Using Mathematical Models to Investigate Phenotypic Oscillations in Cichlid Fish: A Case of Frequency-dependent Selection

by
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SIGNED: Sheree Lynn Arpin
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Abstract

*Perissodus microlepis* is a species of cichlid fish endemic to Lake Tanganyika (Africa). Adult *P. microlepis* are lepidophages, feeding on the scales of other living fish. As an adaptation for this feeding behavior *P. microlepis* exhibit lateral asymmetry with respect to jaw morphology: the mouth either opens to the right or left side of the body. Field data illustrate a temporal phenotypic oscillation in the mouth-handedness, and this oscillation is maintained by frequency-dependent selection. To better understand the oscillation, Takahashi and Hori model frequency-dependent selection in *P. microlepis* using a population genetic model. Their results are intriguing, and the purpose of this dissertation is to improve and extend their model, which fails to account for important biological aspects.

We model *P. microlepis* with a novel approach that fuses the disparate modeling traditions of population genetics and population dynamics; we account for both processes since, in the case of *P. microlepis*, they occur on the same time scale. We construct our models using systems of difference equations. We prove the existence and uniqueness of a positive equilibrium, which corresponds to a 1 : 1 phenotypic ratio. Using a local stability and bifurcation analysis, we show that the equilibrium becomes unstable when frequency-dependent selection is sufficiently strong. We determine necessary and sufficient conditions for onset of oscillation. Local bifurcation analysis indicates key features of the oscillation that may suggest critical experiments.

We determine the role of stage structure and the role of strong and weak intraspecific competition. We show that stage-structure is not necessary for, but enhances, oscillatory behavior. Finally we demonstrate the complicated interplay between population dynamic and population genetic processes. Our findings indicate that classical population genetic models can fail to elucidate complex dynamics.
Chapter 1

Introduction

The field of population genetics concerns the mechanisms that cause changes in allele frequencies in populations over time (evolution). The state variables of population genetic models are usually genotype or allele frequencies. The field of population dynamics, on the other hand, concerns changes in the numbers (or densities) of individuals in populations or changes in the age structure of populations. The state variables of population dynamic models are absolute numbers or densities. The fields of population genetics and population dynamics were developed independently and, as a result, have distinct theories and models [13], [32]. With rare exceptions (e.g., r, K-selection theory) each field ignores the important variables in the other as a simplifying assumption. This is usually justified by differences in ecological and evolutionary time scales. However, some of the most interesting problems in ecology and evolutionary biology occur on a single time scale. The field of evolutionary ecology addresses these types of problems.

Natural selection is the most fundamental force driving evolution. Selection refers to the differential survival and/or reproductive success of individuals possessing different characteristics [12]. Selection acts on phenotypes, but it is the alleles comprising genotypes that are inherited. Mathematical models of natural selection are typically constructed from a classical population genetics framework. It is population dynamic notions - population growth rates and death rates and intraspecific interactions - however, that dictate the evolutionary trajectory of an organism. That is, organisms with high fitness may produce more offspring, live longer, and compete strongly (or be less affected by competition) than organisms with low fitness. Due to the com-
plex interaction between the environment and genotype, phenotype is not usually the physical manifestation of genotype alone. However, there are instances where phenotypic traits are solely determined by the underlying genotype. Population genetic models assume the genotype-phenotype map is sufficiently simple by assigning fitness values to genotypes (quantitative genetic models are exceptions). Early population genetic models of selection assumed that fitness values assigned to genotypes were constant. In 1971, K. Kojima brought attention to a mounting body of evidence that refuted this mathematically convenient assumption [21]. There are a multitude of reasons why fitness values may not be constant. Examples include temporal variation, spatial variation, density-dependent variation, and frequency-dependent variation.

Frequency-dependent selection (FDS) is a form of natural selection where the fitness of a genotype depends on the frequency of that genotype in the population [12]. A simple population genetic model of FDS for a Mendelian one-locus, two allele trait is constructed as follows. We begin with a few main assumptions: random mating, large and constant population size, non-overlapping generations, no migration, and no mutation. Let \(A_1A_1, A_1A_2, A_2A_2\) be the three possible genotypes. Let \(p_t\) be the frequency of allele \(A_1\) at time \(t\) and \(q_t = 1 - p_t\) be the frequency of allele \(A_2\) at time \(t\) in the population. We assign a (relative) fitness value, \(w_{ij}\), to genotype \(A_{ij}\) where \(i, j = 1, 2\). To account for the fact that selection is frequency-dependent, we make the \(w_{ij}\)'s functions of allele frequency, \(p\). We define the mean fitness, \(\bar{w}\), as the sum of the relative contributions of the different genotypes to the next generation as \(\bar{w} = p^2w_{11}(p) + 2pqw_{12}(p) + q^2w_{22}(p)\). Therefore, we can find the frequency of allele \(A_1\) at time \(t + 1\) as

\[
p_{t+1} = \frac{p_t^2w_{11}(p) + p_tq_tw_{12}(p)}{\bar{w}}
\]  

(1.1)

A nontrivial equilibrium equation for allele frequency is
\[ p = \frac{w_{12}(p) - w_{22}(p)}{[w_{12}(p) - w_{11}(p)] + [w_{12} - w_{22}(p)]}. \] (1.2)

In general, it is not possible to find a solution to (1.2). Furthermore, (1.2) does not account for overlapping generations. Thus, by inspecting this simple case it is clear that models of FDS are challenging to analyze.

There are two main forms of FDS: positive and negative. In the case of positive FDS, the fitness of a genotype is an increasing function of its frequency in the population. Positive FDS is known to occur in pollinator systems and among geographic races of species with distinct color variations. A well-documented example of positive FDS concerns unpalatable *Heliconius* butterflies that are Müllerian mimics of one another [24]. In particular, there are local races with varying color pattern in *H. erato* and *H. melpomene*. Individuals not conforming to the local color variant are preyed upon by birds not recognizing the warning colors. This type of FDS accelerates the loss of deleterious alleles and ultimately leads to monomorphic populations.

In the case of negative FDS the fitness of a genotype is a decreasing function of its frequency in the population. That is, individuals with rare genotypes have greater fitness than individuals with common genotypes. Early evidence of negative FDS selection dates back to 1946 laboratory studies of karyotypes in *Drosophila pseudoobscura* [42]. Negative FDS has been found to operate on pathogen resistance genes, predator-prey systems using search images, self-incompatibility (SI) loci in plants, species competing for limited resources, and mating preferences (i.e., frequency-dependent sexual selection) [1], [5], [9], [23], [30]. An important example of pathogen resistance genes is the major histocompatibility complex (MHC) found in vertebrates, including humans. The MHC is a large gene family that has an extremely high level of genetic variation, which is hypothesized to be maintained, in part, by FDS [15], [35]. Numerous laboratory studies have also shown FDS operating on a plethora of other organisms: *Drosophila, Tribolium*, houseflies, butterflies, wheat, barley, grass, flowering plants,
water boatmen, guppies, house wrens, and mice [2], [11], [12], [14].

Conservation biologists are especially interested in negative FDS as an evolutionary force because it leads to balanced polymorphism (i.e., genetic diversity). Indeed, a fundamental paradox in evolutionary biology is the maintenance of genetic diversity in the presence of genetic drift snuffing out new mutations and directional selection removing deleterious alleles from the population. Genetic diversity plays an important role in ecological and evolutionary theory and practice since inbreeding depression is a devastating consequence of its loss [29]. Moreover, populations require genetic diversity to recover from perturbations and adapt to their stochastic environments [11].

While FDS was well-documented in laboratory populations, convincing evidence of its occurrence in the field was noticeably absent [2], [16]. This was the case until 1993 when Michio Hori reported FDS operating in a natural population of predatory cichlid fish, *Perissodus microlepis* Boulenger [16]. *P. microlepis* are a species of fish belonging to family Cichlidae, which include the commercially important *Tilapia* species. With more than 3000 species distributed from Central and South America, across Africa to Madagascar and southern India, cichlids represent the most species-rich group of vertebrates [20]. *P. microlepis* is endemic to the East African Great Lake, Lake Tanganyika, lying in the Great Rift Valley. Lake Tanganyika is the second deepest lake in the world, housing hundreds of fish species. Upper estimates of age place Lake Tanganyika between nine and twelve million years old [22]. The African Great Lakes, Lakes Victoria, Malawi, and Tanganyika, are ideal environments to study evolution due to their isolation and the fact that cichlids have evolved in the very recent evolutionary past [20]. Cichlids are a striking example of adaptive radiation and convergent evolution and, as such, are used to study the poorly understood process of speciation. Cichlids display enormous variation in coloration, body form and size, mating systems, brood care patterns, and feeding habits. Their highly specialized behaviors and morphologies are hypothesized to account for the large number of
coexisting cichlid species, each occupying a slightly different ecological niche.

Sexually mature, adult *P. microlepis* are predatory scale-eaters (lepidophages), feeding off the scales of other living fish. Lepidophagy is not a unique behavior to the genus *Perissodus*, but has evolved independently in Lakes Tanganyika, Malawi, and Victoria. *P. microlepis* attack their prey from behind and use their spine-like teeth to remove scales [37]. Prey fish are keenly alert to approaching predators, and the rate of hunting success is low, approximately 20% [16]. Interestingly, Hori discovered that *P. microlepis* possess a lateral jaw asymmetry; there is an asymmetrical joint of the jaw to the suspensorium [17]. Populations of *P. microlepis* are polymorphic with respect to jaw morphology; an individual’s jaw either opens to the left (sinistral) side or to the right (dextral) side of the body as depicted in Figure 1.1. No intermediate forms have been observed. Hori discovered that left-handed (sinistral) fish consistently attack the prey’s right flank and right-handed (dextral) fish consistently attack the prey’s left flank. This behavior appears to be adaptive by increasing the contact area between the jaw and the prey’s body [16].

**Figure 1.1.** Asymmetric jaw morphology of *P. microlepis* early and late in development.

(a) Asymmetry early in development: on the left, a sinistral (left-handed) fish and on the right, a dextral (right-handed) fish. This picture is reproduced, with permission, from the lab of R. Craig Albertson.

(b) Asymmetric jaw morphology late in development: above, a sinistral (left-handed) fish and below, a dextral (right-handed) fish.

Handedness can be detected early in development and is heritable. It is deter-
mined by a simple Mendelian one-locus, two-allele \((R, L)\) mode of inheritance where
dextrality is completely dominant over sinistrality: \(RL\) and \(RR\) individuals are phe-
notypically indistinguishable [16]. The frequencies of left-handed and right-handed
fish in a population of \(P. \ microlepis\) were found to oscillate about 0.5 as in Figure 1.2,
with an approximate period of five years and an approximate amplitude of 0.075. By
examining the number of denuded spots on the prey fish in a controlled experiment,
it was determined that the prey aggressively guard against the more frequent morph.
Specifically, when sinistral fish were numerically dominant there were more denuded
spots on the prey’s left flanks from successful right-handed predator attacks and vice
versa [16]. This finding is contrary to what is expected based on the sheer number of
individuals possessing each phenotype.

The distorted hunting success suggests that prey fish are sensitive to the number
of attacks received on each flank (a function of the number of individuals possessing
each phenotype), and respond by guarding the heavily targeted side more aggressively.
This behavior, in turn, gives the fish possessing the opposite morphology a hunting
advantage. The hunting advantage translates into marked differences in reproductive
output (i.e., selection results from differential reproductive success rather than dif-
ferential survival). In fact, field data indicate that the rare type can have twice the
reproductive success of the common type [40]. This differential is remarkable con-
sidering phenotypic frequencies in the field oscillated with amplitude less than 0.1.
Collectively, the data, observations, and experiments indicate that jaw polymorphism
for \(P. \ microlepis\) is maintained by negative FDS, which is mediated by the guarding
behavior of the prey fish.

Takahashi and Hori constructed a model of FDS to describe the phenotypic oscil-
lation and show it is due to the differential hunting success and its delayed effect on
reproduction [40]. Here we will describe their model and results. Later, we will mo-
tivate our model construction based on the successes and shortcomings of the model
of Takahashi and Hori. To begin, the change in total population size of \(P. \ microlepis\)
Figure 1.2. Oscillation in the frequency of handedness of two populations of *P. microlepis*. The open dots represent fish collected from the Luhanga coast and closed dots represent fish collected from the Bemba coast. This picture is reproduced, with permission, from Hori [16].

is neglected due to the fact that fish populations in Lake Tanganyika are highly persistent. The variables $L_t$, $H_t$, and $R_t$ represent the frequencies of homozygous left-handed, heterozygous right-handed, and homozygous right-handed adult fish at time $t$, respectively. The variables $L_{1t}$, $H_{1t}$, and $R_{1t}$ represent the corresponding genotypic frequencies of one-year-old fish, and $s_{1t}$ represents the frequency of the sinistral gene in one-year-old fish. The authors claim the Hardy-Weinberg Law is used to find $L_{1t}$, $H_{1t}$, and $R_{1t}$ as

\[
L_{1t} = (s_{1t})^2,
\]
\[
H_{1t} = 2s_{1t}(1 - s_{1t}),
\]
\[
R_{1t} = (1 - s_{1t})^2.
\]
However, the Hardy-Weinberg Law is not being used to obtain the above frequencies since the absence of selection is an underlying assumption of the law. Instead, some of the assumptions of the Hardy-Weinberg Law, such as random mating, are used to arrive at \( L_{1t}, H_{1t}, \) and \( R_{1t} \).

The ratio \( f(L) : 1 - f(L) \) represents the ratio of reproductive success for sinistral and dextral fish when the frequency of sinistral fish is \( L \), where \( f(0.5) = 0.5 \). To account for negative frequency-dependent selection, \( f(L) \) is assumed to be monotone decreasing. Incorporating the fitness values into the model, the frequency of the sinistral gene at time \( t + 1 \) is

\[
s_{1(t+1)} = \frac{f(L_t) L_t + 0.5 [1 - f(L_t)] H_t}{f(L_t) L_t + [1 - f(L_t)](H_t + R_t)}. \tag{1.3}
\]

Equation (1.3) is flawed when compared to (1.1). It should instead be

\[
s_{1(t+1)} = \frac{f(L_t)(L_t + \frac{H_t}{2})^2 + (1 - f(L_t))(L_t + \frac{H_t}{2})(R_t + \frac{H_t}{2})}{f(L_t)(L_t + \frac{1}{2}H_t)^2 + (1 - f(L_t))\left[2(L_t + \frac{1}{2}H_t)(R_t + \frac{H_t}{2}) + (R_t + \frac{H_t}{2})^2\right]},
\]

since \( L_t, H_t, \) and \( R_t \) represent genotype frequencies rather than allele frequencies. Moreover, equation (1.3) assumes Hardy-Weinberg equilibrium has been met, which cannot be the case when selection occurs.

The genotype frequencies change according to the equations

\[
\begin{align*}
L_{t+1} &= rL_t + (1 - r)L_{1t}, \\
H_{t+1} &= rH_t + (1 - r)H_{1t}, \\
R_{t+1} &= rR_t + (1 - r)R_{1t},
\end{align*}
\tag{1.4}
\]

where a constant annual survival rate, \( r \), is assumed for both one-year-old and adult
sinistral and dextral fish (a very limiting assumption). Takahashi and Hori state that $r$ adults survive in the following year and the remainder (which do not survive), $1 - r$, is replaced with recruits from 1-year-old fish. It is not clear how the survival rate, $r$, relates to changes in genotype frequency. By construction, $r$ should be a probability of survival, not the number of individuals surviving. This illustrates the difficulty of modeling organisms with overlapping generations using frequencies as state variables. The frequency of the sinistral gene among adults at time $t + 1$ is

$$s_{t+1} = rs_t + (1 - r)s_{1t}. \quad (1.5)$$

The authors proceed by perturbing $s_t$ and $s_{1t}$ from a 1 : 1 phenotype ratio (i.e., $s_{1t} = s_t = \frac{\sqrt{2}}{2}$). However, it is not clear why the sinistral allele frequency should be the same in one-year-old and adult fish. Furthermore, the authors state that (1.4) has a stationary solution of $L_t = L_{1t} = 0.5$. By inspecting (1.4) and (1.5), however, it is clear that all solutions with one-year-old and adult frequencies being equal are stationary solutions by construction. This pathology of the model makes it difficult to interpret and weight the results from the special case of the steady-state corresponding to the 1 : 1 phenotypic ratio. The authors’ analysis revealed a phenotypic oscillation when $q = -f'(\frac{1}{2})$ is sufficiently large in magnitude. The parameter $q$ was interpreted as the effect the frequency of sinistrality deviating from 50% has on the reproductive success (fitness), when the deviation is small. The authors attribute the phenotypic oscillation, in part, to the time lag in growth period. All other analysis for this system was approximated by a haploid model.

The findings of Takahashi and Hori are intriguing, and we seek to improve on their modeling approach. In particular, we would like to model the absolute numbers of $P. microlepis$ comprising each genotype to accurately account for overlapping generations. By doing this we can also examine reproductive success and selection at the level of mate pairing without assuming individual fertilities are independent
of one another. That is, viabilities and fertilities are typically assigned to individuals possessing each genotype irrespective of mate pairings that form. It may be the case, however, that the fertility of a mate pairing is a complicated function of the fitnesses of the individuals comprising the pair. This notion is key since differential reproductive success gives rise to the oscillation. Furthermore, we are interested in exploiting the relatively simple genetics of *P. microlepis* to investigate the interplay between the population genetic and population dynamic processes, which were completely neglected by Takahashi and Hori. Our approach is general, and can be applied to other biological systems with similar genetic constraints.
Chapter 2

Model Description and Analysis

2.1. Model construction

In this section we describe a mathematical model motivated by the previous work of Takahashi and Hori [40]. We initially focus on a relatively simple model, which will be extended in later chapters to incorporate further biological realism. Throughout, our modeling effort is unique in that it accounts for both the population dynamics and population genetics of *P. microlepis*.

We begin our approach with a system of three difference equations that predict the number of sexually mature, scale-eating adult fish of each handed genotype at time $t + 1$ based on the number present at time $t$. Recall that previous models predicted the genotype frequencies at time $t + 1$ based on the genotype frequencies at time $t$. While we will not have a dynamical system for genotype frequencies, we can determine frequencies at each time by taking ratios of our state variables. In this way our model accounts for both population and phenotypic frequency dynamics.

