Theor Ecol (2015) 8:175–186
DOI 10.1007/s12080-014-0242-8

ORIGINAL PAPER

Mean and variance of population density and temporal Taylor's law in stochastic stage-structured density-dependent models of exploited fish populations

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Received: 30 July 2014 / Accepted: 14 November 2014 / Published online: 2 December 2014

Abstract How does fishing affect the mean and variance of population density in the presence of environmental fluctuations? Several recent authors have suggested that an increasing ratio of standard deviation to mean (coefficient of variation, or CV) in population density indicates declining population stability. We investigated the relationship between the mean and variance of population density in stochastic, density-dependent, stage-structured fish population models. Our models included either compensatory or overcompensatory density dependence affecting either fertility or juvenile survival. Environmental stochasticity affected either juvenile survival (when density dependence affected fertility) or fertility (when density dependence affected juvenile survival). The mean and variance of population density were compared as fishing mortality changed. In some cases, the relationship between the natural logarithms of mean and variance is linear under some parameters (life history strategy) of some models (the type of density dependence and the timing of density dependence and stochasticity), supporting Taylor's law. In other cases, the relationship can be non-linear, especially when density dependence is overcompensatory, and depends on the stage observed. For example, the variance of adult density may increase with its mean while the variance of juvenile density of the same population may decline, or vice versa. The sequence in which individuals experience stochasticity and density dependence matters because density dependence can attenuate or magnify the fluctuation. In conclusion, the use of the CV as a proxy for population instability is not appropriate, and the CV of population density has to be interpreted carefully for other purposes.

Keywords Coefficient of variation · Density dependence · Environmental stochasticity · Fluctuation scaling · Stage-structured population model · Taylor's law

Introduction

Empirical evidence has suggested that the variance of the population density of a given taxon is often a power function of its mean (Taylor and Woiwod 1980):

$$V = aM^b, \tag{1}$$

where M and V are the mean and variance of population density, respectively, and a and b are coefficient and power (or exponent), respectively. This pattern is called Taylor's law (TL; Taylor 1961). This relationship has been found for hundreds of taxa, among the populations of a single or several related species (Eisler et al. 2008; Ramsayer et al. 2012; Taylor and Woiwod 1980). TL has been verified with both temporal and spatial variance of population densities (Taylor and Woiwod 1982). In this paper, we focus on temporal TL relationships and examine the relationships between V and M at varying levels of exploitation (fishing mortality).

A large number of models have been developed to explain the TL relationship theoretically (e.g., Anderson et al. 1982; Ballantyne 2005; Cohen 2014; Cohen et al. 2012a, b, 2013; Keeling 2000; Kilpatrick and Ives 2003; Perry 1994; Yamamura 1990, 2000). For example, Cohen et al. (2012a)

Electronic supplementary material The online version of this article (doi:10.1007/s12080-014-0242-8) contains supplementary material, which is available to authorized users.

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demonstrated that the TL relationship holds for density (denoted by $u(x,t)$ where x and t are individual size and time, respectively) at given individual size x when $u(x,t)$ is periodic in time t. The authors also demonstrated that the results are insensitive to fishery exploitation as long as fish of different sizes are targeted equally (balanced harvesting). Here, we present new results on the mean and variance of fish density under stochastic density-dependent stage-structured population models experiencing fishery exploitation. We investigate how the mean and variance of stage-specific densities change when different levels of exploitation rates are imposed on a population or populations exhibiting the same life history strategy. The use of stage-specific rather than total density reflects the fact that we rarely observe the total density in practice and often use stage density as a proxy.

Define fishing mortality $\mu=1-f$ as the complement of the fishing escapement rate f. Define $V(\mu)$ and $E(\mu)$ as the variance and mean, respectively, of stage density over time of a specified model population subjected to fishing mortality μ. Cohen (2013, p. 95, his equation (7)) defined the "local exponent" (in the context of this paper, $b(\mu)$) of the variance–mean relationship as

$$b(\mu) = \frac{d\log V(\mu)}{d\log E(\mu)}, \tag{2}$$

assuming differentiability. The local exponent $b(\mu)=b$ is constant at all levels of fishing mortality μ if and only if the power-law form of TL (1) describes the relation between V and M at all μ. Cohen (2013, p. 96, his Fig. 1(d)) demonstrated that when the local exponent is viewed as a function of time, the local exponent experienced very abrupt changes (singularities) in a deterministic model of heterogeneous exponentially growing clones. Jiang et al. (2014, their Fig. 3) showed that very abrupt changes of the local exponent (as a function of time) occurred in a linear birth-and-death population model, where stochasticity was purely demographic (affecting each individual independently in a constant environment). Here, we investigate whether, and under which conditions, the local exponent $b(\mu)$ exhibits singularities in stage-structured, density-dependent fishery models with environmental stochasticity only.

The mean and variance over time of population density are important quantities in population biology in general. For example, in conservation ecology, these two measures were proposed as the major determinants of the viability of a population (Beissinger and McCullough 2002). In fisheries, the coefficient of variation (CV), which is the standard deviation (square root of the variance) divided by the mean, is often used as a measure of population variability (Anderson et al. 2008; Hsieh et al. 2006). When the exponent $b=2$ in TL, the CV of population density is constant. But when $b>2$ and $b<2$ (McArdle et al. 1990), the CV increases and declines, respectively, with increasing mean. Whether the mean and variance of population density are compared directly or other quantities are derived from them, for their proper interpretations, it is crucial to understand how they are related to each other for various underlying population processes.

The models used in this study are two-stage matrix population models with environmental stochasticity, density dependence, and fishery exploitation. A similar model was previously used to demonstrate the effect of fishery exploitation on transient and asymptotic dynamics of fish populations exhibiting various life history strategies (Fujiwara 2012). This model complements previous attempts to understand the TL relationship. For example, Perry (1994) investigated the TL relationship with a population model exhibiting chaotic dynamics, Kilpatrick and Ives (2003) used a competition model, and Cohen et al. (2013) used a stochastic multiplicative model without stage or age groups. Here, we show the effects of (1) the order of stochasticity and density dependence experienced by individuals, (2) type of density dependence (compensatory vs. overcompensatory), (3) the stage targeted by exploitation, and (4) life history strategies on the mean and variance of stage densities. In this analysis, the positive equilibrium point of the models without stochasticity was stable; therefore, fluctuation is primarily driven by the environmental stochasticity although other processes can attenuate or magnify the fluctuation.

