LETTERS

Survival variability and population density in fish populations

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To understand the processes that regulate the abundance and persistence of wild populations is a fundamental goal of ecology and a prerequisite for the management of living resources. Variable abundance data, however, make the demonstration of regulation processes challenging[1–3]. A previously overlooked aspect in understanding how populations are regulated[4–6] is the possibility that the pattern of variability—its strength as a function of population size—may be more than 'noise', thus revealing much about the characteristics of population regulation. Here we show that patterns in survival variability do provide evidence of regulation through density. Using a large, global compilation of marine, anadromous and freshwater fisheries data, we examine the relationship between the variability of survival and population abundance. The interannual variability in progeny survival increases at low adult abundance in an inversely density-dependent fashion. This pattern is consistent with models in which density dependence enters after the larval stage. The findings are compatible with very simple forms of density dependence: even a linear increase of juvenile mortality with adult density adequately explains the results. The model predictions explain why populations with strong regulation may experience large increases in variability at low densities[7]. Furthermore, the inverse relationship between survival variability and the strength of density dependence has important consequences for fisheries management and recovery, and population persistence or extinction[8–10].

Hitherto, the analysis of population density regulation has focused on the mean response of the per-capita rate of population change over population density; empirically manifested in tests of return tendency in abundance data[11]. This approach has considerably increased our understanding of population dynamics[5] and for many taxa, density-dependent regulation is readily discerned; however, highly variable populations (chiefly insects) can often defy attempts to detect density regulation of abundance[12]. Among highly variable taxa, fish populations have been somewhat neglected in the density regulation literature. In fact, the extreme variability of reproductive success in fish populations (Fig. 1) suggests that they provide ideal data for tests of proposed links between variability and the strength of population regulation[13]. We develop an alternative approach to understanding population regulation by focusing on the variance in survival. Using theoretical exposition and a meta-analysis of 147 wild populations, we demonstrate that survival variability in fish populations has a specific and consistent pattern, increasing with decreasing abundance. Moreover, we show that high variability does not preclude simple density regulation[14]. In the process, we demonstrate the viability of using patterns in the variance rather than the mean response to overcome the general ecological hurdle of interpreting markedly variable data.

Fish populations pass through a number of life-history stages, from egg to larval to juvenile, before joining the adult population. To analyse the effect of density dependence on the relationship between variability and reproductive adult abundance we examine models in which density-dependent mortality arises in the juvenile stage, an approach that has been shown to be suitable for many fish populations[15]. Stochastic mortality, independent of density, is assumed to take place during egg, larval and juvenile stages. Using these assumptions and a suite of commonly applied models for survival ranging from no density dependence (constant productivity) to extreme overcompensation[13] (survival continually declines with increasing abundance), we derive predictions for the relationship between survival variability and population density (see derivations in the Methods and Supplementary Information).

Figure 2 shows the predicted relationships between survival variability and adult abundance under different survival model

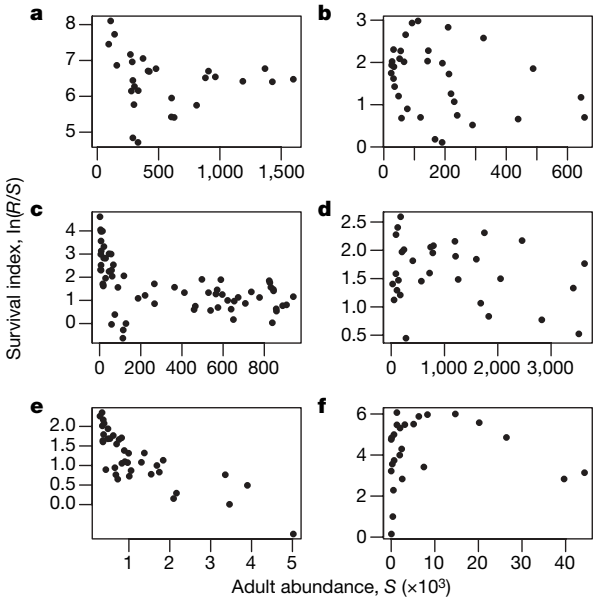

Figure 1 | Example relationships between the survival index and adult abundance. Examples of the Gadidae, Clupeidae and Salmonidae families, chosen to graphically accompany specific points made on the relationship between survival variability and population density. **a**, Cod from Labrador/northeast Newfoundland, Canada. **b**, Silver hake from the Mid-Atlantic Bight. **c**, Herring from Downs stock, North Sea, UK. **d**, Sardine from California, USA. **e**, Atlantic salmon from the Margaree River, Nova Scotia, Canada. **f**, Pink salmon from Sashin Creek, Little Port Walter, Alaska, USA. The greatest variability occurs for populations reduced to very low levels (Downs herring) and Icelandic spring-spawning herring. Extreme variation is shown in the Sashin Creek pink salmon population, for which the highest variation in survival occurs when the number of females spawning was reduced to below 300.

