TECHNICAL REPORT
TO THE
CALIFORNIA COASTAL COMMISSION

M. Bight-wide Effects on Fish: Compensation

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INTRODUCTION

This report deals with a difficult and unsolved question in fisheries biology: how, and to what extent, can populations of marine fish "compensate" for additional negative impacts imposed upon them?

In this case the additional impact is the annual killing by SONGS of billions of early life stages of several fish species. The ability of the populations to compensate for these deaths will determine whether this is one of the most severe, perhaps even the most severe, negative biological effects of SONGS, or whether it is a minor effect.

Unfortunately, unlike other potential effects of SONGS examined by the MRC, direct sampling will not tell us what the effect has been. Because fish, including their immature stages, move a great deal, any effects will be spread out over a large area. Consequently there will be no "Control" area with which the Impact area can be compared; and even a major effect will be so diluted that the change will be indistinguishable from natural variation except by a massive sampling program over many years. Thus the only way for us to assess the effects of the killing of the immature stages is to try to convert the estimated numbers killed into changes in the population size, by means of models.

These models must include factors representing compensation. There is little doubt that compensation occurs to some extent: without it, even the slightest additional source of mortality would cause a population to decline inevitably to extinction. There is also little doubt that compensation is limited: if enough

additional sources of mortality are imposed, any population will eventually be driven extinct. Further, the ability to compensate must vary: some populations may already be compensating for additional mortality imposed by, for example, fishing or other power plants, while others are not. How far can a particular population compensate for mortality such as that imposed by SONGS on immature stages?

Many researchers have examined and failed to answer this question in detail or with precision (e.g., Barnthouse and Van Winkle 1988, Fletcher and Deriso 1988). We can do no better. Instead, we have assembled in this report the arguments behind the broad claim in Chapter 10 that likely compensation mechanisms will not prevent a reduction in the average abundance of adult fish.

We have also attempted to determine the amounts by which some populations seem likely to decline, based on their Adult Equivalent Losses. It is important to note that, unlike the losses given for other species, which are based on estimates of varying precision (which itself can be estimated), the numbers given here are little more than sophisticated guesses whose reliability is quite unknown.

1. THE MEANING AND IMPLICATIONS OF COMPENSATION

Populations of fish fluctuate in density through time. However, few species are observed to go extinct or to increase without any apparent limit within ecological time spans. It can be shown from the study of simple theoretical models that any population in which the *per capita* probabilities of a birth or a death over a specified time interval are unaffected by population density must ultimately behave in one of three ways:

(i) unlimited growth

(ii) decline to extinction

(iii) fluctuations of the sort associated with a "random walk"

Possibility (iii), the random walk, only occurs in the highly unlikely situation where the *per capita* probabilities of a birth or a death happen to balance exactly. If any of the fish populations influenced by SONGS had density-independent birth and death probabilities with parameter values that placed them on this "knife-edge" of viability, then the effect of SONGS would be serious, as *any* additional mortality would start the process of decline towards extinction.

However the fact that the fish populations under study have survived for many generations in spite of natural environmental variability suggests that these populations are to some extent "regulated," which would imply "density dependence" of *per capita* birth and death rates. In their simplest form, such density dependent processes will tend to cause a population that increases above its equilibrium level, or decreases below it, to return to that equilibrium. So the dynamical system of which the fish population is a part can be thought of as being composed of some processes whose effect on the birth or death rate of the population depends on how many fish are present (density dependent processes), and other processes whose effects do not depend on the number of fish present (density independent processes). The latter inevitably vary in their intensity through time and so contribute strongly to fluctuations in the fish population. The population will not remain regulated in the presence of these density independent driving forces unless countervailing density dependent processes occur. Although the detailed dynamics

of real fish populations are typically more complicated than this simplified description, such additional detail will not affect our broad conclusion that density dependent processes must be operating.

We define *compensation* to be the effect on population size of density dependent factors operating on *per capita* birth and death rates. It occurs because the individual fish responds to its environment, which includes other individuals like it: a reduction in the density of like individuals may make the environment more hospitable for those that remain. For example, reducing the density of larvae might cause the remainder to survive or grow better. Density dependent factors may also result in rate processes associated with one life stage being influenced by the population density in another stage; we then use the terminology "compensation *in* stage x *in response to* stage y" to mean that the rate of some process (development, survival or reproduction) for individuals in stage x is affected by the population density of individuals in stage y.

Because of density dependent factors, we do not in general expect the addition of new density independent mortality (such as that imposed on immatures by SONGS) to drive fish populations extinct. Eventually, as a result of the lowered density of either immatures or adults, the remaining individuals (either immatures or adults) can be expected to do better: either the birth rate will increase or the death rate from causes other than SONGS will decrease sufficiently for the population to persist.

However, the average population abundance is likely to decrease. Sardines, white seabass, barracuda, yellowtail, Pacific mackerel, and pelagic sharks are all

California marine fish species that have declined in the face of additional man-imposed mortality, in spite of the probable presence of compensation (CalCOFI 1983). In particular, an increase in the death rate of immatures, such as is caused by SONGS, can be expected to lower the average (or equilibrium) abundance around which the adult population fluctuates.

The remaining two sections of the report are an attempt to justify these assertions and quantify some of them. We do not have the appropriate data to test for compensation in the species potentially affected by SONGS, so we are forced to rely on indirect arguments. In Section 2 we assess the evidence for compensation in three species of marine fish amassed in a recent review, and establish candidate mechanisms through which compensation might operate for fish affected by SONGS. In Section 3, with the aid of a suite of very simple mathematical models, we examine the likely effect of SONGS-induced mortality and compensation on the average abundance of hypothetical fish species in which compensation operates through those mechanisms identified in Section 2.

2. EVIDENCE FOR COMPENSATION IN MARINE FISH

The most recent and thorough review of evidence concerning compensation is presented by Saila *et al.* (1987). We have examined evidence in that review relating to three species of marine fish, to see where and how compensation might operate in the life history. The review, several of the original data papers, and the ecological literature in general, form the basis for our discussion.

Below, the numbers in parenthesis refer to pages in chapter 3 volume 1 of the review by Saila *et al.*

Of the 13 species considered by Saila *et al.*, only three spend all of their lives (including the egg and larval stages) entirely in the ocean: Pacific herring, northern anchovy and Atlantic cod. A fraction of the early stages of a fourth, mainly marine, species (winter flounder) is found in estuaries and rivers. The remainder are either freshwater or anadromous species. Since the species of interest at SONGS are entirely marine, we concentrate on the results for herring, anchovy and cod.