Materials and methods

Two-stage population models

The two stages in the models are for juveniles and adults; the life cycle graph for the models can be found in (Online Resource A). Although the stage structure is simple, the models can incorporate a wide range of life history strategies of organisms by varying parameters as described in Neubert and Caswell (2000). The population matrix is given as

$$\mathbf{A} = \begin{pmatrix} s(1-m) & r \\ sm & p \end{pmatrix}, \tag{3}$$

where r is a fertility rate ($r>0$), s is a juvenile survival rate ($0<s<1$), p is an adult survival rate ($0\le p<1$), and m is a maturation rate ($0<m\le 1$). These rates are finite per-capita rates, and the time unit is assumed to be 1 year. Hereafter, these rates are collectively called life history parameters. State variables are the densities of juveniles and adults, denoted by n_1 and n_2, respectively. The population vector at time t is given as $\mathbf{n}_t = \begin{bmatrix} n_1 & n_2 \end{bmatrix}_t^T$, and it is projected from time t to $t+1$ as

$$\mathbf{n}_{t+1} = \mathbf{A}\mathbf{n}_t. \tag{4}$$

By letting one of the life history parameters be density dependent and another be stochastically fluctuating, we develop stochastic density-dependent models. Fishery exploitation is also incorporated into the model by including the rate of escaping the exploitation (an escapement rate). These are described below.

Density dependence

Density dependence is incorporated into one of two life history parameters: fertility and juvenile survival rates. In the first model, the fertility rate depends on adult density. This is denoted by $r[n_2]$, and this model is called a density-dependent fertility model. In the second model, the juvenile survival rate depends on juvenile density. This is denoted by $s[n_1]$, and this model is called a density-dependent juvenile survival model.

For the density-dependent fertility model, density dependence is expressed as

$$r[n_2] = \frac{\alpha}{1 + (\beta n_2)^{\gamma}}, \tag{5}$$

where α, β, and γ are density-dependent parameters (Fig. 1). Parameter α determines the maximum number of juveniles produced per adult; therefore, the intercepts with the vertical axis in Fig. 1a are given by α. Parameters β and γ determine how fast the fertility rate declines with increasing adult density n_2. The product of $r[n_2]$ and n_2 is generally known as a stock–recruitment (spawner–recruitment) function in fishery literature when n_2 is stock density and stage 1 is the stage to be recruited (Fig. 1b). When γ=1, this function is said to be compensatory and is also known as the Beverton–Holt function (Beverton and Holt 1957). When γ>1, for a sufficiently large n_2, $r[n_2]$ declines faster than an increase in n_2; this type of stock–recruitment relationship is known as overcompensatory (Hilborn and Walters 1992) because recruitment $r[n_2]n_2$ can decline with increasing n_2 (Fig. 1b).

For the density-dependent juvenile survival model, density dependence is expressed as

$$s[n_1] = \frac{1}{1 + (\beta n_1)^{\gamma}}. \tag{6}$$

This is the same as that for the density-dependent fertility rate (Eq. 5) except α is set to 1, which is the maximum possible value of a survival rate.

Fishing mortality

Fishing mortality was incorporated into the density-dependent fertility model as

$$\mathbf{A_n} = \begin{pmatrix} s(1-m) & \dfrac{\alpha f}{1 + (\beta f n_2)^{\gamma}} \\ sm & pf \end{pmatrix}, \tag{7}$$

where f is the annual escapement rate from fishing. Therefore, $1-f$ is an annual fishing mortality rate. Fishing is assumed to target only adults in this model, but the escapement rate also appears in the <1,2> element because the sequence of events is assumed to be (1) fishing, (2) reproduction, and (3) the survival of offspring over 1 year. This order of the events was assumed in order to avoid the situation in which adults do not experience fishing mortality at all before the first reproduction; this is problematic for fish with semelparous life history (p=0). Another way to incorporate fishing mortality is to multiply the <2,1> element by f, rather than the <1,2> element, assuming fishing occurs immediately before time progresses from 1 year to the next.

Similarly, fishing mortality was incorporated into the density-dependent juvenile survival model as

$$\mathbf{A_n} = \begin{pmatrix} \dfrac{1-m}{1 + (\beta n_1)^{\gamma}} & rf \\ \dfrac{m}{1 + (\beta n_1)^{\gamma}} & pf \end{pmatrix}. \tag{8}$$

Fishing often targets adults, but some fisheries, such as Japanese eel (Tsukamoto 2012) and bluefin tuna (Fromentin and Powers 2005) fisheries, also target juveniles. When juveniles are affected by fishing exploitation, the <1,1> and <2,1> elements of the population matrix are multiplied by f. However, qualitative results did not change whether only adults, only juveniles, or both juveniles and adults are targeted by fisheries. Therefore, we present selected results when adults are targeted by fisheries in the main text, and the other results are presented in Online Resource B.

Stability of deterministic models

Under the deterministic density-dependent fertility model, if the dominant eigenvalue of the population matrix after replacing $r[n_2]$ with α was greater than 1, the model has a single positive equilibrium point because the dominant eigenvalue is a decreasing function of $r[n_2]$ according to the Perron–Frobenius theorem (see Caswell 2001) and $r[n_2]$ is also a decreasing function of n_2. Similarly for the juvenile survival

