MATHEMATICAL MODELS OF POPULATION BIOLOGY

Living organisms occur in discrete populations, often delimited by specific ecological and/or ethological boundaries. When scientists study organismal biology, at some point they need to consider population structure. Population biology is a quantitative science, whereby the dynamics and kinetics of population distribution, abundance and growth are measured (as prevalence, incidence, density, concentration, intensity, etc.). Many mathematical models have been developed to describe population structures based on past and current trends, and then used to predict future trends.

Co-existence

Scientists recognize many different levels of biological organization, from the miniscule to the majestic. Living things range from single-celled organisms (simple, but by no means primitive) to multicellular organisms (with cellular specialization to form complex tissues and organs). We also recognize several levels of collective co-existence, where organisms live together in:

- populations (all the individuals of a species within a given area);
- communities (all species of living organisms within a given area);
- ecosystems (all living things within a given area, together with all the non-living components in that area with which life interacts); and the
- biosphere (all the environments on Earth inhabited by life).

Ecology is the study of interactions between organisms and their environments; a holistic science involving many disciplines. Ecologists seek to understand organismal biodiversity, distribution and abundance (species richness, temporal and spatial variation) with respect to biotic and abiotic (environmental) influences.

Ecosystem ecology emphasizes energy flow and chemical cycling among the various biotic and abiotic components.

Community ecology deals with the interactions between the whole array of species in a community, including competition, predation, herbivory, symbiosis, and disease.

Population ecology concentrates mainly on factors that affect how many individuals of a particular species live in an area.

The study of parasite ecology is complicated by the fact that it involves two populations:

- that of the parasite; and
- that of the host.

It is not enough to know how the temporal and spatial distribution and abundance of the parasite changes without considering host population dynamics, kinetics and demography. For example, how many hosts are infected, how severe/intense are the infections, are infected hosts delimited by age, gender, behaviour, etc.?

It is important that interactions between the two populations be carefully examined; basically in order to answer the following question: Do the partner organisms gain benefit or detriment from the interaction? The answer effectively defines the inter-species biological relationship between the two organisms.

In this instance, parasitism is defined as a type of relationship beneficial to the parasite but detrimental to the host (see following Table for different types of relationships).

Type of relationship	Organism 1 (usually larger)	Organism 2 (usually smaller)	Examples
SYMBIOSIS ("living together") (direct contact between organisms) (usually long term)			
mutualism	host benefits	symbiont benefits	ruminants/ciliates
cleaning symbiosis	host benefits	cleaner benefits	fish/cleaner wrasse
commensalism	host unaffected	commensal benefits	reptiles/trichomonads
phoresis	host unaffected	phoront benefits	mollusc/anemone
inquilinism	host unaffected	inquiline benefits	worm tubes/bacteria
neutralism	host unaffected	symbiont unaffected	difficult to prove
parasitism	host harmed	parasite benefits	humans/nematodes
parasitoidism	host harmed	parasitoid benefits	caterpillar/wasp larva
amensalism	unaffected	harmed	mould/bacteria
OTHER ECOLOGICAL INTERACTIONS (direct/indirect contact) (often short term)			
PREDATION (involving feeding, consumption)			
herbivory	herbivore benefits	plant harmed	ruminants/grass
carnivorism	predator benefits	prey harmed	felids/rodents
INTERSPECIFIC COMPETITION (interference/exploitative/apparent/scramble competition) (competitive exclusion, niche differentiation, local extinction, change in community structures)			
competition	harmed	harmed	lions/cheetahs
synnecrosis	killed	killed	extinctions
DECEPTION (crypsis (hard to see), mimesis (masquerade), motion dazzle)			
mimicry	unaffected	mimic benefits	wasp/moth
camouflage	unaffected	benefits	leaf/katydid

It is essential that the type of relationship be determined in order to select the most appropriate methodology to quantitate and model population growth over time, for example:

- predator-prey populations are best examined by Lotka-Volterra models;
- unconstrained microbial growth by exponential models;
- constrained growth by logistic models;
- microparasites by SIR models; and
- macroparasites by HPW models.

Population ecology

Population ecology is the study of populations in relation to the environment, including abiotic/biotic influences on population density, distribution, age structure, and size. Demography is the study of the vital statistics of populations and how they change over time. Populations wax and wane in size as individuals join (births and immigration) and leave (deaths and emigration). The population growth rate ordinarily refers to the change in population over a specific time period expressed as a percentage of the number of individuals in the population at the beginning of that period; given by the formula:

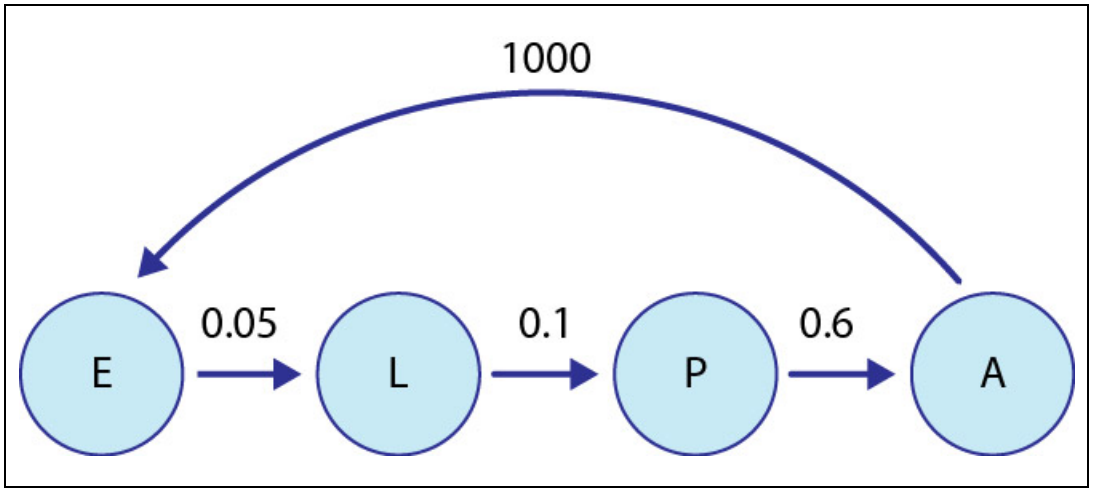
Growth rate = [(births + immigration) - (deaths + emigration)] / population