The projection time interval for the model is two years: the time it takes from birth to sexual maturity and scale-eating. We begin with an unstructured model in order to elucidate the role that structure and time delays play. Specifically, we address the hypothesis that the time delay in development is, in part, responsible for the phenotypic oscillation observed by Hori [16]. Our initial model utilizes a weak density-dependent nonlinearity so as to exclude oscillations known to occur due to strong intraspecific competition. We consider a stronger nonlinearity in Section 4.1.
2.2. Model definition and parameters

The model state variables are given by $A_{LL}$, $A_{RL}$, and $A_{RR}$, the number of (homozygous) left-handed, heterozygous right-handed, and homozygous right-handed sexually mature, scale-eating adult fish. Incorporating both population dynamic and population genetic processes, we write our model as,

$$A_{LL}(t+1) = \frac{nbN(t)}{1+cN(t)} \left[ \left( \frac{A_{LL}(t)}{N(t)} \right)^2 f_1 \left( \frac{A_{LL}(t)}{N(t)} \right) + \left( \frac{A_{LL}(t)A_{RL}(t)}{N(t)^2} \right) f_2 \left( \frac{A_{LL}(t)}{N(t)} \right) \right]$$

$$+ \frac{1}{4} \left( \frac{A_{RL}(t)}{N(t)} \right)^2 f_1 \left( 1 - \frac{A_{LL}(t)}{N(t)} \right) + (1 - \mu_a) A_{LL}(t),$$

$$A_{RL}(t+1) = \frac{nbN(t)}{1+cN(t)} \left[ \left( \frac{1}{2} A_{RL}(t)^2 \right) + A_{RR}(t)A_{RL}(t) \right) f_1 \left( 1 - \frac{A_{LL}(t)}{N(t)} \right)$$

$$+ \left( \frac{A_{LL}(t)A_{RL}(t) + 2A_{RR}(t)A_{LL}(t)}{N(t)^2} \right) f_2 \left( \frac{A_{LL}(t)}{N(t)} \right) \right]$$

$$+ (1 - \mu_a) A_{RL}(t),$$

$$A_{RR}(t+1) = \frac{nbN(t)}{1+cN(t)} \left[ \frac{A_{RR}(t)^2 + A_{RR}(t)A_{RL}(t) + \frac{1}{2} A_{RL}(t)^2}{N(t)^2} \right] f_1 \left( 1 - \frac{A_{LL}(t)}{N(t)} \right)$$

$$+ (1 - \mu_a) A_{RR}(t).$$

The total adult population size is represented by $N(t) = A_{LL}(t) + A_{RL}(t) + A_{RR}(t)$. In each model equation in (2.1) adults originate from the birth of new fish (recruitment).
and the survival of old adult fish. The number of new recruits produced in one time step in the absence of selection and competition is $nbN(t)$. We assume the total number of matings that occur within one time step is a linear function of total population size, $nN(t)$. The constant $b > 0$ is interpreted as the physiological maximum for the number of offspring produced per mating. This maximum number is approached through matings between rare phenotypes as the population becomes monomorphic. If the population is polymorphic, the total number of new recruits produced per time step is reduced by frequency-dependent selection, which is accounted for by the terms in the brackets. We will discuss these terms shortly.

The total number of new recruits produced per time step, $nbN(t)$, is reduced by the fraction,

$$\frac{1}{1 + c(A_{LL}(t) + A_{RL}(t) + A_{RR}(t))}, \quad (2.2)$$

due to intraspecific competition. This density-dependent nonlinearity, the so-called Beverton-Holt or discrete logistic nonlinearity, prevents solutions from becoming unbounded. All orbits of the one-dimensional discrete logistic map,

$$N(t + 1) = \frac{bN(t)}{1 + cN(t)},$$
asymptotically approach a unique equilibrium when $b > 1$. Prototypical for logistic-like dynamics, we eliminate oscillations produced by stronger nonlinearities, such as an exponential or Ricker nonlinearity. We do this to determine if frequency-dependent selection alone can account for the observed phenotypic oscillations. In Chapter 4, we will investigate the interplay of frequency-dependent selection and stronger intraspecific competition. The constant $c > 0$ in (2.2) and (2.1) is the per capita density-dependent coefficient. The constant $0 < \mu_a < 1$ is the probability of adult death in one time interval.

New recruits arise from all possible mating combinations between adults possessing
different genotypes as shown in Table 2.1. Mating is assumed to be random, and we do not distinguish between the matings $A_{RR}$ (male) × $A_{RL}$ (female) and $A_{RR}$ (female) × $A_{RL}$ (male), for example [40]. We assume there is an equal sex ratio and that both sexes have identical allele frequencies. The genotypic frequencies at each time are determined by the following ratios:

$$\frac{A_{LL}(t)}{N(t)}, \quad \frac{A_{RL}(t)}{N(t)}, \quad \frac{A_{RR}(t)}{N(t)}.$$ 

The phenotypic frequencies of left-handed and right-handed fish at time $t$ are respectively

$$l(t) = \frac{A_{LL}(t)}{N(t)}, \quad 1 - l(t) = \frac{A_{RL}(t) + A_{RR}(t)}{N(t)},$$

since the dextral allele ($R$) is completely dominant over the sinistral allele ($L$).

In the absence of selection and genetic drift the probability that a mating event will produce an offspring with a particular genotype can be determined by the Hardy-Weinberg Law [12]. For example, the probability that an individual in the next generation has genotype $A_{ij}$, $P(A_{ij}, t + 1)$, in a two allele system is given by,

<table>
<thead>
<tr>
<th>Table 2.1. Mate pairing combinations and their resulting genotypes.</th>
</tr>
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<tbody>
<tr>
<td>$LL \times LL$ → $100% LL.$</td>
</tr>
<tr>
<td>$LL \times RL$ → $50% LL, 50% RL.$</td>
</tr>
<tr>
<td>$LL \times RR$ → $100% RL.$</td>
</tr>
<tr>
<td>$RL \times LL$ → $50% LL, 50% RL.$</td>
</tr>
<tr>
<td>$RL \times RL$ → $25% LL, 50% RL, 25% RR.$</td>
</tr>
<tr>
<td>$RL \times RR$ → $50% RL, 50% RR.$</td>
</tr>
<tr>
<td>$RR \times LL$ → $100% RL.$</td>
</tr>
<tr>
<td>$RR \times RL$ → $50% RL, 50% RR.$</td>
</tr>
<tr>
<td>$RR \times RR$ → $100% RR.$</td>
</tr>
</tbody>
</table>
\[ P(A_{ij}, t + 1) = \begin{cases} 
 p_i(t)^2, & i = j, \\
 2p_i(t)p_j(t), & i \neq j, 
\end{cases} \]

where,

\[ p_i(t) = \frac{A_{ii}(t)}{N(t)} + \frac{1}{2} \frac{A_{ij}(t)}{N(t)} \quad \text{and} \quad p_j(t) = \frac{A_{jj}(t)}{N(t)} + \frac{1}{2} \frac{A_{ij}(t)}{N(t)}, \]

are the frequencies of alleles \( A_i \) and \( A_j \) at time \( t \), respectively. For instance, the probability that an offspring has genotype \( RL \) at time \( t + 1 \) is,

\[
P(RL, t + 1) = 2 \left( \frac{A_{RR}(t)}{N(t)} + \frac{1}{2} \frac{A_{RL}(t)}{N(t)} \right) \left( \frac{A_{LL}(t)}{N(t)} + \frac{1}{2} \frac{A_{RL}(t)}{N(t)} \right) \]

\[
= \left[ 2A_{RR}(t)A_{LL}(t)
\frac{1}{N(t)^2} + A_{RR}(t)A_{RL}(t)
\frac{1}{N(t)^2} + A_{RL}(t)A_{LL}(t)
\frac{1}{N(t)^2} + \frac{1}{2} \left( \frac{A_{RL}(t)}{N(t)} \right)^2 \right].
\]

For the species in which we are interested the Hardy-Weinberg equilibrium cannot be reached, however, because the assumption that there is no selection is violated \cite{12}. The functions \( f_1 \) and \( f_2 \) in (2.1) account for frequency-dependent selection as fitness functions. Although we know frequency-dependent selection operates in our model system, we do not specify the particular type of selection (e.g., viability selection, mating success, fecundity selection). The state variables, model parameters and functions are listed in Table 2.2.

In order to model frequency-dependent selection, we assign an intrinsic fitness to individuals of each phenotype based on the frequency of that phenotype in the population. Fitness values are assigned to phenotypes, rather than genotypes, since selection acts on phenotypes. Phenotype for handedness in \( P. \ microlepis \) is the physical manifestation of genotype. Individuals with \( RR \) and \( RL \) genotypes display the same phenotype and, thus, have the same fitness. An individual’s fitness value dic-
Table 2.2. State variables, parameters, and functions for model (2.1).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$A_{LL}$</td>
<td>Number of adult left-handed fish at time $t$.</td>
</tr>
<tr>
<td>$A_{RL}$</td>
<td>Number of adult heterozygous right-handed fish at time $t$.</td>
</tr>
<tr>
<td>$A_{RR}$</td>
<td>Number of adult homozygous right-handed fish at time $t$.</td>
</tr>
<tr>
<td>$n$</td>
<td>Number of matings per capita in one time step.</td>
</tr>
<tr>
<td>$b$</td>
<td>Maximum number of offspring per mating.</td>
</tr>
<tr>
<td>$c$</td>
<td>Per capita density-dependent coefficient.</td>
</tr>
<tr>
<td>$\mu_a$</td>
<td>Probability of death in one time step.</td>
</tr>
<tr>
<td>$f_1(l)$</td>
<td>Fitness function for $L \times L$ matings.</td>
</tr>
<tr>
<td>$f_1(1-l)$</td>
<td>Fitness function for $R \times R$ matings.</td>
</tr>
<tr>
<td>$f_2(l)$</td>
<td>Fitness function for $L \times R$ or $R \times L$ matings.</td>
</tr>
</tbody>
</table>

states its reproductive success. Reproductive success is maximally $b$; $b$ is reduced by the frequency-dependent fitness functions ($f_1$ and $f_2$) in accordance with the phenotypic frequencies. In our model of negative frequency-dependent selection, the fitness value assigned to individuals possessing the rare phenotype is greater than the fitness value assigned to individuals possessing the common phenotype (fitness values assigned to the two phenotypes equal one another when $l = \frac{1}{2}$). For a fixed value of $l \neq \frac{1}{2}$, we assume that the reproductive advantage given to the rare phenotype equals in magnitude the reproductive disadvantage given to the common phenotype (i.e., there is symmetry about $l = \frac{1}{2}$).

For reasons of generality, we do not specifically relate the fitnesses of individuals to the fitnesses of matings between phenotypes (like or unlike). Instead, we simply require that the reproductive success of matings between rare phenotypes be greater than or equal to the reproductive success of matings between rare and common phenotypes. In turn, the reproductive success of matings between rare and common phenotypes must be greater than or equal to the reproductive success of matings between common phenotypes. Furthermore, symmetry about $l = \frac{1}{2}$ is maintained for the reproductive advantage and disadvantage apportioned to matings between the phenotypes. To this end, the fitness values of the individuals taking part in a mating...
are reflected in the overall fitness (reproductive success) of the mating.

In model (2.1) we implicitly model the guarding behavior of the prey species through the fitness functions, $f_1$ and $f_2$. As such,

$$0 \leq f_1(l) \leq 1, \quad 0 \leq f_1(1-l) \leq 1, \quad 0 \leq f_2 \leq 1;$$

$b$ is the physiological maximum number of offspring per mating, which can only be reduced when ideal conditions (i.e., when $\frac{A_{LL}(t)}{A_{RL}(t)+A_{RR}(t)} \approx 0$ or $\frac{A_{RL}(t)+A_{RR}(t)}{A_{LL}(t)} \approx 0$) are not met. We require that $f_1(l)$ ($f_1(1-l)$) be a smooth, monotone decreasing (increasing) function of $l$ that describes the reproductive success of $L \times L$ ($R \times R$ matings) where $L$ and $R$ are phenotypically sinistral and dextral fish, respectively.

The monotonicity assumption encompasses the fact that the prey species assesses the phenotypic ratio (based on the number of attacks received on each flank), and guards the appropriate flank. For example, as $l$ increases, the prey strategically guard their right flanks resulting in a reduced (increased) fitness for the sinistral (dextral) fish.

We also require that the fitness function $f_2(l)$ be a smooth function that describes the reproductive success of $R \times L$ and $L \times R$ matings. When the phenotypic ratio is 1 : 1, all matings are equally fit yielding,

$$f_1\left(\frac{1}{2}\right) = f_2\left(\frac{1}{2}\right) = f \left(\frac{1}{2}\right).$$

By constructing our model with absolute numbers as state variables, it is possible to account for selection at the level of mating events (e.g., fecundity, mating success). Fertility is addressed in classical population genetic models by multiplying viabilities and fertilities [32]. That is, each genotype has a viability and fertility. However, it may not be biologically reasonable to assign fertilities to genotypes; this assumes that individual fertilities are independent of one another. Fertility or mating success may be determined by both genotypes comprising the mate pair itself. For instance, offspring with genotype $LL$ can result from matings between two $LL$ individuals, two
RL individuals, or an RL and LL individual. While all of these matings result in LL offspring, they may not be equally successful at producing offspring (e.g., \( LL \times LL \) matings occur between left-handed individuals and \( RL \times LL \) matings occur between left- and right-handed individuals). The success of the \( RL \times LL \) mating may be resolved by the more fit (or less fit) individual in the pairing. In our model, by apportioning the total number of matings among the different genotypes based on genotypic frequencies in the population, and by using the general fitness functions, \( f_1 \) and \( f_2 \), we assign mating successes to mate pairings between like and unlike genotypes. Modeling allele or genotype frequencies alone does not make it possible to determine the genotypes of the individuals contributing the alleles; this is the case of the model by Takahashi and Hori [40]. It is critical to be judicious about accounting for selection at the level of mating success since data suggest that selection takes place early in the life history, at the mating events themselves or early in the zygotic phase [16]. Moreover, modeling absolute numbers is conducive to populations with overlapping generations, which are a confounding difficulty of frequency dynamic models.

In order to simplify the fitness functions and our calculations, we re-scale the equations in model (2.1). In particular, we define new fitness functions, \( g \) and \( h \), by dividing through by \( f(\frac{1}{2}) \) as

\[
A_{LL}(t + 1) = \frac{nBN(t)}{1 + cN(t)} \left[ \left( \frac{A_{LL}(t)}{N(t)} \right)^2 g\left( \frac{A_{LL}(t)}{N(t)} \right) + \left( \frac{A_{LL}(t)A_{RL}(t)}{N(t)^2} \right) h\left( \frac{A_{LL}(t)}{N(t)} \right) \right] + \frac{1}{4} \left( \frac{A_{RL}(t)}{N(t)} \right)^2 g\left( 1 - \frac{A_{LL}(t)}{N(t)} \right) + (1 - \mu_a) A_{LL}(t),
\]
$$A_{RL}(t + 1) = \frac{nBN(t)}{1 + cN(t)} \left[ \left( \frac{1}{2}A_{RL}(t)^2 + A_{RR}(t)A_{RL}(t) \right) g\left(1 - \frac{A_{LL}(t)}{N(t)}\right) + \left( A_{LL}(t)A_{RL}(t) + 2A_{RR}(t)A_{LL}(t) \right) h\left(\frac{A_{LL}(t)}{N(t)}\right) \right]$$

$$+ \left( A_{LL}(t)A_{RL}(t) + 2A_{RR}(t)A_{LL}(t) \right) h\left(\frac{A_{LL}(t)}{N(t)}\right)$$

$$+ (1 - \mu_a) A_{RL}(t),$$

$$A_{RR}(t + 1) = \frac{nBN(t)}{1 + cN(t)} \left[ A_{RR}(t)^2 + A_{RR}(t)A_{RL}(t) + \frac{1}{2}A_{RL}(t)^2 \right] g\left(1 - \frac{A_{LL}(t)}{N(t)}\right)$$

$$+ (1 - \mu_a) A_{RR}(t).$$

where,

$$B = bf\left(\frac{1}{2}\right),$$

$$g(l) = g\left(\frac{A_{LL}(t)}{N(t)}\right) = \frac{f_1(l)}{f(\frac{1}{2})},$$

$$h(l) = h\left(\frac{A_{LL}(t)}{N(t)}\right) = \frac{f_2(l)}{f(\frac{1}{2})}. $$

Notice that the above new assignment gives $g\left(\frac{1}{2}\right) = h\left(\frac{1}{2}\right) = 1.$

There are some additional assumptions we make on the frequency-dependent fitness functions, $g$ and $h$, in model (2.3). We require,

$$h(l) \leq \max\{g(l), g(1 - l)\}, \quad h(l) \geq \min\{g(l), g(1 - l)\}, \quad (2.4)$$
since matings between individuals possessing different phenotypes are comprised of one individual that is more fit and one individual that is less fit than the other. Thus, the less (more) fit individual confers a lower (higher) fitness. At any given time the phenotypic frequency ratio of sinistral to dextral fish is $1/2 - \delta : 1/2 + \delta$, for some $\delta$ satisfying $-1/2 \leq \delta \leq 1/2$. Notice that the frequency ratios $1/2 - \delta : 1/2 + \delta$ and $1/2 + \delta : 1/2 - \delta$ reflect the same deviation from the 1:1 phenotypic ratio (in one case sinistral fish are more frequent and in the other dextral fish are more frequent). In either case, the change in fitness must be the same at the points $l = 1/2 \pm \delta$ and $1 - l = 1/2 \mp \delta$, since we are assuming the reproductive advantage given to the rare phenotype equals in magnitude the reproductive disadvantage given to the common phenotype. Therefore, we require the following condition to be met,

$$\left. \frac{dg}{dx} \right|_{x=l} = \left. \frac{dg}{dx} \right|_{x=1-l}. \tag{2.5}$$

As a consequence, $g'' \left( \frac{1}{2} \right) = 0$. We also require,

$$h(l) = h(1 - l), \tag{2.6}$$

since a deviation from the 1:1 phenotypic ratio in either direction will produce one common phenotype and one rare phenotype; the fitness of matings between individuals with different phenotypes should not be affected by which phenotype is numerically dominant. We have assumed that $h$ is a smooth function, and (2.6) implies $h' \left( \frac{1}{2} \right) = 0$. Equations (2.5) and (2.6) dictate specific symmetry properties of the functions $g(l)$ and $h(l)$ about $l = 1/2$.