Saila *et al.* (1987) concluded that compensation occurs in both the adult and immature stages of fish in general. They concluded there was compensation in the adults of all three purely marine species, although the evidence is weak for herring. They also argued that there was compensation in the immature stages of cod and anchovy. Overall, they concluded there was only weak evidence for density dependence in herring, perhaps occurring via **reproduction**, and that the populations are very sensitive to increased negative impacts (12 *et seq.*). For anchovy they concluded that **cannibalism** [of immatures] "is clearly density dependent," and speculated that **predation** upon the eggs and larvae (27, 28), **fecundity, immature growth** and **starvation**, and **fishing** on the adults (28) may be important density dependent factors. In cod (35 *et seq.*), **adult growth** has been shown to be density dependent. There is also evidence for **earlier maturation** at lower adult densities, and density dependent fecundity. They conclude that "density dependent mechanisms determining recruitment in species such as cod must be taking place at the juvenile and late juvenile stages" (42).

We agree with Saila *et al.*, that there is adequate evidence for compensation by the adults of these species.

However, we disagree about the adequacy of the evidence for compensation by the immature stages, mainly because we differ concerning what we can take for granted *a priori*. We shall argue that there is no convincing evidence for immature compensation in their three marine species, and further that there are *a priori* grounds for suspecting that compensation in the immature stages may be absent or weak.

We do not claim that there is no compensation in immatures; there may be. Our point is, rather, to illustrate how little is known about these processes and how little can safely be assumed. In fact, in Section 3 we examine the probable consequences of compensation by immatures as well as by adults.

2.1 Compensation in Immature Fish

Saila *et al.*'s reasons for concluding that the immatures of the three marine species compensate are both general and particular. The general arguments amount to a claim that food, predation and cannibalism may be expected *a priori* to affect the immatures in a density dependent way, and that reductions in the density of immatures will lead to:

(1) increases in their food intake and hence increases in their growth rate and survival, and

(2) decreases in the predation and cannibalism rates.

Claim (1): increase in food. Our rejection of this claim is based on the known dynamics of feeding in relation to food supply. Saila *et al.* appear not to accept, or to be uncertain of the validity of, the broad generalization that the rate of intake of food depends primarily on the density of the food, and not *directly* on the instantaneous number of individuals searching for the food. Even where interference among individuals may influence feeding rates, this effect is likely to be insignificant in comparison with the effect of variations in food density (see Crowley *et al.* 1986 for a sample calculation).

We believe, on the basis of extensive existing ecological studies of feeding behavior in many different organisms, that the rate of intake of food, for an organism of a given physiological state, is determined by the rate of encounter with potential food items and hence by the density of food (Peters 1983). In other words, the amount of food consumed by an individual depends on how much food it encounters, and this depends almost entirely on how much food is in the environment and hardly at all on how much time the individual wastes in encounters with other individuals. Thus, if the supply of food in the habitat is constant, then the amount consumed by any individual will not be increased by removing other individuals from the habitat, unless these removed individuals were causing a reduction in the amount of food in the habitat.

An opposing view has been expressed (Arditi and Ginsburg 1989), but we disagree with some of the theoretical arguments and note that the evidence offered

in support applies only to rather special systems which are very different from fish in the open ocean.

It follows that *a reduction in the density of immature fish, owing to intake mortality in a power plant, will lead to an increase in the rate of intake of food by the remaining immatures only if the density of food particles increases. This will occur only if (i) food density had previously been suppressed by the immatures, and (ii) the release from this suppression is not cancelled by the introduction of another source of food suppression.*

It is possible for these conditions to be satisfied. But for the species considered by Saila *et al.*, the food particles come from such a variety of sources and are eaten by such a variety of species that the burden of proof seems to rest with those who argue that the affected fish species had been suppressing the food supply. In addition, condition (ii) will be violated if SONGS removes the food of the immatures in about the same proportion as the immatures themselves.

We turn now from these general arguments to the evidence on compensation, via the feeding response of immatures, of the three purely marine fish species reviewed.

Saila *et al.* provide no evidence that immatures of the species in question suppress their food supply. Evidence that the food supply may often be below that required for immature development does *not* demonstrate that the immatures themselves suppress their food supply: there are many possible reasons for this

suppression. There is no evidence for herring or anchovy; indeed MacCall (1980) argued that such suppression might not occur in anchovy even at a local scale.

Nor does the review provide evidence that the growth rate or survivorship of the immatures of any of the three species increases as their own density declines. In the cod, Saila *et al.* concluded there was little or no evidence for density dependent growth in the first year of life. There *is* some evidence for density dependent decrease in age at maturity - apparently over several to many decades (36). This does not reflect an increase in immature growth rate, however, but rather maturation at a smaller size.

The absence of a change in growth rate is important, since Saila *et al.* suggest that faster maturation at lower immature density may cause a density dependent reduction in losses to predators - because the fish escape more quickly the higher predation rates associated with immaturity. However, predation rates are likely to be related to maturity only because mature fish are bigger; i.e., it is size, not sexual maturity, that determines vulnerability to predation, so only an increase in immature growth rate is likely to lead to reduced predation. In sum, there seems to be no good evidence for density dependence acting on immatures via the food supply.

(We note that earlier maturation can have a compensatory effect even when there is no change in growth rate. If there are no other changes, earlier maturation implies a greater probability of reaching the reproductive stage, and a longer period in this stage, for each individual. However, it is most unlikely that there will be no other changes. Earlier maturation is likely to lead to smaller adults, since a period previously devoted to growth is now devoted to reproduction. This is likely to lead

to reductions in survival, fecundity (e.g., the number of eggs an individual can produce) and biomass. This is a very complicated set of possibilities, not suggested by Saila et al., difficult to model and, we feel, unlikely to lead to a significant increase in population size or biomass. We therefore do not consider it further.)

Claim (2): Predation and Cannibalism. Saila *et al.*'s claims that these processes are density dependent rest mainly on an assertion that this is true in general.

There is, however, an enormous literature on predation. In our view, the weight of that body of evidence points to the short-term effects of predation (i.e., the response of individual predators) as typically either density independent or even inversely density dependent (see Murdoch and Bence (1987) for a review). Inverse density dependence would lead to depensatory (the opposite of compensatory) mortality, i.e., mortality rates would increase as the population decreased.

The long-term effect of predation may be compensatory. As their food supply decreases, the number of predators may decline, thus decreasing the predation pressure. However, although very little is known about predation on immature fish, it seems unlikely that any predator population depends heavily on this source of food: for most predators, immature fish are probably not particularly different from any other planktonic material. Thus we would not expect a significant decline in the predators of immatures in response to increased losses caused by SONGS.