The survival pattern of a population can be summarized in a life table, best constructed by following the fate of a specific cohort (a group of individuals of the same age) from birth until death. For example, the following hypothetical life table may apply to flea populations, showing the survival of the different developmental stages over time.

Time period	Developmental stage	Number alive
1	Eggs (E)	10,000
2	Larvae (L)	500
3	Pupae (P)	50
4	Adults (A)*#	30

*The male:female ratio of adult fleas is 1:1
#Adult female fleas lay a total of 2,000 eggs

Stage-structured diagrams can be used to depict the life-cycles of organisms, with arrows connecting different stages showing the proportion of the population transitioning between those stages within any single time interval. For example, the stage-structured life-cycle model based on data from the hypothetical flea life table would be:



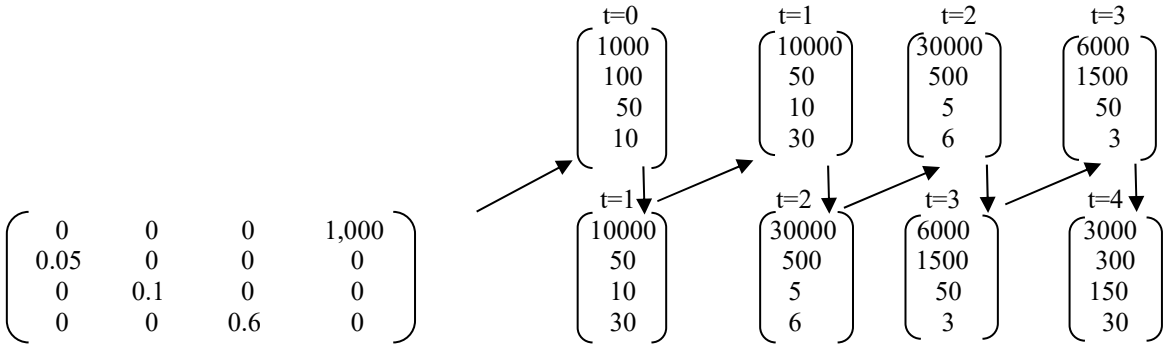
Simultaneous equations (SEs) can be developed to model population structures, allowing the calculation of the numbers of individuals within particular stages over time. The relevant SEs for the hypothetical flea model from time t to time $t+1$ would be:

$E_{t+1} = 0\,E_t + 0\,L_t + 0\,P_t + 1{,}000\,A_t$ [which simplifies to = $1{,}000\,A_t$]
$L_{t+1} = 0.05\,E_t + 0\,L_t + 0\,P_t + 0\,A_t$ [which simplifies to = $0.05\,E_t$]
$P_{t+1} = 0\,E_t + 0.1\,L_t + 0\,P_t + 0\,A_t$ [which simplifies to = $0.1\,L_t$]
$A_{t+1} = 0\,E_t + 0\,L_t + 0.6\,P_t + 0\,A_t$ [which simplifies to = $0.6\,P_t$]

The constants derived for the SEs can be incorporated into a transition matrix (delete letters but retain numbers) so that matrix operations can be used to streamline mathematical calculations. The 4x4 transition matrix for the hypothetical flea model would be:

$$\begin{pmatrix} 0 & 0 & 0 & 1{,}000 \\ 0.05 & 0 & 0 & 0 \\ 0 & 0.1 & 0 & 0 \\ 0 & 0 & 0.6 & 0 \end{pmatrix}$$

The transition matrix can be used to determine the numbers of individuals within each life cycle stage over time. For example, if you currently have 1000 eggs, 100 larvae, 50 pupae and 10 adult fleas, you can use the transition matrix to determine how many of each stage you will have after each time period has passed (begin with t = 0), calculated as follows:



Thus, after four time periods, there will be 3000 eggs, 300 larvae, 150 pupae and 30 adult fleas.

Numerous mathematical processes can be applied to quantitate the numbers of individuals within a population, but they require sound knowledge of population developmental biology as well as reasonable counts or estimates of current population numbers. More frequently, models developed for populations of single species of organisms are based on their growth rates (calculated from births, deaths, immigrations, and emigrations). Two common models describe exponential growth (where population size is unconstrained as resources are unlimited) and logistic growth (where population size is constrained to a carrying capacity due to resource limitations).

