b, and c in (2.1)) causes the two-locus, two-allele problem to typically result in a stable solution, such as the stable equilibrium point illustrated in Figures 2.4 and 2.5.

Giving the "weakest" genotype, usually the genotype of type $aabb$ (expected phenotype is ab), an advantage with a low catchability rate with respect to the other genotypes, such as $l = 0.8$, helps this genotype to persist, even with relatively low population size/density. Regardless, in this simulation the genotype of type $AaBb$ again has the highest population density as illustrated in Figure 2.6.

From Figure 2.6 we see the stabilization points (or **equilibrium** or **rest points**) for the predator and prey populations. In Figure 2.7, the population density for the prey (black) converges to $x = 3$ and the population density of the predator (grey) converges to $y = 2.1$.
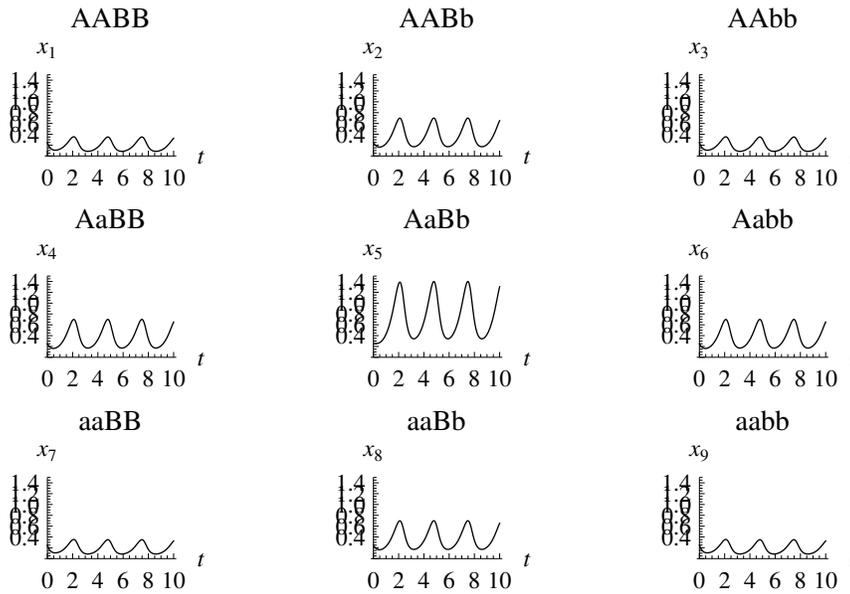
Figure 2.3: Because the genotype of type $AaBb$ has an advantage over the others, all genotypes coexist because random matings of this genotype produce all other genotypes. (Refer to the **Appendix**.)
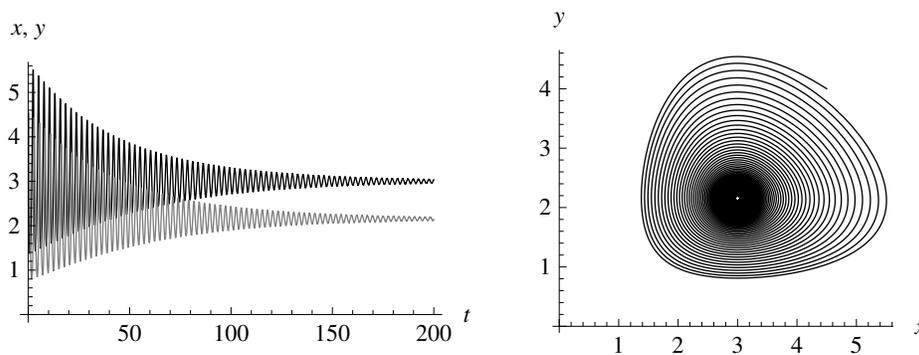
Figure 2.4: The parameter values used are $k = 1$, $m = 2$, $f = 0.8$ and $l = 0.8$, which result in stabilization.
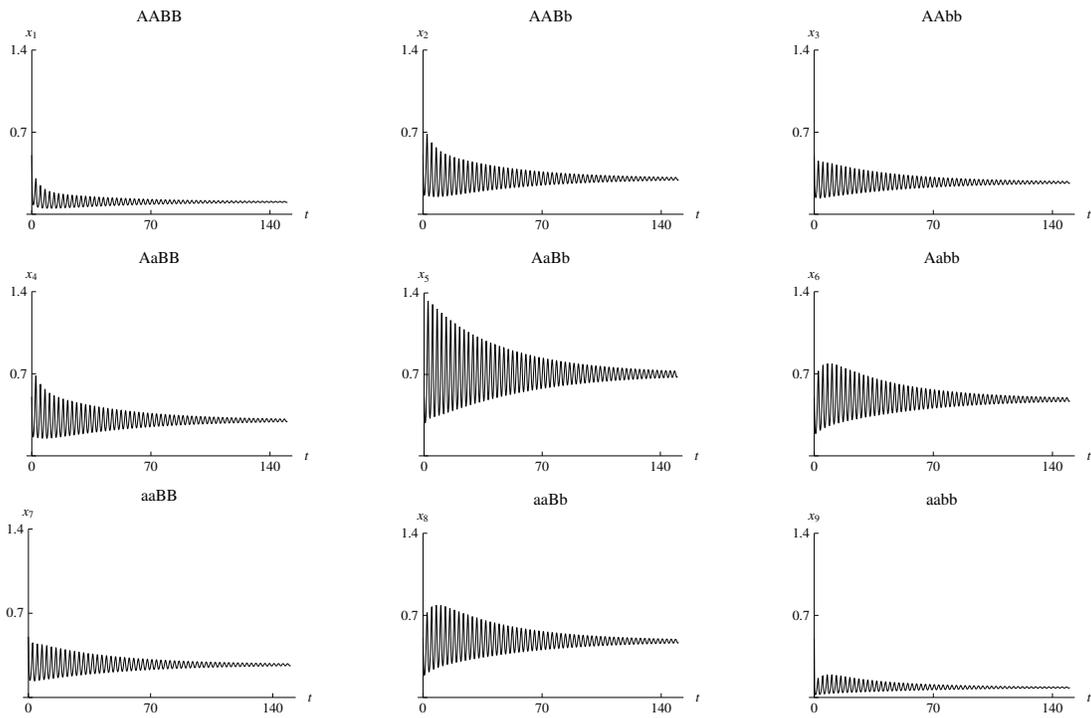
Figure 2.5: Stabilization with $k = 1$, $m = 2$, $f = 0.8$ and $l = 0.8$. The size/density of the population of each expected phenotype stabilizes.
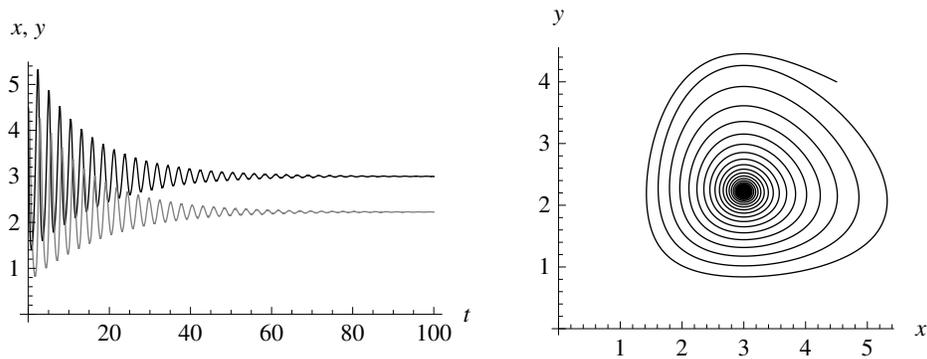
Figure 2.6: Stabilization with $k = 1.2$, $m = 3$, $f = 0.7$ and $l = 0.7$.
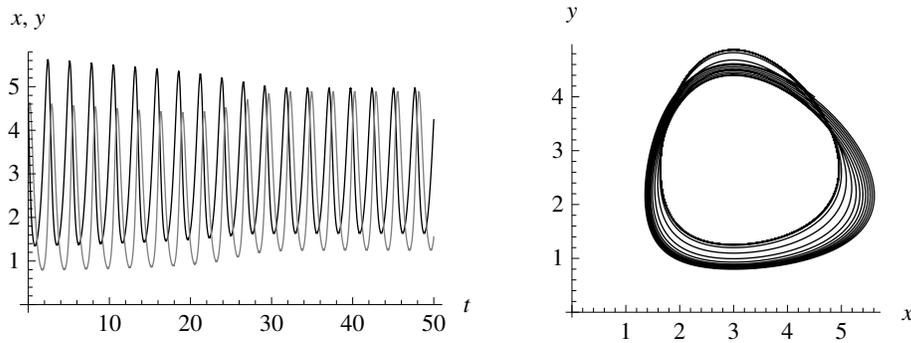
Figure 2.7: A limit cycle with $k = 0.9$, $m = f = 1$ and $l = 0.75$

Our final example using this model illustrates that when the expected weakest genotype of type, *aabb*, dominates, both the a and b alleles may go to fixation. (In the gene pool, **fixation** means that of two variants of a particular allele (gene), only one of the alleles remains after a period of time.) Refer to Figure 2.7. Both prey and predator populations/densities remain at almost oscillatory rates as shown in the graph on the left in Figure 2.7).

