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Individual variation in life history characteristics can influence extinction risk

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Abstract

The white sturgeon (*Acipenser transmontanus*) shows great individual variation in the age at maturation. This study examines the consequences of model assumptions about individual variation in the age at maturation on predicted population viability. I considered: (1) the effects of variation in age at maturation alone; (2) the effects of heritability; and (3) the influence of a stable and an altered selective regime. Two selective regimes represented conditions before and after the impoundment of a river, blocking access of anadromous white sturgeon populations to the ocean. In contrast to previous simulation studies, I found that increased individual variation in the age at maturity did not necessarily lead to a higher likelihood of persistence. Individual variation increased the simulated likelihood of persistence when the variation was heritable and the selective regime had changed such that the mean age at maturity was no longer optimal. © 2001 Elsevier Science B.V. All rights reserved.

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1. Introduction

The relationship between individual variation and population viability remains an unresolved question in ecology (Uchmanski, 1999). As a practical matter, we need to understand this relationship to decide when it is necessary to represent individual variation in models used to assess population viability (for example, Pfister, 1999). As a theoretical matter, this relationship is at the very interface between ecology and evolution.

Different sub-disciplines of ecology and evolutionary biology offer different perspectives on the question of how individual variation influences persistence. Most modeling studies, coming from an ecological perspective, suggest that individual variation improves the odds for population persistence (Rice et al., 1993; Conner and White, 1999). Yet conservation biologists claim that a related phenomenon, demographic stochasticity, increases extinction risk in very small populations (Fox, 1993; Lande, 1993). Those coming from an evolutionary perspective suggest that heritable variation can have either positive or negative population-level consequences, depending on the selective context (Lande and Shannon, 1996).

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This study addresses three questions. First, how does individual variation influence persistence when the variation is not heritable? Second, how does heritability influence the relationship between individual variation and persistence? Finally, what is the role of selection in mediating the relationship between individual variation and population persistence? These three questions are evaluated in the context of white sturgeon populations before and after impoundment of their river habitat using an individual-based model.

1.1. The role of individual variation

Most theoretical studies coming from an ecological perspective conclude that populations made up of uniform individuals are more extinction-prone than populations made up of variable individuals (Rice et al., 1993; Conner and White, 1999; Uchmanski, 1999). In the simplest case, an extreme phenotype has the highest fitness, and the distribution of fitnesses is such that the initial presence of at least some individuals with high fitness is enough to make the difference between population growth or decline. For example, Conner and White (1999) found that increasing the simulated individual heterogeneity in birth and death rates increased the probability of persistence of small populations.

On the other hand, it is well known that demographic stochasticity, when defined as chance variation in demographic traits present in a population, increases extinction risk in small populations (Goodman, 1987). Kendall and Fox (2001) suggested that demographic variation should be interpreted as individual variation in demographic traits, rather than as sampling error. In contrast to the results obtained by models that assign identical survival probabilities to individuals, Kendall and Fox (2001) showed that small populations had lower extinction risks when individuals were assigned different probabilities of survival. They concluded that the effect of variation in a given trait depends on the relationship between its mean and variance.

The population-level benefits of initial individual variation can be greatly amplified if a phenotype with an initial fitness advantage is able to develop into a phenotype that is even more extreme and more fit. This phenomenon has been demonstrated in the growth of individuals belonging to species that are able to capitalize on an early growth advantage. Allometric growth (DeAngelis et al., 1993; Rice et al., 1993; Pfister, 1999) or dominance hierarchies (Lomnicki, 1988) can amplify differences in size. When individuals differ in their abilities to acquire resources, populations are able to persist at much smaller population sizes. If resource levels become low enough that it threatens the population, a small number of dominant individuals can monopolize those resources and survive (van Noordwijk, 1994). Uchmanski (2000) analyzed four resource allocation strategies: even distribution, scramble competition, contest competition, and resource monopolization. He found one apparent discrepancy from the rule that persistence times increase as resource allocation become uneven: resource monopolization resulted in shorter extinction times as the number of dominant individuals increased, probably because the distribution of resources became more even again. The specific example of growth is unusual because of this positive feedback between size and growth that amplifies the population's variability in individual sizes. When combined with density-dependent selection favoring individuals of large size, it is easy to see how higher population persistence can result.

The first question addressed by this study is 'How does individual variation influence persistence?'. I focused on a fish with an extreme and variable life history. The white sturgeon, (*Acipenser transmontanus*), the largest fish in North America, is long-lived and iteroparous. Because individuals of this species vary greatly in the age at maturation, I focused on the role of individual variation in this trait.

1.2. The role of genetic inheritance

Evolutionary theory suggests that the ability to respond to selection should increase a population's ability to persist. Does this imply that we need to understand the genetic underpinnings of

traits to predict population persistence in population viability analysis (PVA)?

To address this, the second question addressed by this study asks, 'How does heritability influence the relationship between individual variation and population persistence?'. In this study, I simulate heritable variation in the age at maturation for a range of heritabilities. This required a modeling approach that could represent the genetic underpinnings of age at maturation.

Before the advent of high-powered computing, three classes of analytical models were tractable for predicting the evolution of phenotypic traits: (1) simple analytical models involving small numbers of loci and alleles; (2) game theoretic models that assumed phenotypic optimality without regard for genetic underpinnings; and (3) quantitative genetic models that assume a normal distribution of trait values produced by many loci and/or alleles. For the specific life history trait considered here, age at maturity, game theory has been used to find an optimal age and to study delayed reproduction in constant and variable environments (Tuljapurkar, 1990; Calsina and Cuadrado, 2000).