Exponential model of population growth

The exponential model describes population growth in an idealized, unlimited environment. While this may not frequently occur naturally, it can be used to examine the capacity for a species to increase in population size and the conditions for that to occur. Population growth rate is based on influx (births and immigration) and efflux (deaths and emigration). The population increases in size when birth rates exceed death rates, and when immigrations exceed emigrations. If we ignore migrations for simplicity's sake (as in closed populations), we can express the change in population size mathematically as:

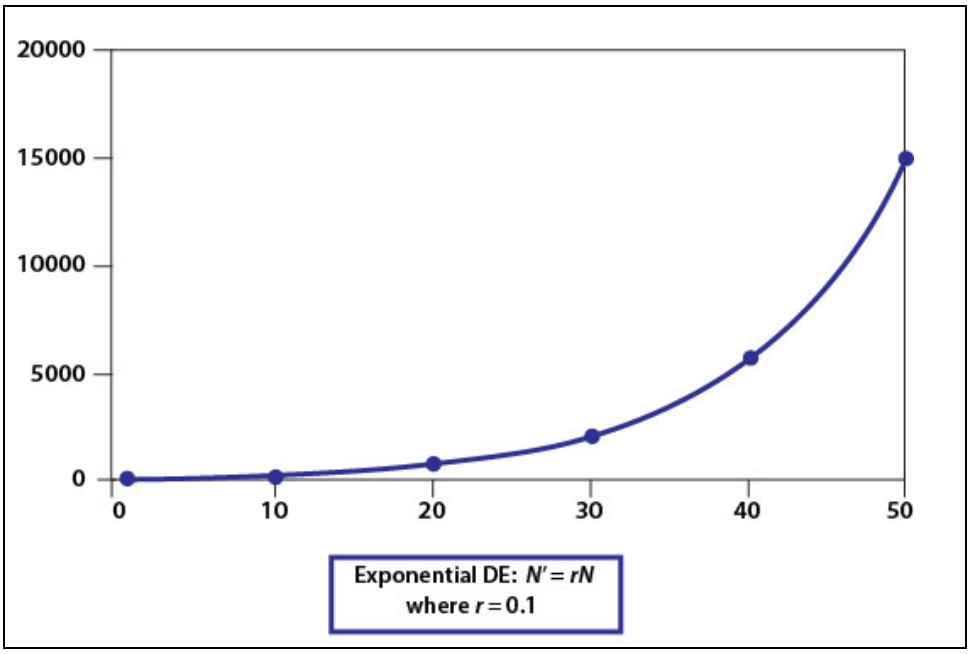
$\Delta N/\Delta t = B - D$ where ΔN = change in population size;
Δt = time interval;
B = births; and
D = deaths.

Births and deaths can also be expressed as "per capita rates", by calculating the average number occurring per individual during the specified time period. For example, if there are 34 births per year in a population of 1,000 individuals, the annual per capita birth rate, b = 34/1000 (= 0.034). This rate can be used to calculate the expected number of births per year in a population of any size by using the formula $B = bN$. Similarly, if there are 24 deaths in the population over the same period, the annual per capita death (mortality) rate, m = 24/1000 (= 0.024), and $D = mN$. The equation for population change over time can be simplified as:

$\Delta N/\Delta t = bN - mN = (b - m)N$

The birth and death rates can be amalgamated into the "per capita rate of increase (r)" [= (b - m)]. Obviously, the population is growing when $r > 0$, declining when $r < 0$, and stable when $r = 0$. The equation for change in population size is therefore simplified to:

$\Delta N/\Delta t = rN$ [expressed as $dN/dt = rN$ or $N' = rN$ using differential calculus]



This differential equation (DE) states that the rate of change of the population (N') depends on the growth rate (r) and the current population size (N). The solution to this differential equation is the exponential function:

$N = Ae^{rt}$ [proven by differentiating the function to give $N' = r.Ae^{rt} = rN$]

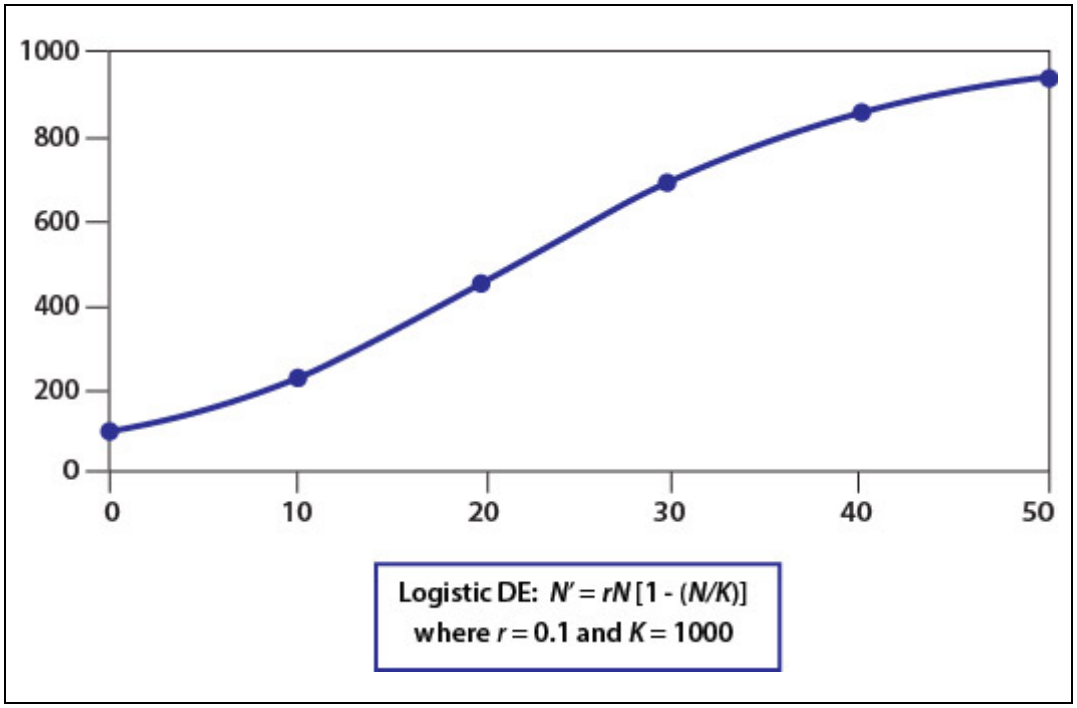
Under ideal conditions, population growth can become exponential. Births exceed deaths, so the population grows and accumulates more new individuals per unit of time when it is large. When population size is plotted over time, exponential growth is seen as a characteristic J-curve (increase becoming steeper with time). J-shaped curves are characteristic of populations that are introduced into new or unfilled environments (especially in the microscopic world of microbes).