In Figure 2.8, observe that we expected the "strongest" (lowest mortality rate than the other prey genotypes experience with the predator) genotypes to survive, but their population rates were continuously decreasing. At some point, around $x_i = 30$, all vanish. At the same time, the population density of the *aabb* genotype type starts to increase and stabilizes in the range from 1.8 to 5: the a and b alleles go to fixation.
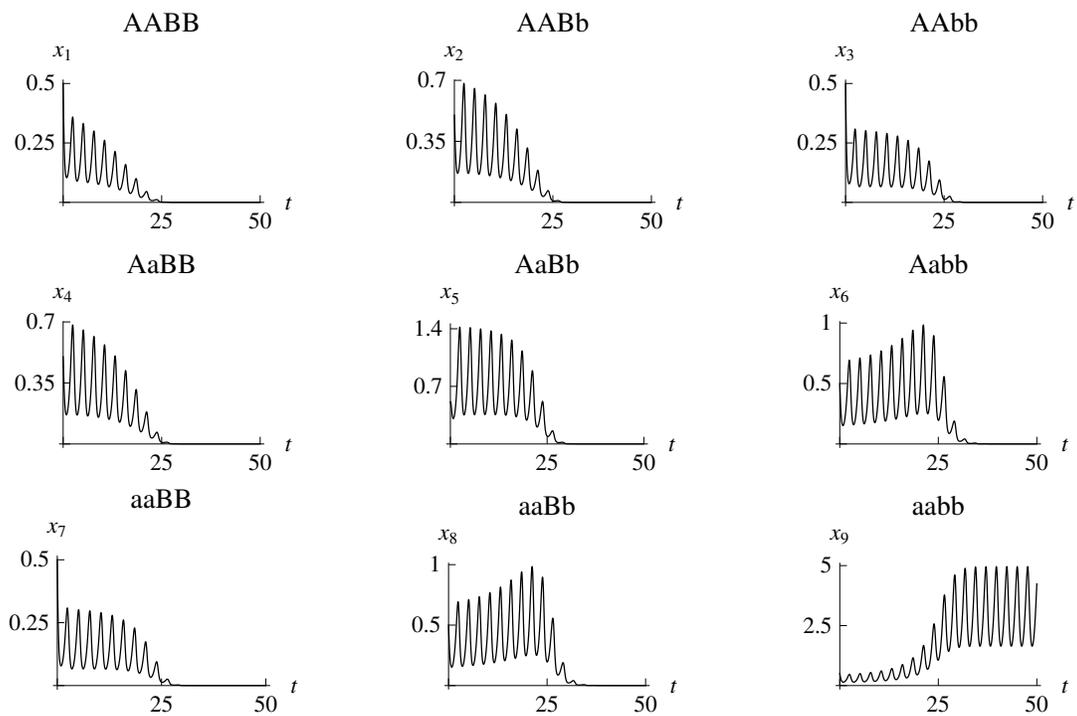
Figure 2.8: Limit cycles with $k = 0.9, m = f = 1$ and $l = 0.75$. The organism of genotype $aabb$ survives; the a and b alleles go to fixation.

<div align="center">

CHAPTER 3

EPISTASIS

</div>

As discussed previously, **epistasis** occurs when the genotype results in a phenotype different from that expected. We model epistasis in a predator-prey relationship by forcing the *catchability* (given by the k_i, m_i, or f_i terms) of one genotype of a particular phenotype to be greater or smaller than other organisms with the same expected phenotype. Recall that the x_i (or x_i') (prey) and y (predator) population densities are given by equations (2.3) and that a is the growth rate of species x_i (prey) while c is the death (or emigration) rate of species y (predator).

We use the same initial conditions and parameter values for a, b, and c as in the previous simulations.

3.1 Example 1. k_5 and l_9 are the greatest

Setting k_5 greater than the other k_i values models epistasis by giving the genotype *AaBb* (expected phenotype *AB*) a higher prey-induced death rate than the other organisms with phenotype *AB*. We choose l_9 to be large as well because some would argue that the *ab* phenotype would often be the weakest, which we continue to assume throughout the examples unless otherwise stated. Figure 3.2 illustrates that the population rate of the type *AABB* genotype (expected phenotype *AB*) stabilizes around the point ($x_1 = 0.006$). This happens because of several factors: the catchability of this genotype is high ($k_1 = 1.2$) and population rates for the genotypes of type *AABb*, *AaBB* and *AaBb* (same expected phenotype *AB*) are low. The population size rate for the genotype of type *aabb* with high catchability parameter $l_9 = 3$ stabilizes at the point around $x_5 = 0.15$. The organisms with genotypes *Aabb* and *aaBB* have the highest population rates (around 0.9).
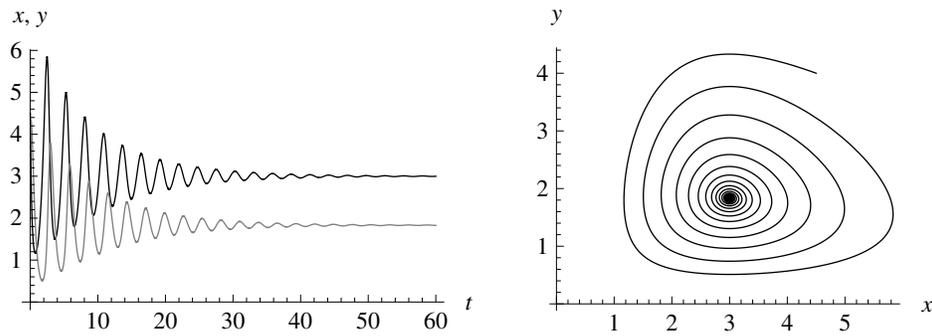
Figure 3.1: Stabilization with $k_1 = 1.2, k_2 = 1.2, k_4 = 1.2, k_5 = 3, l_9 = 3, m_3 = 0.8, m_6 = 0.8, f_7 = 0.8, f_8 = 0.8$
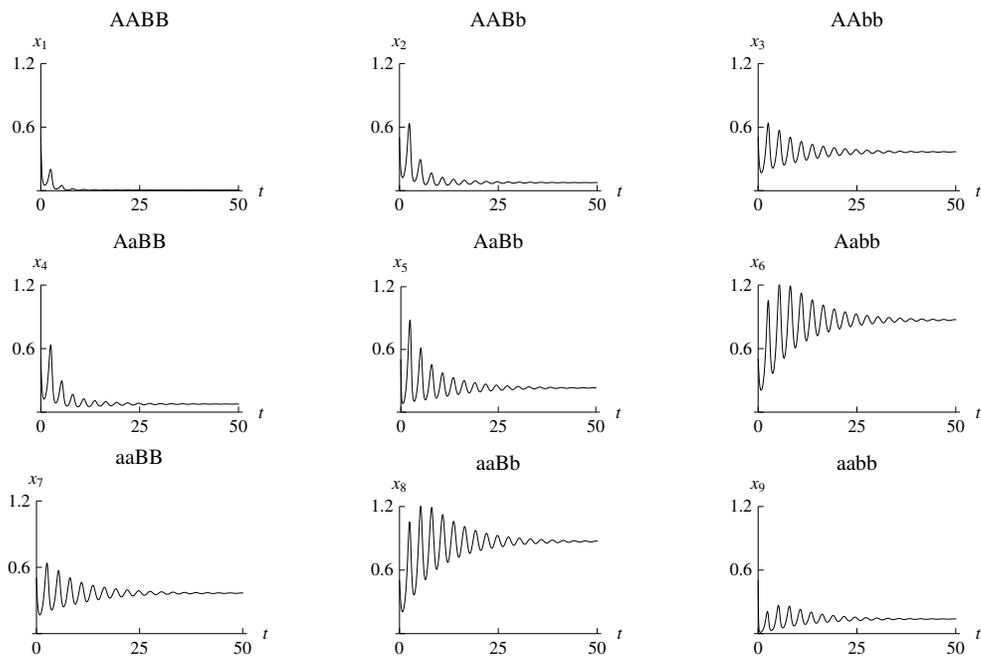
Figure 3.2: Stabilization with $k_1 = 1.2, k_2 = 1.2, k_4 = 1.2, k_5 = 3, l_9 = 3, m_3 = 0.8, m_6 = 0.8, f_7 = 0.8, f_8 = 0.8$
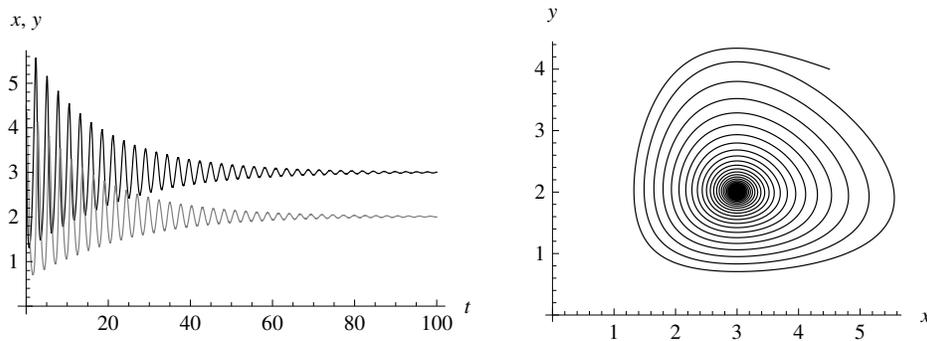
Figure 3.3: Stabilization with $k_1 = 3$, $k_2 = 1.2$, $k_4 = 1.2$, $k_5 = 1.2$, $l_9 = 3$, $m_3 = 0.8$, $m_6 = 0.8$, $f_7 = 0.8$, $f_8 = 0.8$

3.2   Example 2. (a) k_1 and l_9 are the greatest

Setting k_1 greater than the other k_i values models epistasis by giving the genotype $AABB$ (expected phenotype AB) a higher prey-induced death rate than the other organisms with phenotype AB. Using these parameters, stabilization took nearly twice as long as in the previous model. Refer to Figures 3.3 and 3.4.

The high value of the catchability parameter $k_1 = 3$ forces the organism of genotype $AABB$ to very small levels, faster than in the previous example. The population size rate for the organisms of type $aabb$ with catchability parameter $l_9 = 3$ is smaller too (around 0.1). On the other hand, the organisms with lower catchability values (genotypes $AAbb$, $Aabb$, $aaBB$, and $aaBb$) have the highest population densities.

