Intro to the Topic
Discrete Models
Growth and Decay
Linear & Non-Linear Interaction Models

Applications of Non-Linear Models: Logistic Growth

- Linear difference equations are useful in that they permit closed-form solutions to be easily obtained.
- However, solutions often have the disadvantage that they do not agree with observation.
- In many areas of biology, especially population biology, non-linear models are better.
- This section focuses on the development of some simple non-linear models for the growth of populations over time.
- The simplest model is the logistic equation.
- Introduce the logistic equation in discrete form in this chapter and study in detail in continuous form in the next.

Simple linear model $M_{n+1} = aM_n$ is generally unsuitable for real populations due to unbounded growth as $n$ increases.

So if we express it in the form:

$$M_{n+1} = M_n + r \times M_n \quad (2.56)$$

$r \equiv$ no. of births $-$ no. of deaths per time period.

and replace $r$ by $R(M_n)$ (non-constant growth rate affected by population size) get the Logistic Growth Eqn:

$$M_{n+1} = M_n + kM_n \left(1 - \frac{M_n}{K}\right) \quad (2.57)$$

where

$$R(M_n) = k - \frac{k}{K}M_n,$$

for growth rate $k$ & carrying capacity of the population $K$ (a large number that limits the growth rate $k$).
In the Logistic Growth Eqn (2.57)

\[ M_{n+1} = M_n + kM_n \left( 1 - \frac{M_n}{K} \right) \]

the term in brackets behaves as follows:

- For small \( M_n \), \( \frac{M_n}{K} \) is small & growth is unbounded.
- For large \( M_n \), \( \frac{M_n}{K} \to 1 \) & Eqn (2.57) behaves like

\[ M_{n+1} = M_n + \epsilon \]

for small \( \epsilon \).

In other words for large \( M_n \) overcrowding kicks in and the growth rate slows to zero.

By writing Eqn.(2.57) in the form:

\[ M_{n+1} - M_n = kM_n \left( 1 - \frac{M_n}{K} \right) \] (2.58)

notice some important features; as LHS of eqn(2.58) is population change between successive times.....

- follows that if \( M_n < K \) then population in the next time interval will increase & decrease for \( M_n > K \).
- expect a steady increase in population for small \( M_n \) but small oscillations above & below \( K \) for large populations.

Model is realistic in this respect mirroring real populations.

Numerical problems (i.e. predicted -ve populations) happen for certain \( k \) in Eqn.(2.58) & reduce applicability of this model.
In order to see how the Logistic Growth model performs in practice examine plots of $M_n$, $V_n$ for various $k$.

$k$ corresponds to the average fertility of an individual in the population.

$k$ varies in the range $0 < k \leq 3$ for fixed carrying capacity $K = 1000$ & initial population $M_0 = 100$.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{logistic_plot.png}
\caption{Logistic Equation: Stable Growth}
\end{figure}

- Populations in Fig 2.7 (a),(b) approach $K$ as $n$ increases.
- Growth initially exp'l but $K$ causes pop'n to level out.
- Larger $k$ causes steeper growth, more rapid overcrowding.
Figure 2.8: Logistic Equation: Damped Oscillations

- Early on, pop’n growth is so rapid that it overshoots \( K \) before overcrowding is felt.
- Note: the higher the \( k \) value greater than 1, the longer for oscillations to die out.

Figure 2.9: Logistic Equation: Cyclic Growth

- In Fig 2.9(a,b) plot populations with \( 2 < k < 2.57 \).
- Pop’n not damped but fluctuates above & below \( K \), comes back every 2nd breeding season, a 2-cycle.
As \( k \to 2.57 \), pop’n repeats every 4th breeding season, a 4-cycle.

Beyond \( k = 2.5 \) (& for \( k < 2.57 \)), 4-cycles become 8-cycles - period doubling.

For \( 2.57 < k < 3 \) (Fig. 2.11 a,b), even though behaviour is still predicted by simple difference eqn, pattern of growth appears to become more random for increasing \( k \).

Discovery of chaotic behaviour was important in modelling as it questioned the fact that external events always caused complex population fluctuations.

Often difficult to tell whether they are chaotic or simply long-term periodic.

One feature of chaotic behaviour is its sensitivity on the initial population; if \( M_0 \) is varied slightly, can cause wide variations in pop’n in subsequent times.
Applications of Non-Linear Models: Logistic Growth cont’d

Why does pop’n oscillate at all? Answer is twofold:
- pop’n is self-regulating through pop’n-dependent growth rate,
- regulating effect is felt in next time interval but determined in current time interval.

Gives rise to a natural time delay when pop’n is responding to overcrowding & a corresponding over-compensation when growth rate is sufficiently high. Over-compensation can lead to oscillatory behaviour & chaos.

Note: if pop’n is large enough (or time step small enough) that breeding can be assumed to be continuous, growth rate can respond instant’ly to pop’n & oscillations do not normally occur, except due to external or seasonal factors.

Differential equations will be used to show this below.