Logistic model of population growth

In most natural systems, resources are limited therefore population growth is constrained. The logistic model of population growth incorporates the concept of 'carrying capacity' to account for limited resources. As population density increases, each individual has access to a smaller share of the total resources available. Ultimately, there is an upper limit to population size; the 'carrying capacity' defined as the maximum stable population size that a particular environment can support. Carrying capacity is not fixed but varies over time and space. Energy limitation often determines carrying capacity, although other factors, such as shelters, refuges, nutrients, water, breeding sites etc, can be limiting. If individuals cannot obtain sufficient resources to reproduce, the per capita birth rate (b) will decline. If they cannot find and consume enough energy to maintain themselves, the per capita death rate (m) may increase. A decrease in b or an increase in m results in a lower per capita rate of increase (r). In the logistic population growth model, growth declines as carrying capacity is reached.

Mathematically, the logistic model begins with the equation for exponential growth, adding an expression that reduces the rate of increase as N increases. If the carrying capacity is K, then $(K - N)$ is the number of additional individuals the environment can accommodate, and $(K - N)/K$ is the fraction of K that is still available for population growth. Multiplying r by this fraction gives the growth rate of the population as:

$dN/dt = rN\,[(K - N)/K]$ [alternatively expressed as $N' = rN\,[1 - (N/K)]$



When N is small compared to K, the term $[(K - N)/K]$ is large and the rate of increase is steep. But when N is large and approaches K as resources become limited, the term $[(K - N)/K]$ is small and so is the rate of population growth. When N is plotted over time, the logistic model of population growth produces an S-shaped (sigmoid) curve. Population growth rate slows dramatically as N approaches K.

The solution to the logistic differential equation is the function:

$$N(t) = K.N_o / [N_o + (K - N_o)\, e^{-rt}]$$

In practice, knowledge of this function is not required to calculate new population sizes as the DE can be solved approximately using Euler's method where the new population size (N_{new}) can be calculated by knowing the old population size (N_{old}) and the rate of change (N') of the population over known time steps (h):

$$N_{new} = N_{old} + N'.h$$

***r/K* selection theory**

The fitness of a species can be fundamentally enhanced by two different strategies: fast reproduction (multiplication, replication) or prolonged development (long-life, stability). These strategies are not independent, but form a continuum from one extreme to another for different species. How much one species invests in one strategy over the other depends on the selective environment, and in biology is called *r/K* selection. The mathematical principles are derived from the logistic growth model of population biology which indicates the traits that favour either quantity or quality of offspring in a species. That is, *r*-selected species invest in reproduction (quantity) while *K*-selected species invest in prolonged development and long-life (quality).

$$dN/dt = rN\,[(K - N)/K]$$

where r = growth rate (reproduction) (quantity)
K = carrying capacity (development) (quality)
N = population size, and
t = time

***r*-selected species**	***K*-selected species**
population grows exponentially (abundant resources) but never reaches carrying capacity (predators, droughts, etc)	population approaching carrying capacity, slow growth due to competition for limited resources
colonizers (opportunists)	competitors
unstable environments	stable environments
exploit less-crowded ecological niches	strong competitors in crowded niches
small organisms	large organisms
short-lived	long-lived
weak competitors	strong competitors
numerous offspring	few offspring
offspring with low probability of survival	offspring with high probability of survival
little parental care	significant parental care
fast maturation	slow maturation
rapid dispersal	slow dispersal
broad range	territorial
Examples: bacteria, protists, insects, weeds, rodents	Examples: terns, whales, elephants, trees, humans

Epidemiology

Epidemiology literally translates from Greek terms to mean "the study of what is upon the people", suggesting that it applies only to human populations. However, the term is widely used in zoology (sometimes supplanted by the term epizootiology), botany and microbiology. Epidemiology is the study of factors affecting the health and illness of populations. It provides a quantitative foundation for evidence-based medicine for identifying risk factors for disease and determining optimal treatment approaches. Studies may be conducted on communicable (infectious) diseases (caused by viruses, bacteria, fungi, protozoa, helminths, and arthropods) and non-communicable diseases (usually classified as inflammatory, cardiovascular, neoplastic, genetic, developmental, endocrine, nutritional, autoimmune, traumatic, senescence, iatrogenic, or idiopathic in origin). Diseases may be evident in a population as sporadic (occasional), endemic (established/persistent), epidemic (outbreak) or pandemic (global in distribution) diseases.

To study the occurrence, spread and control of diseases, epidemiologists employ a range of study designs generally categorized as descriptive (observational), analytic (testing for significant relationships) and experimental (testing treatments and other interventions). Generally speaking, epidemiologists study relationships between cause (exposure) and effect (morbidity and mortality). Morbidity is defined as the incidence of illness, and mortality as the incidence of death. Public health agencies often combine these measurements into a single equation:

$$DALY = YLD + YLL$$

where DALY = Disability Adjusted Life Years (combined burden);
YLD = Years Life lost due to Disability (morbidity burden); and
YLL = Years Life Lost due to premature death (mortality burden).