The situation with cannibalism is more complex. The predatory behavior of individual cannibals (larger immatures) feeding on their own species presumably obeys similar rules to predation: the short-term ("functional") response of predators is typically a "type 2," which causes depensatory mortality because of satiation or handling time effects; this is typically the case even when the predator feeds on several species. However, we *can* expect a longer-term density dependent feedback process to operate since losses of immatures to SONGS that are not compensated for should lead to fewer cannibals, which in turn will reduce the cannibalism rate on smaller immatures. We explore this effect with the aid of a model in Section 3.

Turning to the evidence specific to the three purely marine species, we were not able to find reliable evidence for density dependent predation or cannibalism. Saila *et al.* make no claim of finding density dependence in herring. In the case of the anchovy, the paper confuses temporal density dependence and spatial density dependence. Temporal density dependence occurs when an overall rate (of death, birth, growth, etc.) varies with the density of the population as a whole. It leads to compensation. Spatial density dependence occurs when the population is patchily distributed, and rates in dense areas are different from rates in sparse areas. This is as likely to cause depensation as compensation - see Murdoch and Stewart-Oaten (1989). At least one piece of evidence presented on anchovy shows predation to be depensatory (23), and the remaining evidence (24) does not specify whether the *number* or the *fraction* of larvae eaten increased with their density (only the latter implies density dependence). Saila *et al.* state that predation upon immature cod is believed to be the most important density dependent mechanism controlling year-class strength (38), but no evidence is presented.

One form of compensation is potentially particularly important because it could completely nullify the SONGS effect or even lead to an increase in the adult population. This is where the compensation acts in a late stage in response to a decrease in an earlier stage. It can arise in more than one way, but a simple mechanism is the "constant yield" condition. The hypothesis here is that there is a bottleneck at the late juvenile stage, so that, over a wide range of larval densities, a more or less constant number passes through the late juvenile stage to adulthood, regardless of the number entering the stage. This could occur, for example, if there were a fixed number of refuges or territories for late juveniles. There is no evidence for this type of compensation in the three species under discussion, and nothing in the biology of the species of concern at SONGS indicates that this mechanism could be operating.

2.2 Compensation by Adult Fish

There is evidence for density dependent *growth* of adults in some herring populations but not in others (13, 14). Density dependent adult growth has also been found in both the anchovy (20) and cod (35). This can be expected to lead to density dependent *fecundity*, which has been separately observed in anchovy (21, 22) but has not been well-established in cod, except via increased growth (37).

There is no evidence for increased adult *survival* at lower stock sizes in the three species studied. The relationship between adult survival and adult density is notoriously difficult to estimate, however, and it is possible that there might be such a response to increased food intake. On the other hand, McCall (*pers. comm.*) suggests that at least one source of adult mortality is likely to be depensatory:

marine birds and mammals that feed on adult fish are such information-rich feeders that they are likely to take a constant *amount* of food over a very wide range of fish densities. (He notes that in this sense these predators are analogous to modern information-rich fishing that uses technology such as sonar and aerial surveys to achieve similar constant yields regardless of fish density.)

If the loss of immatures leads to a decrease in recruitment to the adult population, this may have an effect on the predators of the adults. However, this may make the effect of SONGS more serious, by transferring it from fodder fish to sport and commercial species. We discuss this as "Case 4" in our set of models in Section 3.

These results suggest that adult cod and anchovy populations, and some herring populations, were indeed suppressing their food supply, which presumably increased in response to reduced adult abundance. Overall, there seems to be quite good evidence for density dependent growth and fecundity in adult marine fish, while effects on survival are still unknown.

2.3 Summary

In summary, there seems to be adequate evidence in wholly marine species for various compensation mechanisms involving a response to adult density. These include density dependent growth, fecundity and time-to-maturation, and possibly survival, that depend on adult density. The fraction of immatures cannibalized may also increase with increasing adult density, though there does not seem to be good evidence for this. The evidence is at best weak that immature stages (larval or

juvenile) compensate for reductions in immature density, for example by faster growth or higher survival.

3. CONSEQUENCES OF IMMATURE MORTALITY UPON FISH STOCKS

In this section, we use simple models of fish populations to evaluate how a SONGS-induced increase in immature mortality might affect the average density of adult fish. Two types of fish are killed as immatures by SONGS. One is "fodder fish" (e.g., queenfish), which are important mainly as food for the other type, namely piscivorous sport and/or commercial species. Compensation can affect these two types of fish differently.

3.1 Aims of the Models

Ideally, we would like to write detailed models of the dynamics of the fish populations in the Bight, based upon extensive information about the real populations. The information required to develop such models would include, for example, fecundity, development rate, and death rate for each age class, annual variation in these rates, and any dependence on density. Such information is not available for any of the species of concern, and believable, detailed models are thus out of the question. We therefore use the simplest models that incorporate what we regard as the essential features of fish life history.

The models are intended to provide a more rigorous guide than mere intuition to the likely consequences, for adult stocks, of various possible forms of

compensation. By making assumptions explicit they also allow the reader to judge whether they are acceptable. They do *not* aim to be realistic portrayals of the detailed dynamics of any particular fish populations, nor are they intended to provide a precise measure of potential changes in stock size. They recognize only two classes of each fish species: immatures and adults; they are deterministic; they do not recognize spatial variation; and they look only at effects on numbers, not on biomass.

Three conclusions are reached:

(1) Most forms of compensation examined fail to prevent a decline in adult stock in response to SONGS-induced mortality of immatures; the fractional decline might plausibly be about the same size as the proportion of immatures killed by SONGS.

(2) The adult population is unlikely to be destabilized by the action of SONGS. The most important exception to this generalization is the situation (which we believe rare) where there is sufficient compensation in the late juvenile stage (i.e., *after* SONGS impact but before maturity) to prevent an appreciable fractional decline in adult stock.

(3) The most likely effect of entrainment of immatures of a "fodder" fish that is eaten by sport/commercial species is a reduction in the equilibrium adult abundances of the latter, with only a small change in the abundance of the fodder fish.

3.2. Compensation Mechanisms Modelled

Following Section 2 we recognize only two stages in the models below: adults and immatures.

Adults can compensate by increased survival, growth or fecundity. Since our discussion focuses on individuals, not biomass, we do not discuss growth *per se*: compensation by increased size becomes an implicit part of our discussion of compensation by increased survival or fecundity. Adult compensation will be in response to the density of adults only, since immature individuals do not compete with adults or prey on them.

Immatures can compensate by increased survival or reduced time to maturation. As discussed above, an adequate model of reduced time to maturation involves too many complexities and too much species-specific detail, compared to its likely importance, for it to be modelled here. However, we do consider immature compensation in response to the density of immatures (e.g., via decreased food suppression) and to the density of adults (e.g., via decreased cannibalism).

Guided by section 2, we have classified the major potential mechanisms as follows:

Case 1. *Responses by adults.*

(a) Adults experience higher fecundity at lower adult density.