Recall that $h(l)$ describes the reproductive success (fitness) of $L \times R$ and $R \times L$ matings. When individuals of different phenotypes mate, one individual possesses a higher fitness, greater than $f \left( \frac{1}{2} \right)$, and one possesses a lower fitness, less than $f \left( \frac{1}{2} \right)$. While we have constructed the magnitude of the advantage and disadvantage to be
the same, these differences may or may not cancel one another out biologically. If the fitness differences cancel one another out (the neutral case), $h(l)$ is a constant function: $h(l) = f\left(\frac{1}{2}\right)$. However, it may be the case that the reproductive advantage, conferred by the individual with the rare phenotype, biologically outweighs the reproductive disadvantage, conferred by the individual with the common phenotype, or vice versa.

If the reproductive advantage associated with the rare phenotype in an $L \times R$ or $R \times L$ mating biologically outweighs the reproductive disadvantage associated with the common phenotype (the advantage case), then $h(l)$ is strictly decreasing to the left of $l = \frac{1}{2}$ and strictly increasing to the right, i.e., is concave up in a neighborhood of $l = \frac{1}{2}$. If, on the other hand, the reproductive disadvantage associated with the common phenotype biologically outweighs the reproductive advantage associated with the rare phenotype (the disadvantage case), then $h(l)$ is strictly increasing to the left of $l = \frac{1}{2}$ and strictly decreasing to the right, i.e., is concave down in a neighborhood of $l = \frac{1}{2}$.

In summary, the assumptions we make on $g$ and $h$ are as follows. By re-scaling model (2.1) we have $g\left(\frac{1}{2}\right) = h\left(\frac{1}{2}\right) = f\left(\frac{1}{2}\right) = 1$, and $0 \leq g, h \leq \frac{1}{f\left(\frac{1}{2}\right)}$. We assume that $g(l)$ is smooth, and monotone decreasing with $\frac{dg}{dx}\bigg|_{x=l} = \frac{dg}{dx}\bigg|_{x=1-l}$ (as a consequence, $g''\left(\frac{1}{2}\right) = 0$). We assume that $h(l)$ is smooth and monotone to the left and right of $l = \frac{1}{2}$ with $h(l) \leq \max\{g(l), g(1-l)\}$, $h(l) \geq \min\{g(l), g(1-l)\}$, and $h(l) = h(1-l)$. The monotonicity assumption to the left and right of $l = \frac{1}{2}$ means that $h(l)$ either increases to the left and decreases to the right of $l = \frac{1}{2}$ or decreases to the left and increases to the right of $l = \frac{1}{2}$. As will be discussed later, smoothness of $h(l)$ near $l = \frac{1}{2}$ allows for a local stability analysis of the positive equilibrium. If this condition is not met the analysis fails.

Throughout the sections that follow we define orbits of model (2.3) as those that are biologically well-posed. That is, we assume orbits are non-negative (i.e., $A_{LL} \geq 0, A_{RL} \geq 0, A_{RR} \geq 0$) and the parameters $n, b, c$ are positive, and $0 < \mu_a < 1$. 
By inspection, the non-negative octant is invariant under model (2.3).

We end this section with an example of a suitable function for \( g(l) \),

\[
g(l) = -\tanh \left[ p \left( 1 - l - \frac{1}{2} \right) \right] + 1,
\]

(2.7)

based on the properties of \( g \) outlined in this section, where \( p = g'(\frac{1}{2}) < 0 \) and \( f(\frac{1}{2}) = \frac{1}{2} \). We will use this frequency-dependent fitness function in many of the numerical simulations presented as figures.

2.3. Analysis at the origin

We will now determine the conditions under which the population goes extinct (i.e., \((A_{LL}, A_{RL}, A_{RR}) \to (0, 0, 0) \text{ as } t \to \infty\)). By inspection, our model is not defined at the origin; the origin is not an equilibrium. Therefore, we will discuss the origin as a limit point.

**Theorem 2.3.1.** Orbits approach \((0, 0, 0)\) as \( t \to \infty \) when \( \frac{nb}{\mu_a} = \frac{nB}{f(\frac{1}{2})\mu_a} < 1 \) in model (2.1).

**Proof.** Summing the equations in model (2.1) we obtain

\[
N(t + 1) = \frac{nbN(t)}{1 + cN(t)} \left[ \left( A_{LL}(t) \right)^2 f_1(A_{LL}(t)) + \frac{2A_{LL}(t)}{N(t)} \left( 1 - A_{LL}(t) \right) f_2(A_{LL}(t)) \right]
\]

\[
+ \left( 1 - \frac{A_{LL}(t)}{N(t)} \right)^2 f_1 \left( 1 - \frac{A_{LL}(t)}{N(t)} \right) + (1 - \mu_a)N(t),
\]

(2.8)

where \( l = \frac{A_{LL}(t)}{N(t)} \) is the fraction of left-handed fish in the population at time \( t \). We place a bound on (2.8) as follows,
\[ N(t + 1) = \frac{nbN(t)}{1 + cN(t)} \left[ l^2 f_1(l) + 2l(1-l)f_2(l) + (1-l)^2 f_1(1-l) \right] + (1-\mu_a)N(t), \]

\[ \leq nbN(t) \left[ l^2 f_1(l) + 2l(1-l)f_2(l) + (1-l)^2 f_1(1-l) \right] + (1-\mu_a)N(t), \]

\[ \leq nbN(t) \left[ l^2 + 2l(1-l) + (1-l)^2 \right] + (1-\mu_a)N(t), \]

\[ = [nb + (1-\mu_a)] N(t) = \rho N(t). \]

By inspecting the above inequality, it is clear that \( N(t) \to 0 \) when \( \rho < 1 \). Therefore,

\[ \frac{nb}{\mu_a} = \frac{nB}{f(\frac{1}{2}) \mu_a} < 1, \]

implies \((A_{LL}(t), A_{RL}(t), A_{RR}(t)) \to (0, 0, 0)\) as \( t \to \infty \). \hfill \Box

Due to the singularity at the origin in model (2.3), we cannot define \( R_0 \), the inherent net reproductive number, as the expected number of offspring per individual per lifetime [7]. In fact, numerical simulations suggest that the bifurcation point of nontrivial equilibria from the extinction equilibrium depends on the nonlinear term \( q \).

2.4. Analysis along the axes

Model (2.3) has two equilibrium points that lie along the axes. In particular, one axis equilibrium is characterized by the presence of (homozygous) left-handed fish only, while the other axis equilibrium is characterized by the presence of homozygous right-handed fish only. Notice that there is no axis equilibrium characterized by the
presence of heterozygous right-handed fish since all three genotypes arise from matings between heterozygous individuals. The axes equilibria are given by the following formulae,

\[
(A_{LL}^*, A_{RL}^*, A_{RR}^*) = \left( \frac{nBg(1) - \mu_a}{c \mu_a}, 0, 0 \right),
\]

\[
(A_{LL}^*, A_{RL}^*, A_{RR}^*) = \left( 0, 0, \frac{nBg(1) - \mu_a}{c \mu_a} \right).
\]

The axes equilibria are biologically meaningful and distinct from the origin provided \(nBg(1) > \mu_a\).

We can determine the local asymptotic stability of the axes equilibria from the modulus of the eigenvalues of the Jacobian matrix evaluated at each equilibrium. The eigenvalues associated with \((A_{LL}^*, A_{RL}^*, A_{RR}^*) = \left( \frac{nBg(1) - \mu_a}{c \mu_a}, 0, 0 \right)\) are:

\[
\lambda_1 = 1 - \mu_a,
\]

\[
\lambda_2 = \frac{nBg(1) (1 - \mu_a) + \mu_a^2}{nBg(1)},
\]

\[
\lambda_3 = \frac{g(1) (1 - \mu_a) + h(1) \mu_a}{g(1)}.
\]

By inspecting the eigenvalues, we find that \(\left( \frac{nBg(1) - \mu_a}{c \mu_a}, 0, 0 \right)\) is locally asymptotically stable provided \(h(1) < g(1)\), and unstable if the inequality is reversed. However, the condition \(h(1) < g(1)\) cannot be met under assumption (2.4) on \(h\). Recall that assumption (2.4) implies \(h(1) \geq \min\{g(1), g(0)\} = g(1)\). Therefore, we conclude that \(\left( \frac{nBg(1) - \mu_a}{c \mu_a}, 0, 0 \right)\) is unstable.
The eigenvalues of the Jacobian matrix evaluated at \( (0, 0, \frac{nBg(1) - \mu_a}{c\mu_a}) \) are:

\[
\begin{align*}
\lambda_1 &= 1 - \mu_a, \\
\lambda_2 &= \frac{nBg(1)(1 - \mu_a) + \mu_a^2}{nBg(1)}, \\
\lambda_3 &= 1.
\end{align*}
\]

The eigenvalue \( \lambda_2 \) must be in modulus less than one for the axis equilibrium to be non-trivial. Thus, \( \lambda_3 = 1 \) indicates \( (0, 0, \frac{nBg(1) - \mu_a}{c\mu_a}) \) is, at best, marginally stable.

2.5. Positive equilibria

Now that we have investigated the trivial equilibrium as well as equilibria lying along the axes, we turn our attention to the existence of positive equilibria.

**Theorem 2.5.1.** If \( \frac{nB}{\mu_a} > 1 \) model (2.3) has a unique positive equilibrium given by

\[
(A^*_{LL}, A^*_{RL}, A^*_{RR}) = N^* \left( \frac{1}{2}, \sqrt{2} - 1, \frac{3}{2} - \sqrt{2} \right)
\]

where

\[
N^* = A^*_{LL} + A^*_{RL} + A^*_{RR} = \frac{nB - \mu_a}{c\mu_a}.
\]

Left- and right-handed fish are equally frequent at equilibrium.

**Proof.** By inspection, \( \frac{nB}{\mu_a} > 1 \) implies that \( (A^*_{LL}, A^*_{RL}, A^*_{RR}) \) is positive. We will now show that \( (A^*_{LL}, A^*_{RL}, A^*_{RR}) \) is an equilibrium of model (2.3) by substituting it into each of the model equations:
\[A_{LL}(t + 1) = \frac{nBN^*}{1 + cN^*}\left[\left(\frac{1}{2}\right)^2 g\left(\frac{1}{2}\right) + \left(\frac{1}{2}\right)(\sqrt{2} - 1) h\left(\frac{1}{2}\right) + \frac{(\sqrt{2} - 1)^2}{4} g\left(\frac{1}{2}\right)\right] + \frac{(1 - \mu_a)N^*}{2},\]

\[= \frac{nBN^*}{2[1 + cN^*]} + \frac{(1 - \mu_a)N^*}{2} = \frac{nBN^*}{2\left(1 + \frac{nB - \mu_a}{\mu_a}\right)} + \frac{(1 - \mu_a)N^*}{2},\]

\[= \frac{\mu_a N^*}{2} + \frac{(1 - \mu_a)N^*}{2} = \frac{N^*}{2} = A_{LL},\]

\[A_{RL}(t + 1) = \frac{nBN^*}{1 + cN^*}\left[\left(\frac{1}{2}\right)^2 g\left(\frac{1}{2}\right) + \left(\frac{1}{2}\right)(\sqrt{2} - 1) h\left(\frac{1}{2}\right) + \frac{(\sqrt{2} - 1)^2}{4} g\left(\frac{1}{2}\right)\right]

+ \left(\left(\frac{1}{2}\right)(\sqrt{2} - 1) + 2\left(\frac{1}{2}\right)\left(\frac{3}{2} - \sqrt{2}\right)\right) h\left(\frac{1}{2}\right) + (1 - \mu_a)\left(\sqrt{2} - 1\right)N^*,\]

\[= \frac{nBN^*(\sqrt{2} - 1)}{[1 + cN^*]} + (1 - \mu_a)(\sqrt{2} - 1)N^*,\]

\[= \frac{nBN^*(\sqrt{2} - 1)}{\left(1 + \frac{nB - \mu_a}{\mu_a}\right)} + (1 - \mu_a)(\sqrt{2} - 1)N^*,\]

\[= \mu_a N^*(\sqrt{2} - 1) + (1 - \mu_a)(\sqrt{2} - 1)N^*,\]
\[ A_{RR}(t + 1) = \frac{nBN^*}{1 + cN^*} \left[ \left( \frac{3}{2} - \sqrt{2} \right)^2 + \left( \frac{3}{2} - \sqrt{2} \right) (\sqrt{2} - 1) + \frac{1}{4} (\sqrt{2} - 1)^2 \right] \phi \left( \frac{1}{2} \right) + \left( 1 - \mu_a \right) \left( \frac{3}{2} - \sqrt{2} \right) N^*, \]

\[ = \frac{nBN^*}{1 + cN^*} \left[ \left( \frac{3}{2} - \sqrt{2} \right)^2 + \left( \frac{3}{2} - \sqrt{2} \right) (\sqrt{2} - 1) + \frac{1}{4} (\sqrt{2} - 1)^2 \right] \phi \left( \frac{1}{2} \right) + \left( 1 - \mu_a \right) \left( \frac{3}{2} - \sqrt{2} \right) N^*, \]

\[ = \frac{nBN^*}{1 + cN^*} \left[ \left( \frac{3}{2} - \sqrt{2} \right)^2 + \left( \frac{3}{2} - \sqrt{2} \right) (\sqrt{2} - 1) + \frac{1}{4} (\sqrt{2} - 1)^2 \right] \phi \left( \frac{1}{2} \right) + \left( 1 - \mu_a \right) \left( \frac{3}{2} - \sqrt{2} \right) N^*, \]

\[ = \frac{nBN^*}{1 + cN^*} \left[ \left( \frac{3}{2} - \sqrt{2} \right)^2 + \left( \frac{3}{2} - \sqrt{2} \right) (\sqrt{2} - 1) + \frac{1}{4} (\sqrt{2} - 1)^2 \right] \phi \left( \frac{1}{2} \right) + \left( 1 - \mu_a \right) \left( \frac{3}{2} - \sqrt{2} \right) N^*, \]

\[ = \left( \frac{3}{2} - \sqrt{2} \right) N^* = A_{RR}^*. \]

We next show that this equilibrium is the only positive equilibrium.

Let \((A_{LL}', A_{RL}', A_{RR}')\) be a positive equilibrium of (2.3) and let
\[ N' = A'_{LL} + A'_{RL} + A'_{RR}, \]
\[ \alpha = \frac{A'_{LL}}{N'}, \]
\[ \beta = \frac{A'_{RL}}{N'}, \]
\[ 1 - \alpha - \beta = \frac{A'_{RR}}{N'}, \]
\[ 1 - \alpha = \frac{A'_{RL} + A'_{RR}}{N'}. \]

Summing the three equations of model (2.3) evaluated at the equilibrium we obtain

\[ N' = \frac{nBN'}{1 + cN'} \left[ \left( \frac{A'_{LL}}{N'} \right)^2 g \left( \frac{A'_{LL}}{N'} \right) + \frac{2A'_{LL}}{N'} \left( 1 - \frac{A'_{LL}}{N'} \right) h \left( \frac{A'_{LL}}{N'} \right) + \left( 1 - \frac{A'_{LL}}{N'} \right)^2 g \left( 1 - \frac{A'_{LL}}{N'} \right) \right] \]
\[ + (1 - \mu_a)N', \]
\[ \mu_a N' = \frac{nBN'}{1 + cN'} \left[ \left( \frac{A'_{LL}}{N'} \right)^2 g \left( \frac{A'_{LL}}{N'} \right) + \frac{2A'_{LL}}{N'} \left( 1 - \frac{A'_{LL}}{N'} \right) h \left( \frac{A'_{LL}}{N'} \right) + \left( 1 - \frac{A'_{LL}}{N'} \right)^2 g \left( 1 - \frac{A'_{LL}}{N'} \right) \right], \]
\[ \mu_a = \frac{nB}{1 + cN'} \left[ \alpha^2 g(\alpha) + 2\alpha(1 - \alpha) h(\alpha) + (1 - \alpha)^2 g(1 - \alpha) \right]. \]

Solving for \( \frac{1}{1 + cN'} \) we find that

\[ \frac{1}{1 + cN'} = \frac{\mu_a}{nB \left[ \alpha^2 g(\alpha) + 2\alpha(1 - \alpha) h(\alpha) + (1 - \alpha)^2 g(1 - \alpha) \right]}. \] (2.9)
We now substitute $\alpha = \frac{A'}{N}$, $\beta = \frac{A''}{N}$, and $1 - \alpha - \beta = \frac{A'}{N}$ into the three equations of model (2.3) and, into the following results

\[ \mu_{a\alpha} = \frac{nB}{1 + cn} \left[ \alpha^2 g(\alpha) + \alpha \beta h(\alpha) + \frac{1}{4} \beta^2 g(1-\alpha) \right], \]
\[ \mu_{a\beta} = \frac{nB}{1 + cn} \left[ \left( \frac{1}{2} \beta^2 + (1-\alpha-\beta)\beta \right) g(1-\alpha) + (\alpha \beta + 2(1-\alpha-\beta)\alpha) h(\alpha) \right], \]
\[ \mu_{a(1-\alpha-\beta)} = \frac{nB}{1 + cn} \left[ (1-\alpha-\beta)^2 + (1-\alpha-\beta)\beta + \frac{1}{4} \beta^2 \right] g(1-\alpha), \]

we substitute (2.9). This substitution yields the following three quadratic functions of $\beta$,

\[ \beta^2 + \frac{4\alpha h(\alpha)}{g(1-\alpha)} \beta + \frac{4\alpha}{g(1-\alpha)} \left[ \alpha(1-\alpha)g(\alpha) - 2\alpha(1-\alpha)h(\alpha) - (1-\alpha)^2 g(1-\alpha) \right] = 0, \]
\[ \beta^2 + \frac{2\alpha}{g(1-\alpha)} \left[ \alpha g(\alpha) + (3-2\alpha)h(\alpha) - (1-\alpha)g(1-\alpha) \right] \beta - \frac{4\alpha(1-\alpha)h(\alpha)}{g(1-\alpha)} = 0, \]
\[ \beta^2 + \frac{4\alpha}{g(1-\alpha)} \left[ \alpha(1-\alpha)g(\alpha) + 2(1-\alpha)h(\alpha) - (1-\alpha)^2 g(1-\alpha) \right] \beta \]
\[ - \frac{4\alpha}{g(1-\alpha)} \left[ \alpha(1-\alpha)g(\alpha) + 2(1-\alpha)^2 h(\alpha) - (1-\alpha)^2 g(1-\alpha) \right] = 0. \]

We can solve for $\beta$ in each of the above equations and we require the $\beta$’s equal one another. For this to happen it is necessary that
\[ G(\alpha) = \alpha g(\alpha) + (1 - 2\alpha)h(\alpha) - (1 - \alpha)g(1 - \alpha) = 0. \]

Clearly the above equation holds if \( \alpha = \frac{1}{2} \). Now consider the following two cases:

Case 1: Suppose \( \alpha > \frac{1}{2} \). Then we know \( h(\alpha) \geq \min\{g(\alpha), g(1 - \alpha)\} = g(\alpha) \), and we have

\[
G(\alpha) = \alpha g(\alpha) + (1 - 2\alpha)h(\alpha) - (1 - \alpha)g(1 - \alpha),
\]

\[
\leq \alpha g(\alpha) + (1 - 2\alpha)g(\alpha) - (1 - \alpha)g(1 - \alpha),
\]

\[
= (1 - \alpha)g(\alpha) - (1 - \alpha)g(1 - \alpha),
\]

\[
= (1 - \alpha)[g(\alpha) - g(1 - \alpha)] < 0.
\]

Case 2: Suppose \( \alpha < \frac{1}{2} \). Then we know \( h(\alpha) \geq \min\{g(\alpha), g(1 - \alpha)\} = g(1 - \alpha) \), and we have

\[
G(\alpha) = \alpha g(\alpha) + (1 - 2\alpha)h(\alpha) - (1 - \alpha)g(1 - \alpha),
\]

\[
\geq \alpha g(\alpha) + (1 - 2\alpha)g(1 - \alpha) - (1 - \alpha)g(1 - \alpha),
\]

\[
= \alpha g(\alpha) - \alpha g(1 - \alpha),
\]

\[
= \alpha [g(\alpha) - g(1 - \alpha)] > 0.
\]
Therefore, we have shown that $\alpha = \frac{A'_{LL}}{N'} = \frac{1}{2}$ for any equilibrium, and thus, $1 - \alpha = \frac{A'_{RL} + A'_{RR}}{N'} = \frac{1}{2}$ (i.e., a positive equilibrium must consist of 50% left-handed fish and 50% right-handed fish).

