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FOOD AND THE STABILIZATION MECHANISM IN FISHES

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ABSTRACT

A study of stock/recruitment processes in model form suggests that the natural mortality of fishes is a density dependent function of age. With an estimate of the larval mortality of the plaice in the Southern North Sea, the trend of natural mortality with age was estimated. A critical age was defined as that at which the specific growth rates and specific mortality rates become equal and it was suggested that in older age groups mortality might become senescent. In the models used, mortality rate in the larval stage depends upon the variability of food and there is a sense in which subsequent recruitment is determined by the patchiness of the food. There is a distinction between such an exploratory character in the density dependent mortality that may establish the magnitude of recruitment and the conservative character of the stock dependent on which the initial numbers are based. Differences in the ratio of specific growth rate and specific mortality rate during larval life might express competitive differences which would affect the magnitude of recruitment and eventually the quantity in the stock. A consequence is that possibly high diversity is the result of low competitive pressures and vice versa.

It is a truism that the numbers of animals depend upon the supply of food, and it was the central theme of Lack's (1954) book on the "Natural regulation of animal numbers". The reproductive rate was considered to be adapted by natural selection to the food supply and numbers were controlled by density-dependent mortality. The same principles apply to fishes, where the mechanism of stabilization operates during juvenile life through density-dependent mortality (which may be linked to density-dependent growth). Fecundity cannot be the driving force during adult life because density-dependent growth is by then more or less absent. Variation in stock is expressed through fecundity in the initial numbers of eggs and larvae. However, the mechanism of stabilization itself is linked to fecundity (Cushing, 1971) in that density-dependent mortality is probably higher in the more fecund fishes (Cushing and Harris, in press, *a*).

The very large reduction in numbers during immature life is often regarded as a loss or a wastage in the face of a hostile environment. Because the fecund fishes can stabilize their population well, it does not follow that they must live in adverse conditions. Further, if the numbers are really wasted, the evolutionary processes which operate across the food chains must appear to be inefficient. In this paper another explanation is put forward which may be more relevant to the mild and benign environment in which fishes live.

There are two developing models of the growth and mortality of fishes during larval life. Jones (in press) shows that haddock larvae grow at 12%/d and die at 10%/d, and he suggests that they grow through a succession of predatory fields and that they live on 'private' food sources which develop at the same time as the larvae. A density-dependent mortality is generated by varying the numbers of larvae. Jones suggests that the coefficient of variation of gadoid year-classes in the North Sea is correlated with probable ranges of food available to the different populations.
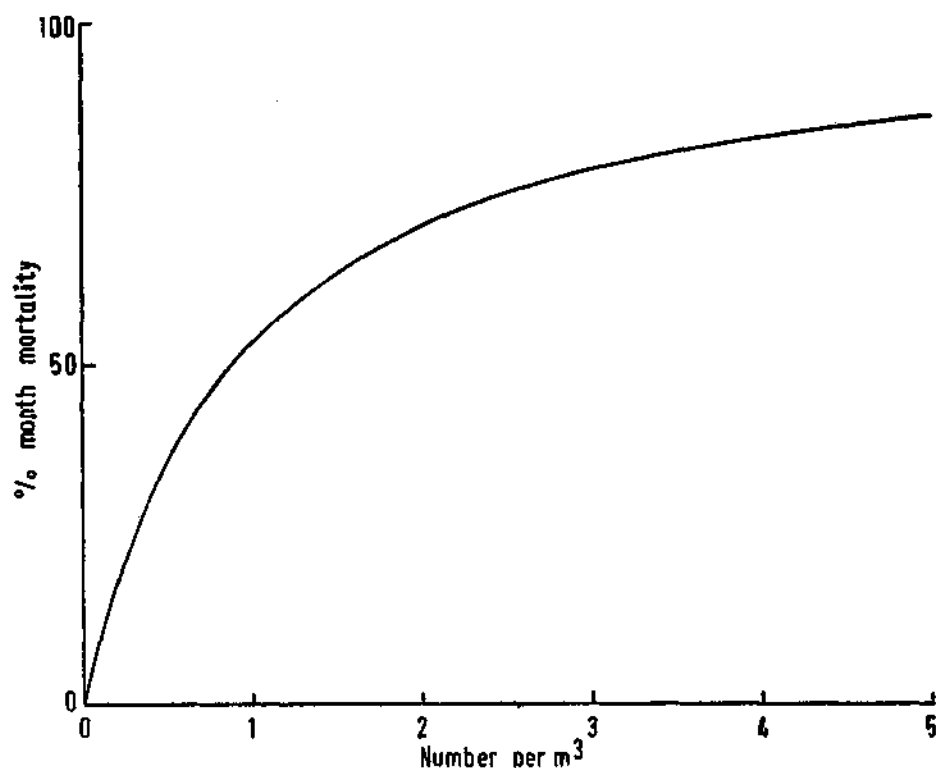