Since $k_5 = 1.2$ and mating between the organism of type $AaBb$ produces all the other genotypes, $Aabb$ and $aaBb$ (expected phenotype is Ab and aB respectively) have the highest rates and more chances for survival.
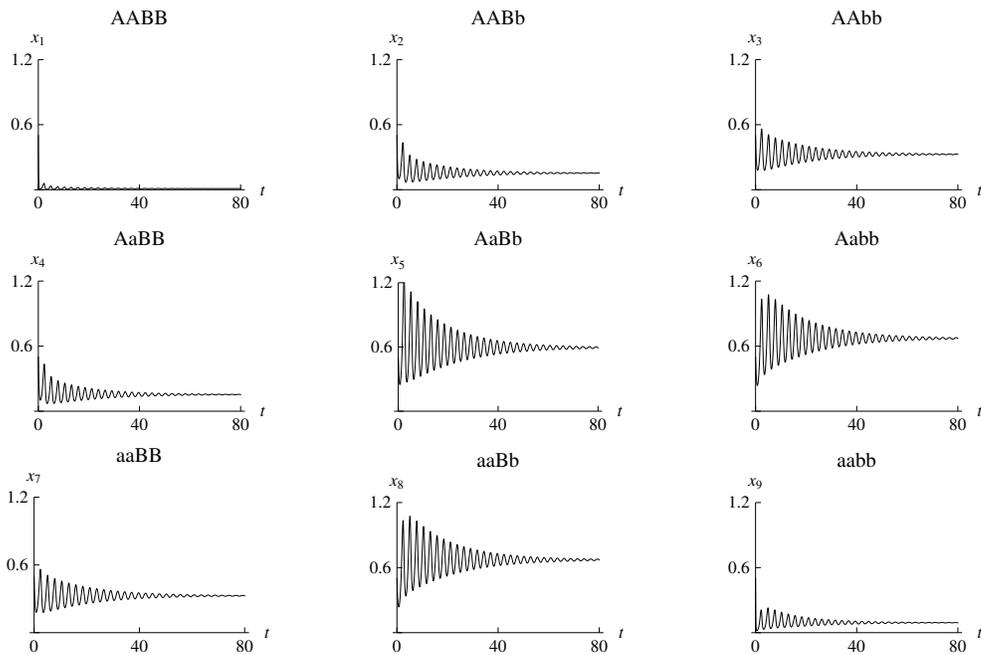
Figure 3.4: Stabilization with $k_1 = 3$, $k_2 = 1.2$, $k_4 = 1.2$, $k_5 = 1.2$, $l_9 = 3$, $m_3 = 0.8$, $m_6 = 0.8$, $f_7 = 0.8$, $f_8 = 0.8$
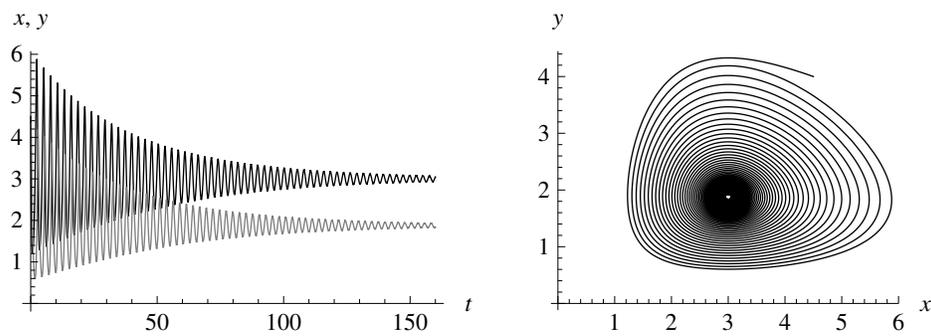
Figure 3.5: Stabilization with $k_1 = 3, k_2 = 1, k_4 = 1, k_5 = 1, l_9 = 3, m_3 = 1, m_6 = 1, f_7 = 1, f_8 = 1$
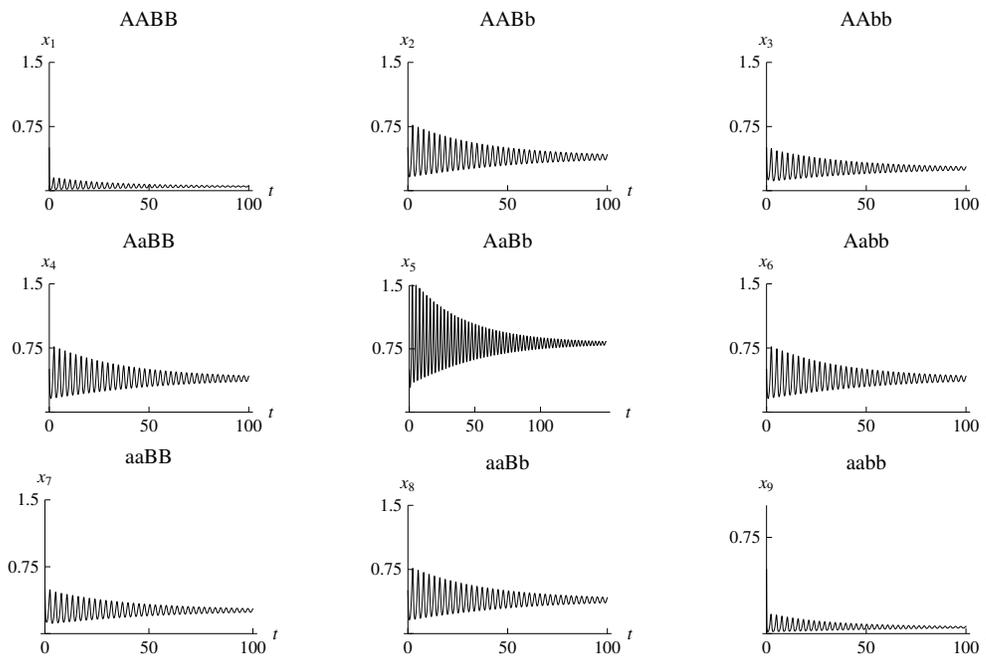
Figure 3.6: Stabilization with $k_1 = 3, k_2 = 1, k_4 = 1, k_5 = 1, l_9 = 3, m_3 = 1, m_6 = 1, f_7 = 1, f_8 = 1$

3.3 Example 3. (b) k_1 and l_9 are the greatest while all other parameter values are 1.

This case is interesting by the "slowness" in the rate at which the system stabilizes when compared to the previous two examples. From Figure 3.5 we see that both populations need around 170 steps to stabilize at one point.

This example numerically indicates that all genotypes except $AABB$ and $aabb$ find stabilization at the hight value population rate point. Both types $AABB$ with dominant alleles and the weakest genotype $aabb$ with recessive alleles have high catchability value $k_1 = l_9 = 3$ and stabilize at the point close to zero (that is, they are close to extinction).
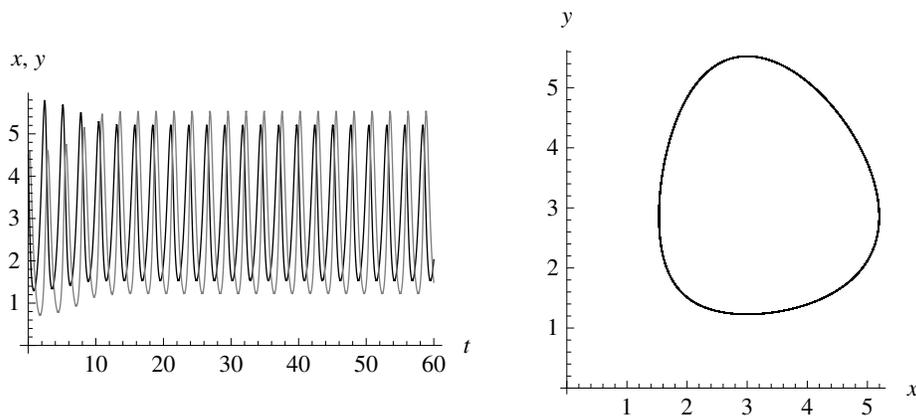
Figure 3.7: Limit cycle with $k_1 = 0.7, k_2 = 1, k_4 = 1, k_5 = 1, l_9 = 1, m_3 = 1, m_6 = 1, f_7 = 1, f_8 = 1$. The A and B alleles go to fixation so the genotype $AABB$ is the only one to survive.

3.4   Example 3. k_1 is the smallest: the catchability of the organism with genotype $AABB$ is the lowest.

Setting k_1 smaller than the other k_i values models epistasis by giving the genotype $AABB$ (expected phenotype AB) a lower prey-induced death rate than the other organisms with phenotype AB. Figure 3.7 illustrates that the population/density rate of the prey changes from approximately $x = 1.5$ to $x = 5.3$ while the predator population/density rate changes from approximately $y = 1.2$ to $y = 5.6$.

Setting k_5 smaller than the other k_i values models epistasis by giving the genotype $AaBb$ (expected phenotype AB) a lower prey-induced death rate than the other organisms with phenotype AB. The example illustrates an interesting situation. The small catchability rate of the species with genotype $AABB$ with dominant alleles A and B and the same catchability rates for the other species with genotypes $AABB$ survive the competition between the other genotypes and forces them to extinction. A critical point around $t = 10$ can be observed. In this case, the A and B alleles go
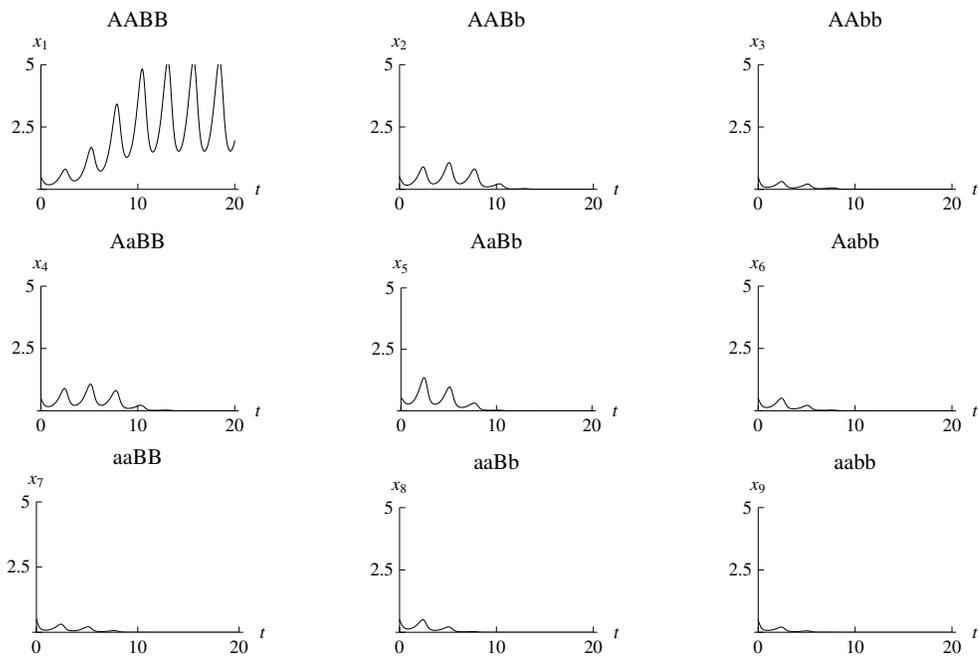
Figure 3.8: Limit cycle with $k_1 = 0.7, k_2 = 1, k_4 = 1, k_5 = 1, l_9 = 1, m_3 = 1, m_6 = 1, f_7 = 1, f_8 = 1$. The A and B alleles go to fixation to the genotype $AABB$ is the only one to survive.
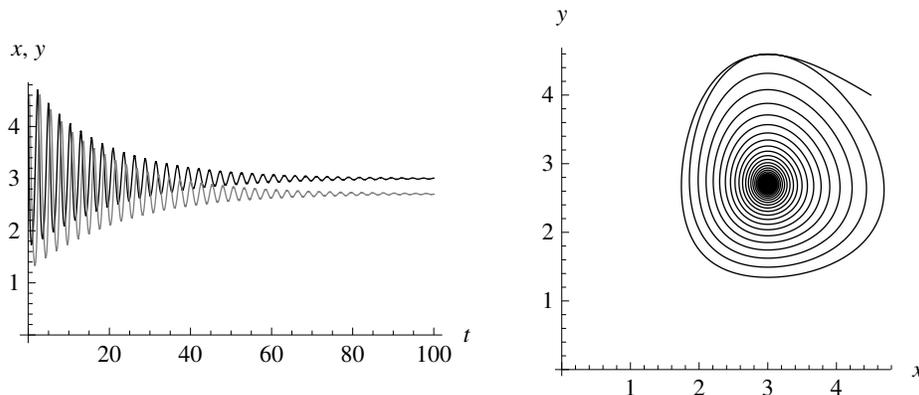
Figure 3.9: Stabilization with $k_1 = 1$, $k_2 = 1$, $k_4 = 1$, $k_5 = 0.5$, $l_9 = 3$, $m_3 = 0.75$, $m_6 = 0.75$, $f_7 = 0.75$, $f_8 = 0.75$

to fixation.