Clearly there are infinitely many equilibria, $(A'_{LL}, A'_{RL}, A'_{RR})$, such that $\frac{A'_{LL}}{N'} = \frac{1}{2}$ and $\frac{A'_{RL} + A'_{RR}}{N'} = \frac{1}{2}$. However, all three of the quadratic equations (in $\beta$) with $\alpha = \frac{1}{2}$ reduce to

$$\beta^2 + 2\beta - 1 = 0,$$

and we conclude that $\beta = \frac{A'_{RL}}{N'} = \sqrt{2} - 1$. We can solve for $N'$ with $\alpha = \frac{1}{2}$ using (2.9) as

$$\frac{1}{1 + cN'} = \frac{\mu_a}{nB \left[ \alpha^2 g(\alpha) + 2\alpha(1-\alpha) h(\alpha) + (1-\alpha)^2 g(1-\alpha) \right]} = \frac{\mu_a}{nB},$$

and we find $N'$ as

$$N' = \frac{nB - \mu_a}{c\mu_a}.$$

Therefore, we have shown that any positive equilibrium of model (2.3) must have the form

$$\left(A'_{LL}, A'_{RL}, A'_{RR}\right) = N' \left(\frac{1}{2}, (\sqrt{2} - 1), \left(\frac{3}{2} - \sqrt{2}\right)\right)$$

where

$$N' = \frac{nB - \mu_a}{c\mu_a}.$$

Thus, $\alpha = \frac{1}{2}$ and we have shown $(A'_{LL}, A'_{RL}, A'_{RR}) = (A^*_{LL}, A^*_{RL}, A^*_{RR})$, i.e., we have shown uniqueness. $\square$
Our finding of a positive equilibrium corresponding to a 1 : 1 phenotypic ratio corroborates the conclusion of Takahashi and Hori [40]. Beyond this, we have shown that such an equilibrium is unique.

2.6. Stability of positive equilibrium

The stability of the positive equilibrium in Theorem 2.5.1 can be determined by the modulus of the eigenvalues of the Jacobian matrix evaluated at the equilibrium. With \( h'(\frac{1}{2}) = 0 \), the eigenvalues of the Jacobian evaluated at the positive equilibrium are

\[
\begin{align*}
\lambda_1 &= 1 - \mu_a, \\
\lambda_2 &= \frac{nB(1 - \mu_a) + \mu_a^2}{nB}, \\
\lambda_3 &= \frac{\mu_ag'(\frac{1}{2}) (\sqrt{2} - 1) + 2}{2}.
\end{align*}
\]

By inspecting the eigenvalues we find that the positive equilibrium is locally asymptotically stable provided,

\[
g'(\frac{1}{2}) > -\frac{4}{\mu_a(\sqrt{2} - 1)}.
\]

Notice that \( g'(\frac{1}{2}) = -\frac{4}{\mu_a(\sqrt{2} - 1)} \) forces \( \lambda_3 \) to escape the unit disk at \(-1\), suggesting the occurrence of a period doubling bifurcation. Recalling the scaling factor in the function \( g \), we see that the positive equilibrium destabilizes for

\[
q \doteq f'(\frac{1}{2}) < -\frac{4f'(\frac{1}{2})}{\mu_a(\sqrt{2} - 1)}.
\] (2.10)
Figure 2.1 shows an example of a stable two-cycle that results from such a period doubling bifurcation.

**Figure 2.1.** Time series plot showing the stable two-cycle. We used \( h = f\left(\frac{1}{2}\right) \) (mean fitness), and \( g \) was defined as in equation (2.7). The initial condition is: \((A_{LL}, A_{RL}, A_{RR}) = (20, 2, 6)\). The parameter values are: \( nb = 50, \ c = 0.1, \ q = -8.5, \) and \( \mu_a = 0.6. \)

We see that the quantity \( q \) is a critical value for the destabilization of the positive equilibrium [40]. Biologically, we interpret \( q \) as a measure of the prey response sensitivity. Recall that \( f_1(l) \) and \( f_1(1 - l) \) determine the fitness of the matings \( L \times L \) and \( R \times R \), respectively; fitness (and ultimately reproductive success) is a function of phenotypic frequency. The quantity \( q = f'_1(\frac{1}{2}) \) indicates how small deviations from the 1 : 1 phenotypic ratio translate to changes in fitness for mating between like phenotypes. If \( q \) is large in magnitude, a small departure from the 1 : 1 phenotypic ratio results in a large difference in reproductive success. Matings between unlike
phenotypes, accounted for by the function, $h$ or $f_2$, do not alter the stability of the unique positive equilibrium. In particular, recall that we have assumed that $h$ is smooth with $h'(\frac{1}{2}) = 0$. This assumption implies that small deviations from the 1 : 1 phenotypic ratio cannot drastically affect the reproductive success of mate pairings between unlike phenotypes.

The prey fish are implicitly modeled through the fitness functions $f_1$ and $f_2$: a large decrease in fitness signifies prey with a powerful guard. In particular, the derivative functions of the fitness functions indicate how strongly the prey affect the reproductive output of $P.\ microlepis$ due to changes in the phenotypic ratio. Prey that respond strongly to the ratio

$$\frac{A_{LL}(t)}{A_{RL}(t) + A_{RR}(t)} = 1 + \epsilon,$$

where $\epsilon$ is small in magnitude, correspond to values of $q$ that are large in magnitude. Our stability analysis indicates that a sensitive and strong prey response destabilizes the positive equilibrium; prey fish must be able to detect small values of $\epsilon$ and respond aggressively. From an evolutionary perspective, it may be adaptive for the prey to be sensitive to small deviations from the 1 : 1 phenotypic ratio. Sensitivity may protect the prey species from suffering a greater number of attacks as the phenotypic ratio shifts.

The parameter region for stability is depicted in Figure 2.2. Notice that the probability of death ($\mu_a$) must be sufficiently large for the destabilization of the positive equilibrium; Takahashi and Hori also reached this conclusion [40]. As $\mu_a$ increases it becomes possible to destabilize the positive equilibrium with biologically reasonable values of $q$. This result suggests that a strongly iteroparous life cycle (i.e., a high probability of survival) stabilizes $P.\ microlepis$. Our finding that large values of $|q|$ promote equilibrium destabilization confirms the discovery of Takahashi and Hori [40]. Our analysis, however, explicitly indicates that the destabilization of the
positive equilibrium in model (2.3) is the result of a period doubling bifurcation.

Under the assumptions of our model, we have shown that the fitness function associated with $R \times L$ and $L \times R$ matings ($f_2$) does not affect the stability of the positive equilibrium. Critical to this finding is the assumption that $h'(\frac{1}{2}) = 0$; we will explore what happens when this assumption is violated in a later chapter. Furthermore, our analysis of model (2.3), which lacked a developmental time lag, demonstrated that a time delay is not necessary for oscillatory behavior. This finding is novel, since Takahashi and Hori hypothesized that the time delay in development was, in part, responsible for the phenotypic oscillation [40]. We explore the effect of time delays in Chapter 3.

The destabilization of the positive equilibrium, as $q$ becomes sufficiently large in
magnitude, is shown as a bifurcation diagram in Figure 2.3. Interestingly, Figure 2.3 also shows that the time-averaged frequency of sinistral fish is greater than the time-averaged frequency of dextral fish. This phenomenon seems counterintuitive because the right-handed allele \((R)\) is completely dominant over the left-handed allele \((L)\). We investigate this finding in the next section.

**Figure 2.3.** Bifurcation diagram showing the period doubling bifurcation in phenotypic frequency as \(q\) becomes sufficiently large in magnitude. We used \(h = f \left( \frac{1}{2} \right)\) (mean fitness), and \(g\) was defined as in (2.7). The initial condition is: \((A_{LL}, A_{RL}, A_{RR}) = (20, 2, 6)\). The parameter values are: \(nb = 50, c = 0.1,\) and \(\mu_a = 0.6\).

2.7. Local bifurcation analysis

Following the period doubling bifurcation depicted in Figure 2.3, the time-averaged frequency of sinistral fish is greater than the time-averaged frequency of dextral fish.
We investigate this phenomenon using a local bifurcation analysis called a Lyapunov-Schmidt expansion [7]. This approach provides us with information about the two-cycle near the period doubling bifurcation, since we cannot find explicit formulae for the two-cycle.

Theorem 2.7.1. The branch of two-cycles of model (2.3) can be parameterized near the bifurcation point by the following expansions:

\[
A_{LL} = A_{LL}^* + x = \frac{N^*}{2} + (\sqrt{2} + 1)(-1)^t \epsilon + x_2^* \epsilon^2 + \ldots,
\]

\[
A_{RL} = A_{RL}^* + y = (\sqrt{2} - 1)N^* + \sqrt{2}(1)^t \epsilon + y_2^* \epsilon^2 + \ldots,
\]

\[
A_{RR} = A_{RR}^* + z = \left(\frac{3}{2} - \sqrt{2}\right)N^* + (-1)^t \epsilon + z_2^* \epsilon^2 + \ldots,
\]

where \(x_2^*, y_2^*,\) and \(z_2^*\) are given by

\[
\begin{pmatrix}
x_2^* \\
y_2^* \\
z_2^*
\end{pmatrix} =
\begin{pmatrix}
\frac{-c[4nBg'(\frac{1}{2})^2(2\sqrt{2} - 3) + 4nBg'(\frac{1}{2})(7 - 5\sqrt{2}) + 8(4\sqrt{2} - 6)]}{4(7 - 5\sqrt{2}) + nBg'(\frac{1}{2})(12\sqrt{2} - 17)}
\\
\frac{2c[nBg'(\frac{1}{2})^2(27\sqrt{2} - 38) + 2nBg'(\frac{1}{2})(52 - 37\sqrt{2}) + 4(15\sqrt{2} - 22) + 4g'(\frac{1}{2})(1 - 3\sqrt{2})]}{4(7 - 5\sqrt{2}) + nBg'(\frac{1}{2})(12\sqrt{2} - 17)}
\\
\frac{-c[2nBg'(\frac{1}{2})^2(31\sqrt{2} - 44) + 2nBg'(\frac{1}{2})(38 - 27\sqrt{2}) + 8(11\sqrt{2} - 16) + 8g'(\frac{1}{2})(1 - 3\sqrt{2})]}{4(7 - 5\sqrt{2}) + nBg'(\frac{1}{2})(12\sqrt{2} - 17)}
\end{pmatrix}.
\]

The time-averaged frequency of sinistral fish is greater than the time-averaged frequency of dextral fish in a neighborhood of the period doubling bifurcation point in model (2.3).

Proof. We begin by centering model (2.3) on the positive equilibrium by using vari-
ables

\[ x(t) = A_{LL}(t) - A_{LL}^*, \]
\[ y(t) = A_{RL}(t) - A_{RL}^*, \]
\[ z(t) = A_{RR}(t) - A_{RR}^*, \]
\[ n(t) = x(t) + y(t) + z(t), \]

where \( N(t) = n(t) + N^* \), and \((x^*, y^*, z^*) = (0, 0, 0)\) is the new equilibrium. We parameterize the branch of two-cycles by expanding \(x, y, \text{ and } z\) about 0. We expand the bifurcation parameter, \(s = 1 - \mu_a\), about

\[ 1 - \mu_a^{\text{crit}} = 1 + \frac{4}{g'(\frac{1}{2}) (\sqrt{2} - 1)}. \]

We introduce a small parameter \(\epsilon\) as

\[ x(t) = x_1(t)\epsilon + x_2(t)\epsilon^2 + \ldots, \]
\[ y(t) = y_1(t)\epsilon + y_2(t)\epsilon^2 + \ldots, \]
\[ z(t) = z_1(t)\epsilon + z_2(t)\epsilon^2 + \ldots, \]

and,

\[ s = 1 + \frac{4}{g'(\frac{1}{2}) (\sqrt{2} - 1)} + s_1\epsilon + s_2\epsilon^2 + \ldots. \]

We deduce that \(s_1 = 0\), since the bifurcation is period doubling; the two-cycle exists to one side of criticality only. Substituting the above expansions into the model equations and matching powers of \(\epsilon\) we obtain
\[
\begin{pmatrix}
  x_1(t+1) \\
  y_1(t+1) \\
  z_1(t+1)
\end{pmatrix}
= J
\begin{pmatrix}
  x_1(t) \\
  y_1(t) \\
  z_1(t)
\end{pmatrix},
\]

where \( J \) is the Jacobian matrix evaluated at the equilibrium. The two-cycle solution of this equation consists of the eigenvector that corresponds to \( \lambda = -1 \) and its image

\[
\begin{pmatrix}
  x_1(t) \\
  y_1(t) \\
  z_1(t)
\end{pmatrix}
= (-1)^t v,
\]

where

\[
v = \begin{pmatrix}
  \sqrt{2} + 1 \\
  -\sqrt{2} \\
  -1
\end{pmatrix}.
\]

Determining the second order terms in the expansion equates to finding the two-cycle solution of

\[
\begin{pmatrix}
  x_2(t+1) \\
  y_2(t+1) \\
  z_2(t+1)
\end{pmatrix}
= J
\begin{pmatrix}
  x_2(t) \\
  y_2(t) \\
  z_2(t)
\end{pmatrix} + u,
\]  \hspace{1cm} \text{(2.11)}

where \( u \) is a constant given by
\[
\begin{pmatrix}
\frac{4c}{g'(\frac{1}{2})} \left[ g'\left(\frac{1}{2}\right)(1+\sqrt{2}) + 2g(\frac{1}{2}) \left(4+\sqrt{2}\right) + 2(\sqrt{2}+1) \right] \\
g'(\frac{1}{2})\left(\sqrt{2}-1\right)^2 \left[ nBg'\left(\frac{1}{2}\right)(\sqrt{2}-1)+4 \right] \\
8c \cdot g'(\frac{1}{2}) + g'(\frac{1}{2}) \left[ 3\sqrt{2}-2 \right] - 2\left(\sqrt{2}+1\right) \\
g'(\frac{1}{2})\left(\sqrt{2}-1\right)^2 \left[ nBg'\left(\frac{1}{2}\right)(\sqrt{2}-1)+4 \right]
\end{pmatrix}
= \begin{pmatrix}
\frac{4c}{g'(\frac{1}{2})} \left[ g'\left(\frac{1}{2}\right)(1+\sqrt{2}) + 2g(\frac{1}{2}) \left(4+\sqrt{2}\right) + 2(\sqrt{2}+1) \right] \\
\frac{8c}{g'(\frac{1}{2})} \left[ 3\sqrt{2}-2 \right] - 2\left(\sqrt{2}+1\right) \\
\frac{4c}{g'(\frac{1}{2})} \left[ 3\sqrt{2}-2 \right] - 2\left(\sqrt{2}+1\right) \\
\frac{4c}{g'(\frac{1}{2})} \left[ 3\sqrt{2}-2 \right] - 2\left(\sqrt{2}+1\right)
\end{pmatrix}.
\]

The second order coefficients: \(x_2, y_2,\) and \(z_2,\) are constants found as the equilibrium solution of (2.11):

\[
\begin{pmatrix}
x_2^s \\
y_2^s \\
z_2^s
\end{pmatrix}
= J \begin{pmatrix}
x_2^s \\
y_2^s \\
z_2^s
\end{pmatrix} + u,
\]

\[
(I - J) \begin{pmatrix}
x_2^s \\
y_2^s \\
z_2^s
\end{pmatrix}
= u,
\]

\[
\begin{pmatrix}
x_2^s \\
y_2^s \\
z_2^s
\end{pmatrix}
= (I - J)^{-1} u.
\]

We find \(x_2 = x_2^s,\ y_2 = y_2^s,\) and \(z_2 = z_2^s\) to be
\[
\begin{pmatrix}
    x^*_2 \\
    y^*_2 \\
    z^*_2
\end{pmatrix} = \begin{pmatrix}
    x_1(t) \\
    y_1(t) \\
    z_1(t)
\end{pmatrix} = \begin{pmatrix}
    -c [nBg'(\frac{1}{2}) (2\sqrt{2} - 3) + 4nBg'(\frac{1}{2}) (7 - 5\sqrt{2}) + 8(4\sqrt{2} - 6)] \\
    [4(7 - 5\sqrt{2}) + nBg'(\frac{1}{2}) (12\sqrt{2} - 17)] [nBg'(\frac{1}{2}) (\sqrt{2} - 1) + 4]
\end{pmatrix}.
\]