Fig. 1. Dependence of larval mortality on numbers of plaice larvae in the model described.

The second model, that of Cushing and Harris (in press, *a*) and Cushing (in press), proposes that, in rich food, larvae eat well, grow well, swim quickly and minimize predation; in scarce food, however, for converse reasons predation is greater. Variations in the numbers of larvae generate density-dependent mortality and variations in food alter the degree of density dependence. Fig. 1 shows how the larval mortality (as percentage per month) depends upon the numbers of plaice larvae in the sea, in model terms. The numbers per m³ correspond to those in the sea and the observed mortality rate of plaice larvae is 80% per month (Harding and Talbot, in press), so the external constraints to the model are approximately right. Fig. 2 shows how in the model the weight
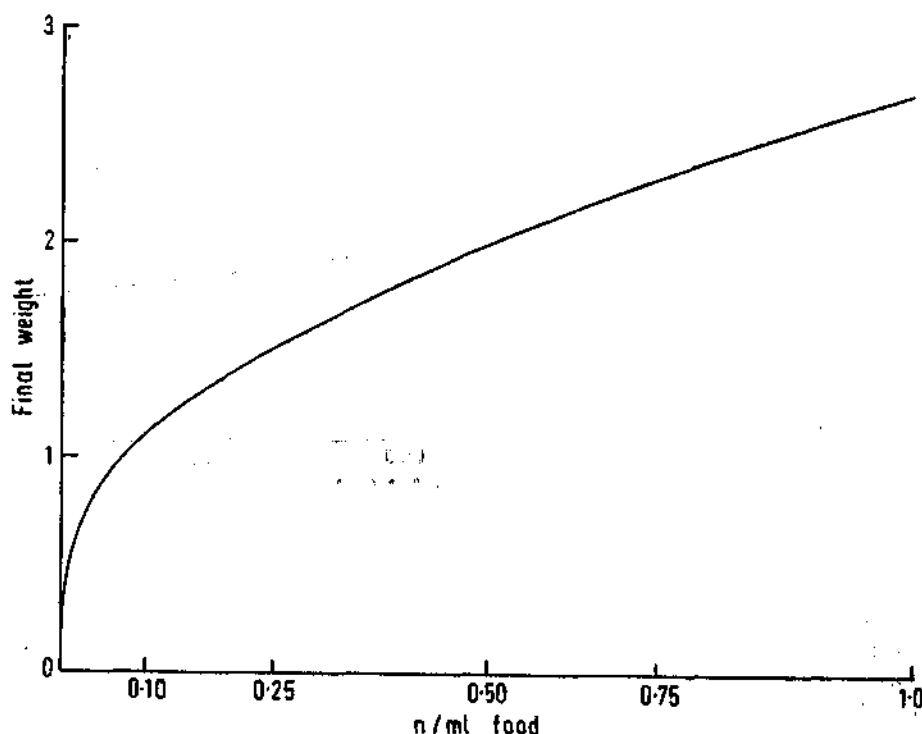