As simulation has become more feasible, individual-based models are more frequently used, and they now provide an intuitive basis for merging genetics with demography (Chambers, 1993). To date, individual-based genetic models have been used in theoretical studies, including studies to evaluate the importance of mutation (Lynch and Gabriel, 1990), the size-selectivity of fishing pressure (Martinez-Garmendia, 1998), and the effects of predator–prey co-evolution on their dynamics (Haefner and Dugaw, 2000). In this study, an individual based model was used to evaluate the role of individual variation in age at maturation by representing the genetic underpinnings of this trait. Because the genetic model developed here is capable of representing traits controlled by an intermediate number of loci, it bridges the gap between simple one-locus, two-allele models and quantitative genetic models.

1.3. The role of selection

Evolutionary theory suggests that the effect of individual variation on persistence must depend on the selective regime. Lande and Shannon (1996) found that genetic variation had different effects on population persistence under different selective regimes. This has important implications for PVA, because it suggests that we must simulate the genetic underpinnings of fitness-related traits. Although PVA models sometimes include a genetic component to address genetic risks, they typically focus on random genetic drift and not selection (Lacy, 1993; van Noordwijk, 1994). This assumption of selective neutrality is not appropriate for ecological applications that involve anthropogenic changes in the environment that are likely to impose a strong and directional selective force (Lynch and Lande, 1993). To understand how natural populations will fare in the face of anthropogenic shifts in the environment, it may be important to know the potential for a selective response, if not to predict the response. The evolution of life history traits in a changing environment has practical implications in pest control, climate change, and other areas of ecological research (Kareiva et al., 1993). For example, many fisheries scientists contend that size-selective fishing mortality has resulted in the evolution of an earlier ages at maturation (Rowell et al., 1989; Reznick et al., 1990; Rodd and Reznick, 1991; Miller and Kapuscinski, 1994; Harris and McGovern, 1997; Martinez-Garmendia, 1998).

The final question addressed by this study is 'What is the role of selection in mediating the relationship between individual variation and population persistence?'. To explore the role of selection, this study compares two hypothesized selective regimes for white sturgeon in the Columbia River basin. Historically, white sturgeon followed an anadromous life history, periodically moving into large rivers to spawn and returning to estuaries along the Pacific coast. The selective regime of this species has been altered by dam-building activities that blocked most populations from the ocean. This final question is addressed by comparing the relationships between individual variation and persistence for a pre-impoundment and a post-impoundment selection regime.

2. Case study: white sturgeon in the Snake River

2.1. White sturgeon demography

In this study, I use a model to evaluate the effect of individual variation, both inherited and not, on the likelihood of persistence of land-locked white sturgeon populations. The white sturgeon (*Acipenser transmontanus*; Fig. 1) is perfectly suited as a model for exploring this relationship for three reasons. First, this species represents a class of fishes following a life history that is highly vulnerable to extinction. Anadromy, dependence on large rivers, large body size, and delayed maturation all contribute to the white sturgeon's vulnerability (Parent and Schrimi, 1995). Second, the selective regime experience by white sturgeon in the Columbia and Snake rivers has been changed by dam-building activity. Historically, adults migrated long distances from estuaries on the Pacific coast of North America into large river systems to spawn, returning afterward to the estuarine environment (Kohlhorst et al., 1989; Warren and Beckman, 1993; Chapman et al., 1996). Sequential development of hydroelectric projects progressively isolated white sturgeon populations between dams. This transition reduced access to a variety of habitats by converting free-flowing habitat to reservoir. In addition, regulated flows adversely affect reproduction by reducing spring flows associated with larger year classes (Artyukhin, 1978; Kriksunov and Mamina, 1995; Auer, 1996).

2.2. White sturgeon genetics

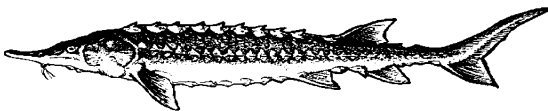The white sturgeon is an ancient species that belongs to the order Acipenseriformes that is thought to have resulted from adaptive radiation following a tetraploidization between 200 and 300 Myr ago (Birstein and Vasiliev, 1987). The number of chromosomes in the karyotype of this fish has been estimated variously between $2n = 240$ and 276 (Van Eenennaam et al., 1998). Its genome is now believed to be functionally diploid (Van Eenennaam, 1997), i.e. copies of loci have been 'silenced' and have no effect on the phenotype expressed. Birstein and Vasiliev (1987) suggested that Acipenseriformes have a mechanism that controls functional diploidization of the genome by compensating for the unusual number of active nucleoli.

Sturgeons exhibit relatively low genetic diversity (Birstein and Vasiliev, 1987). It has been observed that impounded populations of white sturgeon tend to have lower genetic diversity than populations with access to the ocean (Brown et al., 1992b; Bartley et al., 1995). Brown et al. (1992a) suggested that selective pressures associated with impoundment may be responsible for reducing genetic variation in landlocked populations.

In this study, I focus on one trait: age at maturity. The white sturgeon shows extremely high variation in demographic traits such as age at maturation. Females reach maturity between the ages of 13 and 34 years (Semakula and Larkin, 1968). The genetic basis for this trait has not been determined. Although the heritability of age at maturity in white sturgeon is unknown, individuals fed ad libitum rations mature much earlier than wild fish; usually by 5 years. Food availability is therefore one environmental factor that has a considerable influence on the trait. Although little is known about the genetic basis for age at maturity in white sturgeon, quantitative genetic studies have evaluated this trait in other species. Gjerde and Schaeffer (1989) reported a heritability of 0.42 for age at maturity for the semelparous Atlantic salmon and 0.14 for rainbow trout. These values fall within the range reported for life history traits of 0.1–0.5 (Roff, 1997, p. 65). To explore the effect of heritability, I compared simulated heritabilities (narrow-sense = broad-sense), h^2 between 0.0 and 0.55.