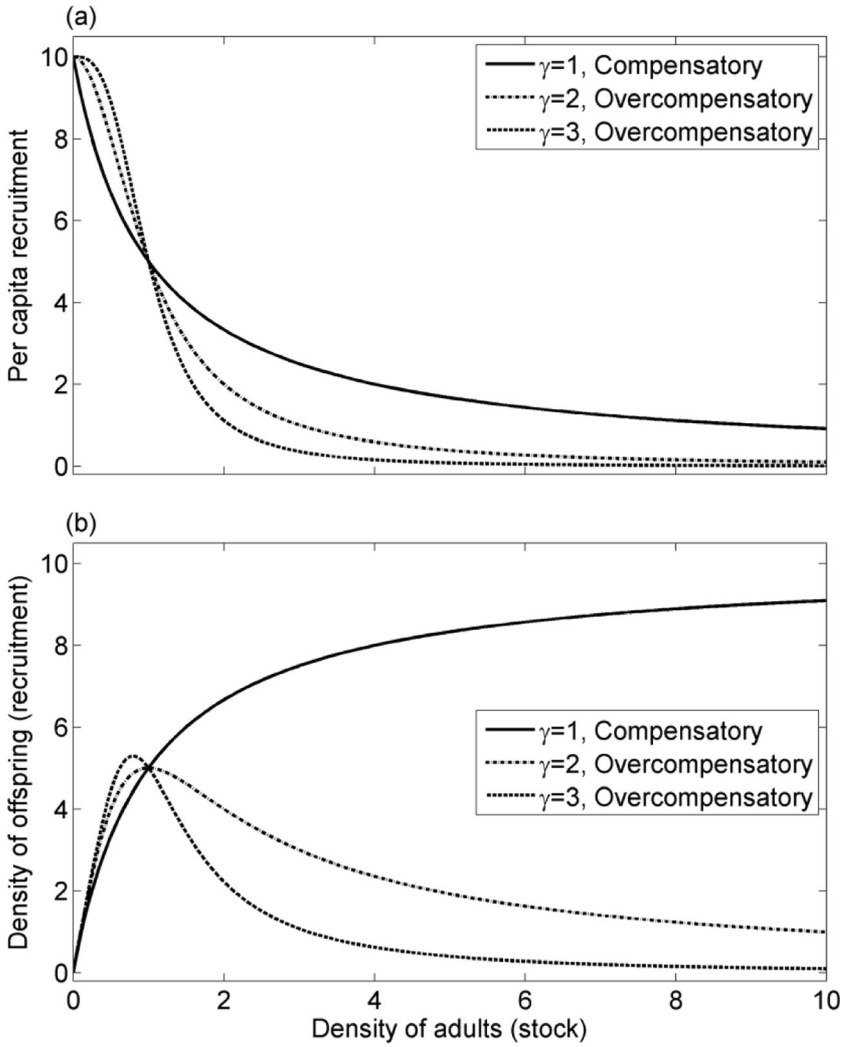

Fig. 1 Density-dependent fertility functions: **a** per-capita recruitment (Eq. 5) as a function of adult density and **b** total recruitment ($r[n_2]n_2$) as a function of adult density. Parameters: α= 10, which appears as intercepts in **a**, and β=1

model, when the dominant eigenvalue of the population matrix after replacing $s[n_1]$ with 1 is greater than 1, the model has a single positive equilibrium point. Hereafter, the positive equilibrium point is denoted by $\mathbf{n}^*$.

The equilibrium point, however, may not be stable. One way to determine its stability is to apply the Jury Criteria (Jury 1974) to a linearized density-dependent population matrix around the equilibrium point:

$$\mathbf{J} = \mathbf{A}_{\mathbf{n}^*} + \left(\frac{\partial \mathbf{A}_\mathbf{n}}{\partial n_1}\mathbf{n} \quad \frac{\partial \mathbf{A}_\mathbf{n}}{\partial n_2}\mathbf{n} \right)_{\mathbf{n}^*}. \tag{9}$$

Matrix $\mathbf{J}$ is called Jacobean (see Caswell 2001). According to the Jury Criteria, the equilibrium point is stable when the following conditions are satisfied:

$$\begin{aligned} 1-\mathrm{tr}\mathbf{J}+\det\mathbf{J} &> 0,\\ 1+\mathrm{tr}\mathbf{J}+\det\mathbf{J} &> 0,\\ 1-\det\mathbf{J} &> 0. \end{aligned} \tag{10}$$

In this analysis, we consider the results only when the equilibrium point of the deterministic model is stable (stable models).

Stochasticity

Stochasticity was incorporated into the stable models by allowing one of two life history parameters to fluctuate stochastically. Under the density-dependent fertility model, stochasticity was incorporated into juvenile survival (i.e., stochasticity occurring after density dependence). To simulate juvenile survival, the beta distribution with mean $\bar{s}$ $(0 < \bar{s} \leq 1)$ and the variance of 0.001 was used. Under the density-dependent juvenile survival model, stochasticity was incorporated into the fertility term (i.e., stochasticity occurring before density dependence). To simulate the fertility rates, the gamma distribution with mean $\bar{r}$ $(\bar{r} > 0)$ and the variance of 0.001 was used. Although a variance of 0.001 appears to be small, small variance, depending on the associated mean, can translate into a large fluctuation in a population growth rate, which in turn can

translate into a large fluctuation in stage densities. To investigate the effect of increased variance, we also simulated different levels of variance up to 0.1. Some of the results with higher variance are presented in Online Resource D.

Analysis

When fishing mortality was imposed on the adult stage, changes in the mean and variance of both juvenile and adult densities were investigated for a wide range of life history strategies, which are determined by the juvenile survival, adult survival, and maturation rates. For a given life history strategy (i.e., a given set of s, p, and m), a fertility rate r that makes the dominant eigenvalue of population matrix (3) equal to 1 is

$$r = \frac{(1-p)(ms-s+1)}{ms}. \quad (11)$$

Equation (11) was obtained by writing the equation for the dominant eigenvalue of matrix **A** in Eq. (3), setting the eigenvalue to 1 and solving the equation for r.

For the density-dependent fertility model, α in Eq. (5) was set to 10 times the value of r obtained with Eq. (11). Then, for both the density-dependent fertility and juvenile survival models, the equilibrium adult density when there was no fishing mortality (f= 1) was set to 100, and juvenile density was obtained by calculating the stable stage distribution. Then, for a given value of parameter γ, the other density-dependent parameter β was obtained by substituting the equilibrium stage densities n_2^* or n_1^* when f=1 into the density-dependent Eq. (5) or (6), respectively, and solving the equations for β. Consequently, without any fishing mortality, the adult equilibrium density without stochasticity is always 100, but it changes as a different level of fishing mortality is imposed.

For each set of life history parameters (s, m, and p); density-dependent parameter (γ); and fishing escapement rate (f), the Jury Criteria were applied to determine the stability of the equilibrium point. If the equilibrium point was stable, s was used for the mean survival rate $\bar{s}$ in the stochastic density-dependent fertility model, and r obtained by Eq. (11) was used for the mean fertility rate $\bar{r}$ in the stochastic density-dependent juvenile survival model. Then, a population vector was simulated over 10,100 time steps starting from the equilibrium point of the corresponding deterministic model. Then, the mean and variance of each stage density were calculated after discarding the first 100 time steps. This was repeated for different sets of parameters (Table 1).

Table 1 Parameters used in the analyses

Parameter	Notation	Values evaluated
Juvenile survival rate	s	0.2, 0.5, 0.8[a]
Maturation rate	m	0.1, 0.5, 1.0
Adult survival rate	p	0, 0.5, 0.9
Fertility rate	r	See Eq. (11)[b]
Density-dependent parameters	α	10r
	β	See main text[b]
	γ	1, 2, 3
Fishing escapement	f	0.2–1.0[c]
Variance of stochastic distributions of juvenile survival and fertility		0.001, 0.1

[a] The results with s=0.2 and 0.8 are shown in Online Resource B, C, and D

[b] r and β are functions of other parameters

[c] Evaluated at 501 points equally spaced within the range

To determine the number of time steps to be used in the simulations, different numbers (1000, 5000, 10,000, and 40,000) of time steps were tried under selected parameter sets. Generally, the time steps required for the convergence depended on parameter values, and the variance took more time steps to converge than the mean. From many trials, it was decided that 10,000 time steps were sufficient to see the patterns in the relationships between the mean and variance of stage densities.