Assessments of the impact of disease are based on measurements of its distribution and abundance within a population (incorporating spatial and temporal variation), including:

- prevalence (cross-sectional study of population at selected point or period)
  [if 70 people were infected from a population of 100 examined in July, prevalence = 70%]

- incidence (longitudinal study of population over specified time)
  [if prevalence is 70% in July and 80% in October, incidence = + 10% (increased by 10% over 3 months)]
  [Note, can have negative incidence, when prevalence decreases over time]

- intensity/severity (different categories, often qualitative, sometimes quantitative)
  [disease severity based on range of symptoms/signs, often supported by clinical parameters]
  [infection intensity = number of organisms per infected host]
  [Note, mean infection intensity is different from mean parasite abundance (the latter may include uninfected hosts)]

These parameters may exhibit longitudinal fluctuations (variation over days, weeks, months or years) due to a wide variety of factors (demographic, socioeconomic, behavioural, geographic, and climatic). For example, climatic changes associated with global warming are predicted to cause many diseases to increase in incidence, distribution and severity (due to host translocations, greater susceptibility/diminished resistance, expanded ranges and enhanced survival of pathogens and vectors, ineffective/inappropriate treatment/control). Establishing cause and effect can be problematic, so several guidelines (non-prescriptive criteria) have been proposed for assessing evidence of causation (the Bradford-Hill criteria), involving:

- Strength (a small association does not mean that there is not a causal effect);
- Consistency (replication by different persons in different places with different samples);
- Specificity (the more specific the association, the greater the probability);
- Temporality (the effect must occur after the cause, and include any expected delays);
- Biological gradient (higher exposure should generally lead to higher incidence);
- Plausibility (there should be a plausible mechanism to explain the relationship); and
- Coherence (correlation between field and laboratory findings).

There are four main types of epidemiological studies: case series, case controls, cohorts and outbreaks.

1. Case series describe the experience of a single patient, or a group of patients with a similar diagnosis. They are purely descriptive and often rely on an astute clinician identifying an unusual feature of a patient's disease and formulating a hypothesis. Analytical studies are then done to investigate possible causal factors.

2. Case control studies involve matching cases with disease to comparable controls without disease (from the same local population), and then looking back through time at potential exposures both populations may have encountered. A 2 x 2 matrix is constructed, displaying exposed cases (A), exposed controls (B), unexposed cases (C) and unexposed controls (D).

	CASES	CONTROLS
EXPOSED	A	B
UNEXPOSED	C	D

The parameter used to measure association is the odds ratio (OR), which is the ratio of the odds of exposure in the cases (A/C) to the odds of exposure in the controls (B/D), that is,

$$OR = (AD) / (BC)$$

If OR >> 1, then the conclusion is "those with the disease are more likely to have been exposed".
If OR ~ 1, then exposure and disease were probably not associated.
If OR << 1, this suggests that exposure may have been protective in the causation of disease.

Case control studies are usually faster and more cost effective than cohort studies, but are sensitive to bias (such as recall and selection bias).

3. Cohort studies select subjects based on their exposure status, and then follows them forward through time to assess their outcome status. The results are tabulated in a 2x2 matrix:

	CASES	CONTROLS
EXPOSED	A	B
UNEXPOSED	C	D

The parameter generated to measure association is the relative risk (RR) which is the ratio of the incidence of disease in the exposed group (A/(A+B)) to that in the unexposed group (C/(C+D)), that is,

$$RR = (A/(A+B)) / (C/(C+D))$$

If RR >> 1, then the conclusion is "those with the exposure were more likely to develop disease".
If RR ~ 1, then there was no association.
If RR << 1, then exposure may have even been protective.

Prospective cohort studies have many benefits over retrospective case control studies. The RR is a more powerful effect measure than the OR, as the OR is just an estimation of the RR, since true incidence cannot be calculated in a case control study where subjects are selected based on disease status. Temporality can be established in a prospective study, and confounders are more easily controlled for. However, cohort studies are more costly, and there is a greater chance of losing subjects to follow-up based on the long time period over which the cohort is followed.

4. Outbreak investigations. A disease outbreak is a classification used in epidemiology to describe the sudden appearance, or sudden increase in incidence, of a disease in a specified group or population. Local outbreaks may be confined to a small area or village, while epidemics are outbreaks affecting regions or countries, and pandemics are global disease outbreaks. Outbreaks may originate from single point sources or from multiple common sources. Exposure to infections may be irregular, periodic or continuous and may involve contact with environmental sources (water, food or air-borne), zoonotic (animal-to-human) and anthropogenic (human-to-human) sources. Several modes of transmission are commonly recognized for infectious diseases: venereal, faecal-oral, aerosol, predator-prey, and vector-borne transmission.

When investigating outbreaks, epidemiologists have developed a number of widely accepted steps; these include:

- verify the diagnosis,
- confirm the existence of the outbreak above background levels,
- define inclusion criteria (what constitutes a case),
- conduct descriptive assessment (time, place, people),
- develop a hypothesis (causes?),
- test hypothesis (collect and analyse data),
- refine hypothesis as appropriate and carry out further study,
- develop and implement control and prevention systems, and
- release findings to greater community.

Mathematical epidemiological models

Biological systems are inherently complex, with many interactions occurring between their component parts, some conspicuous but most cryptic. Nonetheless, many attempts have been made to develop mathematical models to reliably monitor and predict disease distribution and abundance. Simulation models are vital to governments and public health agencies to prepare and respond to disease outbreaks through resource allocation, infrastructure development, preparedness training and public education. While such models have frequently been used in sciences such as physics and engineering, they have only relatively recently been applied to infectious diseases. This has necessitated the development of a specific vocabulary to define parameters as well as adopting a de-constructivist approach to identify integral components and mathematical relationships.

