

# Should ecological factors affect the evolution of age at maturity in freshwater clams?

MIKKO HEINO\* and VEIJO KAITALA

*Department of Ecology and Systematics, Division of Population Biology, University of Helsinki, PO Box 17, FIN-00014 Helsinki, Finland*

## Summary

We studied the evolution of age at maturity in freshwater clams of the genus *Anodonta* in relation to their ecology. We analysed an age-structured density-dependent population dynamics model, which we developed for freshwater clams, using several different options for density dependence. As evolutionary optimality criteria we applied both the maximization of a fitness measure (either intrinsic rate of increase or expected lifetime fecundity) and the concept of evolutionarily stable strategies (ESSs). All three evolutionary criteria yielded estimates which were too high for the optimal age at maturity in a deterministic model with a constant survival rate. The predictions are improved when size-selective predation is included in the model. Mature clams also face a risk of infection by castrating parasites, which would select for delayed maturity. Variable newborn survival selects for earlier reproduction, though the observed levels of stochasticity probably have a negligible effect.

*Keywords:* age-structured population dynamics; ESS; age at maturity; size-dependent mortality; freshwater clams; predation; castrating parasitism; stochasticity

## Introduction

Animals with indeterminate growth (e.g. fish, snakes, lizards, crayfish and freshwater clams) experience life history trade-offs between survival and reproduction and between reproduction and growth. Since reproduction requires energy, which is then not available for growth or maintenance, the age at maturity determines, at least partially, the lifetime growth pattern of an individual. On the other hand, the annual reproductive potential of an individual depends on its size. Thus, one can expect that age at maturity is one of the most important life-history traits. Furthermore, the optimal age at maturity may be affected by the environmentally determined overall growth potential and the age- or size-specific mortality or fertility patterns. In particular, the mortality or fertility patterns may be affected by ecological factors, such as intra- and interspecific competition and the risks of predation and castrating parasitism.

In this paper we study the evolution of age at maturity in animals with indeterminate growth. Our aim is to study the optimal age at maturity and its potential sensitivity, in relation to the environment and ecology of the species. The age- and size-dependent mortality patterns and the overall mortality in general are known to affect the optimal maturity (Stearns and Crandall, 1981; Roff, 1984; Kozłowski and Wiegert, 1986; Ferrière and Clobert, 1992; Kaitala and Getz, 1995). There is also field evidence for a relationship between mortality and maturity (e.g. Reznick and Endler, 1982; Lafferty, 1993; Jokela and Lively, 1995). However, the bulk of the literature concerns only organisms with determinate growth.

For the purposes of the analysis, we developed an age-structured density-dependent population dynamics model for freshwater clams, using several different options for density dependence. We

\* To whom correspondence should be addressed.

used data on two species of freshwater clams, the Western Palearctic *Anodonta anatina* (= *Anodonta piscinalis*) and the Nearctic *Anodonta grandis* (Haukioja and Hakala, 1978a; Hanson *et al.*, 1988; Bauer, 1994). No major differences in the life histories of these two species have been reported, except that *A. anatina* is a dioecious species, but *A. grandis* may also be hermaphroditic. The juvenile stage usually lasts 1–5 years after which the clams mature (Haukioja and Hakala, 1978a; Hanson *et al.*, 1989; Jokela, 1993). After maturation the individuals continue growing and reproduce annually. The reproductive cycle begins in summer when glochidia larvae are developed in the outer gill blades of the females (Jokela *et al.*, 1991). The females store and maintain the larvae over winter and release them in spring. They attach themselves to the fins and gills of common fish species, where they remain for some weeks, metamorphose and finally detach and drop to the bottom. The very high mortality in the early life of clams is counterbalanced by high fecundity (Hanson *et al.*, 1989; Jansen and Hanson, 1991; Bauer, 1994) and low adult mortality (Negus, 1966; Payne and Miller, 1989; Jansen and Hanson, 1991). The most important predator of adult clams is the muskrat, which is a highly size-selective predator (Hanson *et al.*, 1989; Jokela, 1993). Moreover, mature clams face a risk of infection by castrating parasites (Taskinen, 1992).

A comparison of the results from studies of the age at maturity in different animals is complicated by the fact that different measures are used as the criteria for evolutionary optimality. In order to evaluate the robustness of our results, in our analyses we applied and compared three different criteria for evolutionary optimality: first of all, the invasibility criterion related to the theory of evolutionarily stable strategies (ESSs) and then the maximization of a density-independent fitness measure, either the intrinsic rate of increase of a clone,  $r$  (derived from the Euler–Lotka equation of life-history theory) or the lifetime production of offspring,  $R_0$ . The relationships between these different approaches under density-independent and density-dependent conditions have recently been clarified by Mylius and Diekmann (1995).

The paper is composed as follows. In the following section the population dynamics model is constructed. In the third section we introduce the different fitness measures, the fourth section presents the results of the simulation studies and the discussion section concludes the paper.

### Modelling the population dynamics

We next developed a discrete-time population dynamics model for an age-structured population. We considered a population of animals with indeterminate growth that reproduce seasonally once a year. We assumed in particular that the newborn offspring can mature at any age between 1 year and the maximum age. We assumed that the individuals which matured at the same age are identical. In particular, they are of the same size. Reproduction is followed by an overwintering period, during which mortality occurs. Density dependence is assumed to affect either the fecundity, survival or growth potential.

The maturation strategy is defined as the age at maturity. It should be noted, however, that in our paper the strategy optimizing the age at maturity is equivalent to the strategy optimizing the size at maturity, since the size of the individual is completely determined by its maturation strategy and age. The reason for dealing with an age-structured model, rather than a size-structured model, is technical.

#### *Age structure dynamics*

Let  $k$  denote the index updating the reproduction–overwintering cycles and let the  $a$  subscripts denote the age class ( $a = 0, 1, \dots, a_{\max}$ ). Further, let the age at maturity be denoted as  $a_M$ . Assume that when reproduction begins during season  $k$  there are  $X_a(k)$  individuals in age class  $a$ . Let us denote the total number of females as

$$X_{\text{tot}}(k) = \sum_{a=1}^{a_{\text{max}}} X_a(k) \quad (1)$$

and let the total number of reproductive females be

$$X_{\text{mat}}(k) = \sum_{a=a_M}^{a_{\text{max}}} X_a(k) \quad (2)$$

*Offspring.* Assume that during reproductive season  $k$  the female parent population  $X_{\text{mat}}(k)$  produces a total of

$$X_0(k) = \sum_{a=a_M}^{a_{\text{max}}} \sigma_a X_a(k) \quad (3)$$

female offspring, where  $\sigma_a$  denotes the age-specific per capita reproductive potential of the mature females (the number of female young produced).