Results

The natural logarithm of the variance of stage densities was plotted against the natural logarithm of the mean stage density (Figs. 2, 3, 4, and 5). In the figures, the variance and mean change as different levels of fishing mortality were imposed on a population. There is 1 point for each of the 501 levels of fishing mortality. Hereafter, these curves are called the variance–mean curves. We present the results first for compensatory density dependence (first with density-dependent fertility, then with density-dependent juvenile survival), followed by the results for overcompensatory density dependence (again, first with density-dependent fertility, then with density-dependent juvenile survival). Then, we describe the effects of fishing mortality on mean stage densities when density dependence was overcompensatory. Finally, we describe the effects of increased variance in the stochastic term on the mean stage densities.

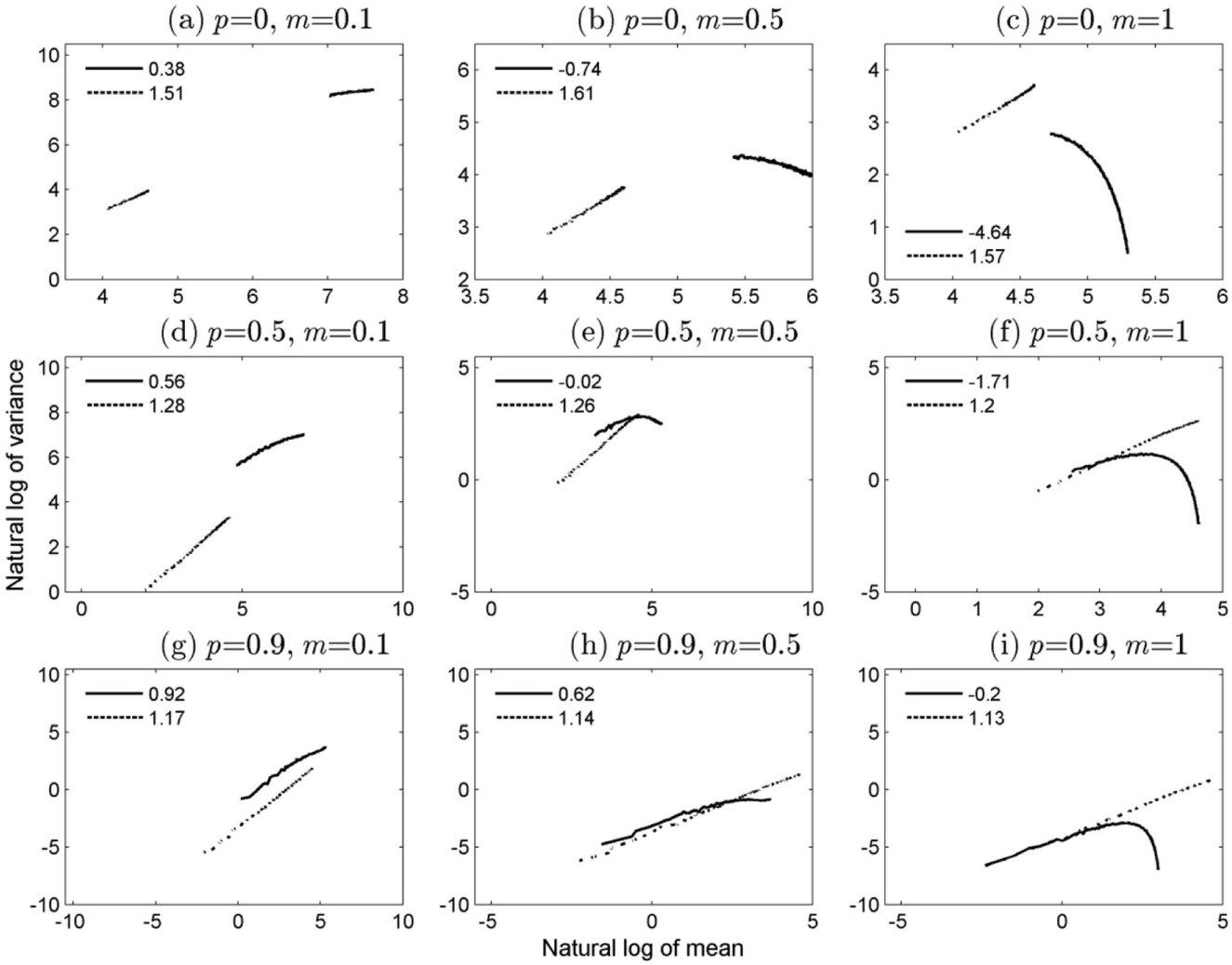

Fig. 2 Natural logarithm of variance and natural logarithm of mean of juvenile (*solid curve*) and adult (*dashed curve*) densities over 10,000 time steps of a stochastic density-dependent fertility model. The slopes b of fitted linear regression lines are also shown in each panel. The simulations are under $\bar{s} = 0.5$, and other life history parameters are shown above each panel. Density dependence is compensatory (γ=1). As we go down the panels, an adult survival rate is increased, and as we go to the *right*, a maturation rate is increased

Mean and variance of stage densities

When density dependence is compensatory (γ=1) and fertility is density dependent (Fig. 2), the variance–mean curves for adults were linear with slopes between 1 and 2. This supports TL. However, the relationships for juveniles were not always linear. The local exponent $b(\mu)$ for juveniles (but not for adults) is very negative when the maturation rate m=1 and fishing mortality μ are small (Fig. 2, in the three panels on the right, solid curves; also see Online Resource C2). The juvenile stage consists of newly produced juveniles and those that survived and remained in the juvenile stage from the previous year. The former fluctuate much less than the latter because stochasticity affects the survival of juveniles and fluctuation in the newly produced is attenuated by density dependence as the density of adults increase. The attenuation can be seen in Fig. 1b where the slope of compensatory density curve declines as density increases; consequently, the same amount of fluctuation in adult density translates into small fluctuation in the density of newly produced juveniles as adult density is increased. As juvenile density increases under the same juvenile survival and maturation rates, the proportion of newly produced juveniles in the stage increases. This in turn reduces the variance of juvenile density.