Fig. 1. The white sturgeon (*Acipenser transmontanus*). (Drawing by Kelly Lepla.)

3. A PVA model for white sturgeon

3.1. Demographic model

The demographic white sturgeon PVA model simulates the growth, reproduction, and mortality of individuals (Fig. 2A). The individual-based population model operates on an annual time step. During each time step, model sturgeon are exposed to two events: reproduction (Fig. 2B) and death. Each of these events is simulated as described in the following.

3.2. Reproduction

Simulated reproduction has four components. First, the model identifies a pool of eligible spawners each year. Second, spawning aggregations form. Third, the fecundity of each female that successfully spawns is determined. Finally, the model simulates the inheritance of parental genetic material by offspring.

The pool of eligible spawners includes all mature adults that reach reproductive readiness in a given year. The timing of reproduction is determined by the age at first maturity and the interval between spawning events. The model assigns these two reproductive parameters to individual fish from specified normal distributions. For females (males), the mean age at maturity is 18 years (14 years). In simulations with individual variation, the standard deviation in age at maturity was 1.5 years. Similarly, the average interval between spawning attempts was 5 years (2 years) for females (males), with a standard deviation of 0.8 years (0.3 years), respectively.

White sturgeon are broadcast spawners that form mating aggregations during spring. The model simulates one aggregation in each river segment. Density-dependent limits are imposed on the density of female spawners allowed to join the mating aggregation. I calculate the maximum number of spawning females as the product of the maximum density per kilometer of river (100 km^{-1}) multiplied by the length of the river segment (50 km). When the ratio of males to females is high, it is unlikely that excess males will fertilize eggs (Domeier and Colin, 1997). Therefore, the model limits the sex ratio of spawning aggregations (< 5 males per female) by randomly excluding excess males.

The fecundity of adult sturgeon increases with size (Cochnauer et al., 1985; Chapman et al., 1996; Van Eenennaam et al., 1996). The model estimates the fecundity of each female in two steps. First, female length is simulated according to a Von Bertallanfy relationship with parameters values estimated for the Snake River. Next, the model predicts fecundity from female length with parameter estimates from DeVore et al. (1995).

After simulating egg production by females in the spawning aggregation, the model simulates the inheritance of alleles by offspring. All males in the spawning aggregation are equally likely to fertilize a given egg. At each locus, one allele is drawn from each parent at random. After offspring inherit alleles from each parent, mutation can occur.

3.3. Death

Simulated mortality is relatively simple. Annual survival is lowest during the first year, increasing from zero at high egg densities ($10^{5.5}$ eggs) to a maximum of $5 \times 10^{-5}\%$ at low egg densities. Annual survival is substantially higher for juveniles and adults (81%). The model simulates random catastrophes that reduce the population by 90%. Catastrophes occur at an average frequency of one every 100 years. In addition, the experiment to address the role of selective regime simulates mortality associated with spawning migration as described in Section 4.

3.4. Genetic model

Here, I describe the genetic components of the model (Fig. 2B). I represent the deviation from mean age at maturation ($Z - \mu$) as the combination of a genetic (Z_G) and an environmental (Z_E) component:

$$Z = \mu + Z_G + Z_E \qquad (1)$$

At the start of the simulation, I specify a gender-specific mean, μ, and standard deviation, σ in the trait (e.g. age at maturation) that applies to
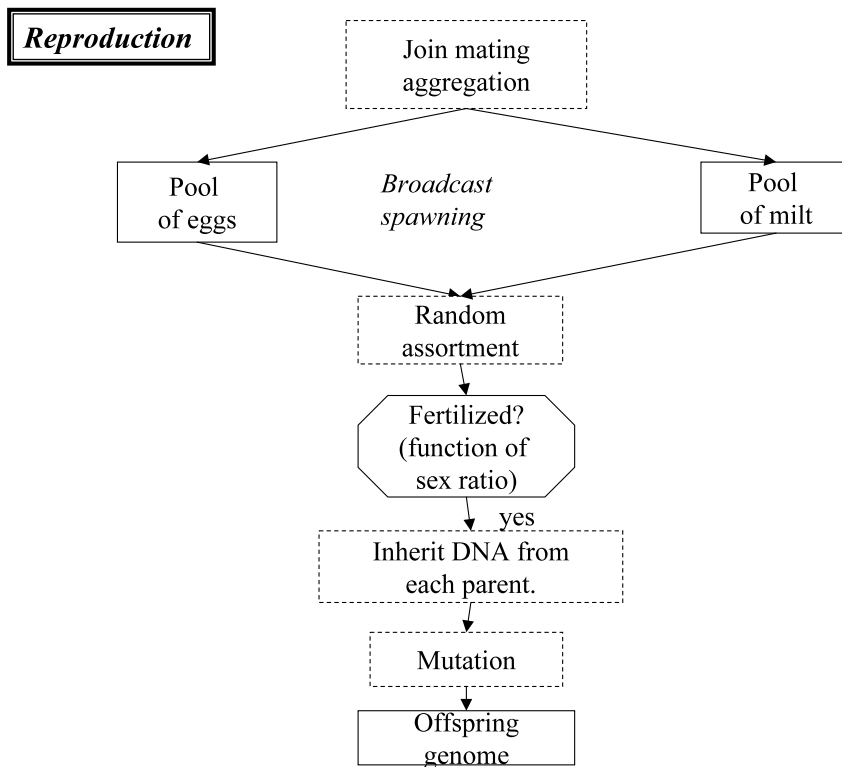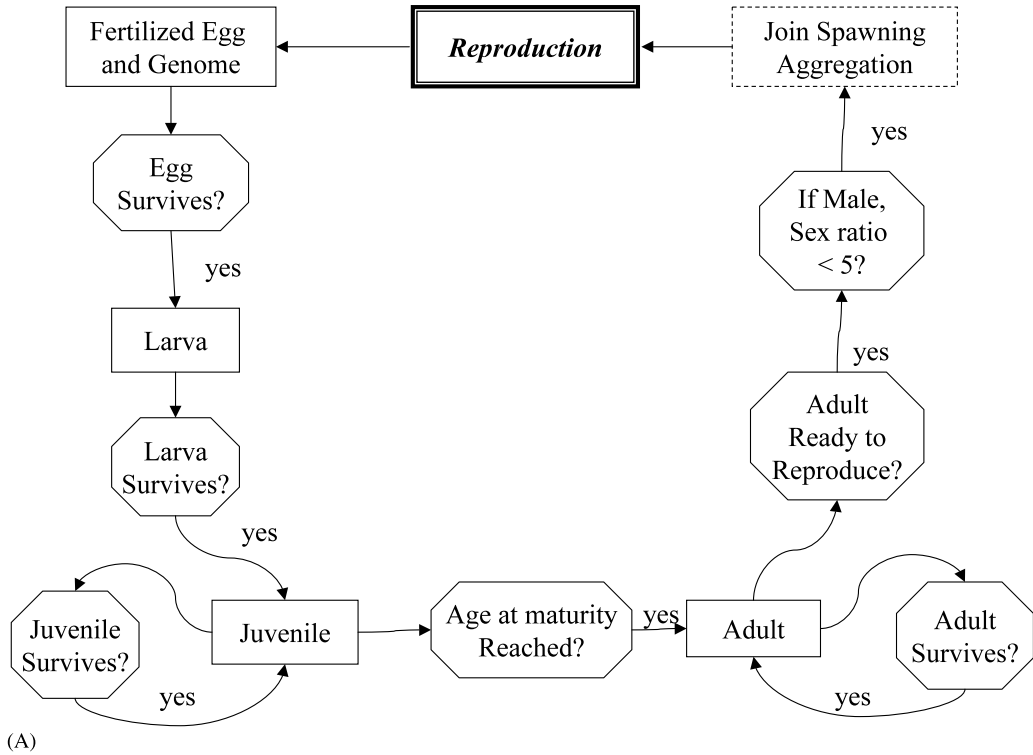
Fig. 2. Flow chart diagram of (A) the demographic individual-based model of white sturgeon and (B) the genetic sub-model.