*Age class dynamics.* Assume next that an age-specific fraction,  $s_a$ , of the overwintering individuals at age  $a$  survive over the overwintering period following reproductive season  $k$  to be alive at the beginning of reproductive season  $k+1$ . Then, the number of the overwintered females is

$$X_a(k+1) = s_{a-1} X_{a-1}(k), \quad a = 1, \dots, a_{\text{max}} \quad (4)$$

Note that we assume no cost of reproduction in the form of reduced survival.

*Density dependence.* Let  $Y(X)$  denote the effect of the interaction between the individuals at a population density  $X$  on a fitness-associated trait such that the effect is identical in each age class. We assume that the scaling factor  $Y(X)$  is a continuous decreasing function of the population size  $X$  responsible for the density dependence

$$Y(X) = \frac{1}{1 + X/\beta} \quad (5)$$

where  $\beta$  is a niche capacity parameter that represents the population density at which the density-independent  $Y$  is reduced by one-half. The optimal strategy does not depend on the choice  $\beta = 1000$ , nor the definition of population size  $X$ , as long as the population dynamics reach an asymptotically stable equilibrium state. The list of fitness-associated traits affected by density dependence is given later.

#### *Size structure and reproduction*

The size of clams is usually reported in terms of length. Further, the annual length increment of juvenile clams is approximately constant. Therefore, the relationship between age and size (length) is modelled by Roff's (1983) growth equation based on energetic considerations:

$$L_0 = 0 \quad (6)$$

$$L_{a+1} = L_a + \Delta(L_a) \text{ for } a < a_M - 1 \quad (7)$$

and

$$L_{a+1} = (L_a + \Delta(L_a)) / \sqrt[3]{1 + G} \text{ for } a \geq a_M - 1 \quad (8)$$

where  $a_M$  is the age at maturity (that is, the age when the larvae are released),  $\Delta(L_a)$  is the maximum (length) growth potential at length  $L_a$  and  $G$  is the ratio of the glochidia weight to the weight of the rest of the body (analogous to the gonadosomatic index used in, e.g. Roff, 1983).

Equations 7–8 implicitly define the weight of offspring produced at age  $a$ . We next show explicitly how the fecundity depends on the parameters  $a_M$ ,  $G$  and  $\Delta$ . We assumed here that  $G$  is

constant (but see Heino and Kaitala, 1996), whereas  $\Delta$  may be constant or density dependent. The model has an asymptotic length at  $\Delta/(\sqrt[3]{1+G} - 1)$ , beyond which growth would be negative. We assumed, however, that negative growth will never take place. It follows that the allocation parameter  $G$  has an upper bound  $G_{\max} = [(L_a + \Delta)/L_a]^3 - 1$ . Equations 7 and 8 can be rewritten as

$$L_a = a\Delta \text{ for } a < a_M \quad (9)$$

and

$$L_a = \left[ \frac{a_M - 1 + \frac{1 - (1+G)^{\frac{a-a_M+1}{3}}}{1 - (1+G)^{1/3}}}{(1+G)^{\frac{a-a_M+1}{3}}} \right] \Delta \text{ for } a \geq a_M \quad (10)$$

The body weight of the animal is assumed to depend on the shell length as follows:

$$W_a = \alpha L_a^3 \quad (11)$$

where  $\alpha$  is a constant, the length is given in millimetres and the weight is given in milligrams. Note that this assumption was implicitly present in Equation 8 already. Thus, premature individuals increase their body weight as follows:

$$\delta W_{L_a} = \alpha [(L_a + \Delta)^3 - L_a^3] \quad (12)$$

where  $\delta W_{L_a}$  is the increase in the weight of a premature individual at size  $L_a$ . Mature individuals allocate to the weight increase as follows:

$$\delta W_{L_a} = \alpha \left[ \frac{(L_a + \Delta)^3}{1 + G} - L_a^3 \right] \quad (13)$$

The unrealized increase in weight, the energy for which is used in reproduction, is

$$\alpha [(L_a + \Delta)^3 - \frac{(L_a + \Delta)^3}{1 + G}] \quad (14)$$

The energy required to produce a unit weight of gonads is a constant proportion,  $c$ , of the energy required to produce a unit mass of body weight. Further, assume that the weight of the offspring is constant,  $w_0$ . Thus, the number of offspring produced at size  $L_a$  and released at size  $L_{a+1}$  is given by

$$\sigma_{L_a} = \frac{\alpha c G}{w_0(1 + G)} (L_a + \Delta)^3 \quad (15)$$

Substituting Equation 10 in Equation 15 yields

$$\sigma_a = \frac{\alpha c G \Delta^3}{w_0(1 + G)} \left[ \frac{a_M - 1 + \frac{1 - (1+G)^{\frac{a-a_M}{3}}}{1 - (1+G)^{1/3}}}{(1+G)^{\frac{a-a_M}{3}}} + 1 \right]^3 \quad (16)$$

## Evolutionary optimality

### *Density dependence and evolutionary stability*

We next introduce three different evolutionary criteria that we use in our study. Traditionally, the lifetime production of offspring is given as

$$R_0 = \sum_{a=a_M}^{a_{\max}} l_a \sigma_a \quad (17)$$

where  $\sigma_a$  is the number of female offspring for a female at age  $a$  (defined by Equation 16) and  $l_a$  is the probability of surviving from birth to the beginning of age class  $a$ , defined as

$$l_0 = 1 \quad (18)$$

and

$$l_a = s_0 s_1 \dots s_{a-1}, \quad a = 1, \dots, a_{\max} \quad (19)$$

The intrinsic rate of increase of a clone,  $r$ , is a parameter that is implicitly determined by the Euler–Lotka equation

$$1 = \sum_{a=a_M}^{a_{\max}} e^{-ra} l_a \sigma_a \quad (20)$$

An ESS is a strategy such that when it is common in the population, then no other strategy can increase (Maynard Smith, 1982). The evolutionary stability of a strategy depends essentially on the way the population is regulated (Mylius and Diekmann, 1995). Unfortunately, no information is available on how freshwater clam populations are regulated. Thus, we consider a few feasible alternatives.