(b) Adult survival may increase when there are fewer adults.

Case 2. *Response by immatures to adult density.*

(a) Cannibalism of immatures by adults decreases as adult density decreases.

(b) Survival of immatures is higher at lower adult density (e.g., each egg may be larger).

Case 3. *Response by immatures to immature density.*

(a) Planktonic and juvenile stages survive better at lower densities.

(b) SONGS-induced mortality on late juveniles is so small it can be ignored, and late juveniles survive better at lower densities.

(c) Cannibalism of planktonic stages by juveniles decreases as juvenile density decreases.

Case 4. *Predation by other species.*

(a) The affected species is a major prey item for a predator (e.g., a sport/commercial) species.

Cases 1(a), 2 and 3(c) cover the mechanisms that emerged from Section 2 as the prime candidate mechanisms for compensation. That section gave little support for compensation via increased adult survival, and none for compensation via increased immature survival in response to reduced immature density, but in view of the inadequacy of the evidence, we have included some discussion of these mechanisms (cases 1(b) and 3). Case 4 was not covered in Section 2, but in view of our knowledge that SONGS entrains fodder fish, this aspect clearly merits investigation. The only candidate mechanism not modelled is dependence of time to maturity on adult density, for reasons given above.

Case 2 will not be separately modelled below. Although it gives responses by immature stages, it is covered by the increased fecundity of adults, Case 1(a): we merely reinterpret "immatures" to mean juveniles only, and "fecundity" as the ability to produce new juveniles rather than eggs or plankton.

3.3. A "Single-Species" Model and its General Properties

In our investigation of most of the forms of compensation, we make extensive use of one particular, "single-species" model. To avoid excessive repetition we now state the model, and establish some of its general mathematical properties, in particular equations for equilibria and conditions for stability of these equilibria. (We include some of the algebra, so interested readers can check the results.) The only cases identified in section 3.2 that cannot be discussed using this model are case 3(c) (Cannibalism) and case 4 (Predation by other species). Models for these cases will be proposed at the point in the text where they are required.

The model recognizes that although there are three stages (adult, planktonic and juvenile) we need only explicitly represent two: adults and immatures. Depending upon the mechanisms we wish to investigate, the immatures can include both the planktonic and juvenile stages, or only the latter. We assume that reproduction occurs in a short period each year and produces a distinct cohort of immatures. An appropriate discrete-time formulation for fish populations consisting of a juvenile stage lasting one year and an adult stage is then:

$$I_t = bf(A_t)A_t \qquad (1)$$

$$A_{t+1} = Sg(I_t)I_t + S_A h(A_t)A_t \qquad (2)$$

where

I_t	=	density of immatures at time t,
A_t	=	density of adults at time t,
b	=	maximum number of "births" per adult, where a "birth" is an egg that hatches and survives through the planktonic stage to become a juvenile (except in case 3(a), where it is a newly hatched planktonic larva);
$f(A_t)$	=	ratio of per capita birth rate when the adult density is A_t to the maximum rate;
S	=	S_I, maximum survival of immatures at low immatures density;
$g(I_t)$	=	ratio of immature survival when immature density is I_t to maximum immature survival:
S_A	=	maximum year to year survival of adult fish;
$h(A_t)$	=	ratio of adult survival when adult density is A_t to maximum adult survival:

SONGS affects b and S. Compensation occurs through f, g and h. We assume these are non-increasing functions, with at least one of them being strictly decreasing, so a SONGS-induced decrease in adults or juveniles will lead to increased (or, at least, not decreased) fertility or survival. Thus $f(0) = g(0) = h(0) = 1$. (It is possible that, in real life, one of these functions actually increases over some range, e.g., the Allee effect, but this would make the effect of SONGS even worse.)

Note that the left side of equ(1) is I_t, not I_{t+1}. For example, the birth process (including hatching and survival through the planktonic stage) could take from April 1 to May 1; the juvenile stage could last until the following March 31, when surviving juveniles would become full adults, and contribute to the next set of births. A_t would be the number of adults on April 1, and I_t the number of juveniles on May 1, of year t.

I_t can therefore be eliminated from the model equations. The numbers of adults in successive years are related by

$$A_{t+1} = F(A_t,b,S)A_t \tag{3}$$

where

$$F(A,b,S) = Sbf(A)g(bAf(A)) + S_Ah(A). \tag{4}$$

The equilibrium population satisfies

$$F(A^*,b,S) = 1. \tag{5}$$

Once this is solved for A^*, the immature density is given by

$$I^* = bA^*f(A^*).$$

Equs (4) and (5) express the fact that the average lifetime production of each adult, $bf(A)/[1 - S_Ah(A)]$, should be the number of juveniles required to produce one adult to replace itself, $1/[Sg(I)]$.

An equilibrium is of little relevance unless it is *locally stable*, i.e., following a small perturbation, the stock returns to its previous level. If $a_t = A_t - A^*$ is the small deviation in year t, then

$$a_{t+1} \approx a_t\{A^*[\partial F/\partial A] + 1\},$$

using equation (5) and Taylor Series expansion of the right side of equ (3).

(Note: all derivatives in this report are evaluated at (A^*,b,S), i.e., the pre-SONGS equilibrium and values of b and S.)

The value

$$z = A^*[\partial F/\partial A] + 1 \tag{6}$$

is the "eigenvalue" of the system. It determines whether, after a small perturbation, the system oscillates ($z < 0$) or changes monotonically ($z > 0$) and also whether it returns to equilibrium ($|z| < 1$) or not ($|z| > 1$). We will see below that, provided there are no depensatory mechanisms (i.e., provided f, g and h are all non-increasing, so that increased populations cannot have increased fertility or survival), z must be less than 1. In other words, monotonic divergence from equilibrium is not possible in this model. The population can be unstable only if it "overcompensates", i.e., if compensation is so strong that A_{t+1} is further above (or below) the equilibrium than A_t was below (or above) it.

From equ (6), $z = \partial(AF)/\partial A$. Writing $I^* = bA^*f(A^*)$, we get

$$z = Sbf(A^*)g(I^*)PQ + S_Ah(A)R$$

$$= (1 - S_Ah(A^*))PQ + S_Ah(A^*)R, \tag{7}$$

using equ (5), where

$$P = 1 + A^*f'(A^*)/f(A^*) \tag{8}$$

$$Q = 1 + I^*g'(I^*)/g(I^*) \tag{9}$$

$$R = 1 + A^*h'(A^*)/h(A^*) \tag{10}$$

Note that if f′, g′ and h′ are all non-positive, then P, Q and R are all ≤ 1; since $S_A h(A)$ = fraction surviving must be ≤ 1, we get $z \leq (1 - S_A h(A)) + S_A h(A) = 1$, as claimed above. The inequality will be strict if at least one of f′, g′ and h′ is strictly negative.