the initial population. Heritability of each trait, h^2, is also specified. I assume that the environmental component is random normal with mean zero and variance $\sigma^2 (1 - h^2)$. The genetic component, following Bulmer (1980) and others, represents additive effects, X, from $M = 10$ loci. Because the white sturgeon genome is considered to be functionally diploid, each parent (represented by one term) contributes $N = 1$ allele for each locus. The identity of each contributed allele, k, is $A(k)$. For each individual, $Z_G = \sigma\, g$, where

$$g = \frac{1}{\sqrt{NM}} \left\{ \sum_{i=1}^{M} \left(\sum_{k=1}^{N} X_{A(k)} \right) + \sum_{j=1}^{M} \left(\sum_{k=1}^{N} X_{A(k)} \right) \right\} \quad (2)$$

Because the X_A are standard normal, Z_G has variance $\sigma^2 h^2$ at the start of the simulation. This model makes the simplifying assumptions that genetic and environmental contributions to the trait are independent, and that genetic variation contributing to the trait is additive.

3.4.1. Initialization

At the start of the simulation, I draw the effects of each allele on each trait from a standard normal distribution. This is carried out for all possible alleles, where a large, but finite, maximum possible number of alleles is specified. In these simulations, I assumed an upper limit of 32 possible alleles. However, only a smaller subset of alleles is initially assumed to occur at each locus in the white sturgeon population. Each individual in the initial population draws a vector of alleles at each locus from a multinomial distribution of initial alleles, where all alleles are equally likely to be selected. In these simulations, I initialized the populations with eight alleles, each with a frequency of 0.125. The remaining 24 alleles may enter the population later through the process of mutation.

I verified that the scenarios produced initial populations with the same, specified, phenotypic means and variances for heritabilities ranging from 0 to 1. I found that simulations could produce initial populations with average trait values that deviated significantly from the specified mean, particularly when the number of alleles and loci was small. Differences in extinction occurred because favorable initial trait values happened to

be present in some, but not other populations. To correct this, the model now calculates the realized trait mean in the initial population and subtracts the mean to remove bias from all allelic effects.

3.4.2. Mutation

Mutation is the source of new genetic information for simulated populations. Mutation rates for white sturgeon have been reported to be low (Birstein et al., 1997). I adopted a stepwise mutation model (Kimura and Ohta, 1978) with a mutation frequency $\mu = 10^{-5}$ per locus and generation. Each mutation results in a one-unit increase or decrease in the allele index. Because allele indices are not ordered by effects on any trait, the allelic effect resulting from mutation is unrelated to the effect of its wild precursor.

4. Simulation experiments

I used the white sturgeon model already described to address the following questions.

1. How does individual variation in age at maturation influence the likelihood of persistence?
2. Does heritability change the relationship between individual variation and the likelihood of persistence?
3. How do shifts in the selective regime influence the relationship between individual variability and persistence?

My design varied each of these three factors (individual variation, heritability, and selective regime) as shown in Fig. 3. Two selective regimes are described in the following, each with a no-variation scenario and a range of scenarios with realistic individual variation that differ in heritability. For each scenario, I simulated 100 replicate populations for 1000 years. The likelihood of persistence to 1000 years is defined as the fraction of replicate populations that had at least one male and one female after 1000 years.