3.5   Example 4. k_5 is the smallest; l_9 is the greatest.

Both predator and prey populations stabilize at almost the same rate (around 2.6 for the predator and 3 for the prey with the parameter values we use) as shown in Figure 3.10.

When the growth advantage is given to the x_5 organism (with genotype $AaBb$) and because random mating of the organism with genotype $AaBb$ produces all of other genotypes, we observe the stabilization shown in Figure 3.11. Since $x_5' = 2ax(ps + rq) - k_5 x_5 y$, all other genotypes stabilize as well.

3.6   Example 5. (a) l_9 is the largest; all other parameter values are equal.

With these parameter values, the example, which is graphically illustrated in Figure 3.11, illustrates how the k-phenotype group (x_1, x_2, x_4, and x_5) can be the strongest (or survive with the highest population/density) with respect to the population sizes/densities of the other genotypes. In this case, the A and B alleles go to fixation.
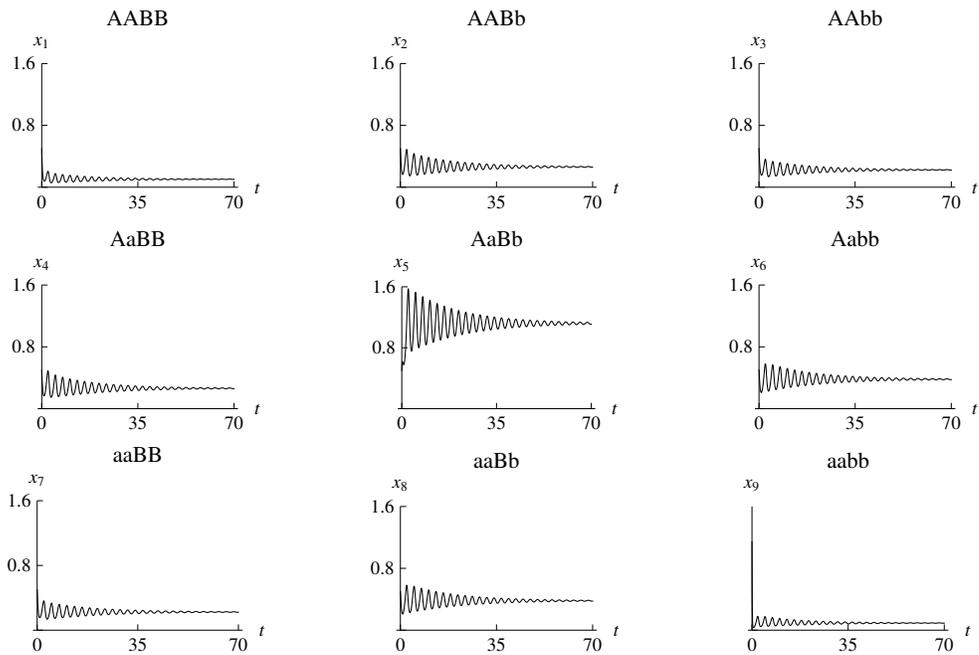
Figure 3.10: Stabilization with $k_1 = 1$, $k_2 = 1$, $k_4 = 1$, $k_5 = 0.5$, $l_9 = 3$, $m_3 = 0.75$, $m_6 = 0.75$, $f_7 = 0.75$, $f_8 = 0.75$
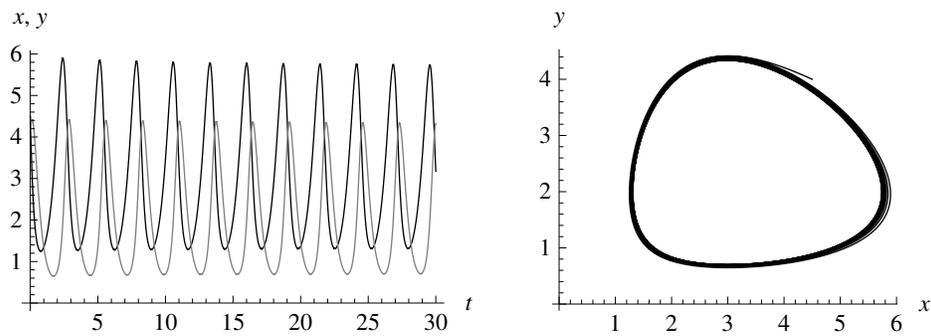
Figure 3.11: Limit cycle with $k_1 = 1$, $k_2 = 1$, $k_4 = 1$, $k_5 = 1$, $l_9 = 3$, $m_3 = 1$, $m_6 = 1$, $f_7 = 1$, $f_8 = 1$.
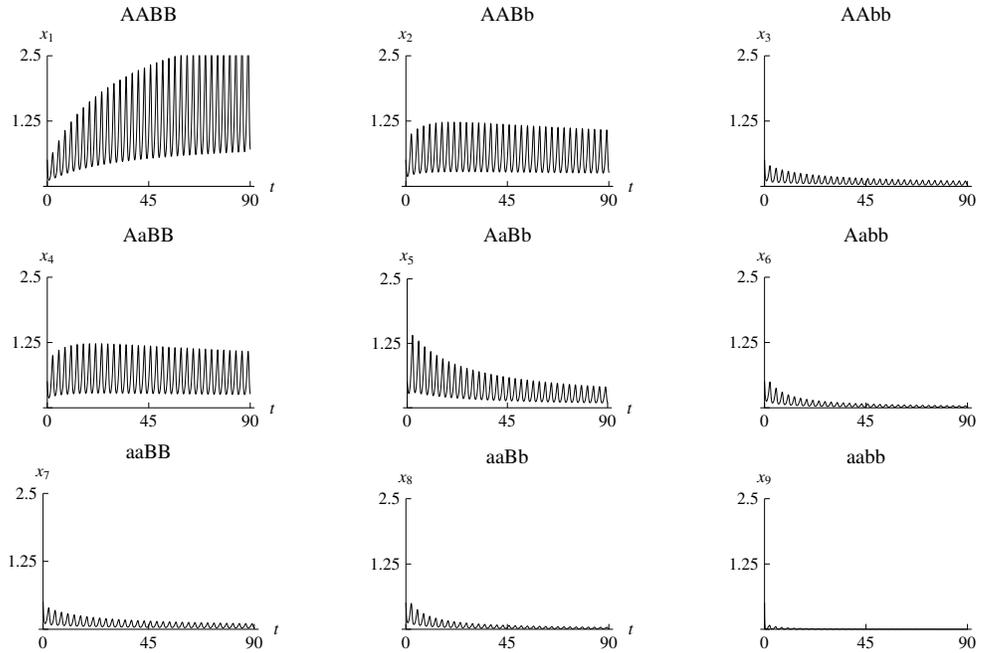
Figure 3.12: Limit cycle with $k_1 = 1$, $k_2 = 1$, $k_4 = 1$, $k_5 = 1$, $l_9 = 3$, $m_3 = 1$, $m_6 = 1$, $f_7 = 1$, $f_8 = 1$

Making the weakest species with genotype $aabb$ (because the genes a and b are recessive) more catchable by choosing large catchability parameter values forces the genotype to extinction. We have also observed that a limit cycle sometimes occurs. Population sizes/densities of the species with genotypes $AABb$ and $AaBB$ decrease extremely slowly with the selected parameter values as shown in Figure 3.12.

3.7 Example 5. (b) Parameter value l_9 is the greatest and we choose the parameter values m_6 and f_8 to be smaller.

As in the previous example (refer to Figures 3.9 and 3.10), high population size/density of the organism with genotype $AaBb$ forces the system to stabilize. In this case, the organisms with genotypes $AaBb$, $Aabb$ and $aaBb$ have the highest values be-
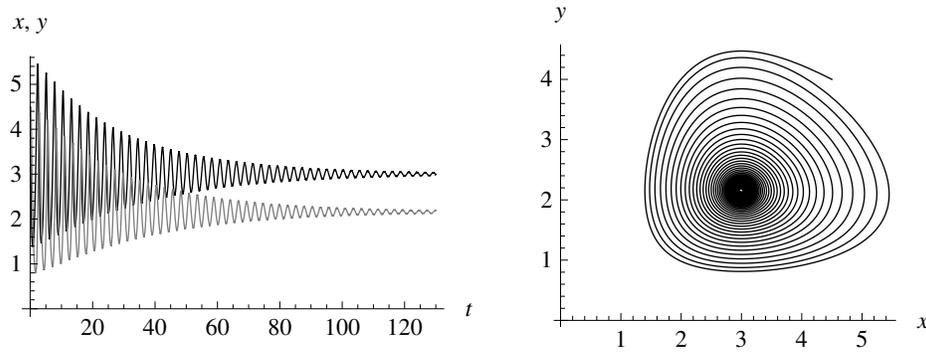
Figure 3.13: Stabilization with $k_1 = 1$, $k_2 = 1$, $k_4 = 1$, $k_5 = 1$, $l_9 = 3$, $m_3 = 1$, $m_6 = 0.7$, $f_7 = 1$, $f_8 = 0.7$