(1) Fecundity: mature females experience intraspecific competition in such a way that their fecundity ( $\sigma_a$ ) is a decreasing function of the population density:  $\sigma_a(X_{\text{tot}}) = Y(X_{\text{tot}})\sigma_a(0)$ , where  $Y$  is given by Equation 5.

(2) Newborn survival: glochidia compete for suitable attachment sites in host fish, which leads to decreased survival ( $s_0$ ) when the number of glochidia is increased:  $s_0(X_0) = Y(X_0)s_0(0)$ .

(3) Overall survival: the population is regulated by predators resulting in reduced survival ( $s_a$ ) in a dense population:  $s_a(X_{\text{tot}}) = Y(X_{\text{tot}})s_a(0)$ .

(4) Growth potential: females experience intraspecific resource competition in such a way that their growth potential ( $\Delta$ ) is a decreasing function of the population density:  $\Delta(X_{\text{tot}}) = Y(X_{\text{tot}})\Delta(0)$ . Thus, both fecundity and growth are affected.

In general, when the lifetime production of offspring,  $R_0$ , depends on the population density, then the density-dependent  $R_0(X)$  is maximized at an ESS in stable populations (Charlesworth, 1994). Mylius and Diekmann (1995), however, posed a specific question whether the density-independent lifetime production of offspring,  $R_0(0)$ , could be used to determine an ESS under density-dependent conditions. Alternatively, could the density-independent fitness measure,  $r$ , be used in predicting evolutionary stability in the same way. In such a case, it would suffice to study the strategy options under ‘virgin’ density-independent conditions: determining the related steady state population levels would be unnecessary. They showed that this is possible provided some assumptions on the density dependence are fulfilled. In particular, if density dependence acts such that the lifetime production of offspring is reduced by a density-dependent multiplication factor, then maximizing the density-independent  $R_0(0)$  determines an ESS. Furthermore, if density dependence causes a uniform, age-independent increase in mortality, then maximizing  $r$  determines an ESS.

We have assumed above that the effects of density dependence are equal for all affected age classes, which is needed in invoking the results of Mylius and Diekmann (1995) on evolutionary stability. In cases (1) and (2) the lifetime production of offspring is reduced by a factor depending on population size  $X$ . If the mortality does not depend on size, this is also true in case (4). In these cases, the strategy maximizing the density-independent lifetime production of offspring,  $R_0(0)$ , is an ESS. Case (3) fulfils the requirement for a uniform increase in mortality, in which case an ESS maximizes the density-independent intrinsic rate of increase of a clone,  $r$ . We apply these results in the following section when determining ESSs.

If the mortality is size dependent, then the density dependence in the growth potential (case 4) will affect both the fecundity and mortality and the results of Mylius and Diekmann (1995) are not easily invoked. We have then sought ESSs by simulations. This was also the case when stochasticity was included in the model.

#### *Procedure for finding an ESS*

Equations 1–5 were used to simulate the dynamics of resident and mutant populations, except that the population size responsible for the density-dependent effect was the sum of the resident and mutant population sizes. The two populations are assumed to consist of similar individuals with the exception of the maturation strategy ( $a_M$ ). Prior to introducing one mutant into the first mature age class, the resident population was left to stabilize for 500 generations. The simulation was then run until either the mutant or the resident population became extinct; the threshold was set at  $10^{-3}$ . The resident strategy which was able to resist invasions of all mutants was an ESS.

## **Simulations**

### *Parameters*

In simulation studies we used data from two clam species: *A. grandis* in Canada and *A. anatina* in Finland. The parameters for the basic model were obtained from an *A. grandis* population studied by Hanson *et al.* (1988, 1989) and Jansen and Hanson (1991), which allows testing of our model.

The maximum age of *A. grandis* reported by Hanson *et al.* (1989) is 15 years, although no clams older than 12 years were caught alive. We assumed here that  $a_{\max} = 15$  years. The growth potential  $\Delta$  varies between different populations, reflecting environmental variation. We assumed here that  $\Delta = 10$  mm, which was the case in the population studied by Hanson *et al.* (1988, 1989).

Jansen and Hanson (1991) estimated that 0.007% of the glochidia produced successfully attach to perch, which is a host during the early parasitic stage of *A. grandis*, and that 0.0027% of the glochidia survive to age 2 years. We assume here that most of the mortality occurs during the first year, thus the survival probability of the newborn individuals to age 1 year is  $s_0 = 0.00003$ . For the other age classes we assumed that survival is either constant or state or size specific (the patterns are given later).

The estimates for  $c$ ,  $w_0$ ,  $G$  and  $\alpha$  are not available. From the data given in Hanson *et al.* (1988, 1989),  $G = 0.33$  and  $\alpha = 0.0025$  can be estimated. The glochidia of *A. anatina* and *A. grandis* are approximately the same size (Bauer, 1994); thus the value  $w_0 = 7.36 \mu\text{g}$  for *A. anatina* is used here (K. Mäntylä, unpublished). The conversion rate  $c$  is assumed to be 1.

### *Results*

*Maturation and growth rate.* The trade-off between age at maturity and the lifetime growth pattern, or between annual reproduction and growth, is a basic one since the reproductive capacity of clams is largely determined by their size. The effect of the age at maturity on the lifetime growth pattern and fecundity can be clearly seen in Fig. 1.