4.1. Initial conditions

The initial populations had densities of 50 individuals km^{-1} in a 50-km reach, distributed according to an exponential age distribution with a
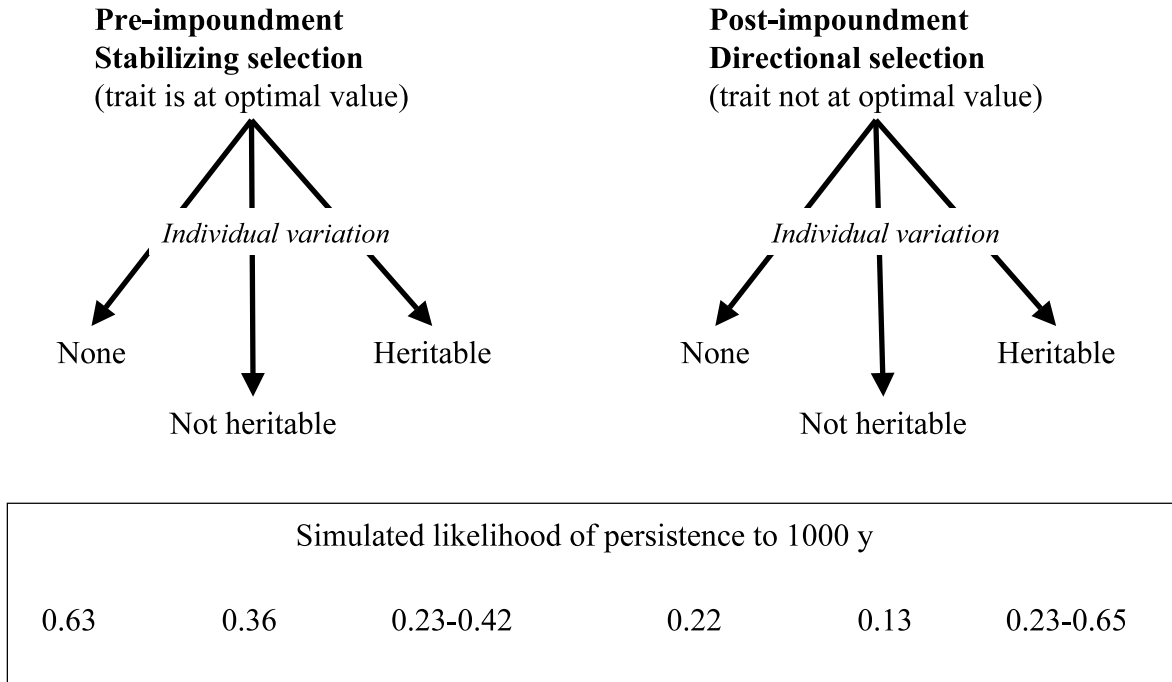
Pre-impoundment
Stabilizing selection
(trait is at optimal value)

Post-impoundment
Directional selection
(trait not at optimal value)

Individual variation   *Individual variation*

None   Heritable   None   Heritable

Not heritable   Not heritable

Simulated likelihood of persistence to 1000 y					
0.63	0.36	0.23-0.42	0.22	0.13	0.23-0.65

Fig. 3. The effects of (1) individual variation in age at maturity, (2) heritability, and (3) selective regime on population persistence: design of simulation experiments and results.

mean age of 8 years. I assigned each individual an age at maturity, with the specified combination of genetically determined and random environmental variation as already described. Scenarios with variation in age at maturity produced ages at maturity ranging between 14 and 23 years for females. Likewise, I assigned each fish a spawning interval from a normal distribution, but assumed that variation in this trait was not heritable. I assumed that the last year of spawning for a given fish was equally likely to have occurred at any time between the previous year and a full spawning interval prior to the start of the simulation.

4.2. Two selective regimes

One of the questions addressed here is how the shift in selective regime influences persistence and the role of individual variation in life history. This leads to the question, 'What adaptive value would delayed maturation have?'. According to life his-

tory theory, several mechanisms may lead to the evolution of delayed maturation (Stearns and Crandall, 1981; Roff, 1984). In the case of the white sturgeon, I hypothesized that two factors may have favored delayed maturation (Fig. 4). First, precocious individuals may have suffered increased mortality resulting from the energetic costs of a long upstream spawning migration. Second, because of relatively low mortality risk while waiting in the marine environment and increases fecundity with increased body size (Chapman et al., 1996), it may not have been worth making the trip until reaching a substantial size. Sturgeon grow quickly and develop an outer covering of scutes, both of which protect them from predators and reduce juvenile mortality.

I simulated two hypothetical selective regimes. For the pre-dam situation with a stable selective regime (Fig. 4A), I calibrated the model to produce an equilibrium age at maturity (that resulting after 1000 years of selection) close to that

observed recently among populations in the Snake River. I calibrated a linear relationship (Eq. (3)) between survival through a spawning migration, $S(t)$, and the age of the migrating adult, t, until it resulted in no shift from the initial average values after 1000 years. Final calibrated parameter values were $S_0 = 0.75$ and $S_A = 0.10$. Variable t_A is the average age at maturity.

$$S(t) = \min\{S_0 + S_A(t - t_A),\ 1\} \qquad (3)$$

For the post-dam situation (Fig. 4B), I simulated a shift in the optimal age at maturity corresponding with a land-locked selective regime by removing mortality associated with spawning migration ($S_0 = 1$). This represents the current situation with land-locked populations that no longer commute between freshwater and the sea. To compensate for this, I decreased survival during the first year of life in the post-dam simulations so that both the pre- and post-dam simulations with heritability of 0.25 produced the same likelihood of persistence. As a result, the absolute predictions of persistence should be compared only within a selective regime.

5. Results

Simulation results for each of the three questions are described in three sections that follow. They are also summarized at the bottom of Fig. 3.