Parasite populations

Infectious disease models distinguish between 'microparasites' and 'macroparasites' *sensu latu*. Microparasites are infectious disease agents which reproduce directly, often at very high rates, within the host. They are generally small, have short generation times, and usually produce long-lasting immunity against re-infection. The duration of infection is usually short relative to the life span of the host, so infections are typically transient. On the other hand, macroparasites have no direct reproduction within the definitive host, producing transmission stages that pass from the host to complete their life-cycle. They are typically large and have longer generation times, which can often be a significant fraction of the host's life expectancy. Immunity tends to be of relatively short duration once the parasites are removed, and infections are often persistent, with hosts being continually re-infected. Thus, two types of models have been developed for host-parasite interactions:

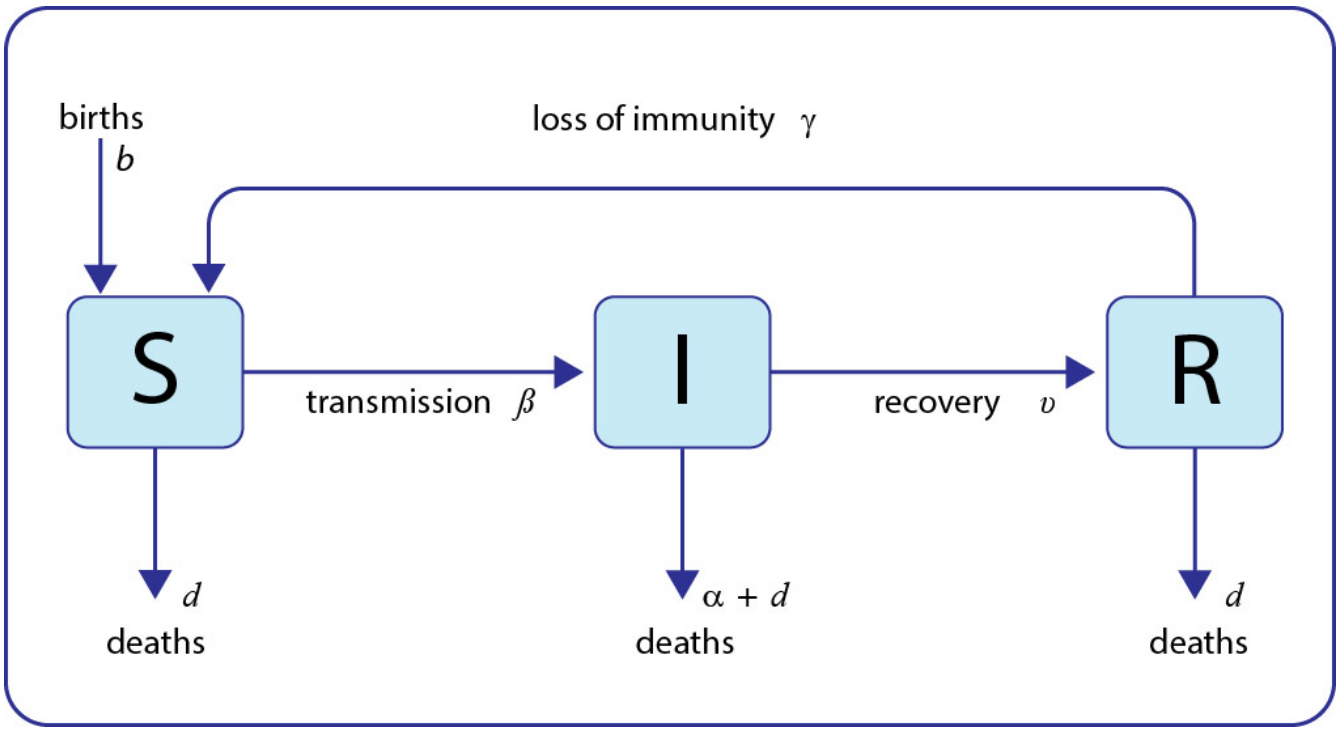
- Microparasite models (usually involving acute, transient, multiplicative infections)
- Macroparasite models (usually involving chronic, prolonged, cumulative infections)

Microparasite models

Microparasites, in the broadest sense, include viral, bacterial and protozoan pathogens. These micro-organisms reproduce quickly, reach very high intensities within individual hosts, cause acute transient infections (short duration compared to host life span), are often limited by host immune responses, and recovered individuals may develop protective immunity against re-infection.

Mathematical models developed for microparasites actually model changes in the number of infections (that is, number of hosts infected), rather than the number of parasites present. The SIR model classifies the total host population (N) into three subpopulations, containing susceptible (S), infected (I) or recovered/resistant (R) individuals, without accounting for within-host abundance/intensity.

Population dynamics of microparasites are driven largely by the rate of transmission between hosts (β), the rate of recovery of infected hosts (ν) and the rate at which any immunity is lost (γ). The model can also take into account changes in host populations, including births (b, all newborns being susceptible) and deaths due to natural causes (d) as well as deaths due to parasitic disease (α). A concept map of the SIR model is shown below.



[An interactive microparasite model is available via Populus (free download at www.cbs.umn.edu/populus/)]

Populations are quantitated as:

N = total host population (number)
S = susceptible host subpopulation
I = infected host subpopulation
R = recovered (immune) host subpopulation

Changes in SIR populations depend on host variables, namely:

b = host birth rate
d = host death rate (natural mortality)

Changes also depend on parasite variables, including:

α = disease-induced mortality rate
β = between-host transmission rate
ν = recovery rate
γ = rate of immunity loss

Movement between S, I and R states are modelled by a series of differential equations (DEs), whereby numbers change over time, that is, dS/dt, dI/dt, dR/dt. Such changes are said to be instantaneous (not discrete) rates of change. When there is no change in rate (for instance, $dS/dt = 0$), that population is stable; but when the rate is > 0 or < 0, the population increases or decreases, respectively.