We will measure the sensitivity of the equilibrium value, A^*, to SONGS-induced changes in S, by asking whether a given small fractional change in S leads to a smaller, larger or equal fractional change in A^*. More precisely, if a change of δS in S (= a fractional change of $\delta S/S$) causes a change of δA^* in A^* (other parameters being unchanged), then the **sensitivity** of A^* to change in S is

$$\sigma_S = [\delta A^*/A^*]/[\delta S/S].$$

From equ (5) and the rules for partial differentiation we have

$$\sigma_S = -S[\partial F/\partial S]/(A^*[\partial F/\partial A])$$

by Taylor Series expansion. From equs (4), (5) and (6), we get

$$\sigma_S = \{1 - S_A h(A^*)\}/(1 - z). \qquad (11)$$

We define the sensitivity of A^* to changes in b similarly: $\sigma_b = [\delta A^*/A^*]/[\delta b/b]$ if a small change δb in b causes a small change δA^* in A^*. We then get

$$\sigma_b = -b[\partial F/\partial b]/(A^*[\partial F/\partial A])$$

$$= Q\sigma_S. \qquad (12)$$

Since $z \leq 1$, equ (11) shows that $\sigma_S > 0$; thus the change in A^* will be in the same direction as the change in S; in particular, a reduction in juvenile survival will lead to a reduction in the equilibrium adult population. If $Q > 0$, the same holds for the fertility, i.e., $\sigma_b > 0$ so a reduction in b leads to a reduction in A^*. However, it is possible that $Q < 0$, so the number of juveniles surviving to adulthood, Sg(I), actually increases when the number of entering juveniles decreases; a decrease in b could then lead to an increase in A^*. This is discussed in Case 3(b) below.

A sensitivity index greater than one implies (for a stable population) that the relative decline in adult stock due to SONGS will *exceed* in magnitude the relative change in b or S. That is, the drop in adult stock will exceed the adult equivalent loss (AEL).

Equations (11) and (12) show that the questions of the impact of SONGS on equilibrium densities and on stability (via z) are not independent. If two populations have the same value of $1 - S_A h(A^*)$ but different values of z (because b, S, f or g is different), then the population with the smaller value of z will be less sensitive to a SONGS-induced change in S, i.e., its equilibrium value will change less. However, if $z < -1$, this advantage has little meaning since the equilibrium will be unstable.

In the following section, we look at some special cases of the model, usually cases in which only one of f, g and h is not constant. Our main interest is in the sensitivity and stability. But the sensitivity indices defined in equations (11) and (12) are strictly valid only for "small" changes in b or S. The practical limitations on what can be regarded as "small" depends on the forms assumed for the functions f, g,

and h; in general the smoother these functions, the larger the range of validity of predictions based on the sensitivity indices. To complement our analysis of stability and sensitivity indices, we shall make one calculation which does take explicit account of nonlinearities: for most of our models we shall attempt to answer the question, "By what fraction must b and/or S be reduced to cause extinction?".

3.4 Predicted Consequences of Immature Mortality

Case 1(a). In this model, "immature" is taken to mean "juvenile" and "birth" means recruitment to the juvenile population, so that fecundity includes the survival of eggs and planktonic larvae. We assume there is no density dependence in either juvenile or adult survival, so the functions $g(I_t)$ and $h(A_t)$ are both identically one. The population then compensates only via $f(A_t)$, i.e., through an increase in fecundity and/or planktonic survival in response to a decrease in adult density. The dynamic consequences are the same, regardless of whether it is adult fecundity or planktonic survival that is density dependent.

Since $g = h = 1$ for all A, we have

$$F(A,b,S) = bSf(A) + S_A. \tag{13}$$

The adult equilibrium density is obtained by solving equ (5):

$$f(A^*) = (1 - S_A)/bS, \tag{14}$$

the eigenvalue determining local stability (equ (7)) is

$$z = 1 + bSA^*f'(A^*), \tag{15}$$

and the equilibrium sensitivity indices (equs (11) and (12)) are

$$\sigma_S = \sigma_b = -f(A^*)/[A^*f'(A^*)]. \tag{16}$$

SONGS kills both planktonic stages and juveniles, so its effect in this model is to reduce both b and S. Equation (14) now tells us two general consequences of this reduction, valid provided only that f(A) is a decreasing function of A:

1. $f(A^*)$ increases, implying that A^*, the adult population, decreases. This agrees with our general remarks earlier: the sensitivity indices are positive, so decreases in survival or fertility lead to reduced populations.

2. The population will go extinct if SONGS reduces bS to $1 - S_A$ or less, i.e., by a factor of $(bS + S_A - 1)/bS$ or more, regardless of the specific functional form for f(A); the most vulnerable populations are those for which this threshold factor is small, e.g., those which are barely viable ($bS + S_A$ barely greater than 1).

3. The sensitivity index is less than 1 only if $\partial[Af(A)]/\partial A < 0$; thus the fractional change in the equilibrium value is less than the fractional change in bS only if there is very strong density dependence: under pre-SONGS conditions, if the number of adults increases from A^* to a value slightly above it, the number of new immatures must actually decrease.

Further quantitative predictions require specification of the function f(A). Unfortunately virtually nothing is known about this function for any of the species affected by SONGS, though we presume that it should decrease from 1 to 0 as A increases from 0 to ∞. The "local" properties (eigenvalue and sensitivity index) involve knowledge of the quantity $f(A^*)/[A^*f'(A^*)]$ whose value can vary greatly among functions which satisfy our broad general conditions for a compensation function. However if we make the not implausible conjecture that the function f(A) is convex (i.e., the straight line between any two points on its graph will lie above the graph) then $-Af'(A)$ has a value between $1 - f(A)$ and 0 and varies slowly. Thus the sensitivity index will be greater than 1 (and potentially very large) if $f(A^*)$ is near 1, i.e., if $(1 - S_A)$ and bS are approximately equal, so the population is barely viable. If A is large and f(A) small, f(A) should behave like cA^{-n} for some constants c and n; then $\sigma_S = -f(A^*)/[A^*f'(A^*)]$ is approximately 1/n. The fractional decrease in A^* is smaller than the corresponding decrease in bS if n is large (strong compensation) and larger if n is small. The borderline case, n = 1, occurs when total recruitment is approximately constant, independent of the size of the adult stock.

However there is a further problem: if $n > 1$, there is the possibility of instability: equations (14) and (15) give $z = 1 - n(1 - S_A)$, so the system is unstable if $n > 2/(1 - S_A)$.