cause their catchability parameters are the lowest with respect to the other population sizes/densities of the species with the other genotypes. The lowest population sizes/densities, as we have seen in the simulation, are the organisms with genotypes *AABB* and *aabb* (both around 0.1).
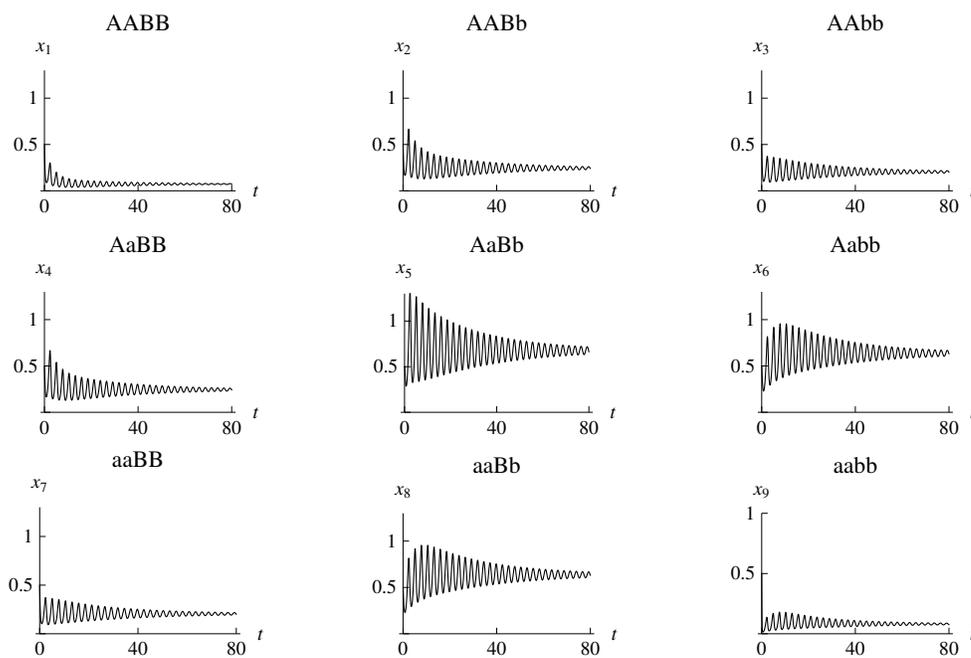
Figure 3.14: Stabilization with $k_1 = 1$, $k_2 = 1$, $k_4 = 1$, $k_5 = 1$, $l_9 = 3$, $m_3 = 1$, $m_6 = 0.7$, $f_7 = 1$, $f_8 = 0.7$

CHAPTER 4

EPISTASIS WITH DANGEROUS PREY

In the previous sections, epistasis was modeled by making the *catchability* (given by the k_i, m_i, or f_i terms) of one genotype different from other organisms with the same expected phenotype.

Another way to model the situation is to examine how the effect of specific genotypes of the prey differ from that of others on the predator. For example, epistasis can manifest itself if some genotypes of the prey population have a negative effect on the growth of the predator population, such as a poisonous prey.

To take into consideration the effect that specific genotypes of the prey have on the growth rate of the predator, the y equation in equation (2.3) becomes:

$$y' = y(-c + (d_1 x_1 + d_2 x_2 + d_3 x_3 + d_4 x_4 + d_5 x_5 + d_6 x_6 + d_7 x_7 + d_8 x_8 + d_9 x_9))$$

$$y' = y \left(-c + \sum_{i=1}^{9} d_i x_i \right). \tag{4.1}$$

As expected, when $d = d_1 = d_2 = \cdots = d_9$, equation (4.1) reduces to $y' = y(-c + dy)$. Of interest is when the d_i values differ and, specifically, when some d_i values are negative. Because the $d_i x_i y$ values indicate how an $x_i - y$ interaction affects the growth of the y (predator) population, negative values of d_i are interpreted to mean that $x_i - y$ interactions have a negative effect on the growth rate of the predator. For example, a negative value of d_i could be interpreted that the prey with population/density x_i is lethal (such as being poisonous) to the predator with population/density y.

In the following simulations, we use the same parameter values for a, b, c and initial conditions as before. Then, the case with lethal (or poisonous) prey can be illustrated with several examples.
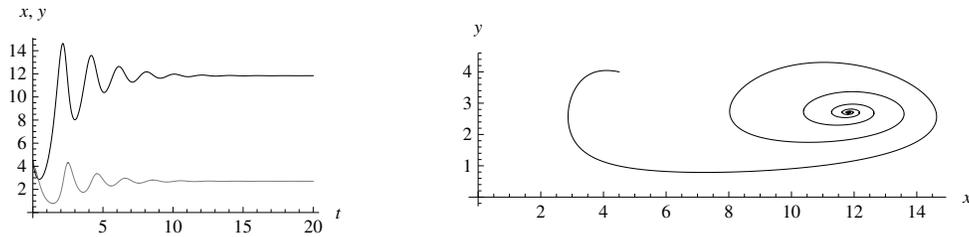
Figure 4.1: Stabilization with a negative value of d_5: $k_1 = k_2 = k_4 = 1$, $k_5 = 0.5$, $l_9 = 3$, $m_3 = m_6 = f_7 = f_8 = 0.75$, $d_5 = -1, d_1 = d_2 = d_3 = d_4 = d_6 = d_7 = d_8 = d_9 = 1$

.

4.1 Example 1. Genotype of type $AaBb$ is poisonous to the prey; parameter value used: $d_5 = -1$

Setting $d_5 = -1$ and leaving the other d_i values positive models epistasis by making the prey of genotype $AaBb$ (expected phenotype AB) poisonous to the predator. On the other hand, the other prey with phenotype AB continue to positively affect the predator's growth rate. In this simulation, the advantage is given to the prey with population/density x_5. The negative value of d_5 result in a negative effect on the growth rate of the predator y. As we see in Figure 4.1, stabilization appears to occur very quickly, at approximately $t = 12$. When $d_5 = -1$ we have much higher population rates of the species with genotype $AaBb$ (expected phenotype AB) as illustrated in Figure 4.2.

In the previous sections, several examples used the same values for all *catchability* parameters (refer to **Example 4**, Figures 3.9 and 3.10) yet the population sizes evolved differently. Now, when the species with genotype $AaBb$ is poisonous to the prey, the phenotype rates for all prey are much higher. We have illustrated that poisonous prey with genotype $AaBb$ increases the population size/density of the prey population/density. Additionally, as with the previous examples both prey and
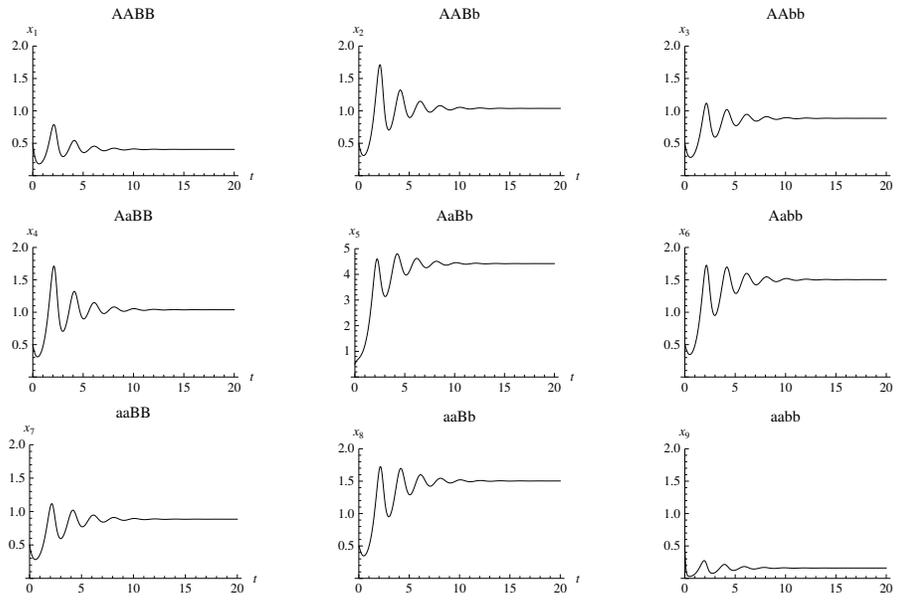
Figure 4.2: Stabilization with a negative value of d_5

predator have close values after stabilization, but the simulation suggests that the difference in the values is much greater.

Because of the influence of poisonous/lethal prey, we see a noticeable difference between population sizes/densities of the genotypes of type $AaBb$ and other types. With the parameter values used here, population sizes/densities of genotype type $AaBb$ are around 2.7 times greater than the population sizes/densities of genotypes $AABb$, $Aabb$ and $aaBb$. These values are more than four times greater than the population sizes/densities of the genotypes of type $AAbb$, $aaBB$, $AABb$ and $AaBB$. Overall, the simulations indicate an increase of more than 10 times greater then the growth rate of the genotype of type $AaBB$ rate and more then 30 times greater than the (weakest) of type $aabb$.
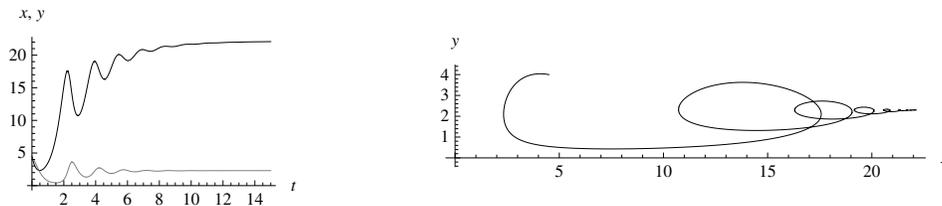
Figure 4.3: Stabilization with $d_5 = -1$ and $k_5 = 0.5$, $k_1 = m_j = f_j = 1.3$, $k_2 = k_4 = l_9 = d_i = 1$ for $i \neq 5$

4.2 Example 2: An interesting case is observed if $d_5 = -1$ and $k_5 = 0.5$, $k_1 = m_j = f_j = 1.3$, $k_2 = k_4 = l_9 = d_i = 1$ for $i \neq 5$.

Again, stabilization occurs very quickly (around $t = 12$), but the difference between populations of prey and predator is much larger: the prey's population is around 10 times greater than that of the predator. In this simulation, poisonous prey of genotype $AaBb$ (i.e, the prey with population x_5 is poisonous to the predator) cause the population size of the prey with this genotype to be many times greater than the population size of other types of the prey with different genotypes.

4.3 Example 3: A Limit Cycle with $d_5 = -1$ and the same parameter values for k_i, m_j, f_j, l_9, d_i for $i \neq 5$ as used in the previous simulations.

Giving a competitive advantage to the prey with genotype $AaBb$ can result in a limit cycle as shown in Figure 4.5.