The freshwater clam life history presupposes a rather low mortality after the first year of life: for basic parameter values, the annual survival probability must be  $> 0.77$  for a viable population. This situation is not changed much in a more favourable environment. If the newborn survival increases 10-fold (to 0.0003), an annual survival probability of 0.54 is needed for older age classes. Doubling the growth capacity has approximately the same effect, while an increase in life span has almost no effect at all. The only published survival rates are consistent with these

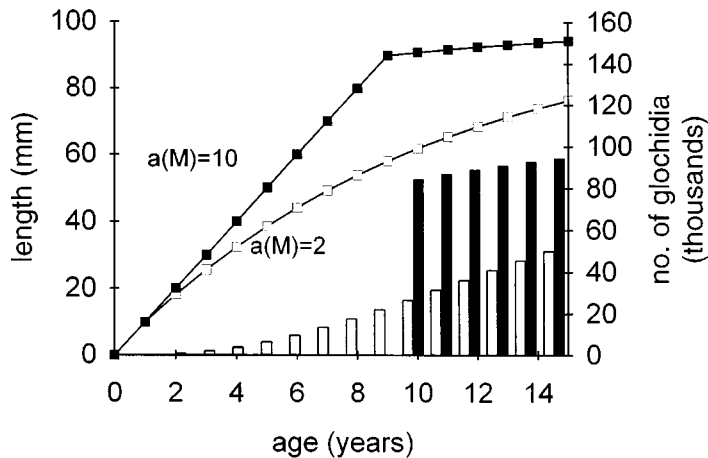


Figure 1. Growth patterns (lines) and fecundity (columns) for two contrasting maturity strategies. An early age at maturity (white) is associated with a long reproductive lifespan, but low fecundity. The opposite is true with delayed maturity (black).

considerations: for 5- to 8-year-old *A. anatina*, Negus (1966) gave values of between 0.61 and 0.86.

The importance of the timing of age at maturity is illustrated in Fig. 2. For  $s_a = 0.95$  and  $0.85$  ( $a > 0$ ), the maximum  $R_0$  is at 11 and 9 years of age, respectively. It appears that the age at maturity is a more important trait in a population with a high survival than with a low survival. The maximum  $r$  is independent of mortality at 7 years. Note, however, that nearby mutants have almost as high values of  $r$  as the one playing the optimal strategy. These results mean that the  $r$  criterion is less sensitive to the survival patterns after the first year of life than the  $R_0$  criterion. As a whole, the above results provide high estimates for the optimal age at maturity as compared with the empirical observations: Hanson *et al.* (1989) found that the *A. grandis* population matured at age 4–5 years.

Not only is age at maturity important, but also the allocation of the resources among growth, maintenance and reproduction. We have assumed that parameter  $G$  describes the allocation of the resources between growth and reproduction. We next attempted to evaluate the sensitivity of the results to the allocation parameter  $G$ . Figure 3 shows the  $R_0$  and  $r$  for  $s_a = 0.85$  ( $a > 0$ ) and  $G = 0.25, 0.33$  and  $0.5$ . The optimal maturation strategy appears to be rather insensitive with respect to the allocation parameter  $G$ . A decrease in  $G$  causes a slightly earlier optimal maturation; nevertheless  $G$  seems not to be responsible for the too high predictions of age at maturity. On the other hand, it appears from Fig. 3 that near the optimum the clam would increase its  $R_0$  and  $r$  by increasing its allocation to reproduction. This result holds until  $G_{\max}$  is reached and no growth after maturity takes place – a pattern not observed in nature. This problem is beyond the scope of this paper (but see Heino and Kaitala, 1996).

*Size-specific mortality.* Muskrats (*Ondatra zibethica*) can prey heavily on adult freshwater clams. In the recent studies of Hanson *et al.* (1989) and Jokela and Mutikainen (1995) a question was raised about whether size-dependent muskrat predation can cause evolutionary responses in clam populations. Although they both failed to verify any evolutionary responses it is interesting to look at how sensitive our model is to different patterns of size-selective predation. We confine

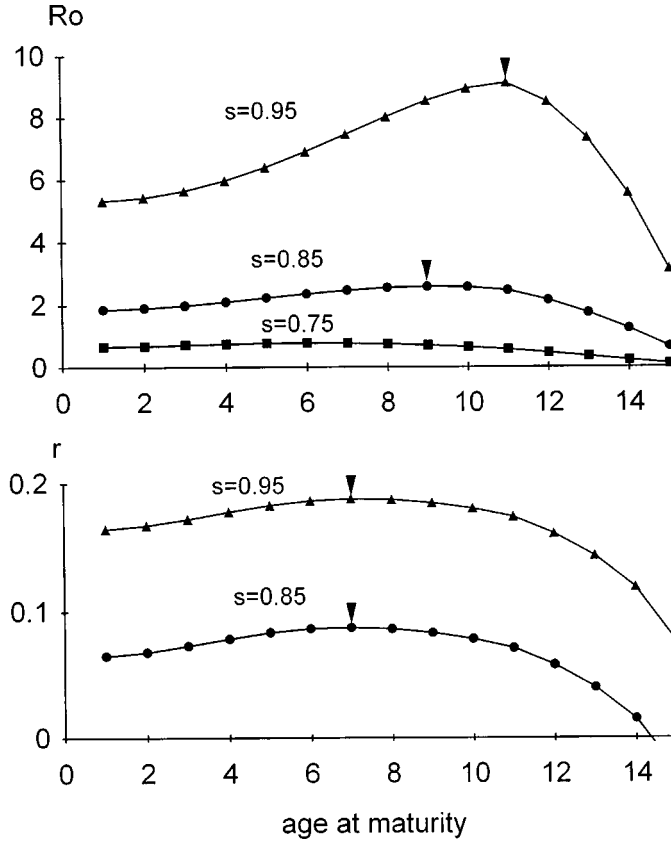


Figure 2.  $R_0$  and  $r$  as functions of the age at maturity. The evolutionary stable age at maturity corresponds either to the maximum (arrowheads) of  $R_0$  or  $r$ , depending on how the density dependence affects the population. The survival is assumed to be constant,  $s_a = 0.85$  or  $0.95$  for  $a > 0$ . The life history is not viable for  $s_a = 0.75$ .

our analysis to the age at maturity realizing that there exist many other forms of evolutionary responses which size-selective predation can affect.

Figure 4 shows the effect of size-specific mortality which increases gradually with size. The size-specific mortality was modelled with a logistic curve. The survival rates were calculated as  $s' = s(1 - p)$ , where  $s$  is the size-independent component of survival and  $p$  is the annual size-specific probability of being predated. Three different predation intensities were used. Regardless of how density dependence is acting in the clam population, the inclusion of size-dependent mortality in the model yields more realistic predictions of the age at maturity. However, density dependence affects the extent to which the predictions are affected: the ESS age at maturity is influenced most when density dependence affects fecundity or newborn survival. In the other two cases, the effect is less pronounced.