When density dependence is compensatory (γ=1) and juvenile survival is density dependent (Fig. 3), the variance of juvenile density increased convexly (faster than linearly) with the mean. The log variance of adults also increased with its log mean with slopes between 1/2 and 2, but when the mean juvenile survival rate was lower, the variance of adult density could decline with increasing mean adult density (Online Resource B9), reversing the pattern observed with the density-dependent fertility model. The variance of juvenile density increased with increasing mean juvenile density with

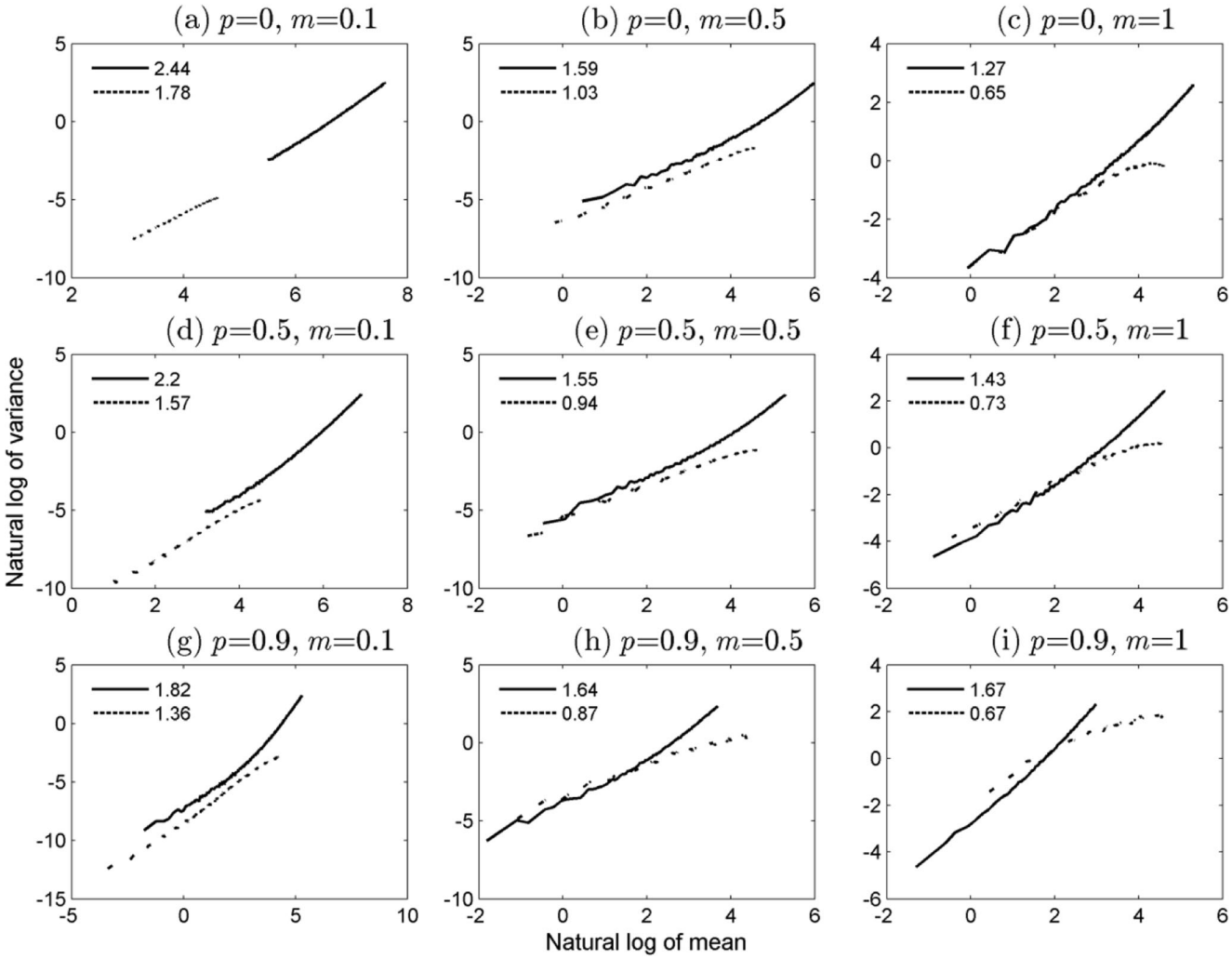

Fig. 3 Natural logarithm of variance and natural logarithm of mean of juvenile (*solid curve*) and adult (*black curve*) densities over 10,000 time steps of a stochastic density-dependent juvenile survival model. The slopes of fitted linear regression lines are also shown in each panel. The simulations are under $s^*=0.5$, and other life history parameters are shown above each panel. Density dependence is compensatory ($\gamma=1$). As we go down the panels, an adult survival rate is increased, and as we go to the *right*, a maturation rate is increased

slopes between 1.3 and 2.5 (Fig. 3), meaning that the CV could increase or decline with increasing mean juvenile density.

When density dependence is overcompensatory ($\gamma=3$) and fertility is density dependent (Fig. 4), the local exponent $b(\mu)$ for juveniles is again very negative for the two higher maturation rates m. However, the variance and mean could have a trough and/or peak at an intermediate fishing mortality rate (Fig. 4). Consequently, the variance–mean curves exhibit the cusp or fold (a singularity) at the density where the stock–recruitment curve is at its peak (asterisks; see Fig. 1b).

When density dependence is overcompensatory ($\gamma=3$) and juvenile survival is density dependent (Fig. 5), the local exponent $b(\mu)$ for adults (but not for juveniles, the reverse of the previous situation) clearly passes through a singularity. For example, in the middle row of Fig. 5, the dotted curve (for adults) has a very negative slope just to the left of the asterisk and a very positive slope just to the right of the asterisk, so the local exponent $b(\mu)$ jumps discontinuously from extremely negative to extremely positive values as fishing mortality decreases (see Online Resource C17) through the peak of the recruitment curve.

Effects of fishing mortality on mean stage densities

The mean and variance of stage densities were not monotonic functions of the fishing mortality rate when density dependence was overcompensatory (Figs. 4 and 5). Counterintuitively, the mean stage density could also increase with an increasing fishing mortality rate (Fig. 6). In particular, juvenile density tended to increase with an increasing fishing mortality rate under the overcompensatory density-dependent fertility model (Fig. C7-C9, C13-C15). This was because, as fishing removed individuals from the adult stage, it was