Figure 4.6 illustrates a situation similar to that illustrated in Figure 4.5. The poisonous prey of genotype $AaBb$ sees its population size/density to finally be more than twice greater than the prey populations of the other genotypes.
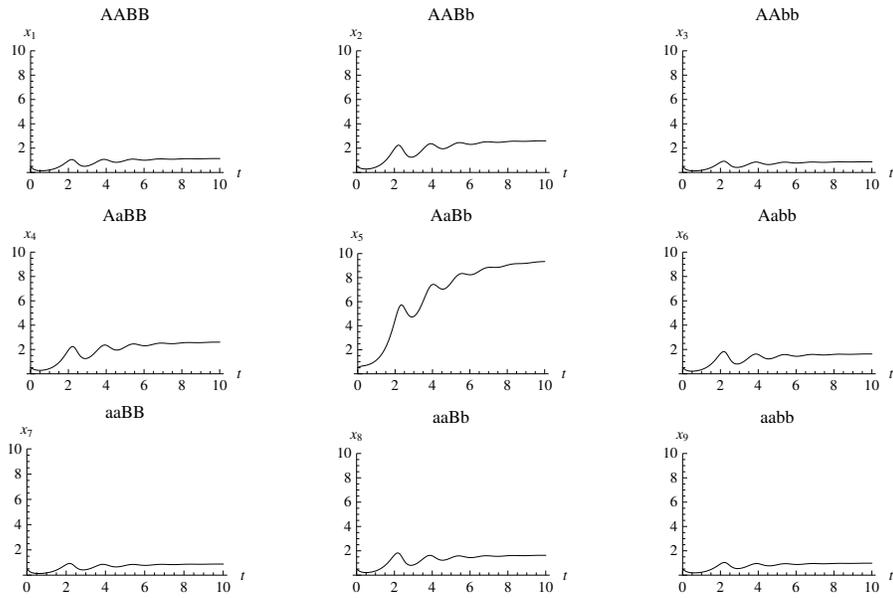
Figure 4.4: Stabilization with $d_5 = -1$ and $k_5 = 0.5$, $k_1 = m_j = f_j = 1.3$, $k_2 = k_4 = l_9 = d_i = 1$ for $i \neq 5$
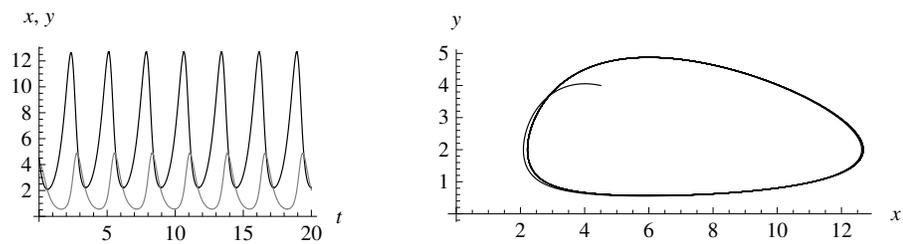
Figure 4.5: A limit cycle with $d_5 = -1$ and all parameter values $k_i = m_j = f_j = l_9 = d_i = 1$ for $i \neq 5$
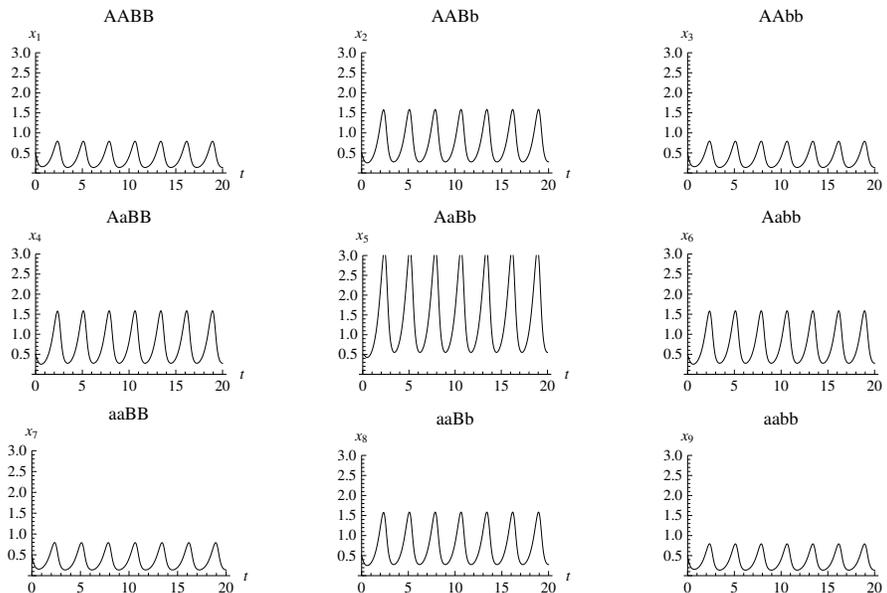
Figure 4.6: Cycle with $d_5 = -1$ and all $k_i = m_j = f_j = l_9 = d_i = 1$ for $i \neq 5$

4.4   Example 4: Predator extinction

Setting more of the d_i parameter values to be negative gives additional advantage to the prey, which is illustrated in Figure 4.7.

In the case illustrated in Figure 4.7, the population/densities of prey of all types increases and forces the predator to extinction. The example illustrates that poisonous prey do not benefit a predator susceptible to the poison of the prey.

Figure 4.8 illustrates that the increase of the prey with genotype $AaBb$ rate causes extreme population/density fluctuations.

4.5   Example 5: d_6 and d_8 are negative

Setting d_6 and d_8 to be negative and leaving the other d_i values positive models epistasis by making the prey of genotypes $Aabb$ (expected phenotype Ab) and $aaBb$ (expected phenotype aB) poisonous to the predator. On the other hand, the other prey with phenotypes Ab and aB continue to positively affect the predator's growth
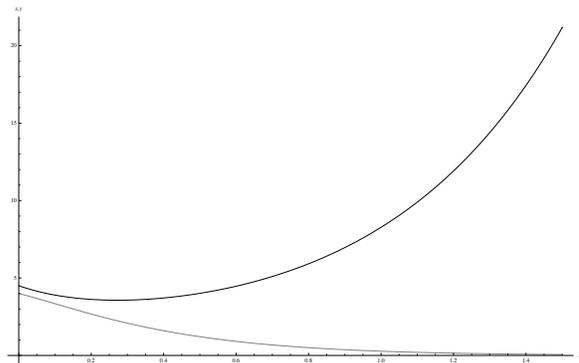
Figure 4.7: We use $d_5 = d_6 = d_8 = -1$ and all $k_i = m_j = f_j = l_9 = d_i = 1$ for $i \neq 5, 6, 8$
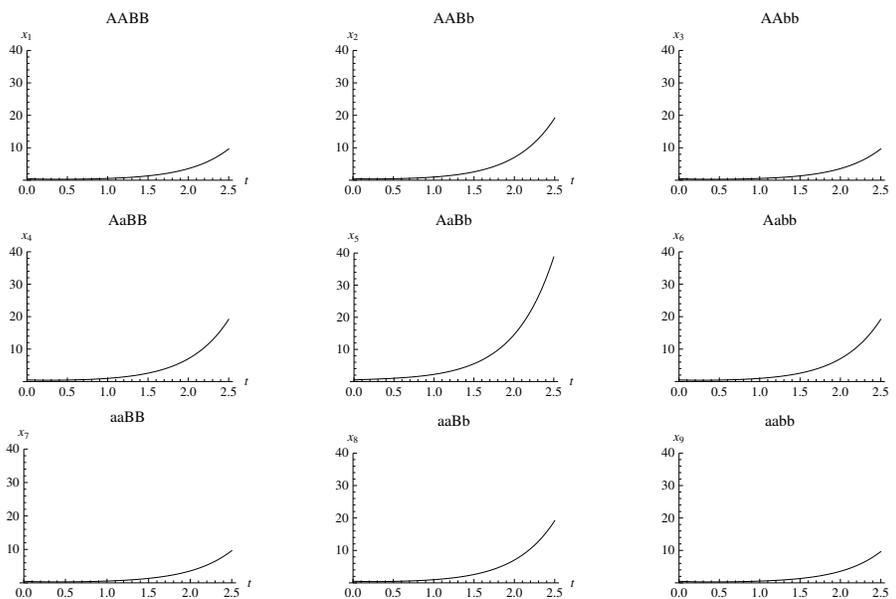
Figure 4.8: Predator extinction when the parameter values are $d_5 = d_6 = d_8 = -1$ and all $k_i = m_j = f_j = l_9 = d_i = 1$ for $i \neq 5, 6, 8$
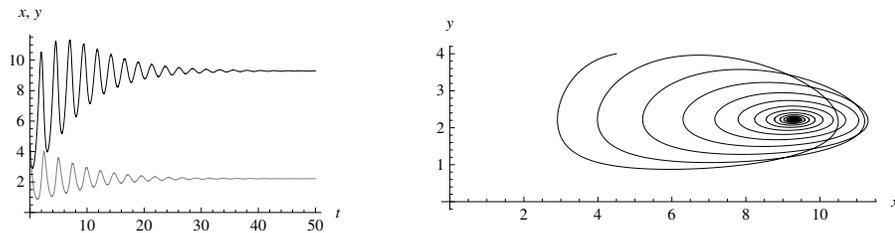
Figure 4.9: Stabilization with $d_6 = d_8 = -1$, $l_9 = 3$ and all $m_j = f_j = 0.75$, $k_i = d_i = 1$, for $i \neq 6, 8$

rate. In Figure 4.9, stabilization of the prey population approachest $x = 9.5$, while the predator population approaches $y = 2.5$.

It is interesting to observe in Figure 4.10 that in spite of the poisonous preys with genotypes $Aabb$ and $aaBb$, the $AaBb$ has the higher population rate. As before, with low catchability ($k_5 = 1$) this genotype has an advantage over other types, even poisonous ones or with same/lower catchability rates.

Because of their poisonous advantage, the two types $Aabb$ (x_6) and $aaBb$ (x_8) have the second (after $AaBb$) highest population rates. The population size of the organism of type $aabb(x_9)$ is close to zero. This is the weakest type because it has the highest catchability parameter $l_9 = 3$.

4.6   Example 6: Both d_6 and d_8 are negative: a limit cycle occurs

Population sizes/densities of all genotypes (except $AABB$) are decreasing. Some of them (genotypes $aabb$, $AAbb$ and $aaBB$) approach zero quickly. Poisonous genotypes ($Aabb$ and $aaBb$) have an advantage, but fail to survive. Refer to Figures 4.11 and 4.12.