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formulations. In comparison to the density-independent form, all density-dependent models predict marked changes in the variance in survival over adult density; including a general increase in variance at low abundance where the models exhibit compensatory survival (increasing survival). The degree of compensation increases from Fig. 2a through to Fig. 2e. The variance in survival declines monotonically for survival models displaying only compensatory survival. For over-compensatory models where survival continually declines with increasing abundance with no asymptote (for example, the Ricker and Schaefer models in Fig. 2d and e) the variance in survival is predicted to initially decrease, then increase with adult abundance.

Maximum likelihood was used to estimate the parameters of a general Deriso–Schnute[16,17] survival model, assuming that the variance is not constant but follows a functional form of the explanatory variable[18] (adult density S) as in $\sigma^2 = \exp(\eta_0 + \eta_1 S)$ (see Methods). This parameterization enables us to estimate a coefficient of heteroscedasticity η_1, which indicates how much and in which direction the variance is changing over adult density in a given population. We then combine these estimates within and across species in a formal meta-analysis (see Methods).

Figure 3 shows the heteroscedastic coefficient estimates combined across populations by species under three different survival model formulations. There is a consistent trend indicated by both the fixed-effects and overall mixed-effects results for an inverse relationship between the variance in survival and adult abundance (see the individual fits in the Supplementary Information). Species for which there are more than four populations emphasize this point in that the decline in survival variability is generally conserved across different survival model formulations. We describe a general mechanism that can explain the changing variability in survival over adult abundance, density-dependent mortality in the juvenile phase following stochastic density-independent mortality in the egg and larval stages (see Methods).

If density-dependent population regulation decreases the variance in survival with increasing density, why then should we see high variability in strongly regulated populations? Assuming that density-dependent mortality is linear in log-abundance, the variance in survival is given by:

$$\mathrm{Var}(\ln(R_t/S_0)) \approx (1-\lambda)^2\sigma_\varepsilon^2 + \sigma_\delta^2 \quad (1)$$

where R_t and S_t are the number of recruits and the number of spawners at time t, λ is density-dependent juvenile mortality and σ_ε^2 is the variance in mortality in the egg and larval stages and σ_δ^2 is the variance in survival during the juvenile phase unrelated to density (see derivation in the Methods section). We choose the density-dependent juvenile mortality to be $\lambda \approx 0.5$ (ref. 15). This corresponds to very strong population regulation in that a hundredfold increase in the abundance of cod entering the juvenile stage would yield only a tenfold increase in the abundance of cod surviving the juvenile stage[15]. Such strong regulation might suggest that recruitment variability of cod should be weak, but this is not the case: cod populations typically have a standard deviation of log recruitment in the 0.5 to 1.0 range[19]. That recruitment variability is strong, despite regulation, is a consequence of the extremely large variability in larval abundance[20,21]. If the variance in the juvenile mortality unrelated to density is ignored[18] then $\sigma_{\ln(R/S)} = (1-\lambda)\sigma_\varepsilon$. Thus, $\sigma_{\ln(R/S)}$ will be reduced to about half of σ_ε; however, despite this attenuation, the large magnitude of σ_ε ensures that there will be strong survival variability. The key to understanding population regulation in this taxon is that although the observed survival variability may be high, this is the result of highly variable stochastic mortality in the larval phase but where density-dependent regulation occurs in the juvenile phase we observe marked patterns of change in survival variability over adult density (Fig. 3).