5.1. How does individual variation in age at maturation influence the likelihood of persistence?

Individual variation in age at maturity decreased the likelihood of persistence to 1000 years predicted by our model for white sturgeon in simulations of the pre-impoundment scenario (0.63 versus 0.36) as shown in Fig. 5A for $h = 0$. In the pre-impoundment scenario, the initial average age at maturity was at or near the selected optimum. Therefore, the absence of individual variation implies that all fish had the optimal value of the trait. Higher individual fitnesses translated into a larger average population sizes and an increased likelihood of persistence at the population level.

Individual variation in age at maturity did not have much influence on the likelihood of persis-
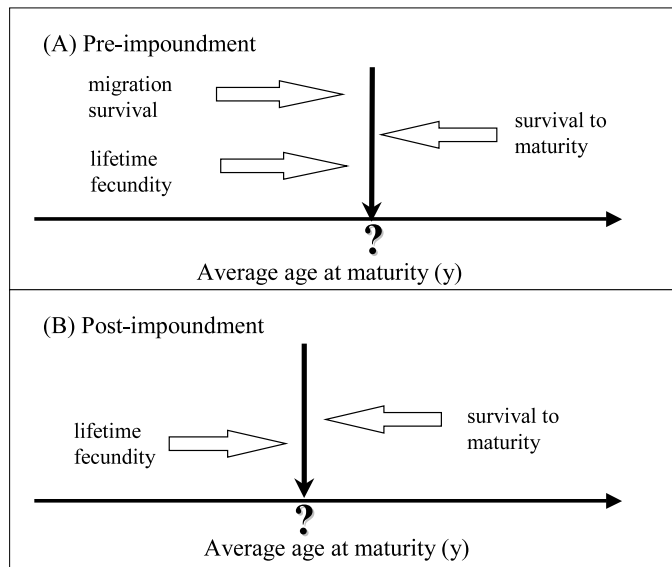

Fig. 4. Diagram of hypothetical forces acting on the evolution of age at maturity for white sturgeon (A) before dams were constructed and (B) after dams were constructed.
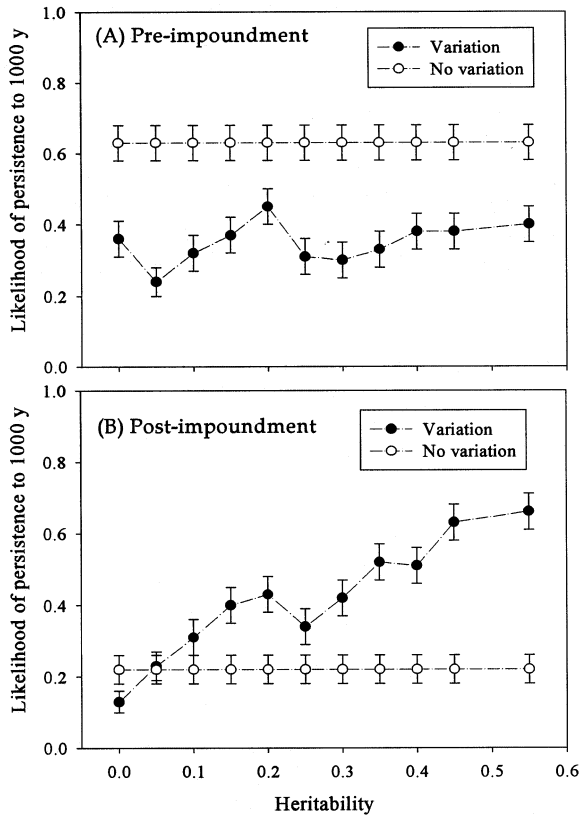
Fig. 5. Changes in the likelihood of persistence to 1000 years simulated with no phenotypic variation and with variation ranging in heritability from completely environmental to a heritability of 0.55 for the (A) pre-impoundment and (B) post-impoundment scenario.

tence to 1000 years predicted by our model for white sturgeon in simulations of the post-impoundment scenario (0.22 versus 0.13) as shown in Fig. 5B for $h = 0$.

5.2. Does heritability change the relationship between individual variation and the likelihood of persistence?

The first experiment addressed the role of individual variation in the life history trait, age at maturity, for white sturgeon. In this second simulation experiment, I asked whether it makes a difference if there is a genetic basis to that variation. Here, the realistic individual variability in

age at maturity is provided with both an environmental and a genetic basis.

The two situations (pre- and post-impoundment) gave different results. The pre-impoundment simulations showed no trend in population viability as heritability changed. In contrast, the post-impoundment simulations showed an increase in population viability with increased heritability (Fig. 5). Because the two scenarios are calibrated to produce the same likelihood of persistence at $h^2 = 0.25$, it is not possible to compare absolute values between scenarios, but it is legitimate to compare patterns.

The final age at maturity in the pre-impoundment simulations did not respond to heritability, consistent with a trait under stabilizing selection (Fig. 6, circles). In contrast, the mean final age of maturity in the post-impoundment scenario decreased with heritability in a way that is consistent with directional selection (Fig. 6, triangles). In these simulations, the mean trait value decreased over the 1000-year period for those populations that persisted.