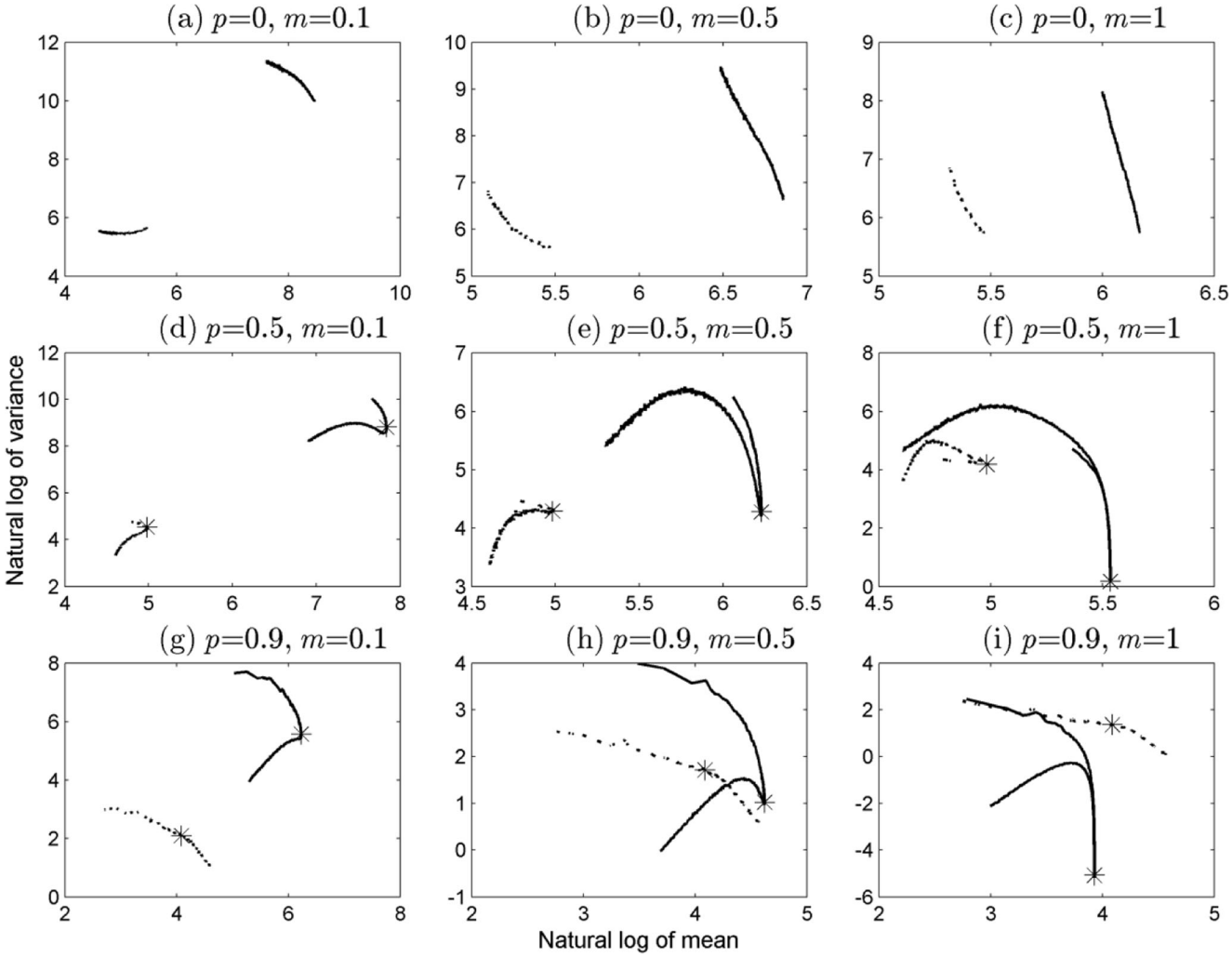

Fig. 4 Natural logarithm of variance and natural logarithm of mean of juvenile (*solid curve*) and adult (*dashed curve*) densities over 10,000 time steps of a stochastic density-dependent fertility model. *Stars* (*) indicate the density at which the stock–recruitment curve $r[n_2]n_2$ is at its peak. The simulations are under $\bar{s} = 0.5$, and other life history parameters are shown above each panel. Density dependence is overcompensatory (γ= 3). As we go down the panels, adult survival rate is increased, and as we go to the *right*, maturation rate is increased

overcompensated by increased reproduction. This, in turn, increased juvenile density. Adult density could also be overcompensated if organisms had a short-lived adult stage (p=0; top row of Fig. 6), but the adult density of organisms with a long-lived adult stage (p=0.9; bottom row of Fig. 6) showed declining adult density with an increasing fishing mortality rate. Finally, when organisms exhibited an intermediate level of adult survival (p=0.5; middle row of Fig. 6), adult density could increase and then decline as the fishing mortality rate was increased from 0 to 0.8. This contributed to creating the folding in the variance–mean relationship (Fig. 4).

Finally, under the density-dependent juvenile survival models, juvenile density declined with an increasing fishing mortality rate in all of the cases we investigated (Online Resource C4-C6, C10-C12, C16-C18). Adult density also tended to decline with fishing mortality, but when organisms had a short-lived adult stage (p=0) and experienced strong overcompensatory density dependence (γ=3), adult density could increase with fishing mortality (top row of figure in Online Resource C16).

In many cases, the stability of the positive equilibrium point was lost as the fishing mortality rate was increased (e.g., panels (g)–(i) in Fig. C1 of Online Resource C). This occurred when 0 (extinction) became stable. On the other hand, under the density-dependent fertility model with strongly overcompensatory density dependence (γ=3; panels (a)–(c) of Fig. C13 and panels (b) and (c) of Fig. C14 and C15 in Online Resource C), the positive equilibrium became stable as fishing mortality was increased and as the dynamics changed from periodic to a stable equilibrium point (this was confirmed by plotting bifurcation diagrams).

When the variance of the life history parameters was increased from 0.001 to 0.1, the mean stage densities generally declined (cf. Online Resources C and D). On the other hand,