Our treatment does not amount to demographic stochasticity[22] alone, where individual fitness variance increases at greatly reduced abundances, accompanying population-level Allee effects. In fact, including depressed survival at very low abundances (depensation) only serves to exacerbate survival variability (Fig. 2f, depensatory Beverton–Holt model) on top of underlying changes across the whole

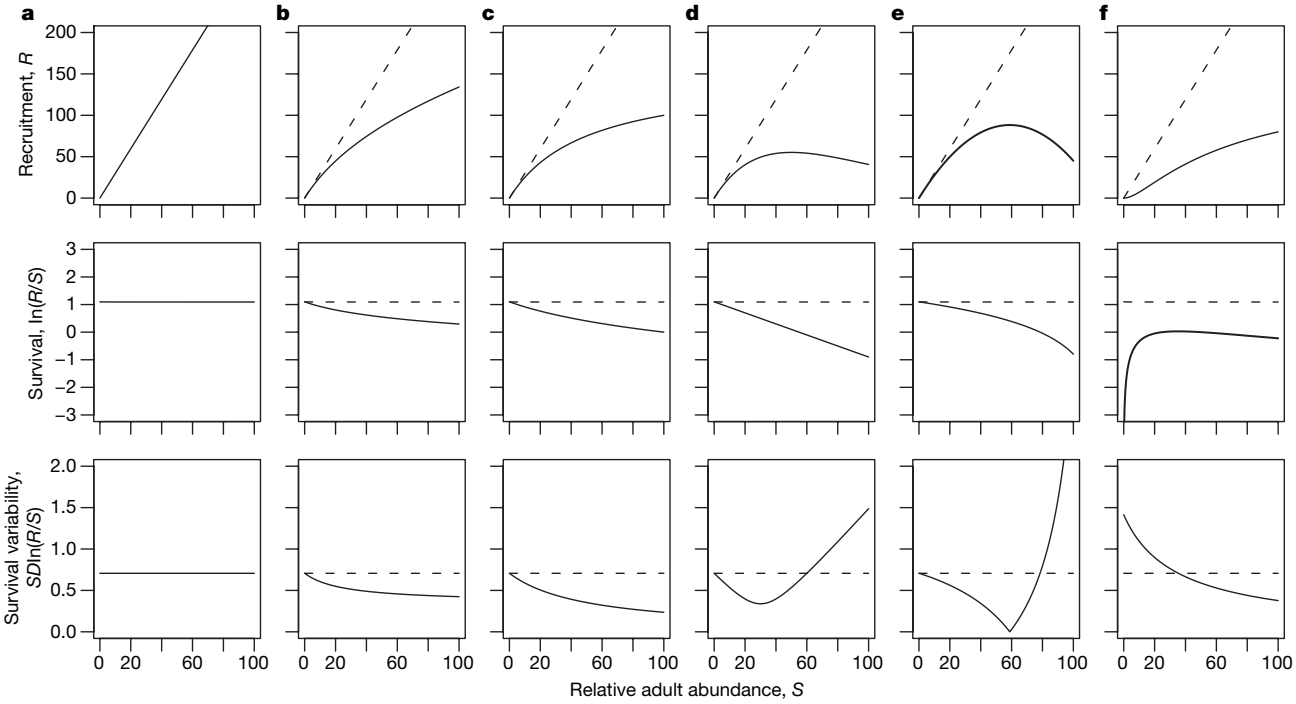

Figure 2 | The predicted relationships between adult abundance and recruitment, survival and survival variability. The models are realizations of the Deriso–Schnute general stock-recruitment model[16,17] with the shape parameter γ corresponding to: $\gamma = -1{,}000$ (**a**, no density dependence), $\gamma = -2$ (**b**, Cushing-like), $\gamma = -1$ (**c**, Beverton–Holt), $\gamma = 0$ (**d**, Ricker), $\gamma = 1$ (**e**, Schaefer) and $\gamma = -1$ (**f**, for the depensatory Beverton–Holt model; see Supplementary Information). The other parameters chosen were $\alpha = 3$ and $\beta = 0.02$ for all models except the Schaefer model, for which $\beta = 0.0085$ (Supplementary Information). The dotted lines are realizations in the absence of density dependence.

range of densities (Fig. 2c, the usual Beverton–Holt model). In addition, using the same data set, the presence of characteristic 'downward hooks' of recruitment over the adult abundance, indicating depensation, is debated[23,24].