The parameters for the medium predation intensity were adjusted to give an approximately similar predation pressure to that reported by Hanson *et al.* (1989). When a 'medium' risk of predation is used, the maxima of  $R_0$  and  $r$  predict the age at maturity falling within the range they observed in the field, that is 4–5 years. A remarkable feature now is that the ESS age at maturity

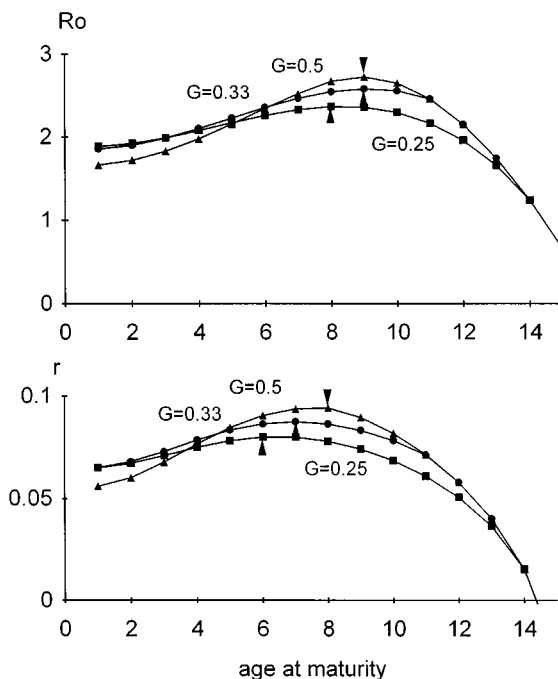


Figure 3.  $R_0$  and  $r$  as functions of the age at maturity for different values of  $G$  ( $s_a = 0.85$  for  $a > 0$ ). See Fig. 2 for other details.

under the density-dependent growth potential departs clearly from the age predicted by the maximization of  $R_0$ .

As a whole, our results show that the evolutionary effect of size-selective predation can be strong. The predictions can be tested empirically by comparing populations which differ in predation pressure. The testability does not depend on the survival rates under no predation. We emphasize that the age at maturity is only one trait under evolutionary pressure and that other types of evolutionary changes may occur under size-selective predation. Note also that if predation does not seem to explain the variation in the age at maturity among populations, then one should look at other possible ecological factors such as parasitism, which is considered next.

**Parasitism.** The castrating trematode parasite, *Rhipidocotyle fennica*, uses *A. anatina* as the first intermediate host (Taskinen, 1992). The parasite uses the energy allocated by the host to growth and reproduction but does not interfere with the host's maintenance energy. Although the trematodes do not necessarily kill their molluscan hosts, they commonly cause infertility. At the late stages of infection, the gonad tissue is almost entirely replaced by the parasite sporocysts and clams are unable to reproduce (Taskinen, 1992; Jokela *et al.*, 1993).

The decreased fertility does not differ much from the increased mortality from the reproductive point of view. Thus, modelling of the effect of the rate of parasitism on the age at maturity can be considered in terms of different mortality patterns. If the rate of parasitism does not depend on age or size, then increased infection rates can be modelled as the uniformly increased mortality rate throughout the lifetime. We showed above that high mortality rates lead to the evolution of an early age at maturity. On the other hand, if the parasites are size selective, then the problem

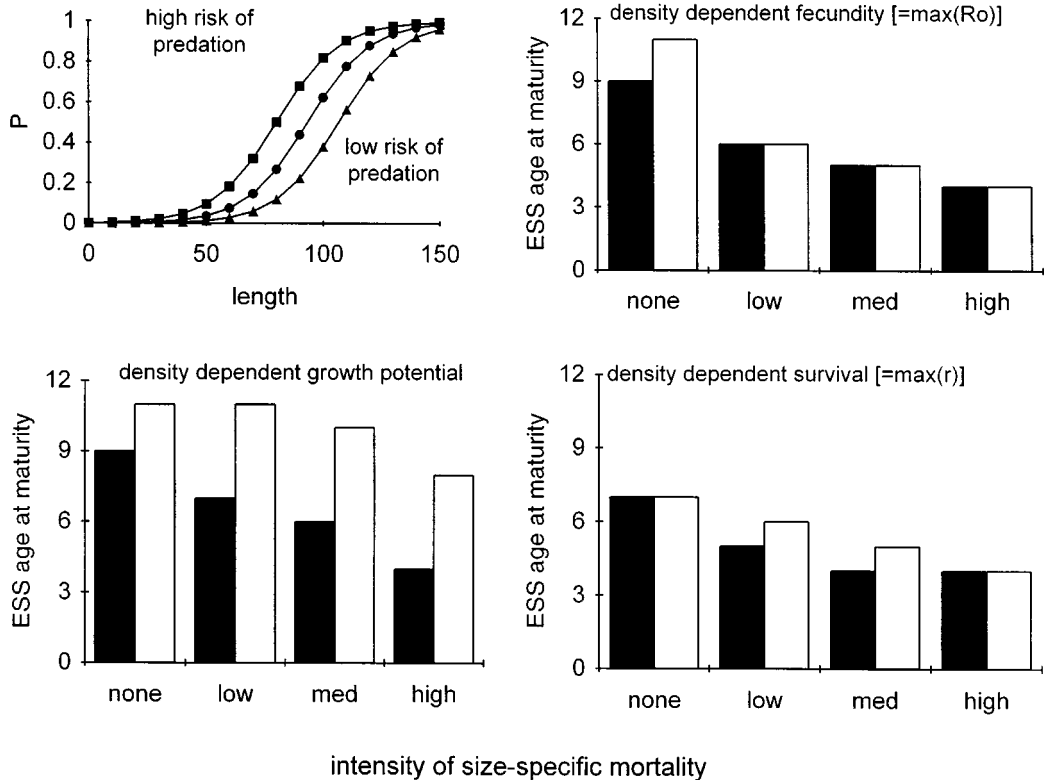


Figure 4. Optimal age at maturity under size-selective predation. The predation risks are given in the upper left panel; the dotted line illustrates the predation risk reported by Hanson *et al.* (1989). The other panels illustrate the optimal age at maturity under different assumptions about density dependence. The black and white columns denote the two levels of the size-independent component of mortality,  $s_a = 0.85$  and  $0.95$  for  $a > 0$ , respectively.

of age at maturity can be considered in terms of size-specific mortality. This was done above in the case of the size-selective predation of clams. In particular, it was shown that when the annual mortality increases, then the age at first reproduction decreases. The third option is that the risk of parasitism is stage dependent and this is considered next.