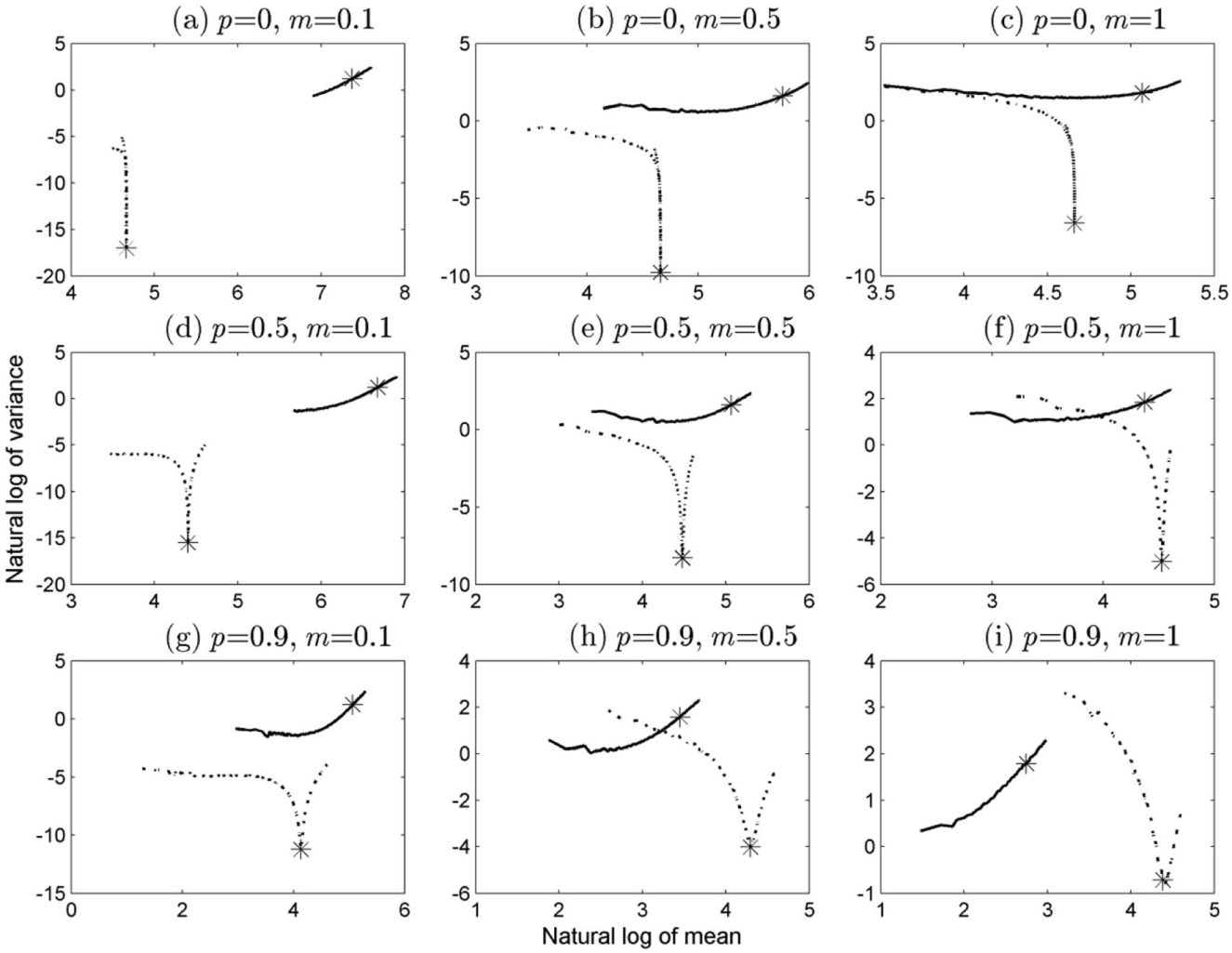

Fig. 5 Natural logarithm of variance and natural logarithm of mean of juvenile (*solid curve*) and adult (*dashed curve*) densities over 10,000 time steps of a stochastic density-dependent juvenile survival model. *Stars* (*) indicate the density at which the stock–recruitment curve $r[n_1]n_1$ is at its peak. The simulations are under $s^*=0.5$, and other life history parameters are shown above each panel. Density dependence is overcompensatory ($\gamma=3$). As we go down the panels, adult survival rate is increased, and as we go to the *right*, maturation rate is increased

the qualitative results described under the low variance remained the same in the majority of the cases investigated. Estimating mean densities became difficult under some parameters because computational rounding errors were introduced as mean density was reduced toward 0. This effect was particularly clear in Fig. D1, D7, and D13 in Online Resource D because population densities tended to be close to 0 and the frequency distribution of the survival rate was U shaped (i.e., the rate was frequently close to 0) because of high variance relative to its mean.

Discussion

Our analysis examined the changes in the mean and variance of stage densities at different levels of fishery exploitation. This can be viewed as comparison of different populations exhibiting the same life history strategy when different levels of fishery exploitation, or more generally additional mortality, were imposed. The comparison can also be between the periods of experiencing different levels of fishing pressure for the same population. The temporal TL was supported under some parameter values (life history strategies) of some models (the type of density dependence and the timing of density dependence and stochasticity). Specifically, under compensatory density dependence, both juvenile and adult density, depending on the timing of density dependence and stochasticity, supported TL and gave estimates of the slope that were reasonable compared to empirical observations (Figs. 2 and 3; Online Resource B1-B16). However, under overcompensatory density dependence, the variance and mean relationships were more complicated (Figs. 4 and 5, Online Resource B17-B50). Our results show that a cusp or