We can now write the expansions for the bifurcating two-cycle:

\[
x(t) = x_1(t)\epsilon + x_2\epsilon^2 + \ldots = (\sqrt{2} + 1)(-1)^t\epsilon + x^*_2\epsilon^2 + \ldots,
\]

\[
y(t) = y_1(t)\epsilon + y_2\epsilon^2 + \ldots = \sqrt{2}(-1)^t\epsilon + y^*_2\epsilon^2 + \ldots,
\]

\[
z(t) = z_1(t)\epsilon + z_2\epsilon^2 + \ldots = (-1)^t\epsilon + z^*_2\epsilon^2 + \ldots.
\]

Translating the expansions to the original state variables: \( A_{LL}, A_{RL}, \) and \( A_{RR} \) we obtain

\[
A_{LL} = A_{LL}^* + x = \frac{N^*}{2} + (\sqrt{2} + 1)(-1)^t\epsilon + x^*_2\epsilon^2 + \ldots,
\]

\[
A_{RL} = A_{RL}^* + y = (\sqrt{2} - 1)N^* + \sqrt{2}(-1)^t\epsilon + y^*_2\epsilon^2 + \ldots,
\]

\[
A_{RR} = A_{RR}^* + z = \left(\frac{3}{2} - \sqrt{2}\right)N^* + (-1)^t\epsilon + z^*_2\epsilon^2 + \ldots.
\]
We now use the parameterized branch of two-cycles to rigorously show that the time-averaged frequency of sinistral fish is greater than the time-averaged frequency of dextral fish. Specifically, we demonstrate that

$$\frac{A_{LL}(t+1) + A_{LL}(t)}{N(t+1) + N(t)} > \frac{A_{RL}(t+1) + A_{RL}(t) + A_{RR}(t+1) + A_{RR}(t)}{N(t+1) + N(t)}.$$ (2.12)

For $\epsilon$ sufficiently small, this inequality is equivalent to

$$A_{LL}^* + x_2^* \epsilon^2 > A_{RL}^* + A_{RR}^* + y_2^* \epsilon^2 + z_2^* \epsilon^2,$$

which in turn is equivalent to

$$\frac{N^*}{2} + x_2^* \epsilon^2 > (\sqrt{2} - 1)N^* + \left(\frac{3}{2} - \sqrt{2}\right)N^* + (y_2^* + z_2^*) \epsilon^2,$$

or

$$x_2^* - y_2^* - z_2^* > 0.$$ (2.13)

Substituting the known values of $x_2^*$, $y_2^*$, and $z_2^*$ into (2.13) we arrive finally with the inequality

$$8c \left[ nBg\left(\frac{1}{2}\right) (5\sqrt{2} - 7) - 4(2\sqrt{2} - 3) \right] \left[ 4(7 - 5\sqrt{2}) + nBg\left(\frac{1}{2}\right) (12\sqrt{2} - 17) \right] \left[ nBg\left(\frac{1}{2}\right) (\sqrt{2} - 1) + 4 \right] > 0.$$ (2.14)

The two-cycle emerges due to the destabilization of the positive equilibrium for

$$g^\prime\left(\frac{1}{2}\right) < -\frac{4}{\mu_a(\sqrt{2} - 1)} < -\frac{4}{nB(\sqrt{2} - 1)}.$$ (2.15)

Using (2.15), we demonstrate that the denominator of (2.14) is negative. From (2.15)
we have that

$$g'\left(\frac{1}{2}\right) < -\frac{4}{nB(\sqrt{2} - 1)} = -\frac{4(7 - 5\sqrt{2})}{nB(12\sqrt{2} - 17)},$$

$$\left[nB(12\sqrt{2} - 17)\right]g'\left(\frac{1}{2}\right) > -4(7 - 5\sqrt{2}),$$

$$\left[4(7 - 5\sqrt{2}) + nBg'\left(\frac{1}{2}\right)(12\sqrt{2} - 17)\right] > 0,$$

and hence the first factor of the denominator is positive. The second factor is negative, which is immediately obvious from (2.15). Thus, the denominator of (2.14) is negative. Furthermore, the numerator of (2.14) is negative as the following string of inequalities show:

$$g'\left(\frac{1}{2}\right) < \frac{4(2\sqrt{2} - 3)}{nB(5\sqrt{2} - 7)} = -\frac{4}{nB(\sqrt{2} - 1)},$$

$$nBg'\left(\frac{1}{2}\right)(5\sqrt{2} - 7) < 4(2\sqrt{2} - 3),$$

$$nBg'\left(\frac{1}{2}\right)(5\sqrt{2} - 7) - 4(2\sqrt{2} - 3) < 0.$$

Thus, (2.12) holds and the time-averaged frequency of sinistral fish is greater than the time-averaged frequency of dextral fish, at least for small values of \(\epsilon\) where we know the two-cycle expansion is valid.

Numerical simulations suggest that the phenomenon described in Theorem 2.7.1 is not confined near the bifurcation point. In fact, we were not able to find a numerical example where the time-averaged frequency of dextral fish was greater than the time-


averaged frequency of sinistral fish for model (2.3). The proof of Theorem 2.7.1 identifies the phenomenon as second order in nature and, as such, would likely be difficult to replicate experimentally, at least near the bifurcation point. *A priori*, we may have expected the time-average frequency of sinistral and dextral fish to be equal. However, our model has a natural asymmetry: $R$ is completely dominant over $L$. Therefore, it seems counterintuitive that the genotype with two recessive alleles is numerically dominant (on average). Upon closer consideration our finding may be expected since the $L$ allele is not subject to strong frequency-dependent selection in the heterozygous state when sinistral fish are numerically dominant. This notion is analogous to the persistence of recessive diseases because detrimental recessive alleles are not exposed to purifying selection in the heterozygous state.
3.1. Model extension

In this section we extend our initial (three-dimensional) model by incorporating further biological realism. In particular, we increase the dimension of (2.3) by introducing stage structure. We will present two models: one with an additional stage class and one with two additional stages classes. By increasing the number of stage classes, we shorten the projection time interval and control the nonlinear terms affecting each stage. This is important since *P. microlepis* possesses different behaviors at the various life stages. Adult *P. microlepis* are monogamous mouthbrooders: a female broods the eggs and embryos in her mouth and the mating pair jointly care for and defend their fry [4], [28]. Parental care is absent as fry mature into the juvenile stage, where juveniles are free-swimming, sexually immature and feed on plankton [16]. Maturation to the adult stage (approximately two years from birth) is characterized by sexually maturity and scale-eating. Based on these biological facts, it is natural to model each *P. microlepis* genotype with three life stages: fry, juveniles, and adults. We begin by extending (2.3) to six dimensions in order to determine how increasing dimensionality, from three to six to nine dimensions, changes the dynamics of the system. We also take this intermediate step to better compare our model analysis and results to the six-dimensional model of Takahashi and Hori [40].
3.2. Six-dimensional, juvenile-adult model definition

We begin the construction of our six-dimensional model by introducing three new state variables, $J_{LL}(t)$, $J_{RL}(t)$, and $J_{RR}(t)$, which represent the number of (homozygous) left-handed, heterozygous right-handed, and homozygous right-handed sexually immature, plankton-feeding juvenile fish at time $t$, respectively. Handedness is a well defined characteristic in juvenile fish: it is detectable and absolute in fry fish [16]. As described in (2.3), $A_{LL}(t)$, $A_{RL}(t)$, and $A_{RR}(t)$ represent the number of (homozygous) left-handed, heterozygous right-handed, and homozygous right-handed sexually mature, scale-eating adult fish at time $t$, respectively. The projection time interval is one year, and $nN(t)$ is the number of matings that occur within this time period. Let $B$, $\mu_a$, $c$, and $l(t)$ be defined as for model (2.3). The total adult population size is represented by, $N(t) = A_{LL}(t) + A_{RL}(t) + A_{RR}(t)$. We introduce the new parameter $0 < \mu_j < 1$ as the probability of juvenile death in one time step. Incorporating all six state variables, we write the new extension of (2.3) as,

\begin{align*}
J_{LL}(t+1) &= \frac{nBN(t)}{1+cN(t)} \left[ \left( \frac{A_{LL}(t)}{N(t)} \right)^2 g \left( \frac{A_{LL}(t)}{N(t)} \right) + \left( \frac{A_{LL}(t)A_{RL}(t)}{N(t)^2} \right) h \left( \frac{A_{LL}(t)}{N(t)} \right) \right] \\
&\quad + \frac{1}{4} \left( \frac{A_{RL}(t)}{N(t)} \right)^2 g \left( 1 - \frac{A_{LL}(t)}{N(t)} \right) \\
A_{LL}(t+1) &= (1 - \mu_j) J_{LL}(t) + (1 - \mu_a) A_{LL}(t),
\end{align*}

\begin{align*}
J_{RL}(t+1) &= \frac{nBN(t)}{1+cN(t)} \left[ \left( \frac{1}{2} A_{RL}(t)^2 + A_{RR}(t)A_{RL}(t) \right) g \left( 1 - \frac{A_{LL}(t)}{N(t)} \right) \\
&\quad + \left( \frac{A_{LL}(t)A_{RL}(t) + 2A_{RR}(t)A_{LL}(t)}{N(t)^2} \right) h \left( \frac{A_{LL}(t)}{N(t)} \right) \right],
\end{align*}

(3.1)
\[ A_{RL}(t + 1) = (1 - \mu_j) J_{RL}(t) + (1 - \mu_a) A_{RL}(t), \]

\[ J_{RR}(t + 1) = \frac{nBN(t)}{1 + cN(t)} \left[ \frac{A_{RR}(t)^2 + A_{RR}(t)A_{RL}(t) + \frac{1}{4}A_{RL}(t)^2}{N(t)^2} \right] g \left( 1 - \frac{A_{LL}(t)}{N(t)} \right), \]

\[ A_{RR}(t + 1) = (1 - \mu_j) J_{RR}(t) + (1 - \mu_a) A_{RR}(t). \]

We assume that only fry are subject to density-dependent effects. This type of assumption is common when intraspecific competition for resources is intense due a large number of breeding adults and their large number resultant offspring. The adult stage is composed of new adult recruits, those that survived the juvenile stage, and old adults. Adult and juvenile survivorships are assumed to be density independent. We make this assumption since both juvenile and adult fish can swim and defend themselves; eggs, wrigglers, and fry fish are dependent on parental care and limited by the resources provided to them. These assumptions on density-dependence and survivorship are similar to those made in the well-known LPA model of *Tribolium castaneum*; the defenseless eggs and pupae are subject to density-dependent effects while mobile larvae and adults are not [6]. As with model (2.3) we look for a positive equilibrium of (3.1).

3.3. Positive equilibria

In the case of model (2.3), we proved the existence and uniqueness of a positive equilibrium corresponding to a 1 : 1 phenotypic ratio. We approach the analysis for model (3.1) in a similar way.
Theorem 3.3.1. If \( \frac{nB(1-\mu_j)}{\mu_a} > 1 \) model (3.1) has a unique positive equilibrium given by

\[
(J^*_L, J^*_R, J^*_R, A^*_L, A^*_R, A^*_R) = \left( \sigma A^*_L, \sigma A^*_R, \frac{N^*}{\sigma}, (\sqrt{2}-1)N^*, \left( \frac{3}{2} - \sqrt{2} \right)N^* \right)
\]

where

\[
\sigma = \frac{\mu_a}{1 - \mu_j}, \quad N^* = A^*_L + A^*_R + A^*_R = \frac{nB(1-\mu_j) - \mu_a}{c\mu_a}.
\]

Left- and right-handed fish are equally frequent at equilibrium.

**Proof.** By inspection, \( \frac{nB(1-\mu_j)}{\mu_a} > 1 \) implies that \( (J^*_L, J^*_R, J^*_R, A^*_L, A^*_R, A^*_R) \) is positive. We will now show that \( (J^*_L, J^*_R, J^*_R, A^*_L, A^*_R, A^*_R) \) is an equilibrium. By inspecting the equations in (3.1), it is clear that:

\[
J^*_L = \frac{\mu_a}{1 - \mu_j} A^*_L, \quad J^*_R = \frac{\mu_a}{1 - \mu_j} A^*_R, \quad J^*_R = \frac{\mu_a}{1 - \mu_j} A^*_R.
\]

Substituting \( A^*_L, A^*_R, \) and \( A^*_R \) into the juvenile equations of model (3.1) yields,

\[
J^*_L = \frac{nBN^*}{1+cN^*} \left[ \left( \frac{1}{2} \right) g\left( \frac{1}{2} \right) + \left( \frac{1}{2} \right) (\sqrt{2} - 1) g\left( \frac{1}{2} \right) + \frac{1}{4} (\sqrt{2} - 1)^2 g\left( \frac{1}{2} \right) \right],
\]

\[
= \frac{nBN^*}{2 [1 + cN^*]} = \frac{nB}{2} \left( \frac{\frac{nB(1-\mu_j)}{\mu_a} - \mu_a}{2c(1-\mu_j)} \right) = \frac{nB(1-\mu_j) - \mu_a}{2c(1-\mu_j)},
\]
\[ J_{RL}^* = \frac{nBN^*}{1+cN^*} \left[ \left( \frac{1}{2} \left( \sqrt{2} - 1 \right) ^2 + \left( \sqrt{2} - 1 \right) \left( \frac{3}{2} - \sqrt{2} \right) \right) g \left( \frac{1}{2} \right) \right] + \left( \left( \frac{1}{2} \right) \left( \sqrt{2} - 1 \right) + 2 \left( \frac{1}{2} \right) \left( \frac{3}{2} - \sqrt{2} \right) \right) h \left( \frac{1}{2} \right) , \]

\[ = \frac{nB(\sqrt{2} - 1)N^*}{1 + cN^*} \left[ \frac{nB(1-\mu_j) - \mu_a}{\mu_a} \right] \left( 1 + \frac{nB(1-\mu_j) - \mu_a}{\mu_a} \right) , \]

\[ = \frac{[nB (1-\mu_j) - \mu_a](\sqrt{2} - 1)}{c(1-\mu_j)} , \]

\[ J_{RR}^* = \frac{nBN^*}{1+cN^*} \left[ \left( \frac{3}{2} - \sqrt{2} \right) ^2 + \left( \frac{3}{2} - \sqrt{2} \right)(\sqrt{2} - 1) + \frac{1}{4}(\sqrt{2} - 1)^2 \right] g \left( \frac{1}{2} \right) , \]

\[ = \frac{nB(\frac{3}{2} - \sqrt{2}) N^*}{1 + cN^*} \left[ \frac{nB(1-\mu_j) - \mu_a}{\mu_a} \right] \left( 1 + \frac{nB(1-\mu_j) - \mu_a}{\mu_a} \right) , \]

\[ = \frac{[nB (1-\mu_j) - \mu_a](\frac{3}{2} - \sqrt{2})}{c(1-\mu_j)} , \]

Equating (3.2) with the above gives,
\[ A_{LL}' = \left( \frac{nB (1 - \mu_j) - \mu_a}{2c (1 - \mu_j)} \right) \left( \frac{1 - \mu_j}{\mu_a} \right) = \frac{1}{2} N^*; \]

\[ A_{RL}' = \left( \frac{[nB (1 - \mu_j) - \mu_a] (\sqrt{2} - 1)}{c (1 - \mu_j)} \right) \left( \frac{1 - \mu_j}{\mu_a} \right) = (\sqrt{2} - 1) N^*; \]

\[ A_{RR}' = \left( \frac{[nB (1 - \mu_j) - \mu_a] (\frac{3}{2} - \sqrt{2})}{c (1 - \mu_j)} \right) \left( \frac{1 - \mu_j}{\mu_a} \right) = \left( \frac{3}{2} - \sqrt{2} \right) N^*, \]

where \( N^* = \frac{nB (1 - \mu_j) - \mu_a}{c \mu_a} \). We next show that this equilibrium is the only positive equilibrium.

Let \((J_{LL}', J_{RL}', J_{RR}', A_{LL}', A_{RL}', A_{RR}')\) be a positive equilibrium of (3.1) and let

\[ N' = A_{LL}' + A_{RL}' + A_{RR}'; \]

\[ \alpha = \frac{A_{LL}'}{N'}, \]

\[ \beta = \frac{A_{RL}'}{N'}, \]

\[ 1 - \alpha - \beta = \frac{A_{RR}'}{N'}, \]

\[ 1 - \alpha = \frac{A_{RL}' + A_{RR}'}{N'}. \]
Summing the three juvenile equations of model (3.1) evaluated at the equilibrium we obtain

\[
\frac{\mu_a}{1 - \mu_j} = \frac{nB}{1 + cN'} \left[ \alpha^2 g(\alpha) + 2\alpha(1 - \alpha) h(\alpha) + (1 - \alpha)^2 g(1 - \alpha) \right],
\]

Solving for \(1 + cN'\) we find that

\[
1 + cN' = \frac{nB}{\mu_a} \left[ \alpha^2 g(\alpha) + 2\alpha(1 - \alpha) h(\alpha) + (1 - \alpha)^2 g(1 - \alpha) \right] (1 - \mu_j).
\] (3.3)

We now substitute \(\alpha = \frac{A'_{LL}}{N}, \beta = \frac{A'_{RL}}{N},\) and \(1 - \alpha - \beta = \frac{A'_{RR}}{N}\) into the three juvenile equations of model (3.1)

\[
\frac{\mu_a \alpha}{1 - \mu_j} = \frac{nB}{1 + cN'} \left[ \alpha^2 g(\alpha) + \alpha \beta h(\alpha) + \frac{1}{4} \beta^2 g(1 - \alpha) \right],
\]

\[
\frac{\mu_a \beta}{1 - \mu_j} = \frac{nB}{1 + cN'} \left[ \left( \frac{1}{2} \beta^2 + (1 - \alpha - \beta) \beta \right) g(1 - \alpha) + (\alpha \beta + 2(1 - \alpha - \beta) \alpha) h(\alpha) \right],
\]

\[
\frac{\mu_a (1 - \alpha - \beta)}{1 - \mu_j} = \frac{nB}{1 + cN'} \left[ (1 - \alpha - \beta)^2 + (1 - \alpha - \beta) \beta + \frac{1}{4} \beta^2 \right] g(1 - \alpha).
\]

This yields the same three quadratic functions of \(\beta\) that we found in the proof of Theorem 2.5.1. From the analysis carried out in the proof of Theorem 2.5.1, we find \(\alpha = \frac{1}{2}\) and \(\beta = \sqrt{2} - 1\). Furthermore, we can find \(N'\) using (3.3) to be

\[
N' = \frac{nB (1 - \mu_j) - \mu_a}{c \mu_a}.
\]

Thus, we have shown,
\[ \left( J'_{LL}, J'_{RL}, J'_{RR}, A'_{LL}, A'_{RL}, A'_{RR} \right) = \left( J^*_r, J^*_r, J^*_r, A^*_r, A^*_r, A^*_r \right), \]
i.e., we have shown uniqueness.