The populations sizes/densities for the species with genotypes of $AABb$ and $AaBB$ decrease very slowly and have strong oscillations. Further calculations that are not illustrated in the figures show that the rates of x_2 and x_4 become lower than
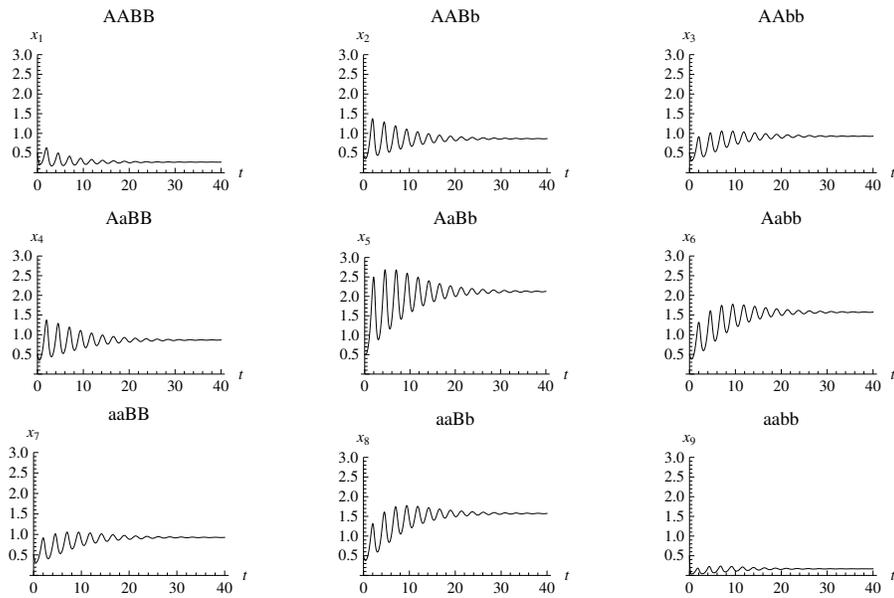
Figure 4.10: Stabilization with $d_6 = d_8 = -1$, $l_9 = 3$ and all $m_j = f_j = 0.75$, $k_i = d_i = 1$, for $i \neq 6, 8$
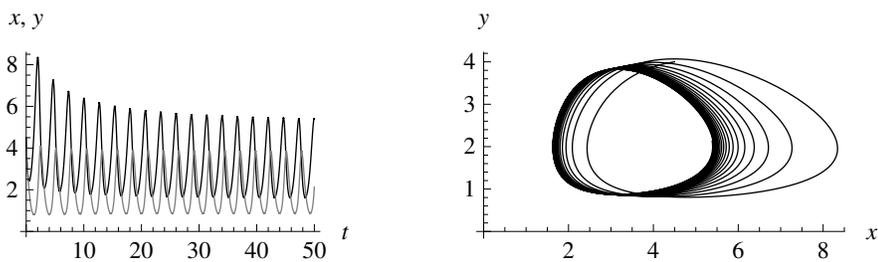
Figure 4.11: A limit cycle with $d_6 = d_8 = -1$, $l_9 = 3$ and all $k_i = m_j = f_j = d_i = 1$ for $i \neq 6, 8$
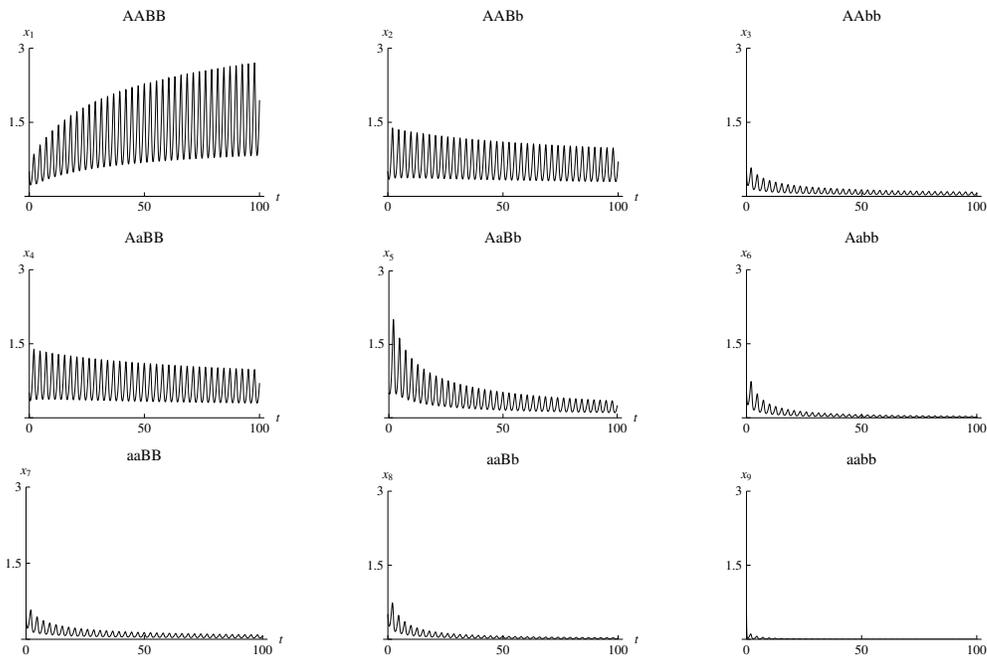
Figure 4.12: A limit cycle with $d_6 = d_8 = -1$, $l_9 = 3$ and all $k_i = m_j = f_j = d_i = 1$ for $i \neq 6, 8$

1 only around the point $t = 400$. When $t = 700$ the situation does not change: they continue decreasing.

4.7   Example 7.

In the case when one of the types has $d_i = -2$, we can observe a sudden growth of the prey population. The growth is so extreme, that considerable difficulties arise in the numerical algorithms after some point in the calculations ($t = 35$ for the situation illustrated in Figure 4.13). Figure 4.13 illustrates behavior similar to that seen in previous examples with population rates increasing to some critical point. When t is close to 20, both rates change their behavior. The predator's population starts decreasing rapidly, while the prey's population increases.

Figure 4.14 gives a closer look on the situation in the model. The point around
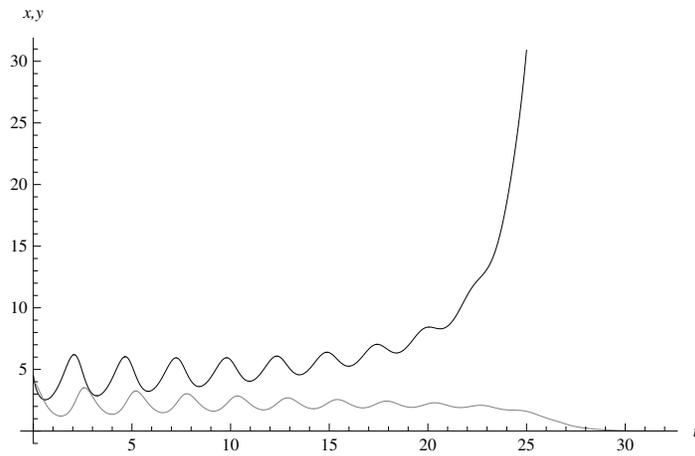
Figure 4.13: Prey population growth with $k_1 = m_3 = m_6 = f_7 = f_8 = 0.75$, $k_2 = k_4 = k_5 = 1$, $l_9 = 3$, $d_1 = -2$ and $d_i = 1$ for $i \neq 1$
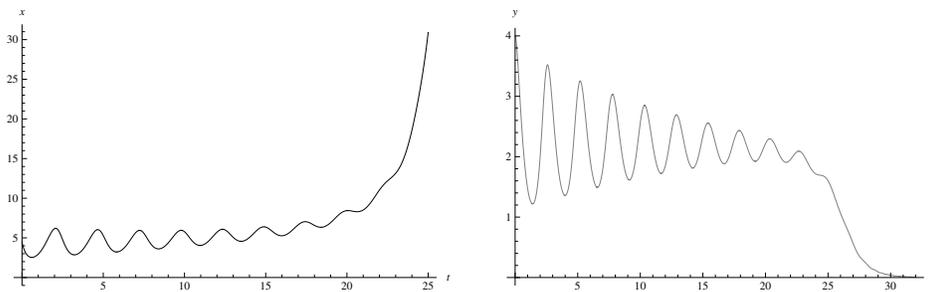
Figure 4.14: Prey population growth with $k_1 = m_3 = m_6 = f_7 = f_8 = 0.75$, $k_2 = k_4 = k_5 = 1$, $l_9 = 3$, $d_1 = -2$ and $d_i = 1$ for $i \neq 1$

$t = 20$ is critical. The predator population size decreases to a very small size for a very short period of time.

Figure 4.15 illustrates the case when the organism of type $AABB$ is both poisonous and has low catchability so it makes a greater impact on the predator's extinction than the other genotypes.
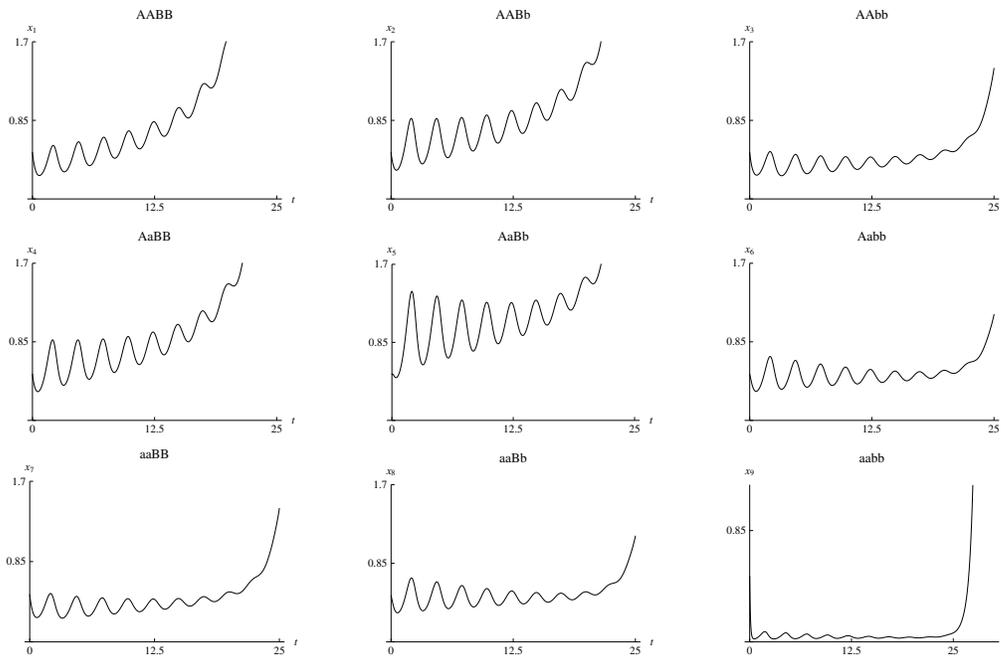
Figure 4.15: Prey population growth with $k_1 = m_3 = m_6 = f_7 = f_8 = 0.75$, $k_2 = k_4 = k_5 = 1$, $l_9 = 3$, $d_1 = -2$ and $d_i = 1$ for $i \neq 1$

4.8   Example 8.

A limit cycle resulting in extreme populaton sizes/densities of both the predator and prey are found in the model with negative parameters as shown in Figure 4.17.