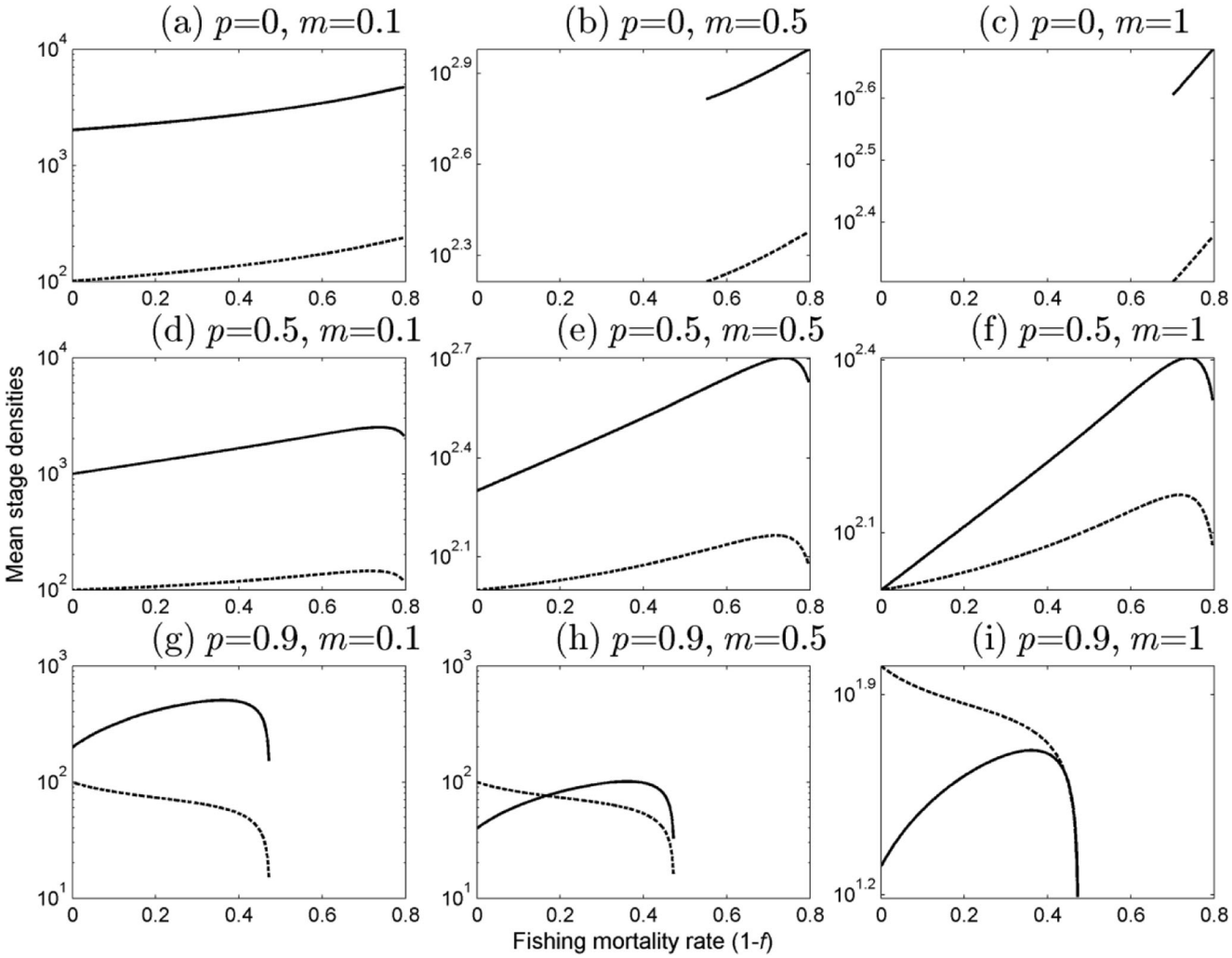

Fig. 6 Mean densities of juvenile (*solid curve*) and adult (*dashed curve*) stages as a function of a fishing mortality rate under a stochastic density-dependent fertility model. The simulations are under $\bar{s} = 0.5$, and other life history parameters are shown above each panel. Density dependence is overcompensatory γ=3. As we go down the panels, the adult survival rate is increased, and as we go to the *right*, a maturation rate is increased

fold (a singularity) can be observed in the variance–mean function of stage-structured density-dependent stochastic models. Similar singularities were also demonstrated with other population models previously (Cohen 2013, 2014; Jiang et al. 2014).

When fish populations are sampled in the field, a certain stage or a set of stages is often targeted; we rarely observe the entire population. Our results suggested that the stage observed matters because the mean and variance of stage densities could change differently among different stages as fishery exploitation was imposed (Figs. 2, 3, 4, and 5). In some cases, variance–mean curves had a positive slope for one stage and a negative slope for the other (Figs. 2, 4, and 5). This suggests that how we measure population density is very important in understanding its variance–mean relationship.

Similarly, fishery exploitation can also target different stages. However, the general patterns between the mean and variance relationships were similar whether juveniles, adults, or both were targeted by fishing (Online Resource B) although the ranges of a fishing mortality rate for stable positive equilibrium were different. This result is different from that in Cohen et al. (2012a). They showed that the mean–variance relationship of the size spectrum was insensitive to fishing mortality under "balanced harvesting," but it was sensitive when only larger individuals were targeted. There are two major differences in the models used in our study and Cohen et al. (2012a). First, we compared the mean and variance of the same stage of populations experiencing different levels of the fishery exploitation rates whereas Cohen et al. (2012a) calculated the mean and variance of the density of individuals of a given size and compared how the mean and variance changed with the individual size within the same population. Second, in our models, the source of fluctuation is stochasticity whereas, in the model of Cohen et al. (2012a), the source of fluctuation is deterministic periodic cycles.

With fish and other populations, the relative timing of density dependence and environmental fluctuation affecting a population is highly uncertain (Fujiwara et al. 2014; Ralston and O'Farrell 2008). In our results, the order in which individuals experienced density dependence and stochasticity was an important determinant of the mean–variance relationship. Under compensatory density dependence, if density dependence came before stochasticity, adult density followed the temporal TL, but juvenile density did not (Fig. 2, Fig. B1-B2 in Online Resource B). On the other hand, if stochasticity came before density dependence, juvenile density followed the temporal TL (Fig. 3, Fig. B9-B10 in Online Resource B). Under the latter model, adult density also followed TL when the annual juvenile survival rate was 0.5 (Fig. 3), but with another juvenile survival rate, adult stage might or might not follow TL (Fig. B9-B10 in Online Resource B). We suggest that the variance–mean relation may be used to elucidate the relative timing of the density dependence and environmental fluctuation affecting population rates by comparing the patterns in empirical data and theoretical models.

The type of density dependence also mattered. Under overcompensatory density dependence, the same mean could be associated with two different values of the variance in population density (and vice versa). The folding of the variance–mean curve occurred at the peak of the stock–recruitment relationship. This suggests that when formulating a population model, the density dependence chosen for the model needs to approximate the true process. If overcompensatory density dependence is used and the population density is estimated to be near the peak of the density-dependent curve (Fig. 1b), the variance of population density can have a non-monotonic relationship with its mean.

When the temporal TL was supported, the slopes of the variance–mean curves tended to be less than 2 (Figs. 2 and 3), so that, with increasing mean density, the CV decreased, i.e., the standard deviation did not increase as fast as the mean. In other cases, the log mean and log standard deviation were not linearly related (Figs. 4 and 5) or had a negative slope (Figs. 3, 4, and 5). These results suggest the CV is not generally an informative proxy for fluctuations in population density, or instability, and should be interpreted carefully.

We did not include age structure in the current analysis, but it would be interesting to extend the analyses to include age structure in future research. Age is often a strong determinant of survival, maturation, and reproduction for many natural populations. Age-structured and stage-structured population models differ because the latter can reduce the delay in the response of population density to environmental fluctuation and/or density dependence. This reduction arises because a stage-structured model may lump different age classes together in one stage. For example, in our density-dependent fertility model, increase in juvenile density by increasing fertility rate can increase population maturation rate immediately. However, in reality, individuals may need to spend a certain period of time in a juvenile stage before maturing, thus creating delay. Such delay along with density dependence can produce population cycles (e.g., Gurney et al. 1980), which are common dynamics with overcompensatory density dependence. Therefore, we speculate that incorporating age structure into a model with compensatory density dependence would cause the variance–mean relationship to become similar to the current model with overcompensatory density dependence, and incorporating age structure into the current model with overcompensatory density dependence would exacerbate the complexity of the variance–mean relationship. However, when multiple age classes in juvenile or adult stages are lumped together to calculate stage density, the effect of statistical averaging must also be considered.

Acknowledgments We thank A. Hastings and an anonymous reviewer for valuable feedback on a previous version of this manuscript. This project was developed during The Keyfitz Centennial Symposium on Mathematical Demography organized by John Bongaarts, Hal Caswell, Noreen Goldman, Josh Goldstein, Ron Lee, and Shripad Tuljapurkar in 2013. MF was funded in part by an Institutional Grant (NA10OAR4170099) to the Texas Sea Grant College Program from the National Sea Grant Office, National Oceanic and Atmospheric Administration, US Department of Commerce. JEC was funded in part by US National Science Foundation grants EF-1038337 and DMS-1225529. JEC thanks Priscilla K. Rogerson for assistance.

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