Taskinen and Valtonen (1995) found that only mature clams were infected with *R. fennica*. The prevalence of infection was 50% among mature females, which also includes the early stages of infection with insignificant effects on fertility. We assumed in our simulations that the annual survival is stage dependent such that the survival decreases from 0.95 to 0.85 or 0.75 after maturity; in other words, there is a survival cost of reproduction (Fig. 5). These rates correspond approximately to 10 or 20% of the annual risk of parasitic castration, respectively. The evolutionary response to such parasitic castration is delayed maturity (Fig. 5); this effect is more prominent in situations where  $r$  is maximized. However, the clam population Taskinen and Valtonen (1995) studied matured early (at age 2–4 years). They state that the risk of infection increases with both size and age, which seems probable in the light of the previous result. The alternative explanation is that immature clams may also become infected, but that the parasite remains inactive until its host becomes mature.

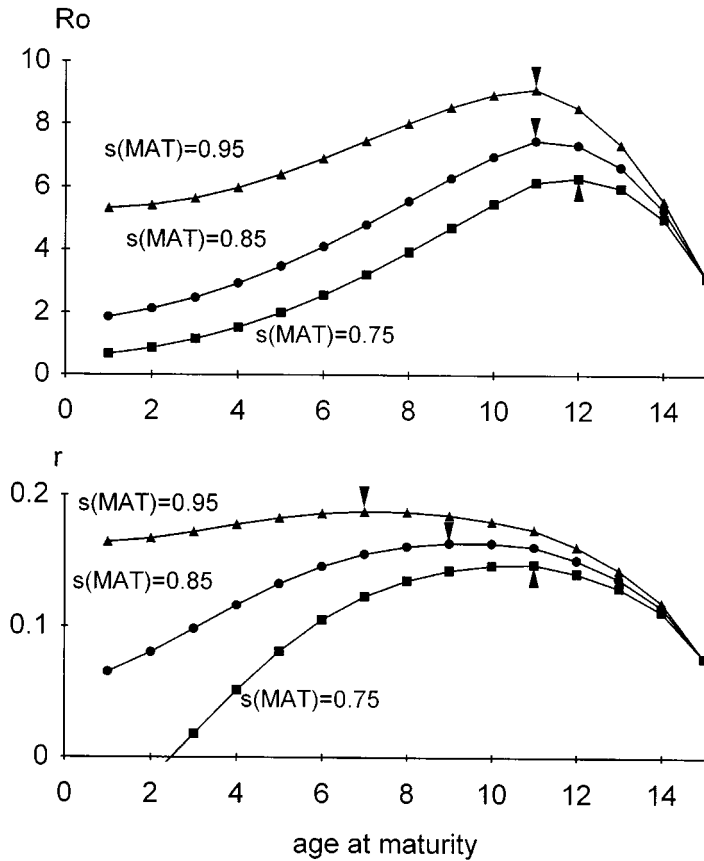


Figure 5.  $R_0$  and  $r$  as functions of age at maturity under stage-specific mortality or parasitic castration. The stage-specific mortality means that the survival declines from 0.95 to 0.85 or 0.75 after maturity. See Fig. 2 for other details.

*Stochasticity.* A high annual variation in recruitment is typical for freshwater clams (Negus, 1966; Haukioja and Hakala, 1978b; Hanson *et al.*, 1988; Payne and Miller, 1989). We next consider the stochastic variation in newborn survival. The stochasticity is introduced to newborn survival by multiplication with either a random variate with a uniform distribution on  $[0,2]$  (abbreviated  $U(0,2)$ ) or with a random variate  $e^{N(0,\sigma)}$ , where  $N(0,\sigma)$  denotes a normally distributed random variate with a zero mean and standard deviation  $\sigma$ . In the former case, neither the mean or median of  $s_0$  is affected, but in the latter case the mean will increase with  $\sigma$ . The simulations were repeated 20 times.

The evolutionary response to unpredictable newborn survival is earlier maturity and an increased reproductive lifespan (Fig. 6), although rather high variation is needed for any significant change. It is only in the case of density-dependent overall mortality and highly variable newborn survival, that there is a prominent decrease in the age at maturity. This response is partially caused by the increased mean newborn survival. For example, when  $e^{N(0,2)}$  reflects environmental variation, the mean newborn survival is 0.0026 (but the median is unchanged). In a deterministic environment with  $s_0 = 0.0026$  and a density-dependent overall survival, the ESS