Stabilization of this limit cycle goes slowly. The cycle area expands and stabilizes only around the moment when $t = 1000$ as shown in Figure 4.17. These specific examples show that during each cycle both prey and predator populations can become close to extinction. Nevertheless, the populations are growing after every cycle and never stabilize. Extreme situations like these that are illustrated in Figure 4.18. probably are unlikely to happen in reality. However, extreme changes of population size can be triggered by severe circumstances, such as changes of weather or other external factors.

In Figure 4.18, epistasis is modeled when a particular d_i value differs from other
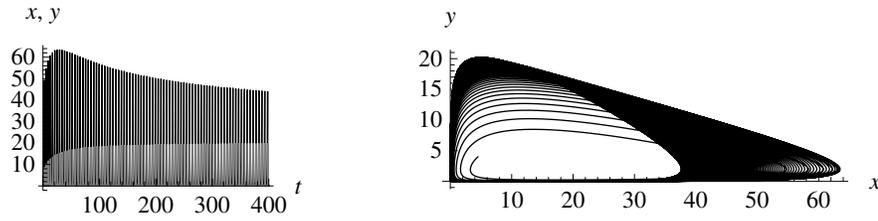
Figure 4.16: Limit cycle with $k_1 = k_2 = k_4 = k_5 = 1$, $l_9 = 3, m_3 = m_6 = f_7 = f_8 = 0.75$, $d_2 = d_6 = d_9 = -2$ and $d_i = 1$ for $i \neq 2, 6, 9$
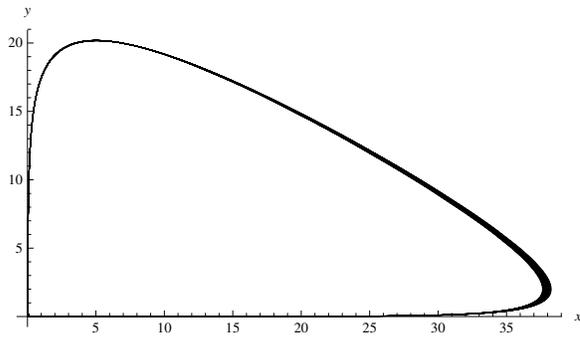
Figure 4.17: Resulting limit cycle with $k_1 = k_2 = k_4 = k_5 = 1$, $l_9 = 3, m_3 = m_6 = f_7 = f_8 = 0.75$, $d_2 = d_6 = d_9 = -2$ and $d_i = 1$ for $i \neq 2, 6, 9$
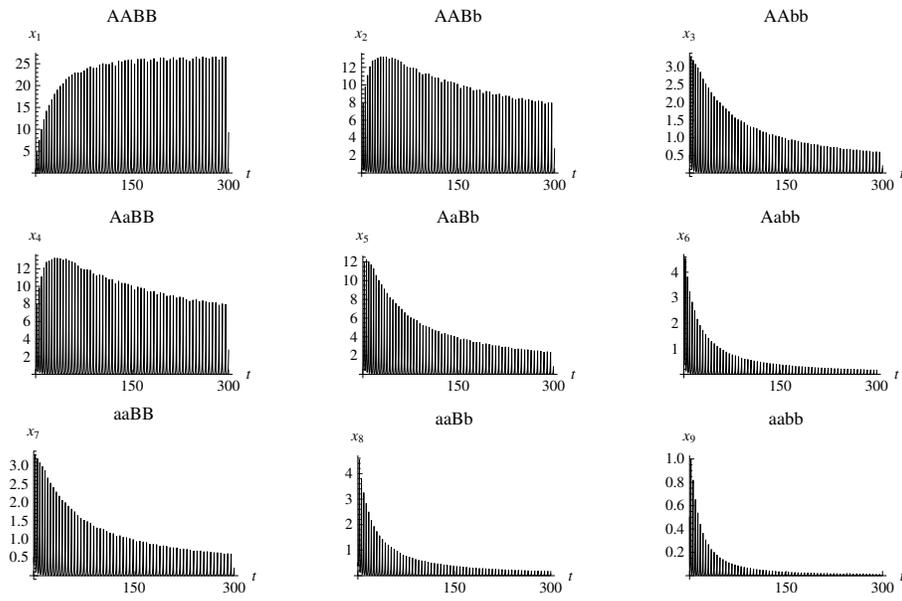
Figure 4.18: Limit cycles with $k_1 = k_2 = k_4 = k_5 = 1$, $l_9 = 3$, $m_3 = m_6 = f_7 = f_8 = 0.75$, $d_2 = d_6 = d_9 = -2$ and $d_i = 1$ for $i \neq 2, 6, 9$. Observe that the A and B alleles go to fixation.

prey that are expected to have the same phenotype.

CHAPTER 5

CONCLUSION

In this paper we have discussed different cases of epistasis of the prey in a two-locus, two-allele problem in a basic predator-prey relationship. After discussing the most famous example of epistasis in humans, *The Bombay Phenotype*, we constructed the main model for a two-locus, two-allele problem with nine genotypes. Then we used different values for "catchability" parameters to examine both population sizes as well as genotypic and phenotypic population/densities.

In our simulations and examples, we saw that in different situations limit cycles or stabilization can occur. The simulations showed that the model was highly sensitive to the different parameters. Some cases illustrated total extinction of the weakest types of the prey, while in other examples all population types survived and a limit cycle occurred. Some interesting examples were observed where the weakest or the strongest types also became extinct. Since random mating of the organism of type $AaBb$ produces all of the other genotypes (and phenotypes), this type had the highest population rate most of the time.

Other models showed situations with dangerous (poisonous) prey. Forcing several types or prey population to be poisonous (lethal to the predator), we modeled the situation where total extinction of the predator population (after some critical moment t) occurred. Other particular examples of dangerous prey situations illustrate cases with relatively rapid stabilization of both populations or cycling of both population rates (with extremely slow speed of stabilization of the cycle).

Future studies might include using different predator-prey models such as epistasis of the predator in a two-locus two-allele problem, epistasis of both predator and prey in the same model, or epistasis in the dangerous predator case. In future studies, we will be able to see how the numerical results obtained here might change or how

the situation might evolve differently if epistasis occurs in both the predator and the prey. Other interesting studies would be to incorporate epistasis into competition, cooperation problems, or host-parasite problems.

Computational Remarks

Mathematica 9.0, [9], was used to create the graphics and perform the computations presented in this paper. Copies of the *Mathematica* notebooks used are available from the thesis director by sending a request for them to Jim Braselton at `jbraselton@georgiasouthern.edu`.

REFERENCES

[1] Martha Abell and James Braselton, *Introductory Differential Equations with Boundary Value Problems*, Third Edition, Academic Press (2010).

[2] Edward Beltrami, *Mathematical Models for Society and Biology*, Second Edition, Academic Press (2013).

[3] Brendan J. M. Bohannan and Richard E. Lenski, Effect of Prey Heterogeneity on the Response of a Model Food Chain to Resource Enrichment, *Am. Nat.* **153**:73-82 (1999).

[4] James Braselton, Martha Abell, and Lorraine Braselton, Selective mating in a continuous model of epistasis, *Applied Mathematics and Computation* **171**:225-241 (2005).

[5] S. Karlin, Some Mathematical Models of Population Genetics, *American Mathematical Monthly* **79**:699-739 (1972).

[6] Alfred J. Lotka, *Elements of Mathematical Biology*, Dover (1956).

[7] James Murray, *Mathematical Biology: I. An Introduction*, Third Edition, Springer (2007).

[8] E. Szathmáry, A Note on the Reduction of the Dynamics of Multilocus Diploid Genetic Systems with Multiplicative Fitness, *J. Theor. Biol.* **164**:351-358 (1993).

[9] S. Wolfram, *The Mathematica Book*, Third Edition, Wolfram Media (1996).

[10] Masato Yamamichi and Akira Sasaki, Single-gene speciation with pleiotropy: effects of allele dominance, population size, and delayed inheritance, *Evolution* **67(6)**:2011-2023 (2013).

Appendix A

RATIOS OF OFFSPRING FOR X_I-X_J MATING COMBINATIONS

	x_1	x_2	x_3	x_4	x_5	x_6	x_7	x_8	x_9
x_1-x_1	1								
x_1-x_2	1/2	1/2							
x_1-x_3		1							
x_1-x_4	1/2			1/2					
x_1-x_5	1/4	1/4		1/4	1/4				
x_1-x_6		1/2			1/2				
x_1-x_7				1					
x_1-x_8				1/2	1/2				
x_1-x_9					1				
x_2-x_2	1/4	1/2	1/4						
x_2-x_3		1/2	1/2						
x_2-x_4	1/4	1/4		1/4	1/4				
x_2-x_5	1/8	1/4	1/8	1/8	1/4	1/8			
x_2-x_6		1/4	1/4		1/4	1/4			
x_2-x_7				1/2	1/2				
x_2-x_8				1/4	1/2	1/4			
x_2-x_9					1/2	1/2			
x_3-x_3			1						
x_3-x_4		1/2			1/2				
x_3-x_5		1/4	1/4		1/4	1/4			
x_3-x_6			1/2			1/2			
x_3-x_7					1				
x_3-x_8					1/2	1/2			
x_3-x_9						1			

x_4-x_4	1/4			1/2			1/4		
x_4-x_5	1/8	1/8		1/4	1/4		1/8	1/8	
x_4-x_6		1/4			1/2			1/4	
x_4-x_7				1/2			1/2		
x_4-x_8				1/4	1/4		1/4	1/4	
x_4-x_9					1/2		1/2		
x_5-x_5	1/16	1/8	1/16	1/8	1/4	1/8	1/16	1/8	1/16
x_5-x_6		1/8	1/8		1/4	1/4		1/8	1/8
x_5-x_7				1/4	1/4		1/4	1/4	
x_5-x_8				1/8	1/4	1/8	1/8	1/4	1/8
x_5-x_9					1/4	1/4		1/4	1/4
x_6-x_6			1/4		1/2				1/4
x_6-x_7					1/2		1/2		
x_6-x_8					1/4	1/4		1/4	1/4
x_6-x_9						1/2			1/2
x_7-x_7							1		
x_7-x_8							1/2	1/2	
x_7-x_9								1	
x_8-x_8							1/4	1/2	1/4
x_8-x_9								1/2	1/2
x_9-x_9									1

Table A.1: Ratios of offspring for x_i-x_j mating combinations