The DEs differ according to whether transmission is density-dependent (DD) or frequency-dependent (FD). DD transmission considers that infections occur in direct proportion to the number of encounters between susceptible and infected individuals, which is simply the product of their densities (SI). FD transmission recognizes that mixing of the population is not homogeneous and that many individuals have limited numbers of contacts. The frequency of susceptible hosts (S/N) therefore determines the number of transmissions rather than the absolute density (S).

The DEs for the SIR-DD model are:

$$dS/dt = b(S+I+R) - dS - \beta SI + \gamma R \qquad \text{[in the SIR-FD model, } \beta SI \text{ is replaced with } (\beta SI)/N\text{]}$$

$$dI/dt = \beta SI - (\alpha+d+\nu)I$$

$$dR/dt = \nu I - (d+\gamma)R$$

The equations can be integrated to obtain the net reproductive rate of disease:

$$R_0 = (\beta S)/(\alpha+d+\nu)$$

Note the effects on R_0 due to:

- variation in parasite transmission [affecting the term βS ($\uparrow$ infections, $\uparrow R_0$)]; and
- the life span of infections [affecting the term $1/(\alpha+d+\nu)$ ($\uparrow$ mortality of host or parasite, $\downarrow R_0$)].

For disease to persist, R_0 must exceed 1, so we can set $R_0 = 1$ to get the threshold susceptible-host population density to sustain the parasite, that is:

$$S_T = (\alpha+d+\nu)/\beta$$

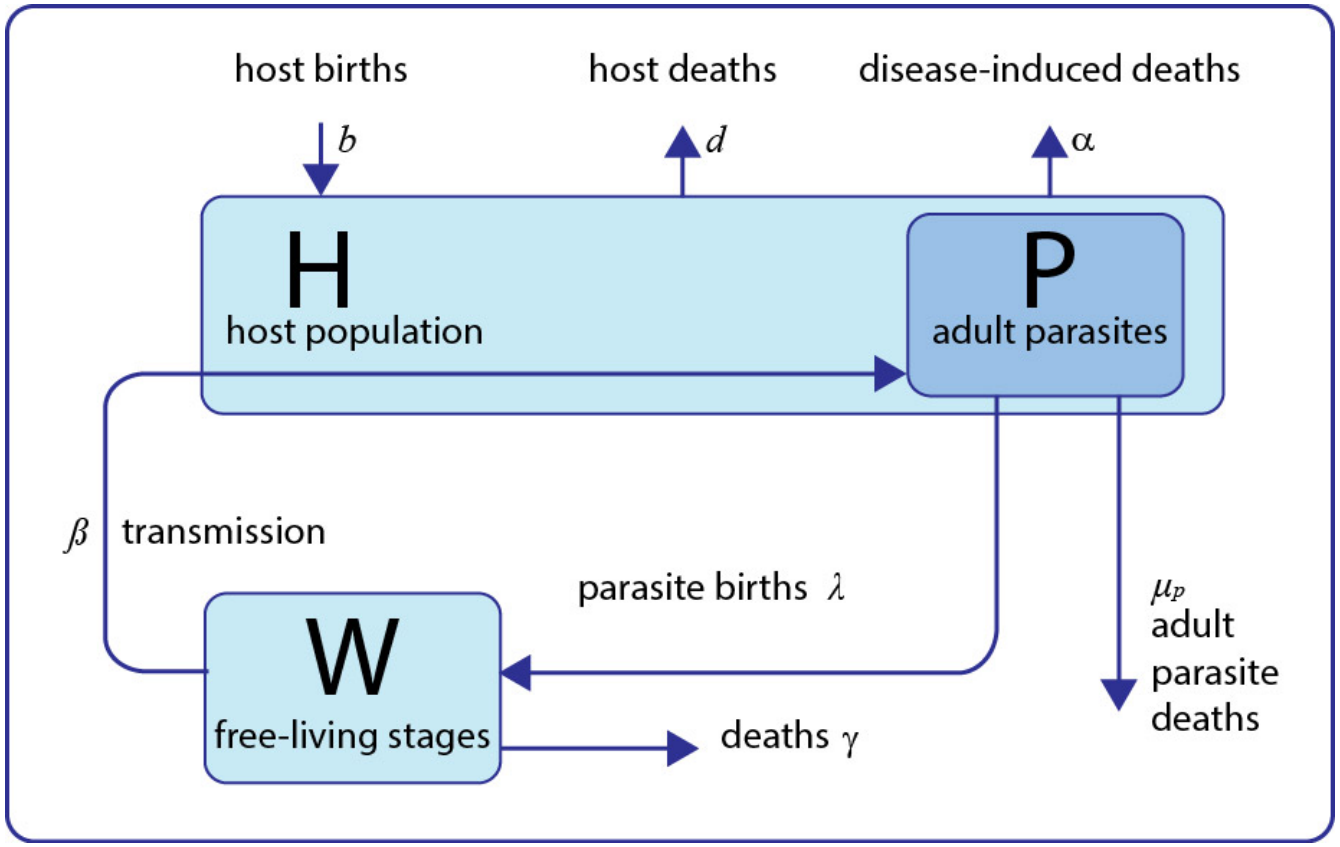
The implication is that the disease will go extinct unless $S > S_T$

Macroparasite Models

Macroparasites generally include helminths and arthropods which usually cause chronic and persistent infections. Disease severity depends on the number of parasites present which in turn depends on exposure to free-living infective stages. Infections are often over-dispersed, as a small fraction of the host population may harbour most of the parasites.

It is not sufficient to divide the host population into *S*, *I* or *R* classes, as macroparasite models must track the intensity of infection in individual hosts (often by assuming a probability distribution for parasite loads). Models take into account host population size (*H*) as well as populations of adult parasites (*P*) (inside hosts) and free-living stages (*W*) (outside hosts).