Using the commonly applied Ricker and Schaefer models, which exhibit overcompensation (survival continually declines at higher adult abundances), we have shown that density-dependent survival variability will actually increase again at larger adult abundances (Fig. 3). This behaviour is not captured in approaches that predict that demographic variance will affect populations only at low abundances[25]. We have empirically found that the variability in survival is greater at low rather than higher abundances (see individual fits in the Supplementary Information); whereas the predictions for the Ricker and Schaefer models are that the variance should reach a minimum and then increase. This apparent discrepancy is because all the data we have used comes from exploited populations (most very highly exploited), so that we simply do not have data at high population levels[26].

Other mechanisms may be important for some species. Populations reduced to low abundances also have altered age and size structure. As cod populations were reduced by fishing, the reduction in older ages resulted in a reduction in the seasonal duration of spawning[27]. The reduction of the seasonal duration of spawning should increase the variability in survival of eggs and larvae because the probability that larval emergence will coincide with environmentally favourable conditions, such as the peak abundance of zooplankton, will be reduced[21].

In contrast to density-independent random walks, which allow populations to plummet to irrecoverably low densities, density dependence has long been considered a safeguard from population extinction[14]. From a fisheries perspective, survival should be sufficiently high at low densities so as to mitigate the effects of driving the population down. However, the greatest survival variability occurs for populations reduced to very low levels, such as Downs herring in the North Sea and Iceland spring-spawning herring, both of which were greatly overexploited[13].

Extreme variation is exhibited by pink salmon from Sashin Creek, Alaska, where the highest variation in survival occurs when the number of females spawning was reduced to below 300 (Fig. 1). The increased variance at lower adult abundance will result in higher extinction risk not accounted for in current projections[8,9]. An immediately practical implication for recovery is that current biological reference points and recovery projections are based upon the maximum reproductive rate at low population sizes, estimated from the slope of the stock-recruitment function at the origin. Recruitment is currently deemed to be lognormally distributed by assuming that the survival rates in each life-history stage are an independent random variable and the sum of these on the log scale is normally distributed[28]. This would imply that recruitment variability would increase with the mean recruitment and equivalently that survival should be normally distributed at a given abundance with a constant variance.

However, our treatment has shown the variance in survival to be in general non-constant over abundance (Fig. 2). If the maximum reproductive rate is estimated from the data here shown to be naturally heteroscedastic (non-constant variance), then erroneous recovery projections could result. Our model results show that survival variability can be inversely density-dependent in that the steepness of the increase in variability of survival as zero density is approached depends on the strength of the density dependence parameter (sensitivity analysis in Supplementary Information). Populations with very strong density dependence may exhibit greatly increased survival variability during population declines.