As is the case for model (2.3), our finding of a positive equilibrium corresponding to a 1 : 1 phenotypic ratio corroborates the conclusion of Takahashi and Hori; moreover, model (3.1) has the same dimensionality as their model [40]. We are now interested in determining the stability of this unique positive equilibrium.

### 3.4. Stability of positive equilibrium

The stability of the positive equilibrium in Theorem 3.3.1 can be determined by the modulus of the six eigenvalues of the Jacobian matrix evaluated at the equilibrium. With \( h'\left( \frac{1}{2} \right) = 0 \), the eigenvalues of the Jacobian evaluated at the positive equilibrium are \( \lambda = 0 \), \( \lambda = 1 - \mu_a \), and the four solutions of the following two quadratic equations:

\[
\lambda^2 - (1 - \mu_a) \lambda - \frac{\mu_a^2}{nB (1 - \mu_B)} = 0,
\]
\[
\lambda^2 - (1 - \mu_a) \lambda - \frac{\mu_a \left[g'\left( \frac{1}{2} \right)(\sqrt{2} - 1) + 2 \right]}{2} = 0.
\]

To determine if the solutions of the above equations (eigenvalues) lie inside the unit disk, we can use the Jury conditions, which are necessary and sufficient conditions for this to occur:

1. \(|a_1| < a_2 + 1\),
2. \(a_2 < 1\),
where $\lambda^2 + a_1 \lambda + a_2 = 0$ [19]. We find that the positive equilibrium is locally asymptotically stable if

$$g'(\frac{1}{2}) > -\frac{2(1 + \mu_a)}{\mu_a(\sqrt{2} - 1)}.$$ 

In fact, $g'(\frac{1}{2}) = -\frac{2(1 + \mu_a)}{\mu_a(\sqrt{2} - 1)}$ forces a pair of complex conjugate eigenvalues to escape the unit disk, suggesting the occurrence of an invariant loop or Neimark-Sacker bifurcation [27], [33]. Figure 3.1 shows an example of a stable invariant loop that results from such an invariant loop bifurcation.

**Figure 3.1.** A sample quasi-periodic time series resulting from the invariant loop bifurcation. We used $h = f(\frac{1}{2})$ (mean fitness), and $g$ was defined as in equation (2.7). The initial condition is: $(J_{LL}, J_{RL}, J_{RR}, A_{LL}, A_{RL}, A_{RR}) = (0, 0, 20, 2, 6)$. The parameter values are: $nb = 50$, $c = 0.1$, $q = -6.8$, $\mu_j = 0.1$ and $\mu_a = 0.6$.

![Time series graph](image)

Recalling the scaling factor in the function $g$, we see that the positive equilibrium
destabilizes for

\[ q = f'_1 \left( \frac{1}{2} \right) < -\frac{2f\left( \frac{1}{2} \right)(1 + \mu_a)}{\mu_a(\sqrt{2} - 1)}. \]  \hspace{1cm} (3.4)

We can now determine the effect a time delay (i.e., stage structure) has on the stability of the positive equilibrium when a juvenile stage is introduced into the basic genetic-population dynamic model (2.3) to obtain (3.1). The stability criterion for both models requires \( q \) to be sufficiently small in magnitude for stability. We can compare the size of the two parameter regions of instability by inspecting (2.10) and (3.4), and it is clear that the two critical values satisfy the inequality

\[ -\frac{4f\left( \frac{1}{2} \right)}{\mu_a(\sqrt{2} - 1)} < -\frac{2f\left( \frac{1}{2} \right)(1 + \mu_a)}{\mu_a(\sqrt{2} - 1)}. \]

Therefore, we conclude that the positive equilibrium remains stable for a larger parameter region for the three-dimensional model, (2.3), than the six-dimensional, juvenile-adult model, (3.1). The parameter regions are compared in Figure 3.2. This result is not surprising, since adding time delays (i.e., life stages) typically has a destabilizing effect. Therefore, we have shown that a time delay is not necessary for a phenotypic oscillation, but promotes oscillatory behavior. This finding corroborates, in part, the hypothesis of Takahashi and Hori that the time lag in growth period plays a role in the destabilization [40]. We qualified their hypothesis by showing that stage structure is not necessary for oscillation, but shrinks the parameter region of equilibrium stability.
Figure 3.2. Plane showing the parameter regions where the positive equilibrium of models (2.3) and (3.1) is stable and unstable \( f\left(\frac{1}{2}\right) = \frac{1}{2} \).

3.5. Nine-dimensional, three life stage model definition

We now construct a model that incorporates the three natural stage classifications for \( P. \ microlepis \): fry, juveniles, and adults. Our new model is nine-dimensional, and \( F_{LL}(t), F_{RL}(t), \) and \( F_{RR}(t) \) represent the number of (homozygous) left-handed, heterozygous right-handed, and homozygous right-handed fry at time \( t \), respectively. The state variables \( J_{LL}(t), J_{RL}(t), J_{RR}(t), A_{LL}(t), A_{RL}(t), \) and \( A_{RR}(t) \) are as described for model (3.1). The projection time interval is eight months, and let \( nN(t) \) be the number of matings that occur within this time period. Let \( B, \mu_j, \mu_a, c, N(t) \) and \( l(t) \) be defined as for model (3.1). We introduce the new parameter \( 0 < \mu_f < 1 \) as the probability of fry death in one time step. Incorporating all nine state variables, we write the nine-dimensional extension of model (2.3) as,
\[ F_{LL}(t+1) = \frac{nBN(t)}{1+cN(t)} \left[ \left( \frac{A_{LL}(t)}{N(t)} \right)^2 g \left( \frac{A_{LL}(t)}{N(t)} \right) + \left( \frac{A_{LL}(t)A_{RL}(t)}{N(t)^2} \right) h \left( \frac{A_{LL}(t)}{N(t)} \right) \right] \\
+ \frac{1}{4} \left( \frac{A_{RL}(t)}{N(t)} \right)^2 g \left( 1 - \frac{A_{LL}(t)}{N(t)} \right) \right], \\
J_{LL}(t+1) = (1 - \mu_f) F_{LL}(t), \\
A_{LL}(t+1) = (1 - \mu_j) J_{LL}(t) + (1 - \mu_a) A_{LL}(t), \\

F_{RL}(t+1) = \frac{nBN(t)}{1+cN(t)} \left[ \left( \frac{1}{2} A_{RL}(t)^2 + A_{RR}(t)A_{RL}(t) \right) \right] g \left( 1 - \frac{A_{LL}(t)}{N(t)} \right) \\
+ \left( \frac{A_{LL}(t)A_{RL}(t) + 2A_{RR}(t)A_{LL}(t)}{N(t)^2} \right) h \left( \frac{A_{LL}(t)}{N(t)} \right) \right], \\
J_{RL}(t+1) = (1 - \mu_f) F_{RL}(t), \\
A_{RL}(t+1) = (1 - \mu_j) J_{RL}(t) + (1 - \mu_a) A_{RL}(t), \\

F_{RR}(t+1) = \frac{nBN(t)}{1+cN(t)} \left[ \left( \frac{A_{RR}(t)^2 + A_{RR}(t)A_{RL}(t) + \frac{1}{4}A_{RL}(t)^2}{N(t)^2} \right) g \left( 1 - \frac{A_{LL}(t)}{N(t)} \right) \right], \\
J_{RR}(t+1) = (1 - \mu_f) F_{RR}(t), \\
A_{RR}(t+1) = (1 - \mu_j) J_{RR}(t) + (1 - \mu_a) A_{RR}(t).\]
As with model (3.1), we introduce density-dependent effects through the birth process only. Fry, juvenile, and adult survivorship are assumed to be density-independent.
As with models (2.3) and (3.1), we look for a positive equilibrium of model (3.5).

3.6. Positive equilibria

In the case of models (2.3) and (3.1), we proved the existence and uniqueness of a positive equilibrium corresponding to a 1 : 1 phenotypic ratio. We approach the analysis for model (3.5) in a similar way.

**Theorem 3.6.1.** If \( \frac{nB(1-\mu_f)(1-\mu_j)}{\mu_a} > 1 \) model (3.5) has a unique positive equilibrium, \((F_{LL}^*, F_{RL}^*, F_{RR}^*, J_{LL}^*, J_{RL}^*, J_{RR}^*, A_{LL}^*, A_{RL}^*, A_{RR}^*)\), given by

\[
\left( \kappa A_{LL}^*, \kappa A_{RL}^*, \kappa A_{RR}^*, \sigma A_{LL}^*, \sigma A_{RL}^*, \sigma A_{RR}^*, \frac{N^*}{2}, (\sqrt{2} - 1)N^*, \left(\frac{3}{2} - \sqrt{2}\right)N^* \right)
\]

where

\[
\kappa = \frac{\mu_a}{(1-\mu_f)(1-\mu_j)},
\]
\[
\sigma = \frac{\mu_a}{1-\mu_j},
\]
\[
N^* = A_{LL}^* + A_{RL}^* + A_{RR}^* = \frac{nB(1-\mu_f)(1-\mu_j) - \mu_a}{c \mu_a}.
\]

Left- and right-handed fish are equally frequent at equilibrium.
Proof. The proof of this theorem is almost identical to the proof of Theorem 3.3.1.

3.7. Stability of positive equilibrium

The stability of the positive equilibrium in Theorem 3.6.1 can be determined by the modulus of the eigenvalues of the Jacobian matrix evaluated at the equilibrium. With \( h'(\frac{1}{2}) = 0 \), the eigenvalues are \( \lambda = 0 \) (double), \( \lambda = 1 - \mu_a \), and the six solutions of the following two cubic equations:

\[
\lambda^3 - (1 - \mu_a) \lambda^2 - \frac{\mu_a^2}{nB(1 - \mu_f)(1 - \mu_j)} = 0,
\]

\[
\lambda^3 - (1 - \mu_a) \lambda^2 - \frac{\mu_a \left(g'(\frac{1}{2}) (\sqrt{2} - 1) + 2\right)}{\mu_a} = 0.
\]

To determine if the solutions of the above equations (eigenvalues) lie inside the unit disk, we can use the Jury conditions,

1. \(|a_1 + a_3| < a_2 + 1|\),

2. \(|a_3| < 1|\),

3. \(|a_2 - a_3 a_1| < |1 - a_3^2|\),

where \( \lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0 \) [19]. We find that the positive equilibrium is locally asymptotically stable if
\[ g'(\frac{1}{2}) = \frac{-3\mu_a + 1 - \sqrt{(1 - \mu_a)^2 + 4}}{\mu_a(\sqrt{2} - 1)}. \]

Similar to the bifurcation associated with model (3.1), \( g'(\frac{1}{2}) = \frac{-3\mu_a + 1 - \sqrt{(1 - \mu_a)^2 + 4}}{\mu_a(\sqrt{2} - 1)} \) forces a pair of complex conjugate eigenvalues to escape the unit disk, suggesting the occurrence of an invariant loop bifurcation. Figure 3.3 shows an example of quasi-periodic time series that results from such an invariant loop bifurcation. Recalling the scaling factor in the function \( g \), we see that the positive equilibrium destabilizes for

\[ q = f'_1\left(\frac{1}{2}\right) < \frac{f\left(\frac{1}{2}\right)\left[-3\mu_a + 1 - \sqrt{(1 - \mu_a)^2 + 4}\right]}{\mu_a(\sqrt{2} - 1)}. \] (3.6)

We can now determine how further increasing dimensionality has affected the stability of the positive equilibrium from model (3.1) to (3.5). In particular, the stability criteria from Sections 2.6 and 3.4 require \( q \) to be sufficiently small in magnitude for stability. We can compare the size of the parameter regions of instability for models (2.3), (3.1), and (3.5) by inspecting (2.10), (3.4), and (3.6), and it is clear the critical values satisfy the inequalities

\[ \frac{-4f\left(\frac{1}{2}\right)}{\mu_a(\sqrt{2} - 1)} < \frac{-2f\left(\frac{1}{2}\right)(1 + \mu_a)}{\mu_a(\sqrt{2} - 1)} < \frac{f\left(\frac{1}{2}\right)\left[-3\mu_a + 1 - \sqrt{(1 - \mu_a)^2 + 4}\right]}{\mu_a(\sqrt{2} - 1)}. \]

Therefore, we conclude that the positive equilibrium remains stable for a larger parameter region in the three-dimensional model, (2.3), than the six-dimensional model, (3.1), which in turn remains stable for a larger parameter region than the nine-dimensional model, (3.5). The sizes of the different regions of stability are compared in Figure 3.4. As dimensionality is increased, destabilization of the positive equilib-
Figure 3.3. A sample quasi-periodic time series resulting from invariant loop bifurcation. We used $h = f\left(\frac{1}{2}\right)$ (mean fitness), and $g$ was defined as in equation (2.7). We used, $(F_{LL}, F_{RL}, F_{RR}, J_{LL}, J_{RL}, J_{RR}, A_{LL}, A_{RL}, A_{RR}) = (0, 0, 0, 0, 0, 20, 2, 6)$ as the initial condition. The parameter values are: $nb = 50$, $c = 0.1$, $q = -6.8$, $\mu_f = 0.1$, $\mu_j = 0.05$, and $\mu_a = 0.6$.

Interestingly, Figure 3.4 indicates that increasing dimensionality does not make it possible to destabilize the equilibrium with values of $q$ that are small in magnitude. This demonstrates that strong frequency-dependent selection is necessary for the destabilization of the positive equilibrium in all three models. Phenotypic oscillations are possible for smaller values of $\mu_a$ when stage-structure is added, but there appears to be an asymptotic limit to how small in magnitude $q$ may be.

The stable invariant loops for adult fish in the six- and nine-dimensional models, (3.1) and (3.5), are plotted in phase space in Figure 3.5 for similar parameter values.
Figure 3.4. Plane showing the parameter regions where the positive equilibrium of models (2.3), (3.1), and (3.5) is stable and unstable \( f\left(\frac{1}{2}\right) = \frac{1}{2} \).

Parameter regions of equilibrium destabilization

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Stable</th>
<th>Unstable</th>
</tr>
</thead>
</table>

The frequencies of left- and right-handed fish can be averaged over the loop. As we found for the three-dimensional model in Section 2.7, the time-averaged frequency of left-handed fish is greater than the time-averaged frequency of right-handed fish. This phenomenon appears to persist as \( q \) increases in magnitude. Furthermore, the difference between the left- and right-handed frequencies seems to increase as either dimensionality or \( |q| \) are increased.
Figure 3.5. Phase space plot showing the two stable invariant loops for models (3.1) and (3.5). We used \( h = f\left(\frac{1}{2}\right) \) (mean fitness), and \( g \) was defined as in equation (2.7). The parameter values are: \( \text{nb} = 50, c = 0.1, q = -6.8, \) and \( \mu_a = 0.6. \) In the case of six-dimensional model, \( \mu_j = 0.1; \) in the case of the nine-dimensional model, \( \mu_f = 0.1 \) and \( \mu_j = 0.05. \)
Chapter 4

Strong Density-Dependent Effects

4.1. Model construction

In this section we return our study to a three-dimensional model (without immature stages). Until now, we have used only the discrete-logistic (or Beverton-Holt) nonlinearity to describe density-dependence. We constructed our initial models this way in order to determine if frequency-dependent selection could account for the phenotypic oscillation in the absence of oscillations induced by the population dynamics. We found in Sections 2.6, 3.4, and 3.7 that strong prey response sensitivity (frequency-dependent selection) is necessary for the phenotypic oscillation. In this chapter we are interested in compounding population dynamic oscillations with phenotypic oscillations by using model (2.3) with the discrete logistic nonlinearity replaced by an exponential or Ricker nonlinearity. Ricker introduced this type of nonlinearity in a model of fish population dynamics

\[ N(t + 1) = bN(t)e^{-cN(t)}, \]

when the species was cannibalistic [31]. Like many fish species, fish in the Family Cichlidae are known to be cannibals [3]. While little is known about \textit{P. microlepis} in particular, we consider the possibility that such behavior exists. A stage-structured extension of the Ricker model has been successfully used to model the complex population dynamics of the cannibalistic flour beetle, \textit{Tribolium castaneum} [8].
4.2. Model definition and parameters

We will now modify model (2.3) by including strong intraspecific competition. We aim to tease out the effects of strong frequency-dependent selection and density-dependence. If, in fact, *P. microlepis* is cannibalistic, it may be more biologically realistic to consider such a model. However, we are also interested in the interplay of population dynamic and population genetic processes generally, as this modeling approach can be applied to other biological systems. We rewrite model (2.3) with the Ricker nonlinearity as,

\[
A_{LL}(t+1) = nBN(t)e^{-cN(t)} \left[ \left( \frac{A_{LL}(t)}{N(t)} \right)^2 g \left( \frac{A_{LL}(t)}{N(t)} \right) + \left( \frac{A_{LL}(t)A_{RL}(t)}{N(t)^2} \right) h \left( \frac{A_{LL}(t)}{N(t)} \right) \right] \\
+ \frac{1}{4} \left( \frac{A_{RL}(t)}{N(t)} \right)^2 g \left( 1 - \frac{A_{LL}(t)}{N(t)} \right) + (1 - \mu_a) A_{LL}(t),
\]

\[
A_{RL}(t+1) = nBN(t)e^{-cN(t)} \left[ \left( \frac{1}{2}A_{RL}(t)^2 + A_{RR}(t)A_{RL}(t) \right) g \left( 1 - \frac{A_{LL}(t)}{N(t)} \right) \right] \\
+ \left( \frac{A_{LL}(t)A_{RL}(t) + 2A_{RR}(t)A_{LL}(t)}{N(t)^2} \right) h \left( \frac{A_{LL}(t)}{N(t)} \right) \\
+ (1 - \mu_a) A_{RL}(t),
\]

\[
A_{RR}(t+1) = nBN(t)e^{-cN(t)} \left[ \frac{A_{RR}(t)^2 + A_{RR}(t)A_{RL}(t) + \frac{1}{4}A_{RL}(t)^2}{N(t)^2} \right] g \left( 1 - \frac{A_{LL}(t)}{N(t)} \right) \\
+ (1 - \mu_a) A_{RR}(t),
\]
where all state variables, functions, and parameters are defined as for model (2.3).
We first look at the existence of a positive equilibrium of model (4.1).