**Figure 2.11**: Logistic Equation: Into the Chaotic
Previously difference equations used to model pop’n changes from one generation to next.

So, as chars of individuals change from one generation to next thru heredity, it seems natural to use diff eqns with laws of probability to predict dominant genes in a pop’n.

Basic laws that use are those based on Mendel’s ideas.

Main result of Mendel’s ideas is that certain traits of plants & animals determined by pairs of genes.

2 genes responsible for same char (eg eye colour) are called alleles of each other & 2 genes are said to form pair of alleles.

Different pairs of alleles, resp for diff characteristics, are located at diff points on chromosome. It is common to denote alleles by letters $A$ and $\alpha$ respectively.

So in any individual in a species, the alleles can occur in just one form:

$$AA, \ A\alpha \ or \ \alpha\alpha$$

called genotypes. (Note that genotype $A\alpha \equiv \alpha A$.)

A given genotype determines a physical char of individual. Which allele of $A$ and $\alpha$ determines the particular char is said to be dominant, (other is recessive).

If $A$ is dominant & causes some char then all genotypes with this allele will have that char (assuming 1 gene only is responsible).
Next generation, assume (as per Mendel’s first law) that allele in egg/sperm is chosen at random from parent’s two alleles.

So if AA genotype mates with Aα, possible outcomes are AA, Aα, AA, αA.

Thus, on average, no. of AAs will be half total number of offspring, or, to generalize, proportion of AA genotypes given by:

\[ G(AA) = \frac{N'(AA)}{N} \tag{2.59} \]

where \( N'(AA) \) is no. with genotype AA & \( N \) is total number of offspring.

We can now introduce gene pool concept to which each member of a pop’n contributes its genotype’s 2 alleles.

As gene pool contains all alleles in pop’n, can talk about proportion of alleles of one kind or another.

If \( P(A) \) & \( P(α) \) are proportions of A and α alleles in pop’n respectively then can say that:

\[ P(A) = \frac{2N'(AA)+N'(Aα)}{2N}, \]
\[ P(α) = \frac{2N'(αα)+N'(Aα)}{2N} \tag{2.60} \]
Note: can view eqns such as Eqn.(2.59, 2.60) as prob’s i.e. $G(AA)$ is proportion of genotype $AA$ in pop’n but also prob that a genotype selected at random is $AA$.

Similarly from Eqn.(2.60), $P(A)$ gives prob that genotype selected at random contains $A$ allele.

As with all applications of probability must have:

$$G(AA) + G(A\alpha) + G(\alpha\alpha) = 1$$

and

$$P(A) + P(\alpha) = 1$$

(2.61)

Random Mating with Equal Survival

Now progress to use these derivations to model changes in gene pool over time. Initially make a number of assumptions to simplify the process:

- Mating occurs at random & does not depend on a mate’s genotype
- Equal survival: each genotype has same chance of surviving from fertilized egg stage to mating stage (at end of generation)
- Equal fertility: each couple produces on average same number of viable sperm and eggs again irrespective of genotype.

Change of terminology:

- $G_k(AA)$ expected prop of genotype $AA$ (say) at end of $k$th generation &
- $G_{k+1}(AA)$ denotes that at start of next generation.

Similar sub-/ superscripts hold for proportions of alleles.
Random Mating with Equal Survival cont’d

- Since prop of a particular genotype (say, again AA) in new generation will depend on prop’s of alleles making up that genotype in previous generation (meeting up and mating) then:

\[ G_{k+1}^*(AA) = P_k(A)P_k(A) = [P_k(A)]^2 \]

\[ G_{k+1}^*(A\alpha) = P_k(\alpha)P_k(A) + P_k(A)P_k(\alpha) = 2P_k(A)P_k(\alpha) \] (2.62)

\[ G_{k+1}^*(\alpha\alpha) = P_k(\alpha\alpha)P_k(\alpha\alpha) = [P_k(\alpha)]^2 \]

Recall that point of this was to model changes in gene pool over time.

This can be done by deriving difference equations for allele proportions using a two step process:

- calculate the probability/fraction of each genotype using Eqn.s(2.62) & constant survival fraction to maturity \( r \),

- count up one of the allele proportions & eliminate the other using \( P_k(A) + P_k(\alpha) = 1 \).
Random Mating with Equal Survival cont’d

If \( N_{k+1}^* (AA) \) is no. of genotype \( AA \) at start of \( k+1 \)th gen’n & \( N_{k+1}^* \) total no. of fertilized eggs which give rise to that gen’n, then, from Eqn.(2.62)

\[
N_{k+1}^* (AA) = [P_k(A)]^2 N_{k+1}^* \\
N_{k+1}^* (A\alpha) = 2P_k(A)P_k(\alpha)N_{k+1}^* \\
N_{k+1}^* (\alpha\alpha) = [P_k(\alpha)]^2 N_{k+1}^*
\]  

(2.63)

\& assuming a survival fraction \( r \) of fertilized egg to end of \( k+1 \)th gen’n & maturity:

\[
N_{k+1} (AA) = r [P_k(A)]^2 N_{k+1}^* \\
N_{k+1} (A\alpha) = 2rP_k(A)P_k(\alpha)N_{k+1}^* \\
N_{k+1} (\alpha\alpha) = r [P_k(\alpha)]^2 N_{k+1}^*
\]  

(2.64)

which, for equal survivals, yields \( P_{k+1}(\alpha) = P_k(\alpha) \), ie const \( \alpha \) allele pop’n over all gen’ns.