The most "optimistic" assumption concerning the function f is that it is sigmoidal in form: only slight compensation at small values of A, but strong compensation (perhaps reflecting pressure on resources) at A^*. In this case a small value of $f(A^*)$ and a large (absolute) value of $A^*f'(A^*)$ (possibly larger than $1 - f(A)$) may combine to give a sensitivity index less than one. However if the slope

$f'(A^*)$ becomes very large and negative (specifically, $f'(A^*) < -2/bSA^*$, see equ (15)), the equilibrium is unstable due to overcompensation. There is no evidence suggesting that any of the species potentially impacted by SONGS are fluctuating around an unstable equilibrium, and it thus appears that while a sigmoidal compensation function may through good fortune minimize the impact on some species with appropriate parameters, it is unsafe to rely on this mechanism.

An instructive example that illustrates these points is obtained by recasting the well-known Beverton-Holt stock-recruitment relation and assuming

$$f(A) = 1/(1 + A/A_0). \qquad \text{Beverton-Holt} \qquad (17)$$

This form gives a stock-recruitment curve in which an increase in the adult population always leads to an increase in total recruitment, but there are diminishing returns: the recruitment increase becomes vanishingly small at high adult densities, so that $bAf(A)$, the total recruitment to the juvenile population, is essentially bA_0, independent of stock, at these densities. At equilibrium, the adult population density is (equ (14))

$$A^* = A_0 [bS/(1-S_A) - 1]. \qquad \text{Beverton-Holt} \qquad (18)$$

whose sensitivity to changes in b or S is (equ (16))

$$\sigma_S = \sigma_b = bS/[bS - (1-S_A)]$$

Thus if SONGS reduces b or S by a small fraction y (i.e., to (1 - y) S or (1 - y) b), the fractional reduction in A^* will be greater than y.

A generalization of the Beverton-Holt form is

$$f(A) = 1/[1 + (A/A_0)^n]. \quad (19)$$

As for eqn (17), $f(0) = 1$ and $f(A_0) = 0.5$. If $n > 0$, per capita birth rates decrease as A increases; if $0 < n < 1$, total recruitment increases without limit as A increases; $n = 1$ is the standard Beverton-Holt model; if $n > 1$, total recruitment peaks when $A = A_0/(n - 1)^{1/n}$, and then declines, so that high stock levels lead to low recruitment. This choice of f gives (equ (14))

$$A^* = A_0\{bS/(1-S_A) - 1\}^{1/n}, \quad (20)$$

the eigenvalue is (equ (15))

$$z = 1 - n(1-S_A)[1 - (1-S_A)/bS], \quad (21)$$

and the sensitivity indices (equ (16)) are given by

$$\sigma_S = \sigma_b = n^{-1}bS/[bS - (1-S_A)]. \quad (22)$$

Equ. (21) shows that, although it is difficult to derive necessary and sufficient conditions for stability for a general function f, with this (rather broad) family of generalized Beverton-Holt functions *a SONGS-induced reduction in the product bS will always enhance the stability of the system* (by increasing z). Note that if n is large, z can be < -1, and the system unstable.

Equ. (22) tells us that *the equilibrium sensitivity indices are always greater than one unless $n > 1$*, i.e., the percentage reduction in population is greater than the percentage reduction in bS unless compensation is stronger than the standard

Beverton-Holt form. In the case $n > 1$, no general comment is possible, as the sensitivity indices may take values greater or less than one depending on the precise values taken by b, S, and S_A.

Note that equs (21) and (22) combine to give $z = 1 - (1 - S_A)/\sigma$, where $\sigma :=$ σ_S or σ_b. Thus increasing n to reduce σ will eventually lead to $z < -1$ and instability.

It is possible to develop similar arguments with countless families of functions, and furthermore to generalize the above results to the situation where the developmental time is greater than one year (see e.g., models in Bergh and Getz 1988, Getz and Haight 1989), but we are aware of no examples that contradict our main conclusions for this model, namely that unless there is overcompensation, SONGS will produce a reduction in adult stock at least as large as the reduction in bS and an increase in stability.

Case 1(b). In this case, h(A) is the only function not identically equal to one. We have

$$F(A^*,b,S) = bS + S_A h(A^*). \tag{23}$$

The equilibrium is given by

$$h(A^*) = [1 - bS]/S_A, \tag{24}$$

and exists only if $bS < 1 < bS + S_A$. The first of these inequalities reflects the fact that adult mortality (i.e., mortality after the year of growth from egg to adult) cannot control the population if each adult produces more than one replacement in

its first year; the population will then grow without limit. The second inequality is simply the requirement that at low densities (where h(A) is approximately one) the population should be viable: on average, the new population each year will consist of a fraction S_A of the adults of the old population plus bS new adults for each adult in the old population. Where there is an equilibrium, the eigenvalue z determining its stability is given by (equ (6))

$$z = 1 + S_A A^* h'(A^*), \tag{25}$$

and the equilibrium sensitivity indices (equs (11) and (12)) are

$$\sigma_S = \sigma_b = -bS/(S_A A^* h'(A^*). \tag{26}$$

Further discussion requires assumptions about the function h(A). If, as seems plausible, this function is convex, it turns out that the eigenvalue must lie in the range (2 - bS - S_A,1), implying exponential stability (i.e., a steady return to equilibrium), and that

$$\sigma_S = \sigma_b > bS/S_A(1-h(A^*)) = bS/(bS + S_A - 1) > 1, \tag{27}$$

implying that the percentage decrease in adult stock will be *more* than that in b and/or S.

If h is sigmoidal, as might happen if density dependence is weak until the population grows large enough to strain resources, it is possible that $-A^*h'(A^*) > 1 - h(A^*)$, so that σ_S and σ_b are smaller than 1, implying a smaller fractional change in A^* than in bS. But if $-A^*h'(A^*) > 2/S_A$, the equilibrium will be unstable because of overcompensation.

Case 3. For cases 3(a) and 3(b), f = h = 1, so compensation acts through juvenile survivorship, $g(I_t) = g(bA_t)$. Thus we have

$$F(A,b,S) = bSg(bA) + S_A, \tag{28}$$

so the equilibrium is given by

$$g(bA^*) = (1 - S_A)/bS, \tag{29}$$

and the stability is determined by

$$z = 1 + b^2SA^*g'(bA^*). \tag{30}$$

In both cases, if bS is decreased to $1 - S_A$, by a decrease in either S or b, the population goes extinct; and the equilibrium is stable in both cases if $g'(bA^*) > -2/b^2SA^*$. The cases differ in other respects.

The remaining case, 3(c), requires a new model, since it involves two juvenile stages occurring simultaneously.