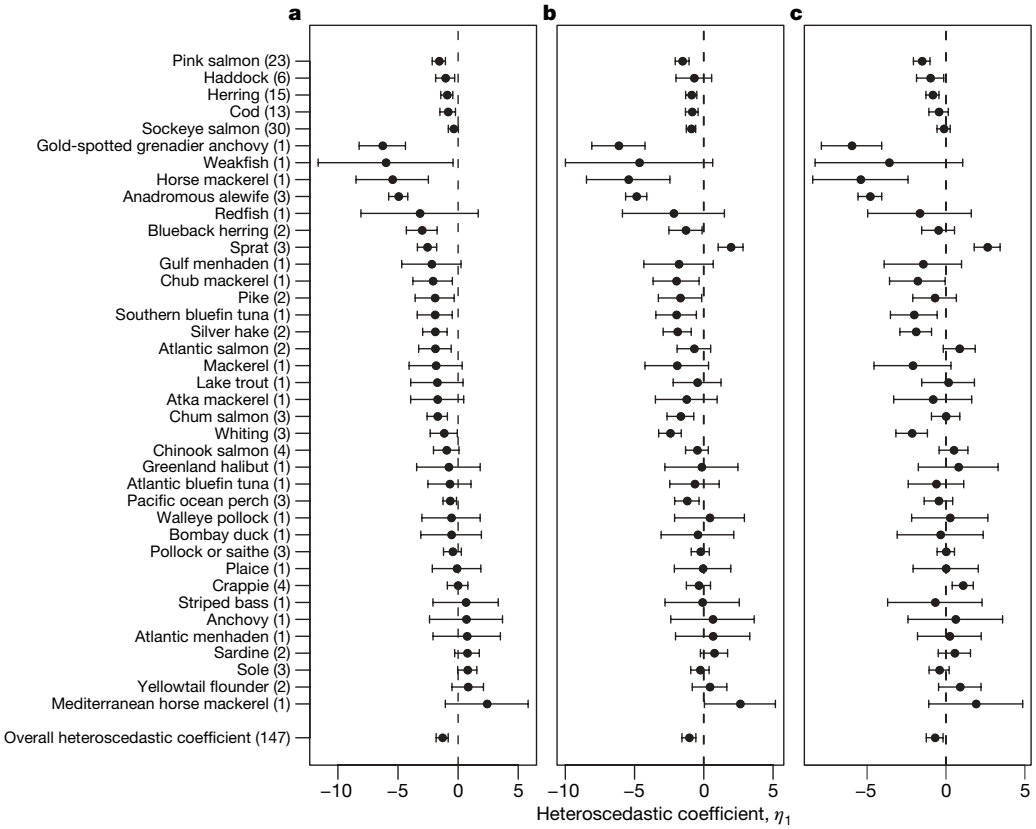

Figure 3 | Estimates of the heteroscedastic coefficient η_1 in survival across available fish species. Survival models: **a**, Schaefer ($\gamma = 1$); **b**, Ricker ($\gamma = 0$); and **c**, Beverton–Holt ($\gamma = -1$). The number of populations per species is given in parentheses and the error bars represent the 95% confidence intervals on the estimate. For species where the number of populations is greater than four, the estimate represents a fixed-effects estimate using all populations simultaneously. For species with four or less populations a weighted average of the individual population estimates is provided. An overall estimate of the heteroscedastic coefficient is provided by a random-effects meta-analysis (Supplementary Information).

Incorporating this heteroscedastic component by weighting will affect estimates of the slope at the origin and thus alter recovery projections for severely depleted populations.

METHODS SUMMARY

Models for the variance in survival. A derivation of the survival variability model is presented in the Methods and fully expanded upon in the accompanying Supplementary Information.

Data. The data come from a standardized global compilation of stock-recruitment data for over 500 species[29]. The data are standardized so that recruits and spawners have the same units[13]. To avoid the subsequent meta-analytical means being dominated by populations with large ranges of adult abundance and thus small standard errors, the recruits and spawners were further standardized to range between 0 and 1. Only data sets with at least 15 pairs of spawner recruit observations and where the ratio of the maximum observed adult abundance to the minimum was at least five were used. This was done to eliminate data sets which had little power to address the question[30] and resulted in the analysis of 147 populations of 39 species.

Likelihood. A log-likelihood function for a regression of survival $\ln(R/S)$ on spawning stock biomass S with normally distributed errors and a fixed survival mean $\mu_i = \ln(\alpha) + \ln(1 - \beta_\gamma S_i)^{1/\gamma}$ from the three-parameter Deriso–Schnute[16,17] stock-recruitment model (see Methods) at a given S_i and variance σ^2 is given by:

$$l(\mu, \sigma^2) \propto -\frac{1}{2}\sum_{i=1}^{n} \ln\sigma^2 - \frac{1}{2}\sum_{i=1}^{n} \frac{\left(\ln\left(\frac{R_i}{S_i}\right) - \mu_i\right)^2}{\sigma^2} \quad (2)$$

To investigate the relationship between survival variability and population density, the variance term can be re-parameterized as a functional form of adult abundance[10,18]. The log-likelihood is now written:

$$l(\mu, \eta_0, \eta_1) \propto -\frac{1}{2}\sum_{i=1}^{n} (\eta_0 + \eta_1 S_i) - \frac{1}{2}\sum_{i=1}^{n} \frac{\left(\ln\left(\frac{R_i}{S_i}\right) - \mu_i\right)^2}{e^{\eta_0 + \eta_1 S_i}} \quad (3)$$

If the variance is constant over adult abundance, the heteroscedastic coefficient $\eta_1 = 0$ and a constant variance is recovered at $\sigma^2 = e^{\eta 0}$.

Meta-analysis. A full description of the fixed and mixed-effects meta-analytical methods, used to estimate the heteroscedastic coefficients is provided in the Supplementary Information.

Full Methods and any associated references are available in the online version of the paper at www.nature.com/nature.

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Supplementary Information is linked to the online version of the paper at www.nature.com/nature.