The variation in the population around the mean value decreased in both scenarios (Fig. 7). Under the pre-impoundment regime (Fig. 7, circles), stabilizing selection reduced phenotypic variation as expected. Under the post-impoundment regime (Fig. 7, triangles), directional selec-
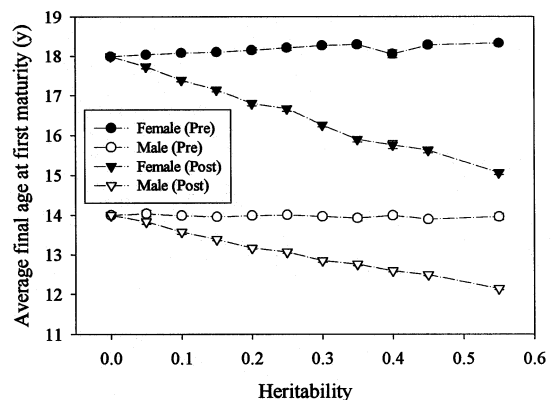

Fig. 6. Changes in mean age at maturation simulated with no phenotypic variation and with variation ranging in heritability from completely environmental to a heritability of 0.55 for a pre- and a post-impoundment scenario.
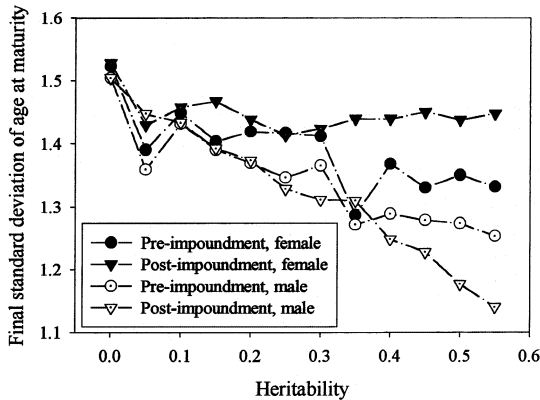
Fig. 7. The standard deviation in male and female ages at maturity among individuals remaining in the simulated populations in year 1000 is shown as a function of heritability for the pre- and post-impoundment selective regimes.

Table 1
Simplifying assumptions made in the genetic individual-based model

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1. The white sturgeon genome is functionally diploid
2. Evolution in the trait age at maturation is unopposed by environmental correlations (e.g. reduced growth leading to increased mortality) or genetic correlations (pleiotropy with other traits)
3. The number of loci and alleles contributing to this trait is similar to the arbitrary intermediate values selected here
4. Genetic variation is additive, and contributions from dominance and epistasis are not important
5. Mutations are not inherently different from wild alleles in effects, their effects are unrelated to prior state, and there is no asymmetry in the rates of forward versus backward mutation
6. Allelic effects are symmetric normal with zero mean and no kurtosis
7. There are no genotype-by-environmental interactions

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tion decreased the variation among males more than it did variation among females.

5.3. How does the selective regime influence the relationship between individual variability and persistence?

The results reported in the previous two sections indicate that the answer to each question differed completely under the two selective

regimes considered. When the mean age at maturity was optimal under the selective regime, individual variation was detrimental to the population and heritability had no effect. In contrast, a mean age at maturity away from the optimum led to the conclusion that non-heritable individual variation has no significant effect, but that heritable variation is beneficial.

6. Discussion

These simulation experiments demonstrate the ability to represent heritable variation in traits in an individual-based population model without a great deal of specific information about genetic architecture. I made a number of simplifications (Table 1), the effects of which are not explored here. Evaluating sensitivity to these assumptions may be a fruitful direction for future research.

The results of this study answer the third question unequivocally. They indicate that it is not possible to predict the effect of individual variation, heritable or not, on persistence without understanding the selective regime. The first two questions are discussed in the following.

6.1. How does individual variation in age at maturation influence the likelihood of persistence?

The first simulation experiment showed that population viability results can be sensitive to whether variation observed among individuals in life history traits is portrayed. The main result, however, was that the effect of individual variation on population extinction risk depended very much on the juxtaposition of trait values with respect to an optimal trait value. An 'optimal' age at maturity under a given selective regime is defined as a value yielding the highest fitness within a specified region of possible trait values. In the situation addressed here, the fitness surface was smooth enough to neglect the issue of multiple local optima. Operationally, an optimal trait value can be estimated as the stable average reached in genetic simulations after a long period of time from a particular initial state. One implication of this is that a population model can be

checked to see whether its demographic parameters evolve. If they do not, then the parameters are in equilibrium with the selective regime simulated. The results give some indication of how individual variation in fitness-related traits will influence simulated population persistence.

To put these results in the context of previous studies, these results are unusual in that I did not find an increased chance of persistence associated with adding (non-heritable) individual variation. In the pre-impoundment selective regime, the constant age at maturity of individuals was at the optimal value. Therefore, in the absence of overcompensating density dependence, higher fitness of equivalent individuals translated directly into higher population sizes and persistence. When variation was added, causing some individuals to deviate from the optimal age at maturity, lower individual fitnesses were reflected by reduced persistence at the population level.

Variation in age at maturity did not lead to a higher chance of persistence under the post-impoundment selective regime. One would expect that the presence of some individuals with extreme phenotypes having higher fitness (i.e. those with shorter generation times) might reduce extinction risk at the population level. That is, one would expect that maximum fitness of the population (i.e. the minimum age at maturity present) would be the best predictor of persistence. Instead, these results showed that non-heritable variation in age at maturity among individuals did not greatly influence persistence at the population level. I conducted some additional simulations with: (1) a larger number of replicates; (2) no density dependence; and (3) a wider range of variation in age at maturity. I found a slight, but still not significant, increase in persistence.

The results from this study with an individual-based model contrast with those obtained from an age-based model. I developed an age-based model with the proportion mature at each age drawn from an inverse normal distribution. I compared the likelihood of persistence for simulations with a standard deviation of zero (all females achieved maturity at exactly 18 years) and a standard deviation of 1.5 years (the proportion reproducing at each age gradually increased). The likelihood of persistence in the age-based model increased significantly with increased variation in aged at maturity.

6.2. Does heritability change the relationship between individual variation and the likelihood of persistence?

To address the second question, I compared the effect of simulating this life history variation as having a genetic, as well as an environmental, basis. Under the pre-impoundment selective regime, heritable and non-heritable variation had the same influence. I found that extinction risks were predicted to be lower when the variation was heritable because the trait (age at maturity) distribution shifted downward in response to selection. In reality, such a response may be countered by pleiotropic responses in other traits that were not simulated, which may prevent a response to selection. However, there is evidence that this trait can evolve in response to selection. For example, several studies have measured selective differentials in the ages and sizes at maturity in fish populations exposed to size-selective predation (Rowell et al., 1989; Reznick et al., 1990).