Case 3(a). SONGS affects only S. For this analysis we lump together the planktonic and juvenile stages in the immature population (still denoted by I_t). The parameter b now refers to the (fixed) number of eggs produced per adult. Since b is fixed, σ_b is ignored. The other sensitivity index (equ (11)) is

$$\sigma_S = -g(bA^*)/[bA^*g'(bA^*)]. \tag{31}$$

Equations (28) to (31) are very similar to equs (13) to (16), and much of the discussion of our Case 1(a) applies here. In particular, a reduction in S implies an increase in $g(bA^*)$; this implies a decrease in A^*, since g is a decreasing function and b is fixed. Thus, *even though immature survival increases in response to SONGS effects on the immatures, fewer individuals recruit to the adult population, and hence average adult population is reduced.*

Also, the previous points 2 and 3 are the same: the same fractional reduction in bS (in this case, in survival, S) will bring extinction, and the sensitivity will be > 1 (so the fractional change in A^* will be greater than that in S), unless a decrease in new juveniles (I_t) would lead to an increase in new adults ($A_{t+1} = I_t S g(I_t)$) at equilibrium under pre-SONGS conditions.

The Beverton-Holt form and its generalization are also reasonable here, with $g(I) = 1/(1 + (I/I_0)^n)$; replacing I and I_0 by bA and bA_0 shows that we get exactly the same conclusions.

Case 3(b): We assume that the late juveniles suffer no mortality from SONGS, as an extreme case of very low mortality on this age group. We further assume however that SONGS does increase mortality on previous immature stages. The parameter affected by SONGS is now b, which in this model represents the number of "births" per adult into the late juvenile age class (I_t), and is affected via mortality on all immatures up to the late juvenile stage; S, the maximum survival at low density, is assumed unaffected by SONGS because this stage is assumed to have negligible SONGS-induced mortality. Compensation again acts through $g(I_t)$.

Thus we again have equs (28) to (30): the same equilibrium value, and the same fractional change in bS (in this case, in fertility and early survival, b) will lead to extinction.

But now it is S which is fixed, so σ_S is ignored, and

$$\sigma_b = -1 - g(bA^*)/[bA^* g'(bA^*)]. \qquad (32)$$

If compensation is strong enough (i.e., if g' is large enough) σ_b can be negative, implying that a small decrease in b can lead to an actual increase in the equilibrium population A^*.

Mathematically, the key difference between this case and Case 3(a) is that the parameter affected by SONGS is b and not S. A decrease in b implies an increase in $g(bA^*)$ and thus a decrease in bA^*; but this no longer implies a proportionate decrease in A^*, or even a decrease at all.

Biologically, the difference between this case and all the others is that compensation can operate even when there is no reduction in later stages. In Cases 1 and 2, compensation responds to a decrease in adults; it cannot prevent a decrease, since it cannot operate until some decrease has occurred. In Case 3(a) both SONGS and the compensation work through the same parameter: if SONGS is to cause any reduction, it must reduce the numbers in a stage following the stage on which compensation operates. Thus, although the compensation is responding to a decrease in I, this decrease is itself a consequence of the decrease in A caused by SONGS: as before, if there were no adult decrease, there could be no

compensation. In the present case, the SONGS effect is to reduce the number of recruits to the immature stage; and it is to this reduction, rather than to a subsequent reduction in adults, that compensation responds by increasing the number of individuals "graduating" from this stage. In fact, it is easy to see that this effect can occur even if SONGS reduces S, provided that compensation can be strong enough to more than counter this reduction. The necessary condition is that the survival of a late stage can increase as a result of a reduction in an earlier, pre-adult stage.

A consequence of equation (32) is that if g(I) varies as cI^{-n} for a range of I in the vicinity of the equilibrium, $g(bA^*)/[bA^*g'(bA^*)] = -1/n$ so a sensitivity index less than one is achieved if $n > 0.5$. There is no decrease at all if $n = 1$ (perfect compensation, whereby the number of immatures emerging from the stage is the same regardless of how many entered it, provided this is large enough for the "I^{-n}" behavior) and an actual increase in adult density is possible if $n > 1$. However, from equ (30), we again get $z = 1 - n(1 - S_A)$, so the system is unstable if $n > 2/(1 - S_A)$, i.e., if compensation is too strong.

The generalized Beverton-Holt model, (equ (19) with "g", "I", and "I_O" replacing "f", "A", and "A_O"), is also plausible here. We write $E = (bS + S_A - 1)/bS$; when the population is low (no compensation), this is the "excess" fraction of new adults beyond those needed to replace the adults who died. We then get $z = 1 - n(1 - S_A)E$ and $\sigma_b = -1 + 1/En$. Thus the sensitivity index is less than 1 if $nE > 0.5$ and is negative if $nE > 1$; but the system is unstable if $nE > 2/(1 - S_A)$.

Case 3(c). In this mechanism, small immatures are the victims of predation by large immatures (e.g., planktonic stages eaten by juvenile fish). Our standard model is not suitable for modelling this case, since this type of cannibalism requires two stages to be present simultaneously; we must therefore abandon our discrete time modelling framework. Some insight into the likely effects can be obtained from a very crude model in which we assume "small" and "large" immature stages of duration T_1 and T_2 respectively and let the large individuals cannibalize the small ones. We assume random searching and neglect any beneficial effect of cannibalism on the cannibals. The model then takes the form

$$dS(t)/dt = R_S(t) - R_L(t) - cS(t)L(t) - dS(t) \tag{33}$$

$$dL(t)/dt = R_L(t) - R_A(t) - eL(t) \tag{34}$$

$$dA(t)/dt = R_A(t) - fA(t) \tag{35}$$

in which d, e, and f represent density-independent death rates for the three stages, the term $cS(t)L(t)$ represents cannibalism, and $R_S(t)$, $R_L(t)$, and $R_A(t)$ represent recruitment rates to the three stages. We have:

$$R_S(t) = b\,A(t), \tag{36}$$

i.e., a constant rate of production by the adults;

$$R_L(t) = R_S(t-T_1)\exp\{-\int_{t-T_1}^{t}(cL(x)+d)dx\}, \tag{37}$$

i.e., the survivors of those who recruited to the small stage T_1 time units before; and

$$R_A(t) = R_L(t-T_2) \exp(-eT_2), \tag{38}$$

the survivors of those who recruited to the large stage T_2 time units before. From equs (34) - (38), the equilibrium adult density can be shown to be

$$A^* = \frac{e\,[\ln(b/f) - dT_1 - eT_2]}{fcT_1[\exp\{eT_2\} - 1]} \tag{39}$$

which decreases if either d, the death rate of small immatures, or e, the death rate of large immatures, increases. Thus SONGS-induced mortality on the immature stages, no matter how apportioned, leads to a decrease in the adult population. Sufficient mortality on either stage will lead to extinction. However, the sensitivity indices describing the response to changes in d or e or both, depend on the other parameters in the model, in a messy and unrevealing way. We remark that this model may overestimate the compensatory effect of cannibalism, since it includes the negative effects of cannibalism on the small larvae, but ignores the beneficial effects on the large larvae.