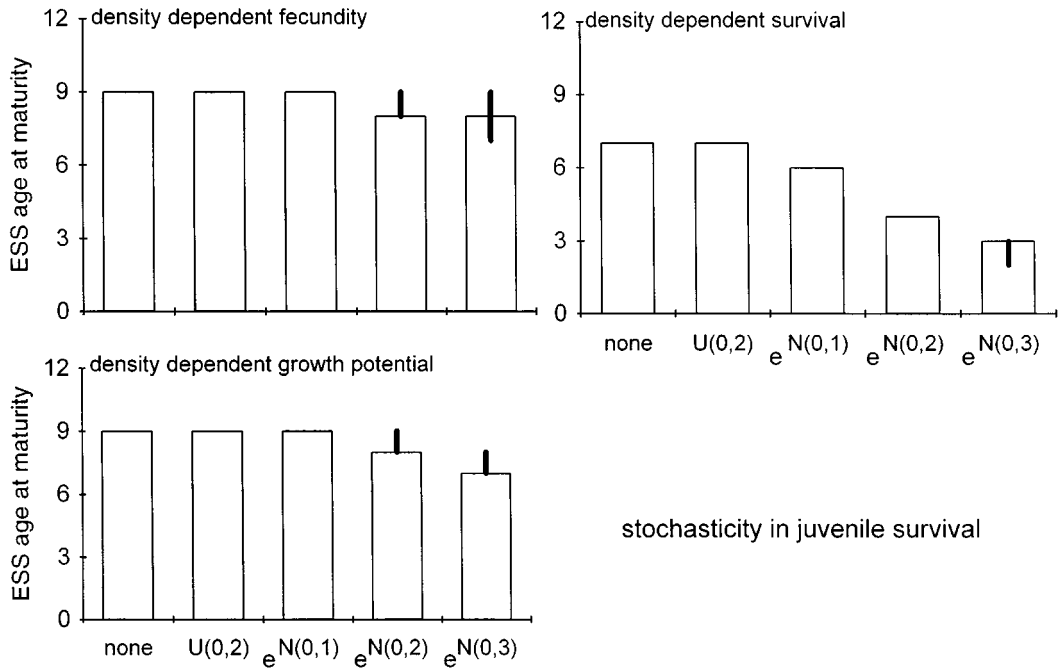


Figure 6. The evolutionary stable age at maturity when there is stochasticity in newborn survival. The variation increases from left (none) to right (coefficient of variation 18).  $U$  denotes the random variate with a uniform and  $e^N$  log-normal distribution. The vertical bars represent strategies that are occasionally able to outcompete other strategies.  $s_a = 0.85$  ( $a > 0$ ).

would be to mature at age 1 year. Thus, neither the mean or median value can be used to predict the outcome of selection.

The evidence of variable recruitment comes from variable age-class strengths. In our model,  $U(0,2)$  produces a similar or more variable age-class distribution to those reported in the studies of Negus (1966), Haukioja and Hakala (1978b) and Hanson *et al.* (1988). However,  $e^{N(0,1)}$  may be appropriate for the *Fusconaia ebena* population studied by Payne and Miller (1989). Although these levels of unpredictability do not cause any significant evolutionary change in our model, environmental stochasticity cannot be discarded as one potential explanation for a low age at maturity.

## Discussion

Hanson *et al.* (1989) failed to verify the hypothesis that slower growth rates have evolved as a response to predation in a clam population. Our model clearly indicates that predation may well have caused the age at maturity to have decreased, which in turn would be realized as decreased growth rates after maturation. However, it is probable that other traits also evolve in response to predation. In the *A. anatina* populations studied by Haukioja and Hakala (1978a), the age at maturity was low and varied only slightly. Furthermore, no significant correlations were observed between the age at maturity and other life-history traits. On the contrary, at a species level, freshwater clams display a substantial variation in the age at maturity, ranging from 1 year in

*Anodonta* to approximately 20 years in *Margaritifera* (Haukioja and Hakala, 1978a; Bauer, 1987).

In an evolutionary sense, parasitic castration is much the same as dying. A castrating parasite attacking only mature individuals would cause a potentially strong selective pressure for delayed maturity and reduced reproductive life span, possibly even to semelparity. However, for a castrating parasite, it is advantageous to also be able to infect immature individuals and to remain inactive until the gonads start to grow. In this case, the host cannot escape the risk of infection by delaying maturation; thus we would expect the evolutionary effects of parasitism to approach those results achieved under constant mortality.

Surprisingly, castrating parasites have received little attention by evolutionary biologists until recently. Minchella and Loverde (1981) were able to show an increased early reproductive effort in snails (*Biomphalaria glabrata*) when exposed to a castrating parasite. Lafferty (1993) and Jokela and Lively (1995) found negative correlations between the size at maturity and the prevalence of parasitic castration in two other snail species. Lafferty (1993) was also able to show that interpopulation differences were partly genetically determined. However, many other examples probably remain unnoticed. A possible one is the sticklebacks with a high parasitic load in Fuller Lake (McPhail and Peacock, 1983). The authors stated that the sticklebacks had not evolved in response to their parasites. However, it is possible that the 1 year life span of the sticklebacks is a response to parasites – much longer life spans are common elsewhere. Further empirical studies are needed to verify the presence of these patterns in other taxa and, on the other hand, to investigate which life-history traits are the most likely to evolve.

We assumed that all age-classes experience the density dependence in the same manner, which often is rather unrealistic. For example, population regulation through a uniform mortality increase in all age classes is rather implausible in animals with great size differences resulting from the indeterminate growth pattern. At least newborns often have a different feeding biology and predators from adults and are unlikely to face the same density-dependent processes as the adults.

The traditional way of maximization of either the density-independent  $r$  or  $R_0$  is consistent with the theory of ESSs when an appropriate assumption on density dependence is made. In our model, the optimal age at maturity according to the  $r$  criterion is less than or seldom equal to the age that is optimal according to the  $R_0$  criterion. Both criteria yield estimates which are too high for the age at maturity in the absence of size-dependent mortality, while the predictions of both criteria are more realistic when size-dependent mortality factors are included. In some cases the qualitative conclusions from the two evolutionary criteria may differ crucially. In our model, the maximum of  $R_0$  is independent of the newborn survival probability and glochidia weight and, in the case of size-independent survival, of growth potential as well. None of these is true for the maximum of  $r$ , which is only rather invariant to changes in the overall survival rates. Thus, when one of these factors is under study, the predictions depend on the fitness measure used.

Our model is fairly similar to that used by Roff (1983) to study optimal age at maturity in a flatfish (*Hippoglossoides platessoides*), except that rather than having fecundity as a part of the output of the model, he used an allometric fecundity function estimated directly from the field data. Ideally, these two methods should produce similar outcomes. In reality this is probably not the case and the latter, 'composite model' violates the energy conservation law, which may result in artefactual results.

Models with separate growth and fecundity functions have proved to be rather successful in predicting correlations between life-history parameters such as age at maturity, mortality and growth rate in fish (Roff, 1984; Mangel, 1996). It would be appealing to apply the biologically more realistic energy allocation model to such questions. However, the drawback of the energy

allocation model is that data on life-history parameters not traditionally reported in fisheries studies are needed.

In conclusion, our model shows that the age at maturity may be sensitive to increased mortality rates in animals with indeterminate growth, although other traits, such as reproductive effort, may evolve as well. Further empirical and theoretical work needs to be carried out to study the optimal allocation of resources to survival, growth and reproduction, when populations face different mortality patterns. Situations in which predation or castrating parasitism have favoured earlier maturation are likely to be more common than has been observed.