4.3. Positive equilibria

In the case of models (2.3), (3.1), and (3.5), we proved the existence and uniqueness of a positive equilibrium corresponding to a 1 : 1 phenotypic ratio. We approach the analysis for model (4.1) in a similar way.

**Theorem 4.3.1.** If \( \frac{n_B}{\mu a} > 1 \) model (4.1) has a unique positive equilibrium given by

\[
(A_{LL}^*, A_{RL}^*, A_{RR}^*) = N^* \left( \frac{1}{2}, \sqrt{2} - 1, \frac{3}{2} - \sqrt{2} \right)
\]

where

\[
N^* = \ln\left(\frac{n_B}{\mu a}\right) \cdot c.
\]

Left- and right-handed fish are equally frequent at equilibrium.

**Proof.** The proof of this theorem is identical to the proof of Theorem 2.5.1.

Now that we know there exists a unique positive equilibrium for model (4.1), we turn our attention to its stability.

4.4. Stability of positive equilibrium

The stability of the positive equilibrium given in Theorem 4.3.1 can be determined by the modulus of the eigenvalues of the Jacobian matrix evaluated at the equilibrium. With \( h'(\frac{1}{2}) = 0 \), the eigenvalues are
\[
\lambda_1 = 1 - \mu_a,
\]
\[
\lambda_2 = 1 - \mu_a \ln \left( \frac{nB}{\mu_a} \right),
\]
\[
\lambda_3 = \frac{\mu_a g' \left( \frac{1}{2} \right) (\sqrt{2} - 1) + 2}{2}.
\]

Notice that \( \lambda_1 \) and \( \lambda_3 \) are identical \( \lambda_1 \) and \( \lambda_3 \) for model (2.3).

By inspecting the eigenvalues we find that there are two possible bifurcations. The first bifurcation occurs when \( \lambda_2 = 1 - \mu_a \ln \left( \frac{nB}{\mu_a} \right) \) escapes the unit disc at \(-1\), suggesting a period doubling bifurcation. The bifurcation occurs when

\[
nB = nB_{\text{crit}} = \mu_a e^{\frac{\sqrt{2}}{\mu_a}}.
\]

This bifurcation is analogous to the period doubling bifurcation that occurs for the one-dimensional Ricker model as \( b \) is increased through criticality. We attribute oscillations from this bifurcation to population dynamic effects, namely to the over-compensatory density effects described by the Ricker exponential nonlinearity.

Another bifurcation occurs when \( \lambda_3 = \frac{\mu_a g' \left( \frac{1}{2} \right) (\sqrt{2} - 1) + 1}{\sqrt{2}} \) escapes the unit disc at \(-1\), suggesting another period doubling bifurcation. The bifurcation occurs when

\[
q = g' \left( \frac{1}{2} \right) f \left( \frac{1}{2} \right) = q_{\text{crit}} = -\frac{4f \left( \frac{1}{2} \right)}{\mu_a(\sqrt{2} - 1)}.
\]

The critical value \( q_{\text{crit}} \) is identical to that for model (2.3) (see (2.10)). Therefore, we attribute oscillations from this bifurcation to population genetic effects (i.e., strong frequency-dependent selection).

Now that we know the positive equilibrium can be destabilized by using two
different influences (one population dynamic, one population genetic), we analyze the predictions of model (4.1) in the $nB$-$q$ plane numerically, as depicted in Figure 4.1. We restrict our attention to the portion of this plane where $nB$ and $q$ are biologically reasonable (i.e., $|q|$ and $nB$ not too large). The bifurcations dictate that the state variables ($A_{LL}$, $A_{RL}$, $A_{RR}$) oscillate with period two. However, to compare the model predictions to the biological data, we are interested in whether the phenotypic frequencies, $\frac{A_{LL}}{N}$ and $\frac{A_{RL}+A_{RR}}{N}$, oscillate. It may be the case that the state variables oscillate, but the phenotypic frequencies do not.

**Figure 4.1.** $nB$-$q$ plane for model (4.1), where $nB_{crit} = \mu_a e^{\mu_a}$ and $q_{crit} = -\frac{4f(\frac{1}{2})}{\mu_a(\sqrt{2}-1)}$. Four distinct quadrants (A, B, C, D) are shown to indicate the two bifurcations. The horizontal ($nB$) axis represents population dynamic effects and the vertical ($q$) axis represents population genetic effects.

Quadrant A in Figure 4.1 is characterized by the following inequalities:
Local analysis in quadrant A is tractable, and the positive equilibrium is locally asymptotically stable within this quadrant.

Quadrant B in Figure 4.1 is characterized by the following inequalities:

\[ n_B < n_B^{\text{crit}}, \quad q > q^{\text{crit}}. \]

The positive equilibrium is unstable in quadrant B; a period doubling bifurcation has occurred due to strong frequency-dependent selection. Both the number of fish and the phenotypic frequencies oscillate with period two. A local bifurcation analysis, paralleling the one conducted for model (2.3) in Section 2.7, reveals that the time-averaged frequency of left-handed fish is greater than the time-averaged frequency of right-handed fish near criticality as shown in Figure 4.2. Numerical simulations support this finding and suggest that this feature is further enhanced as \( q \) increases in magnitude.

As \( n_B \) increases in quadrant B (i.e., as we move closer to quadrant D), and for a sufficiently large value of \( |q| \), multiple two-cycle attractors appear for a small range of \( n_B \). The multiple attractors have similar, but opposite characteristics. In particular, one of the attractors is characterized by the time-averaged frequency of left-handed fish being greater than the time-averaged frequency of right-handed fish and the other is characterized by the time-averaged frequency of right-handed fish being greater than the time-averaged frequency of left-handed fish, as shown in Figure 4.3. Interestingly, the two-cycle attractor characterized by the time-averaged frequency of left-handed fish being numerically dominant is more biologically tangible in the sense that it has a much larger basin of attraction (as observed numerically). The multiple two-cycle attractors eventually collide with one another and form a single two-cycle attractor in quadrant D, as in Figure 4.4. Following (and nearby) the collision, the phenotypic
Figure 4.2. Bifurcation diagram showing the period doubling bifurcation in phenotypic frequency as \( q \) becomes sufficiently large in magnitude in Quadrant B. We used \( h = f\left(\frac{1}{2}\right) \) (mean fitness), and \( g \) was defined as in (2.7). The initial condition is: \( (A_{LL}, A_{RL}, A_{RR}) = (20, 2, 6) \). The parameter values are: \( nb = 20, c = 0.01, \) and \( \mu_a = 0.6; q_{crit} \approx -8.05 \) and \( nB_{crit} = \frac{nb_{crit}}{2} \approx 16.82 \).

frequencies cease to oscillate, and instead, equilibriate at the 1:1 phenotypic ratio. We can attribute the existence of multiple attractors directly to the Ricker nonlinearity, since multiple attractors are absent in model (2.3) with the discrete logistic nonlinearity.

Quadrant C in Figure 4.1 is characterized by the following inequalities:

\[
nB > nB_{crit}, \quad q > q_{crit}.
\]

The positive equilibrium is unstable in quadrant C; a period doubling bifurcation has
Figure 4.3. Multiple two-cycle attractors for phenotypic frequency in quadrant B. We used \( h = f\left(\frac{1}{2}\right) \) (mean fitness), and \( g \) was defined as in (2.7). The initial condition used in the left panel: \( (A_{LL}, A_{RL}, A_{RR}) = (20, 2, 6) \), and on the right panel: \( (A_{LL}, A_{RL}, A_{RR}) = (2, 2, 2) \). The parameter values are: \( nb = 33, q = -9, c = 0.01, \) and \( \mu_a = 0.6; \); \( q_{crit} \approx -8.05 \) and \( nB_{crit} = \frac{nb_{crit}}{2} \approx 16.82 \).

(a) Two-cycle attractor in quadrant B characterized by the time-averaged frequency of left-handed fish (\( \approx 0.5058 \)) being greater than the time-averaged frequency of right-handed fish (\( \approx 0.4942 \)).

(b) Two-cycle attractor in quadrant B characterized by the time-averaged frequency of right-handed fish (\( \approx 0.4957 \)) being greater than the time-averaged frequency of left-handed fish (\( \approx 0.5043 \)).

occurred, which can be attributed to strong intraspecific competition. Specifically, the population dynamics undergo a period doubling cascade with increasing \( nB \). Despite the fact that the state variables (population dynamics) oscillate, the phenotypic frequencies equilibrate at the 1 : 1 phenotypic ratio for \( |q| \) sufficiently small. Figure 4.5 depicts the complex population dynamics and the simple phenotypic frequency dynamics.

For larger values of \( |q| \) in quadrant C, increasing \( nB \) causes the phenotypic frequency dynamics to (slightly) increase in complexity. In particular, for a fixed value of \( q \) sufficiently large in magnitude, but \( q > q_{crit} \), the phenotypic frequencies oscillate with period four, corresponding to the population dynamics undergoing a second period doubling bifurcation (to period four) as seen in Figure 4.6. As we increase
Figure 4.4. Bifurcation diagram showing the phenotypic frequency dynamics as \( nB \) is increased in quadrant B to quadrant D. We used \( h = f \left( \frac{1}{2} \right) \) (mean fitness), and \( g \) was defined as in (2.7). The initial condition is: \( (A_{LL}, A_{RL}, A_{RR}) = (20, 2, 6) \). The parameter values are: \( q = -9 \), \( c = 0.01 \), and \( \mu_a = 0.6 \); \( q_{crit} \approx -8.05 \) and \( n_{B_{crit}} = \frac{n_B q_{crit}}{c} \approx 16.82 \). The bending of the branches indicates the existence of multiple attractors.

\( nB \) further, two locally stable four-cycles appear. These multiple attractors, like those found in quadrant B, are characterized by one of the four-cycles having a time-averaged frequency of left-handed fish being numerically dominant and the other by the time-averaged frequency of right-handed fish being numerically dominant. The attractor with left-handed dominance appears to have a much larger basin of attraction, a phenomenon also seen in quadrant B. With further increases in \( nB \), the branches of the four-cycles collide with one another and become a single four-cycle population dynamic attractor. The phenotypic frequency dynamics, following collision, return to
Figure 4.5. Bifurcation diagrams showing complex population dynamics and simple phenotypic frequency dynamics as \( nB \) is increased in quadrant C when \( |q| \) is sufficiently small. We used \( h = f\left(\frac{1}{2}\right) \) (mean fitness), and \( g \) was defined as in (2.7). The initial condition is: \((A_{LL}, A_{RL}, A_{RR}) = (20, 2, 6)\). The parameter values are: \( q = -5 \), \( c = 0.01 \), and \( \mu_a = 0.6 \); \( q_{crit} \approx -8.05 \) and \( nB_{crit} = \frac{nB_{crit}}{2} \approx 16.82 \). The attractors for left- and right-handed fish are identical.

(a) Bifurcation diagram showing the complex population dynamics as \( nB \) is increased in quadrant C.

(b) Bifurcation diagram showing the simple phenotypic frequency dynamics as \( nB \) is increased in quadrant C.

equilibrium. Figure 4.7 shows the two different frequency four-cycles.

In summary, quadrant C in Figure 4.1 is characterized by complex population dynamics and much less complex phenotypic frequency dynamics. For reasonable values of \( nB \), numerical simulations suggest that the frequency dynamics are only as complex as the four-cycle, which is restricted to a small range of \( nB \). These numerical studies suggest that strong density-dependence alone cannot account for phenotypic frequency fluctuations. In fact, it appears to take sufficiently strong frequency-dependent selection for oscillations to appear (i.e., the subregion of quadrant C where oscillations occur is bounded away from the \( nB \) axis). However, these studies also indicate that strong density-dependence and moderate frequency-dependent selection can compound their effects to cause phenotypic frequency oscillations. These oscillations are not possible in nearby quadrant A or for model 2.3 for values of \( q \) less than
Figure 4.6. Bifurcation diagrams showing the population and phenotypic frequency dynamics for left-handed fish as $nB$ is increased in quadrant C when $|q|$ is sufficiently large. We used $h = f\left(\frac{1}{2}\right)$ (mean fitness), and $g$ was defined as in (2.7). The initial condition is: $(A_{LL}, A_{RL}, A_{RR}) = (20, 2, 6)$. The parameter values are: $q = -7$, $c = 0.01$, and $\mu_a = 0.6$; $q_{crit} \approx -8.05$ and $nB_{crit} = \frac{nB_{crit}}{2} \approx 16.82$.

(a) Bifurcation diagram showing the complex population dynamics as $nB$ is increased in quadrant C.

(b) Bifurcation diagram showing the simpler phenotypic frequency dynamics as $nB$ is increased in quadrant C.

the bifurcation value.

Quadrant D in Figure 4.1 is characterized by the following inequalities:

$$nB > nB_{crit}, \quad q < q_{crit}.$$ 

The positive equilibrium is unstable in quadrant D; two period doubling bifurcations have occurred (one attributed to strong frequency-dependent selection and one to strong intraspecific competition). The phenotypic frequency dynamics appear to equilibriate at the 1 : 1 phenotypic ratio for values of $nB$ near criticality. We observed this phenomenon in Figure 4.4, and we investigate it further in the next section. Increasing $nB$, however, results in the frequency dynamics becoming more complex. In particular, a phenotypic frequency four-cycle emerges; multiple attractors emerge with further increases in $nB$. The multiple attractors disappear for larger values of
Figure 4.7. Multiple four-cycle attractors for phenotypic frequency in quadrant C. We used $h = f\left(\frac{1}{2}\right)$ (mean fitness), and $g$ was defined as in (2.7). The initial condition used in the left panel: $(A_{LL}, A_{RL}, A_{RR}) = (20, 2, 6)$, and on the right panel: $(A_{LL}, A_{RL}, A_{RR}) = (2, 2, 2)$. The parameter values are: $nb = 106$, $q = -7$, $c = 0.01$, and $\mu_a = 0.6$; $q_{\text{crit}} \approx -8.05$ and $nB_{\text{crit}} = \frac{nb_{\text{crit}}}{2} \approx 16.82$.

(a) Four-cycle attractor in quadrant C characterized by the time-averaged frequency of left-handed fish ($\approx 0.5025$) being greater than the time-averaged frequency of right-handed fish ($\approx 0.4975$).

(b) Four-cycle attractor in quadrant C characterized by the time-averaged frequency of right-handed ($\approx 0.5003$) fish being greater than the time-averaged frequency of left-handed fish ($\approx 0.4997$).

$nB$, and a single attractor exists where both phenotypic frequency and population dynamics become chaotic. The phenotypic frequency and population dynamics in quadrant D are depicted in Figure 4.8.

In summary, quadrant D is characterized by complex phenotypic frequency dynamics. Multiple attractors and chaos are possible. Furthermore, there is strong agreement between the population dynamics and the phenotypic frequency dynamics in quadrant D. The behavior of model (4.1) in each of the four quadrants is summarized in Table 4.1.
Figure 4.8. Bifurcation diagrams showing the population and phenotypic frequency dynamics for left-handed fish as $nB$ is increased in quadrant D. We used $h = f\left(\frac{1}{2}\right)$ (mean fitness), and $g$ was defined as in (2.7). The initial condition is: $(A_{LL}, A_{RL}, A_{RR}) = (20, 2, 6)$. The parameter values are: $q = -9$, $c = 0.01$, and $\mu_a = 0.6$; $q_{crit} \approx -8.05$ and $nB_{crit} = \frac{nb_{crit}}{2} \approx 16.82$.

(a) Bifurcation diagram showing the complex population dynamics as $nB$ is increased in quadrant D.

(b) Bifurcation diagram showing the complex phenotypic frequency dynamics as $nB$ is increased in quadrant D.

Table 4.1. Summary of population and phenotypic frequency dynamics for model (4.1).

<table>
<thead>
<tr>
<th>Quadrant</th>
<th>Behavior</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Positive equilibrium LAS.</td>
</tr>
<tr>
<td>B</td>
<td>Positive equilibrium unstable. Population dynamics and phenotypic frequency dynamics oscillate with period two. Multiple two-cycle attractors possible.</td>
</tr>
<tr>
<td>C</td>
<td>Positive equilibrium unstable. Complex population dynamics and simple phenotypic frequency dynamics (only as complicated as a four-cycle). Minimal matching between population and phenotypic frequency dynamics. Multiple four-cycle attractors possible.</td>
</tr>
<tr>
<td>D</td>
<td>Positive equilibrium unstable. Population and phenotypic frequency dynamics are complex and typically agree with one another. Multiple attractors and chaos possible.</td>
</tr>
</tbody>
</table>
4.5. Local bifurcation analysis

In this section we investigate a little more thoroughly the period doubling bifurcation, attributed to strong intraspecific competition, that occurs in quadrant B in Figure 4.1. In Section 4.4 we reported that, near criticality, the state variables (population dynamics) oscillate with period two while the phenotypic frequency dynamics equilibriate. This finding is based on numerical explorations, and to investigate these oscillations analytically we use a Lyapunov-Schmidt expansion similar to that discussed in Section 2.7.