Case 4. Here, the fish species that suffers SONGS-induced immature mortality is a major food item for a predator (e.g., sport/commercial) species. Again, our standard model is not suitable. Since the life cycles of fodder and predator species are probably different, we model this situation in a continuous-time framework.

First, guided by Section 2, we assume that the compensatory mechanisms for the fodder fish operate in response to adult density only. We also assume that the

predatory (sport/commercial) fish species depends entirely upon the fodder fish for food.

A standard type of model is

$$dA/dt = AF(A,b) - Pk(A) \tag{40}$$

$$dP/dt = cPk(A) - mP, \tag{41}$$

where A and P are respectively the densities of adult fodder fish and of predators. The term k(A) is the "functional response" giving the number of prey killed per predator per unit time. These prey are converted into new predators with efficiency c. The term F(A,b) represents the *per capita* rate of increase (decrease if F is negative) of the adult stock, as a function of the stock size; b is the recruitment rate, which may be affected by SONGS. Thus F plays a role similar to that in equ (3). The SONGS effect is to reduce F by reducing b, which combines the roles previously played by b and S. We assume the predator suffers a constant death rate, m.

We assume that, for given b, a reduction in A leads to an increase in F; thus compensation will act if A is reduced. But from equation (41), the equilibrium fodder fish population is given by $k(A^*) = m/c$. Thus this population is completely unaffected by the reduction in F caused by SONGS, so no compensatory mechanism is brought into operation.

But the equilibrium population for the predator species is now given by equation (40):

$$P^* = A^*F(A^*,b)/k(A^*). \qquad (42)$$

A SONGS-induced reduction in b will thus not affect A^* but will cause a reduction in the predator species. How large the reduction is depends on the role of b in F. For example, if $F(A^*,b) = bF_1(A^*) - dF_2(A^*)$, i.e., growth rate = birth rate - death rate, then a change δb in b leads to a change $\delta P^* = [dP^*/db]\delta b$ in P^*, so the ratio of fractional changes is $[\delta P^*/P^*]/[\delta b/b] = bF_1(A^*)/[bF_1(A^*) - dF_2(A^*)] > 1$, i.e., the fractional change in P^* is greater than that in b.

There are *a priori* reasons to doubt the realism of this simple model as it excludes many factors, including some emphasized in the single species models. Furthermore, it turns out that with many forms for the function F (or for F_1 or F_2), the equilibrium is likely to be very unstable, with large amplitude cycles in both prey and predator. However the result that the fodder fish density is set by attributes of the *predator* is rather general and will hold provided there is no dependence on *predator* density of the predator death rate, the efficiency of converting prey into new predators, and/or the functional response. It does however require that the predator is a specialist, regulated exclusively by the availability of one particular prey species.

Modification of the model to cover either of the above weaknesses is beyond the scope of this report. However, it would seem plausible that relaxation of the model assumptions will prevent the complete transfer of losses from the fodder to the predator fish (cf. McCauley, Murdoch and Watson 1988 for a related study on *Daphnia* and a heterogeneous algal food supply). The overall conclusions from the

model are thus rather weak; it suggests a "greater than one" sensitivity index, but plausible modifications are likely to reduce this. It is thus possible that the fractional predator fish losses will be less than the fractional change in fodder fish production caused by SONGS; however acceptance that this is likely involves excessive faith in pure theory backed by rather little experimental evidence.

3.4.1 Combinations of Compensatory Mechanisms

There may be more than one compensatory mechanism operating, and it is of interest to know how they interact. The baseline model with all mechanisms operating is very complex, its properties being summarized in equations (3)-(12). We have made no attempt beyond the discussion around these equations to interpret these general formulae; however it is of interest to study the combined effects of two of the most likely of the mechanisms studied.

We assume that there is no density dependence in adult mortality (i.e., h(A) = 1) and that the equilibrium population occurs in a region of the curves f(A) and g(I) where

$$f(A) = cA^{-n} \tag{43}$$

and

$$g(I) = dI^{-m}. \tag{44}$$

for constants c and d.

Substituting in equ (4) we get

$$F(A,b,S) = Sb^{1-m}DA^{mn-m-n} + S_A \quad (45)$$

where $D = dc^{1-m}$. The sensitivity indices (equs (11) and (12)) are then

$$\sigma_S = 1/[1-(1-n)(1-m)] \quad (46)$$

and

$$\sigma_b = (1-m)/[1-(1-n)(1-m)] \quad (47)$$

It follows that the sensitivity index to changes in juvenile survival (S) is greater than one unless $(1-m)(1-n) < 0$, which requires overcompensation in one of the processes (i.e., $m>1$ or $n>1$). The case of double overcompensation ($m>1$ and $n>1$) is very complex (Rodriguez 1988, Onyiah and Nisbet, *in prep.*) since multiple equilibria can occur; thankfully there is no evidence to suggest that this is likely in the populations under study. The expression for sensitivity to changes in b is of interest since it includes the only case we studied (case 3(b)) where total compensation was plausible. We see that the results of that section appear quite robust: perfect compensation occurs with $m=1$ irrespective of the other compensation via adult fecundity.

The above analysis is rather superficial, but it adds some credibility to the conclusions derived from consideration of one effect at a time. Further support comes from a few further calculations with different functions. The only situations we have found where compensatory mechanisms acting together significantly reduce the adult loss occur in models where one of the mechanisms was density dependent adult mortality, which we concluded in section 2 was unlikely.

3.5. Implications of the Models

The overall message from these models is that compensation will not prevent a reduction in the average abundance of adult fish, unless SONGS affects early stages and late juveniles compensate perfectly. We know of no reason to expect much compensation in late juveniles in the species of concern, and what little is known about their ecology suggests compensation is less than perfect (Section 2).

The present suite of "strategic models" does not provide an unambiguous pointer to the likely magnitude of the decline in adult density caused by SONGS-induced death of a specified fraction of the immatures. However, we cannot escape the conclusion that all "optimistic" outcomes (fractional decline in adult stock that is significantly less than the fractional AEL) appear to demand mechanisms which have not been proved in *any* marine fish *anywhere*. Still, compensation holding the percentage loss in adult stock to about the same as the percentage AEL seems plausible: more plausible if several compensatory mechanisms operate simultaneously, and still more if one of these is adult survival. Although we have not tried to model in detail the case where the affected fish is a fodder fish and there is compensation in the predator fish, it appears that much of the loss may be "transferred" from the fodder fish to the predator, and the predator's percentage loss could be as large as the loss that would be experienced by a single species affected directly.

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