## Acknowledgements

We thank P. Abrams and T. Kawecki for commenting on earlier drafts and O. Diekmann and S.D. Mylius for providing a manuscript prior to publication. The constructive criticism of P.D. Taylor greatly improved the manuscript.

## References

- Bauer, G. (1987) Reproductive strategy of freshwater pearl mussel *Margaritifera margaritifera*. *J. Animal Ecol.* **56**, 691–704.
- Bauer, G. (1994) The adaptive value of offspring size among freshwater mussels (Bivalvia; Unionoidea). *J. Animal Ecol.* **63**, 933–44.
- Charlesworth, B. (1994) *Evolution in Age-structured Populations* (2nd edn). Cambridge University Press, Cambridge.
- Ferrière, R. and Clobert, J. (1992) Evolutionarily stable age at first reproduction in a density-dependent model. *J. Theor. Biol.* **157**, 253–67.
- Hanson, J.M., Mackay, W.C. and Prepas, E.E. (1988) Population size, growth, and production of a unionid clam, *Anodonta grandis simpsoniana*, in a small, deep boreal forest lake in central Alberta. *Can. J. Zool.* **66**, 247–53.
- Hanson, J.M., Mackay, W.C. and Prepas, E.E. (1989) Effect of size-selective predation by muskrats (*Ondatra zibethicus*) on a population of unionid clams (*Anodonta grandis simpsoniana*). *J. Animal Ecol.* **58**, 15–28.
- Haukioja, E. and Hakala, T. (1978a) Life-history evolution in *Anodonta piscinalis* (Mollusca, Pelecypoda). *Oecologia* **35**, 253–66.
- Haukioja, E. and Hakala, T. (1978b) Measuring growth from shell rings in populations of *Anodonta piscinalis* (Pelecypoda, Unionidae). *Ann. Zool. Fenn.* **15**, 60–5.
- Heino, M. and Kaitala, V. (1996) Optimal resource allocation between growth and reproduction in clams: why does indeterminate growth exist? *Funct. Ecol.* **10**.
- Jansen, W.A. and Hanson, J.M. (1991) Estimates of the number of glochidia produced by clams (*Anodonta grandis simpsoniana* Lea), attaching to yellow perch (*Perca flavescens*), and surviving to various ages in Narrow Lake, Alberta (Canada). *Can. J. Zool.* **69**, 973–7.
- Jokela, J. (1993) The selective environment of a freshwater clam: causes of selection and evolution of a life history strategy. PhD dissertation, University of Turku.
- Jokela, J. and Lively, C.M. (1995) Parasites, sex and early reproduction in a mixed population of freshwater snails. *Evolution* **49**, 1268–71.
- Jokela, J. and Mutikainen, P. (1995) Effect of size-dependent muskrat (*Ondatra zibethica*) predation on the spatial distribution of a freshwater clam, *Anodonta piscinalis* Nilss. (Unionidae, Bivalvia). *Can. J. Zool.* **73**, 1085–94.
- Jokela, J., Valtonen, E.T. and Lappalainen, M. (1991) Development of glochidia of *Anodonta piscinalis* and their infection of fish in a small lake in northern Finland. *Arch. Hydrobiol.* **120**, 345–55.
- Jokela, J., Uotila, L. and Taskinen, J. (1993) Effect of the castrating trematode parasite *Rhipidocotyle fennica* on energy allocation of fresh-water clam *Anodonta piscinalis*. *Funct. Ecol.* **7**, 332–8.

- Kaitala, V. and Getz, W.M. (1995) Population dynamics and harvesting of semelparous species with phenotypic and genotypic variability in reproductive age. *J. Math. Biol.* **33**, 521–56.
- Kozłowski, J. and Wiegert, R.G. (1986) Optimal allocation of energy to growth and reproduction. *Theor. Pop. Biol.* **29**, 16–37.
- Lafferty, K.D. (1993) The marine snail, *Cerithidea californica*, matures at smaller sizes where parasitism is high. *Oikos* **68**, 3–11.
- Mangel, M. (1996) Life history invariants, age at maturity and the ferox trout. *Evol. Ecol.* **10**, 249–263.
- Maynard Smith, J. (1982) *Evolution and the Theory of Games*. Cambridge University Press, Cambridge.
- McPhail, J.D. and Peacock, S.D. (1983) Some effects of the cestode (*Schistocephalus solidus*) on reproduction in the threespine stickleback (*Gasterosteus aculeatus*): evolutionary aspects of a host-parasite interaction. *Can. J. Zool.* **61**, 901–8.
- Minchella, D.J. and Loverde, P.T. (1981) A cost of increased early reproductive effort in the snail *Biomphalaria glabrata*. *Am. Nat.* **118**, 876–81.
- Mylius, S.D. and Diekmann, O. (1995) On ESS, optimization and the need to be specific about density dependence. *Oikos* **74**, 218–224.
- Negus, C.L. (1966) A quantitative study of growth and production of unionid mussels in the River Thames at Reading. *J. Animal Ecol.* **35**, 513–32.
- Payne, B.S. and Miller, A.C. (1989) Growth and survival of recent recruits to a population of *Fusconaia ebena* (Bivalvia: Unionidae) in the lower Ohio River. *Am. Midl. Nat.* **121**, 99–104.
- Reznick, D.N. and Endler, J.A. (1982) The impact of predation on the life history evolution in Trinidadian guppies (*Poecilia reticulata*). *Evolution* **36**, 160–77.
- Roff, D.A. (1983) An allocation model of growth and reproduction in fish. *Can. J. Fish. Aquat. Sci.* **40**, 1395–404.
- Roff, D.A. (1984) The evolution of life history parameters in teleosts. *Can. J. Fish. Aquat. Sci.* **41**, 989–1000.
- Stearns, S.C. and Crandall, R.E. (1981) Quantitative predictions of delayed maturity. *Evolution* **35**, 455–63.
- Taskinen, J. (1992) On the ecology of two *Rhipidocotyle* species (Digenea: Bucephalidae) from Finnish lakes. PhD thesis, University of Jyväskylä.
- Taskinen, J. and Valtonen, E.T. (1995) Age, size, and sex-specific infection of *Anodonta piscinalis* (Bivalvia: Unionidae) with *Rhipidocotyle fennica* (Digenea: Bucephalidae) and its influence on host reproduction. *Can. J. Zool.* **73**, 889–97.