We begin by centering model (4.1) on the positive equilibrium by using variables

\[
\begin{align*}
    x(t) &= A_{LL}(t) - A_{LL}^*, \\
    y(t) &= A_{RL}(t) - A_{RL}^*, \\
    z(t) &= A_{RR}(t) - A_{RR}^*, \\
    n(t) &= x(t) + y(t) + z(t),
\end{align*}
\]

where \( N(t) = n(t) + N^* \) and \((x^*, y^*, z^*) = (0, 0, 0)\) is the new equilibrium. We parameterize the branch of two-cycles by expanding \( x, y, \) and \( z \) about 0. We expand the bifurcation parameter, \( nB \), about

\[ nB_{\text{crit}} = \mu_a e^{\frac{2}{\mu_a}}. \]

We introduce a small parameter \( \epsilon \) as
\[ x(t) = x_1(t) \epsilon + x_2(t) \epsilon^2 + \ldots, \]
\[ y(t) = y_1(t) \epsilon + y_2(t) \epsilon^2 + \ldots, \]
\[ z(t) = z_1(t) \epsilon + z_2(t) \epsilon^2 + \ldots, \]

and,

\[ nB = \mu_a \epsilon^2 \mu_b + nB_1 \epsilon + nB_2 \epsilon^2 + \ldots. \]

We deduce that \( nB_1 = 0 \), since the bifurcation is period doubling; the two-cycle exists to one side of criticality only. Substituting the above expansions into the model equations and matching powers of \( \epsilon \) we obtain

\[
\begin{pmatrix}
  x_1(t+1) \\
  y_1(t+1) \\
  z_1(t+1)
\end{pmatrix}
= J
\begin{pmatrix}
  x_1(t) \\
  y_1(t) \\
  z_1(t)
\end{pmatrix},
\]

where \( J \) is the Jacobian matrix evaluated at the equilibrium. The two-cycle solution of this equation consists of the eigenvector that corresponds to \( \lambda = -1 \) and its image

\[
\begin{pmatrix}
  x_1(t) \\
  y_1(t) \\
  z_1(t)
\end{pmatrix}
= (-1)^t v,
\]

where
\[
\mathbf{v} = \begin{pmatrix}
(2\sqrt{2} + 3) \\
2(\sqrt{2} + 1) \\
1
\end{pmatrix}
\]

Determining the second order terms in the expansion equates to finding the two-cycle solution of

\[
\begin{pmatrix}
x_2(t + 1) \\
y_2(t + 1) \\
z_2(t + 1)
\end{pmatrix} = \mathbf{J} \begin{pmatrix}
x_2(t) \\
y_2(t) \\
z_2(t)
\end{pmatrix} + u
\]

(4.2)

where \( u \) is a constant given by

\[
u = \begin{pmatrix}
2c (1 - \mu_a) (12\sqrt{2} + 17) \\
4c (1 - \mu_a) (5\sqrt{2} + 7) \\
2c (1 - \mu_a) (2\sqrt{2} + 3)
\end{pmatrix}.
\]

The second order coefficients: \( x_2, y_2, \) and \( z_2, \) are constants found as the equilibrium solution of 4.2:
\[
\begin{pmatrix}
  x^*_2 \\
  y^*_2 \\
  z^*_2
\end{pmatrix} = J \begin{pmatrix}
  x^*_2 \\
  y^*_2 \\
  z^*_2
\end{pmatrix} + u,
\]

\[
(I - J) \begin{pmatrix}
  x^*_2 \\
  y^*_2 \\
  z^*_2
\end{pmatrix} = u,
\]

\[
\begin{pmatrix}
  x^*_2 \\
  y^*_2 \\
  z^*_2
\end{pmatrix} = (I - J)^{-1} u.
\]

We find \(x_2 = x^*_2, y_2 = y^*_2\), and \(z_2 = z^*_2\) to be

\[
\begin{pmatrix}
  x^*_2 \\
  y^*_2 \\
  z^*_2
\end{pmatrix} = \frac{1}{2} u = \begin{pmatrix}
  c (1 - \mu_a) (12\sqrt{2} + 17) \\
  2c (1 - \mu_a) (5\sqrt{2} + 7) \\
  c (1 - \mu_a) (2\sqrt{2} + 3)
\end{pmatrix}.
\]

We can now write the expansions for the bifurcating two-cycle:

\[
x(t) = x_1(t)\epsilon + x_2\epsilon^2 + \ldots = (-1)^t \left( 2\sqrt{2} + 3 \right) \epsilon + x^*_2\epsilon^2 + \ldots,
\]

\[
y(t) = y_1(t)\epsilon + y_2\epsilon^2 + \ldots = (-1)^t 2(\sqrt{2} + 1)\epsilon + y^*_2\epsilon^2 + \ldots,
\]

\[
z(t) = z_1(t)\epsilon + z_2\epsilon^2 + \ldots = (-1)^t \epsilon + z^*_2 + \ldots.
\]

Translating the expansions to the original state variables: \(A_{LL}, A_{RL}, \) and \(A_{RR}\) we obtain
\begin{align*}
A_{LL} &= A_{LL}^* + x = \frac{N^*}{2} + (-1)^t \left(2\sqrt{2} + 3\right) \epsilon + x_2^* \epsilon^2 + \ldots,
\end{align*}
\begin{align*}
A_{RL} &= A_{RL}^* + y = (\sqrt{2} - 1)N^* + (-1)^t 2(\sqrt{2} + 1)\epsilon + y_2^* \epsilon^2 + \ldots,
\end{align*}
\begin{align*}
A_{RR} &= A_{RR}^* + z = \left(\frac{3}{2} - \sqrt{2}\right)N^* + (-1)^t \epsilon + z_2^* + \ldots.
\end{align*}

To investigate what happens to the phenotypic frequencies near bifurcation, we inspect the fractions, \( \frac{A_{LL}(t)}{N(t)} \) and \( \frac{A_{RL}(t)+A_{RR}(t)}{N(t)} \). In doing this, notice that

\begin{align*}
A_{LL}^* &= A_{RL}^* + A_{RR}^*, \\
x_1(t) &= y_1(t) + z_1(t),
\end{align*}
\begin{equation}
(4.3)
\begin{align*}
x_2^* &= y_2^* + z_2^*.
\end{align*}
\end{equation}

Substituting the two-cycle expansions we obtain

\begin{align*}
\frac{A_{LL}(t)}{N(t)} &= \frac{\frac{N^*}{2} + x_1(t)\epsilon + x_2^* \epsilon^2 + \ldots}{N^* + [x_1(t) + y_1(t) + z_1(t)] \epsilon + [x_2^* + y_2^* + z_2^*] \epsilon^2 + \ldots},
\end{align*}
\begin{align*}
\frac{A_{RL}(t)+A_{RR}(t)}{N(t)} &= \frac{\frac{N^*}{2} + [y_1(t) + z_1(t)] \epsilon + [y_2^* + z_2^*] \epsilon^2 + \ldots}{N^* + [x_1(t) + y_1(t) + z_1(t)] \epsilon + [x_2^* + y_2^* + z_2^*] \epsilon^2 + \ldots}.
\end{align*}

Rearrangement of the terms in the series is permissible since the series converge absolutely [41]. Using (4.3), it is clear that the frequencies of left-handed and right-handed fish equal one another to second order. We cannot prove that the phenotypic frequencies equal one another, but our analysis suggests equality near criticality.
We constructed and analyzed four discrete-time models of varying complexity and biological realism to explain a phenotypic oscillation in mouth-handedness, discovered by Hori, in a population of *P. microlepis*. Our modeling effort was motivated by, and partially based on, the model of Takahashi and Hori [40]. Our modeling approach was novel as it combined both population dynamic and population genetic processes. In particular, our model included terms that accounted for birth and deaths, intraspecific competition, complete dominance, and frequency-dependent selection. Despite the biological complexity, we were able to study the models from both an analytic and numerical perspective.

All four of our models predicted a phenotypic oscillation under similar conditions, which corroborated the findings of Takahashi and Hori [40]. We were able to prove the existence and uniqueness of a positive equilibrium for all four models, which corresponded to a 1 : 1 phenotypic ratio. We rigorously showed that this equilibrium becomes unstable when frequency-dependent selection is sufficiently strong, and determined the necessary and sufficient conditions for the oscillation.

Our first three models included a weak density-dependent term, the so-called discrete logistic nonlinearity. We devised these models to determine if frequency-dependent selection could account for the phenotypic oscillation (in the absence of density-dependent terms known to promote oscillatory behavior). These models differed from one another in their dimensionality: we began with a three-dimensional model lacking stage structure and built-up to a nine-dimensional model with three distinct life stages. By creating stage-structured models we controlled the stages af-
fected by density-dependence and more closely matched the reproductive biology of
*P. microlepis*. Based on their stage-structured model, Takahashi and Hori hypothe-
sized that the time lag of the growth period was, in some way, responsible for the
oscillation.

Our initial three-dimensional model addressed the importance of the time delay
in growth for the phenotypic oscillation, since this model lacked stage structure.
Our analysis showed that such a delay is not necessary for oscillatory behavior. In
fact, we showed that frequency-dependent selection must be sufficiently strong for the
equilibrium to destabilize via a period doubling bifurcation. Specifically, the prey fish
must be sensitive and react strongly to small deviations from the 1 : 1 phenotypic
ratio (a parameter we called $q$). For biologically reasonable values of $q$, we found that
$\mu_a$ must also be sufficiently large for phenotypic oscillations. Furthermore, we showed
that the time-averaged frequency of left-handed fish is greater than the time-averaged
frequency of right-handed fish following bifurcation. Numerical simulations suggest
this phenomenon persists away from the bifurcation point. This finding is somewhat
surprising, since the left-handed allele is recessive.

Six- and nine-dimensional models were constructed by adding life stages in order to
better match the biology of *P. microlepis*. In particular, *P. microlepis* have multiple
life stages characterized by different morphologies and behaviors. We account for
these differences by introducing non-scale eating, sexually immature fish to the model,
and we adjust the length of the time step accordingly. We found that including life
stages in, and thereby increasing the dimensionality of, our model promoted the
destabilization of the equilibrium by decreasing the parameter region of equilibrium
stability. This is not an unexpected result since mathematically the inclusion of life
stages introduces, in effect, time delay into the model. The equilibrium destabilized
via an invariant loop bifurcation for both higher dimensional models.

Next we turned our attention back to a three-dimensional model, this time incor-
porating strong (over-compensatory) density-dependent effects. We can only spec-
ulate as to the importance of over-compensatory effects for cichlids, although we point out that such effects have been reported in many fish species and have a long history in the modeling of fish population dynamics (in particular, with regard to cannibalism) [10], [31]. In fact, filial cannibalism has been widely documented in fishes exhibiting maternal, paternal, and biparental care (as *P. microlepis* does) [34].

We used the Ricker nonlinearity to describe density-dependence. The model analysis demonstrated the complicated interplay between population dynamics and population genetics. Specifically, we found that strong density-dependent effects could cause phenotypic oscillations for values of $q$ that were smaller in magnitude than those realized for the other models. This finding indicates that classical population genetic models can fail to elucidate complex dynamics. Our analysis also showed that population dynamic oscillations were not indicative of phenotypic frequency oscillations. In fact, we found large regions of parameter space where the population dynamics oscillated wildly while the phenotypic frequencies equilibrated at the 1:1 ratio.

The modeling approach we used for *P. microlepis* is quite general, and may be applied to other biological systems for which the underlying genetics are known and are sufficiently simple. Furthermore, the functions used for frequency-dependent selection may be modified to accommodate other forms of selection, or other modes of mediation. By modeling absolute numbers rather than frequencies, we can easily apply our approach to organisms with overlapping generations. In our study we found that neglecting the population dynamics, as is done with the model of Takahashi and Hori, left out some interesting findings. In particular, the compounding effects of complex population dynamics and frequency dependent selection gave rise to phenotypic oscillations that were not otherwise predicted. For this reason we find it critical to incorporate population dynamic processes in population genetic models.

We now compare the predictions of our models with the known biological data. Unfortunately, the data collected describe the phenotypic frequencies, not the population dynamics of the genotypes. Therefore, a comparison of our model predictions
to the data is limited. We will make a comparison by computing the period of the oscillations of the model cycles with the period found in the data. We begin by recognizing that the period doubling bifurcations that occur for models (2.3) and (4.1) result in the period of the cycle being twice that of the projection time interval, or four years. Hori observed a periodic cycle with an approximate period of five years [16]. Roughly speaking, the period of the oscillation for models (2.3) and (4.1) agrees with the data.

The period of the oscillation corresponding to the invariant loop bifurcation that occurs for model (3.1) can be approximated by inspecting where the eigenvalues escape the unit disk and computing the rotation angle. This computation is possible because we have explicit formulae for the eigenvalues. The eigenvalues involved in the destabilization of the positive equilibrium for model (3.1) are

$$\lambda_{+, -} = \frac{(1 - \mu_a) \pm \sqrt{(1 + \mu_a)^2 + 2\mu_a g'\left(\frac{1}{2}\right) (\sqrt{2} - 1)}}{2},$$

where $\lambda_{+, -} = re^{\pm i\theta}$ and $r = |\lambda_{+, -}|$. Clearly, $\lambda_+ + \lambda_- = 2r \cos \theta = (1 - \mu_a)$, which implies $\cos \theta = \frac{1 - \mu_a}{2r}$. With $r > 1$ for equilibrium destabilization, we have

$$0 < \frac{1 - \mu_a}{2r} = \cos \theta < \frac{1}{2}.$$

The above inequality implies

$$\frac{\pi}{3} < \cos^{-1}\left(\frac{1 - \mu_a}{2r}\right) = \theta < \frac{\pi}{2}.$$

Approximating the period by $\frac{2\pi}{\theta}$ we obtain

$$4 < \frac{2\pi}{\theta} < 6.$$

Thus the period of the cycle ($p$) is approximately, $4 < p < 6$, which corresponds to
between four and six years. This result agrees with the phenotypic data.

While we have analyzed four models of varying biological realism, there are many unanswered questions regarding the dynamics of *P. microlepis*. Currently, very little is known about the biology of *P. microlepis*, and a first step would be to obtain more biological information, perhaps using manipulative experiments. Incorporating other important biological facts into our models will likely result in further mathematical complications. We list some of these possible considerations:

1. Biological experiments: Conducting experiments in a laboratory setting would allow parameter value estimation. Furthermore, experiments could be conducted to test the model predictions. For example, we have found the existence of multiple attractors for model (4.1). An experiment could be designed with multiple initial conditions lying in distinct basins of attraction. Furthermore, we could manipulate parameter values, such as \( \mu_a \), to determine if a switch occurs from equilibrium to oscillatory dynamics.

2. Model males and females: Our model assumes an equal male:female sex ratio. An unequal sex ratio will change the model by limiting reproduction by the less frequent sex. Furthermore, male and female consideration may be important if one of the sexes affects the fitness of a mating pair more than the other. For example, females with low fitness may fail to produce viable eggs whereas males with low fitness may not be affected. In this case the fitness of a mating pair is limited by the low fitness of females.

3. Incorporate a non-smooth function for \( h \): We assumed throughout the stability analysis that \( h'(\frac{1}{2}) = 0 \). This assumption, however, may not be reasonable. Recall that \( h \) is the function governing the fitness of \( R \times L \) and \( L \times R \) matings. The assignment of a fitness value to a mating pair consisting of individuals with different phenotypes is ambiguous, since the two individuals possess different
fitness values. It may be the case that the fitness of a mating pair consisting of two individuals with different fitness values is determined by the least fit individual. That is, regardless of how fit one of the individuals is, the less fit individual limits the overall reproductive success of the mating pair. In this case, we would choose \( h = \min \{g(l), g(1-l)\} \). This function is not differentiable at \( l = \frac{1}{2} \), thereby rendering all local stability analysis useless. Preliminary numerical studies indicate that non-smooth functions further promote phenotypic oscillation. In particular, oscillations occur for model (2.3) for much smaller values of \( |q| \) when \( h = \min \{g(l), g(1-l)\} \). The ability to account for selection at the level of the mating pair (e.g., fecundity) is one of the major benefits of our modeling approach.

4. Stochasticity: The models we have studied thus far are completely deterministic. However, we know that models that allow for some stochasticity can be more realistic. To examine the effects of stochasticity, we could construct a model with demographic or environmental stochasticity, or a blending of the two. Demographic noise involves variation in the number of births and deaths, whereas environmental noise involves variation in the environmental conditions (e.g., temperature and rainfall). Furthermore, we could account for genetic drift (i.e., the random sampling of alleles). This is especially prudent when population size is small.

5. Periodic forces: While we have shown that strong frequency-dependent selection can cause the phenotypic frequencies to oscillate, there may external periodic factors that contribute to these oscillations. For example, matings success may be influenced by the seasonal fluctuation in water temperature. In fact, Lake Tanganyika is subject to vast changes in water level due to variations in rainfall, temperature, and evaporation [36]. This type of environmental periodicity would, in theory, affect all individuals in the population similarly. Therefore,
we would not expect this type of external periodic forcing to cause phenotypic oscillations in the absence of frequency-dependent selection. However, periodic parameters in a nonautonomous model may enhance the oscillations we have observed.

6. Model prey explicitly: We account for the prey species in our models through the fitness functions, $g$ and $h$. That is, as the frequency of a phenotype increases, the fitness of that phenotype decreases due to the guarding behavior of the prey species. By modeling the prey’s behavior implicitly, we assume the prey population dynamics are stable. This assumption, while justified by data collected by Hori, may not be biologically reasonable in all environments and local populations [18], [17]. In particular, the prey species may oscillate thereby contributing to the phenotypic oscillation observed for $P. microlepis$. It has been suggested that the a lateral dimorphism also exists for the prey species, and this system was modeled using food web differential equations [26]. We would like to apply our modeling approach to such a case of coevolution. Furthermore, the predatory behavior of $P. microlepis$ may increase the susceptibility of the two prey morphs to pathogens and disease.

7. Species competition: We would like to be able to include several competing species, within the Perissodus genus, to determine how competition affects the underlying dynamics. Takahashi and Hori modeled the competition between $P. microlepis$ and $P. straelani$ using delay differential equations [39]. This model, like the model presented in [40], has phenotypic frequencies as state variables. As we have argued in Chapter 1, it is not clear how to relate the phenotypic frequency at some later time to the phenotypic frequency at some previous time. For this reason we would like to adapt our modeling approach to account for competing species in regions of the lake where they co-occur. Data suggests that $P. microlepis$ and $P. straelani$ exhibit exploitative mutualism
(both species benefit from the presence of the other) [25], [38]. Furthermore, phenotypic frequencies for both species appear to oscillate in phase with one another [39].

8. Introduce life stages to model (4.1): A numerical study could be conducted to determine how increasing dimensionality in model (4.1) changes the dynamics. For instance, we would like to know whether increasing dimensionality results in phenotypic oscillations for smaller values of $|q|$ and $\mu_\alpha$ than for models (3.1) and (3.5).
References