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Author Information All data used are available at the stock-recruitment database www.mathstat.dal.ca/~myers/welcome.html. Reprints and permissions information is available at www.nature.com/reprints. Correspondence and requests for materials should be addressed to C.M. (mintoc@mathstat.dal.ca).

METHODS

Analytical models for survival variability. The number of fish that survive to a specified age (generally the youngest ages at which they are effectively sampled) is termed recruitment. Recruitment can be written as $R_t = E_t\exp[-(C_{1,t}+C_{2,t}+C_{3,t})]$, where E_t is the number of eggs produced in year t and $C_{i,t}$ is the cumulative mortality in stage i: $i=1$ for the egg stage, $i=2$ for the late larval stage, and $i=3$ for the juvenile phase[31]. Population sizes in the egg, late-larval and late juvenile phase (when they join the older population) are given by E_t, N_t and R_t, respectively. To analyse the effect of density dependence on the relationship between variability and reproductive adult abundance we examine a model in which density-dependent mortality arises in the juvenile stage, a treatment shown to be suitable for many fish populations[15].

Stochastic mortality, independent of density, is assumed to take place during both the larval and juvenile stages. The number of late-stage larval fish is $N_t = E_t\exp[-(C_{1,t}+C_{2,t})]$. The number of fish surviving from the late-larval stage through to the end of the juvenile phase is given by $R_t = N_t\exp[-(C_{3,t})]$. It is useful to formulate the above equations in terms of deviations from their means. Letting $C_{3,t} = \overline{C_3} + \Delta C_{3,t}$ and $\ln N_t = \overline{\ln N} + \Delta \ln N_t$ gives $\ln R_t = \overline{\ln R} + \Delta \ln N_t - \Delta C_{3,t}$, where $\overline{\ln R} = \overline{\ln N} - \overline{C_3} = \ln\left(N_*\exp\left(-\overline{C_3}\right)\right)$ and N_* is the geometric mean abundance of the late-larval stage[31]. Rearranging the expression for $\ln R_t$, we obtain $R_t = R_*\exp(\Delta\ln N_t - \Delta C_{3,t})$, where R_* is the geometric mean recruitment (see Supplementary Information for a full derivation). The effect of density-dependent mortality can be incorporated by writing:

$$\Delta C_{3,t} = f(\Delta \ln N_t) + \delta_t - \bar{f} \quad (4)$$

where f is an as-yet-unspecified function representing density dependence, δ_t represents mortality in the juvenile stage unrelated to density and $\bar{f}$ is the time average of $f(\Delta\ln N_t)$. Letting $\varepsilon_t = \sum_{i=1}^{2} \Delta C_{i,t}$ be the sum of the de-meaned mortalities in the egg and larval stages, it has been shown[19] that:

$$\Delta \ln N_t = \Delta \ln E_t - \varepsilon_t \approx \Delta \ln S_t - \varepsilon_t = \ln S_t - \ln S_* - \varepsilon_t = \ln(S_t/S_*) - \varepsilon_t \quad (5)$$

where E_t is egg production, $\Delta \ln S_t = \ln S_t - \overline{\ln S}$, S_* is geometric mean adult abundance, and we have used $\Delta\ln S_t \approx \Delta\ln E_t$, valid when egg production is linearly related to adult abundance. Substituting equations (5) and (4) into the expression for recruitment gives:

$$R_t = R_*\frac{S_t}{S_*}\exp\left(-\left(\varepsilon_t + f(\Delta \ln S_t - \varepsilon_t) + \delta_t - \bar{f}\right)\right) \quad (6)$$

At a given adult abundance, $S_t = S_0$, log survival is $\ln(R_t/S_0)$, so, from equation (6), we have:

$$\ln(R_t/S_0) = \ln(R_*/S_*) - \varepsilon_t - f[\ln(S_0/S_*) - \varepsilon_t] - \delta_t + \bar{f} \quad (7)$$

One conclusion is immediately apparent from equation (7): the variability of survival, $\mathrm{Var}[\ln(R_t/S_0)]$, will be independent of adult abundance only if $f[\ln(S_0/S_*) - \varepsilon_t]$ is linear. Specifically, we can write $f[\ln(S_0/S_*) - \varepsilon_t] = \lambda[\ln(S_0/S_*) - \varepsilon_t]$; prescribing a density-dependent mortality that is linear in log-abundance is the core of key factor analysis[32]. The variability of survival is now given by equation (1). Here survival variability is independent of S_0 because the linearity of density-dependent mortality in log abundance allows adult density to drop out of the variance derivation (Supplementary Information). The effect of density-dependent juvenile mortality λ is clearly to reduce survival variability. An analogous expression was previously obtained for the Gompertz form of population growth[9].