6.3. How does the selective regime influence the relationship between individual variability and persistence?

The importance of the selective regime is a main result. Genetic variation in this study had the greatest effect on population persistence in a changed environment. Lande and Shannon (1996) consider a wider range of selective regimes, including variable regimes. Correspondence of the mean value of demographic parameters with a value leading to an optimal simulated fitness determines how individual variation will influence population persistence. This suggests that it is difficult to interpret comparisons between results produced with and without individual variation without first understanding the fitness surface produced by the model. Note that this result applies equally to genetic and non-genetic population models.

This study only looked at constant selection regimes. A wrinkle in the relationship between

heritable genetic variation and the population persistence was first articulated by Wright (1978). He recognized that stabilizing selection would have the effect of reducing individual variation in the short term, while longer-term persistence of the population might depend on the presence of genetic variation. Therefore, the long- and short-term effects of heritability may differ under a changing selective regime.

6.4. Can life history traits evolve?

One controversy relevant to this study is the extent to which life history traits are able to evolve. It has long been held that the constant exposure of life history traits to strong selection will erode their genetic variability (thus heritability). This view has been challenged by Houle (1992) who surveyed over 200 quantitative genetic studies of animal populations. Houle concluded that traits closely related to fitness had higher additive genetic and non-genetic variability. This is explained in part by the observation that inheritance of life history traits is polygenic (Istock, 1982). In a hierarchical view, one might rank traits according to the degree of aggregation. As one moves down the pyramid, traits are defined to mirror the parameters needed for population models of increasing complexity. In this view, fitness is the single trait at the top of the pyramid; birth and death rates are in the second tier; and the three life history traits, survival, age at maturity, and fecundity form the third tier; and so on. Individual differences in life history traits arise as the integrated result of many specific, lower-level traits that develop during the lifetime of the organism (for example, de Jong and van Noordwijk, 1992). Thus, even a small amount of genetic variation at one locus can add up to a significant overall amount. A different explanation is that selection that appears to be stabilizing is actually a result of short-term fluctuations in directional selection and, therefore, does not erode genetic variation. For example, the fitness associated with one life history genotype may fluctuate in response to either population density or environmental cycles. A third argument is that protected polymorphisms, maintained by antagonistic pleiotropy, tend to evolve among life history traits (Hartt and Haefner, 1998).

6.5. How is individual variation maintained?

This paper addresses only the effect of individual variation on persistence. In situations when individual variation does increase the chances of population persistence, are there feedback mechanisms that promote individual variation within populations? The 'risk-spreading' argument maintains that heterogeneous populations are more likely to persist because they spread the risk of extinction over a variety of phenotypes (den Boer, 1982; van Dijk, 1982; Uchmanski, 1999). Individual variation can promote persistence through density-dependent population regulation, where regulation is viewed in the sense of bounding populations away from very small and very large sizes (Conner and White, 1999; Pfister, 1999; Uchmanski, 1999). This implies that population-level selection is one mechanism for maintaining variation.

Does individual-level selection also provide mechanisms for maintaining variation under these circumstances? The question 'How is genetic variation maintained in natural populations?' has long preoccupied evolutionary biologists. Does individual variation tend to increase the likelihood of persistence under selective regimes that tend to maintain genetic variation (i.e. rugged, fast-changing fitness surfaces)? If so, this would provide a positive feedback mechanism for evolution in the variation in a trait, as well as its mean.

Frequency dependence is one mechanism that can promote individual variation. Individual variation in a trait is promoted when rare phenotypes have higher fitness than common phenotypes. If variation in such a trait improves persistence at the population level (e.g. individuals that specialize on different prey), the feedback cycle is complete.

To avoid giving the impression that individual variation is always a good thing at the population level, recall that there are many examples of traits in which variation is selected against (e.g. canalized early development, synchronized reproduction, frequency dependent behaviors that favor conformity). Hartt and Haefner (1998) predict

that large, predictable changes in the environment will select for populations with life history strategies that maximize population growth, possibly at the expense of phenotypic diversity. Are there feedback mechanisms that reduce variation in life-history traits when such variation has adverse effects on persistence at the population level?

6.6. *Phenotypic plasticity*

A final topic of discussion is the role of phenotypic plasticity. In the simulations reported in the present study, environmental variation among individuals was random. I did not consider the potential role of environmental factors such as temperature or food availability or population density on the age at maturation. A next step would be to consider more realistic reaction norms between age at maturity and these environmental factors. A practical consideration is that this removes the ability to control simulated heritability.

A particular type of plasticity, 'bet-hedging', is particularly relevant to the discussion of how individual variation can enhance individual fitness and, thus, lead to higher chances of persistence at the population level. Parents employing a bet-hedging strategy produce offspring with a variety of phenotypes (Seger and Brockmann, 1987). By adopting this strategy, they are able to hedge against unpredictable environments and the risk of complete reproductive failure. The possibility that genetic mechanisms exist for passing along variation itself as a trait is intriguing (Via, 1993). It seems reasonable to conclude that whenever the production of variable phenotypes is advantageous to the individual, it is probably also advantageous to the population.

7. Conclusions

This study, in contrast to previous simulation studies, found that increased individual variation in the age at maturity did not necessarily lead to a higher likelihood of persistence. Individual variation in this trait only increased the simulated likelihood of persistence under an altered selective regime when the individual variation was heritable. The challenge ahead is to consider how we can predict the future viability of populations with a limited understanding of the relationships between the genetic composition of the population and its potential response to a changing environment.

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