The Anderson & May (A&M) model characterizes between-host variation in parasite burdens with a negative binomial distribution. The decoupled A&M model is a simplified version whereby infective stages are assumed to be short-lived and at equilibrium. The Dobson & Hudson (D&H) model includes a hypobiotic stage of arrested parasite development following infection, before maturation of adult parasites affect host vitality. A concept map for the A&M macroparasite model with direct transmission is shown below:



[An interactive macroparasite model is available via Populus (free download at www.cbs.umn.edu/populus/)]

Parameters used in the model include:

H = total host population size (numbers)
P = total parasite population size (total numbers of adults)
W = population of free-living infective stages (eggs and larvae)

Host parameters include:

b = host birth rate (/time)
d = host death rate (natural mortality) (/time)

Parasite parameters involve:

α = disease-induced mortality rate (/parasite/time)
β = transmission rate per host contact (/host/time)
λ = birth rate of parasite eggs or larvae (/time)
κ = negative binomial aggregation parameter (a dimensionless constant inversely proportional to assumed parasite aggregation among hosts)
μ_P = natural mortality rate of adult parasites (/time)
γ = infective stage mortality rate (/time)

The DEs for movement between H, P and W states in the A&M model are:

$$dH/dt = (b-d)H - \alpha P$$

$$dP/dt = \beta WH - (\mu_P+d+\alpha)P - \alpha P^2/H[(k+1)/k]$$

$$dW/dt = \lambda P - \gamma W - \beta WH$$

The equations can be integrated to obtain the net reproductive rate of disease:

$$R_0 = (\beta\lambda H)/(\mu_P + d + \alpha)(\gamma+\beta H)$$

Note the effects on R_0 due to variation in:

- parasite fecundity [affecting the term $\beta\lambda$] ($\uparrow$ fecundity, $\uparrow R_0$);
- the life span of infections [affecting the term $1/(\mu_P + d + \alpha)$] ($\uparrow$ mortality of host or parasite, $\downarrow R_0$); and
- the life span of free-living stages [affecting the term $1/(\gamma+\beta H)$] ($\uparrow$ parasite mortality, $\downarrow R_0$).

Host and parasite populations may reach equilibrium if $\lambda - (\mu_P+d+\alpha) > (b-d).[(\kappa+1)/\kappa]$

If so, the parasite is capable of regulating host populations to an equilibrium where $P/H = (b-d)/\alpha$

If not, but $\lambda - (\mu_P+d+\alpha) > 0$, then hosts grow at an exponential rate (< disease-free rate).

If not, but $\lambda - (\mu_P+d+\alpha) < 0$, then the parasite cannot be maintained.

Parasite biodiversity and community structures

The distribution and abundance of parasites depends on many biotic and abiotic factors. Traditional measures of parasite biodiversity are species richness (number of species present), relative abundance (numbers present) and distribution (temporal and spatial). However, when examining host-parasite communities, other aspects must be considered: including host range (definitive, intermediate and/or paratenic hosts, including vectors) and host specificity (especially phylospecificity involving obligate, facultative, opportunistic, preferred or permissive hosts). Because parasites adversely affect their hosts, information is also required on their pathogenicity (infectivity, virulence) and immunological interactions (influencing host susceptibility/resistance).

All qualitative and quantitative measures of ecological indices are based on the recognition of individual species (both parasites and hosts), traditionally determined using a constellation of characters encompassing structural and biological variation (morphotypes, biotypes, culture requirements, viability, transmission, growth and developmental cycles) and more recently, genetic variation (subspecies, strains, variants, serotypes, zymodemes, genotypes, clades). If the traditional definition of a species is given as their genetic exclusivity (i.e. living in reproductive isolation), it is notable that the complete developmental cycles of many parasites is unknown, and few interbreeding experiments have ever been attempted. It is therefore often quite subjective as to whether any variation between isolates should be considered inter-specific (separating species) or intra-specific (within species). The recognition of individual species is essential for any quantitative ecological studies as all metrics are based on either prevalence (presence or absence) of parasite species within host species, their abundance or intensity (numbers of parasites present in individual hosts) or their relative abundance or relative intensity (mean values for populations).

Ecological studies may be conducted at several different levels: involving single or multiple parasite populations within single individual hosts, single host populations or multiple host populations. By definition,

- 'infra-population' studies involve one parasite species in one host individual,
- 'infra-community' studies involve all parasite species in one host individual,
- 'component community' studies involve all parasite species found in one host population, and
- 'compound community' (or metapopulation) studies involve all parasite species in all host species within a given area.

Analyses of prevalence and/or abundance data are used to reveal:

- patterns of dominance (core versus satellite species, prevalence-abundance interactions),
- dispersion and aggregation (variance to mean ratio (VMR) and derivatives, rank-abundance plots, species abundance models such as niche pre-emption),
- biodiversity complexity, involving species richness (contingency tables of co-existence, Jaccard's coefficient, Sorensen's coefficient),
- relative abundance influencing evenness and heterogeneity (Shannon's diversity index (H'), Simpson's diversity index (DI) and derivatives),
- host range and specificity (phylospecificity (Rohde's indices, Poulin & Mouillot's index), beta-specificity, phylobetaspecificity), and
- community structures based on similarity/dissimilarity matrices (covariance, nestedness, and cluster analyses).

Meta-population studies also allow observations to be made on:

- the type of diversity (alpha-, beta- or gamma-diversity),
- host-parasite relationships (co-speciation, host switching, ecological fitting), and
- environmental relationships (analyses of variance, correlation/regression analyses, principal components analyses, correspondence analyses involving biotic/abiotic factors such as season, location, climate, host demography).