Random Mating with Lethal Recessives

Taking the case of lethal recessives, no assumption of Equal Survival above rather that carriers of two copies of the allele \( \alpha \) (say) do not survive to maturity & \( N_{k+1}(\alpha\alpha) = 0 \). Now, from Eqn.(2.59)

\[
P_{k+1}(\alpha) = \frac{\text{no. of } \alpha \text{ alleles}}{\text{total no. of alleles in gene pool}}
\]

therefore

\[
P_{k+1}(\alpha) = \frac{2N_{k+1}(\alpha\alpha) + N_{k+1}(\alpha A)}{2N_{k+1}(AA) + 2N_{k+1}(A\alpha) + 2N_{k+1}(\alpha\alpha)}
\]  

(2.65)

substituting from Eqn.(2.64), to get:

\[
P_{k+1}(\alpha) = \frac{P_k(A)P_k(\alpha)}{[P_k(A)]^2 + 2P_k(A)P_k(\alpha)}
\]  

(2.66)
Random Mating with Lethal Recessives cont’d

Which, given \( P_k(A) + P_k(\alpha) = 1 \) gives

\[
X_{k+1} = \frac{X_k}{1 + X_k}
\]

(2.67)

where \( X_k = P_k(\alpha) \). Eqn.(2.67) is a non-linear diff eqn which, has a closed-form solution. It can be seen that

\[
X_1 = \frac{X_0}{1 + X_0}
\]

so

\[
X_2 = \frac{X_1}{1 + X_1} = \frac{\frac{X_0}{1 + X_0}}{1 + \frac{X_0}{1 + X_0}} = \frac{X_0}{1 + 2X_0}
\]

and

\[
X_2 = \frac{X_2}{1 + X_2} = \frac{\frac{X_0}{1 + 2X_0}}{1 + \frac{X_0}{1 + 2X_0}} = \frac{X_0}{1 + 3X_0}
\]

So, from this, can write the general form for the pop’n of \( \alpha \)-alleles in \( k \)th generation:

\[
P_k(\alpha) = X_k = \frac{X_0}{1 + kX_0}
\]

(2.68)

where \( X_0 \) is the initial population of \( \alpha \)-alleles.

From eqn(2.68), if \( P_0(A) = P_0(\alpha) = 0.5 \), will take 8 gen’ns for recessive allele proportion to reduce to 0.1, 98 to reduce to 0.01 and 998 to reduce to 0.001.

This slow elimination of a lethal recessive is due to the *carrying effect* of hybrid genotype \( A\alpha \).
For a more true-to-life model than the above take Natural Selection where a fraction \( r_1 \) newborn AA & Aα populations survive to end of gen’n & a different fraction \( r_2 \) of newborn αα survive. From Eqn.(2.65), have:

\[
P_{k+1}(\alpha) = \frac{2N_{k+1}(\alpha\alpha) + N_{k+1}(\alpha A)}{2N_{k+1}(AA) + 2N_{k+1}(A\alpha) + 2N_{k+1}(\alpha\alpha)}
\]  

(2.69)

& substituting altered form of Eqn.(2.64):

\[
\begin{align*}
N_{k+1}(AA) &= r_1 [P_k(A)]^2 N_{k+1}^* \\
N_{k+1}(A\alpha) &= 2r_1 P_k(A) P_k(\alpha) N_{k+1}^* \\
N_{k+1}(\alpha\alpha) &= r_2 [P_k(\alpha)]^2 N_{k+1}^*
\end{align*}
\]  

(2.70)

simplifying, to get:

\[
P_{k+1}(\alpha) = \frac{2r_2 X^2 + 2r_1 X(1-X)}{2r_1(1-X)^2 + 4r_1 X(1-X) + 2r_2 X^2}
\]  

(2.71)

where \( X = P_{k+1}(\alpha) \) and \( 1 - X = P_{k+1}(A) \). Hence

\[
P_{k+1}(\alpha) = \frac{r_2 X^2 + r_1 X - r_1 X^2}{r_1(1-2X+X^2) + 2r_1 X - 2r_1 X^2 + r_2 X^2}
\]  

\[= \frac{r_1 X^2(\beta-1) + X}{1 + X^2(\beta-1)}
\]  

(2.72)

where \( \beta = r_2/r_1 \) is relative fitness of the genotype αα & measures the fitness to survive of the αα genotype relative to the AA and Aα genotypes.

Note for \( \beta = 0 \) get lethal recessive model & for \( \beta = 1 \) get equal survival model.