Now we consider common models and how the variability in recruitment and survival is a function of egg abundance. For the commonly applied Ricker spawner-recruit function, where survival is a linear function of adult abundance:

$$f[\ln(S_0/S_*) - \varepsilon_t] = \exp[\ln(S_0/S_*) - \varepsilon_t + \kappa] = \beta S_0\exp(-\varepsilon_t) \quad (8)$$

where $\beta = e^{\kappa}/S_*$. From equations (8) and (6), we have:

$$R_t = \alpha S_0\exp(-\beta S_0 e^{-\varepsilon_t} - \varepsilon_t - \delta_t) \quad (9)$$

where several constants have been combined into the parameter α. In the limit of zero noise, we obtain from equation (9) the Ricker form, $R_t = \alpha S_0\exp(-\beta S_0)$. The variability of survival is:

$$\mathrm{Var}[\ln(R_t/S_0)] = \sigma_\varepsilon^2 + \beta^2 S_0^2\mathrm{Var}(\exp(-\varepsilon_t)) + 2\beta S_0\mathrm{Cov}(\varepsilon_t, \exp(-\varepsilon_t)) + \sigma_\delta^2 \quad (10)$$

The variance term on the right side of equation (10) may be obtained from the variance of a log normal distribution, $\mathrm{Var}(\exp(-\varepsilon_t)) = \exp(\sigma_\varepsilon)(\exp(\sigma_\varepsilon) - 1)$, assuming $E[\varepsilon_t] = 0$, and the covariance term may be analytically calculated (see Supplementary Information) to yield, for small ε:

$$\mathrm{Var}[\ln(R_t/S_0)] \approx \sigma_\varepsilon^2(1-\beta S_0)^2 + \sigma_\delta^2 \quad (11)$$

which can also be approximated using the delta method (Supplementary Information). In general, it is apparent from equation (11) that for large $\beta S_0 > 1$, the term containing $\beta^2 S_0^2$ will dominate and the variability of survival will increase with S_0. For $\beta S_0 \ll 1$, the term in $2\beta S_0$ will dominate that containing $\beta^2 S_0^2$ and thus the variability of survival will decrease as S_0 increases. Therefore, the variability of survival will have a bowl-shaped dependence on S_0 (Fig. 2d, $\gamma = 0$ Ricker, bottom panel). As σ_ε increases the bottom of the bowl will be pushed closer to the $S_0 = 0$ line (Supplementary Fig. 1).

Delta method approximations to survival variability. For population growth models that do not exhibit linearity between survival and adult abundance, the variance term is analytically intractable; here we use a delta approximation to the variance of a function[33]. To generalize the results across commonly applied models, the three-parameter Deriso–Schnute[16,17] stock-recruitment model is used:

$$R_t = \alpha S_0(1-\beta\gamma S_0)^{\frac{1}{\gamma}} \quad (12)$$

$$\ln(R_t/S_0) = \ln(\alpha) + \frac{1}{\gamma}\ln(1-\beta\gamma S_0) \quad (13)$$

Here the variance in survival is approximated using a delta method approximation (Supplementary Information) to yield:

$$\mathrm{Var}(\ln(R_t/S_0)) \approx (1-\beta S_0/(1-\gamma\beta S_0))^2\sigma_\varepsilon^2 \quad (14)$$

Possible models of survival range over a degree of compensation continuum between constant productivity (no density dependence) when $\gamma \ll -1$ and high degrees of over-compensation when $\gamma \geq 0$ (for example, the Ricker and Schaefer model). We fix $\gamma \in \{-1{,}000, -2, -1, 0, 1\}$ for cases of no density dependence, Cushing-like density dependence (non-asymptotic recruitment), Beverton–Holt compensation, and Ricker and Schaefer overcompensation models, respectively. $\sigma_\varepsilon^2 = 0.5$ and $\beta = 0.02$ for all models except the Schaefer model for which $\beta = 0.0085$ (Supplementary Information).

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