Fig. 2. Dependence of larval weight on food in the model described.

after 30 days depends upon food density, that is, the weight of larvae increases with food in roughly the form of an Ivlev curve. Fig. 3 shows how the percentage mortality per month varies with food density for a constant number of larvae; the mortality rate is high (but corresponds with that observed) and the differences between food levels are low. The growth rates and mortality rates are roughly those observed. It is shown that density-dependent mortality can be generated in the larval stages of plaice as a function of the food available. Such models have been proposed in the past (Ricker and Foerster, 1948; Beverton and Holt, 1957) for short periods during larval life. However, the processes

described in the two models are very general and could be applied, in principle, to fish of any age; however, such processes must be much sharper in larval life, and conversely in adult or older fish they are slight.
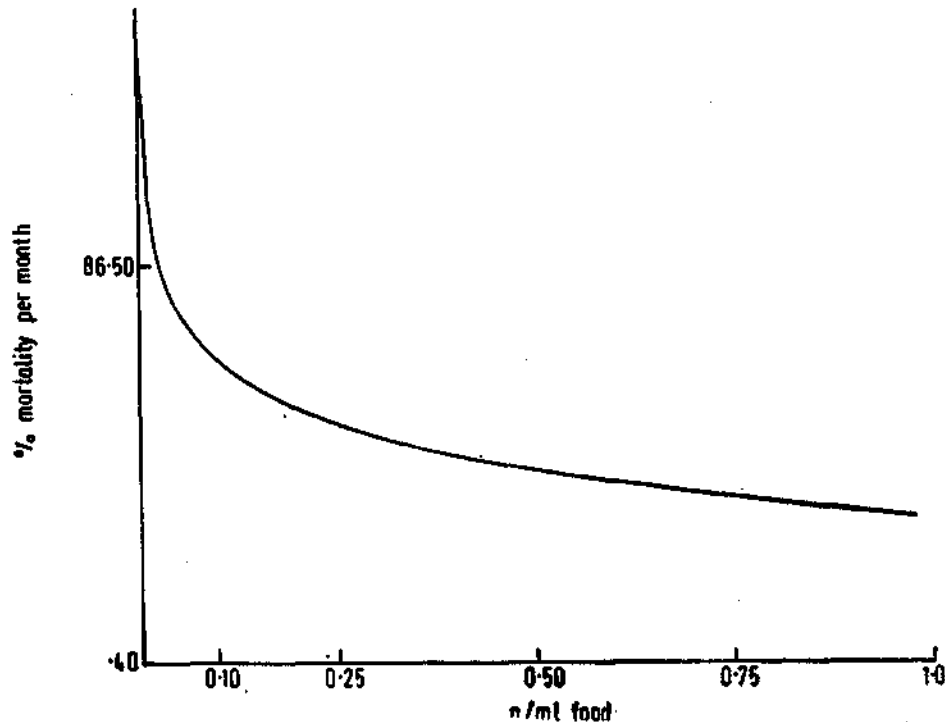

Fig. 3.   Dependence of larval mortality rate on food in the model described.

Fisheries biologists have usually considered that density-dependent mortality was a small proportion of the total.   One of the reasons for this belief is that the variability in recruitment is very high as compared with that in stock (as, for example, in a stock and recruitment curve).   The density-independent mortality is usually considered to be the wastage in a hostile environment.   If, however, we suppose that the natural mortality of fishes were density dependent throughout life (primarily because predation must be considred as a density-dependent process; the time to capture and eat a prey organism is constant, but the time to search is inversely proportional to prey density and as the ratio of the first to second increases with density, predation is density-dependent), the variability of recruitment is considered as the end product of a continuous process during immature life.   Ricker's equation (1958) relating recruitment to parent stock supposes that the tangent to the curve at the origin represents density-independent mortality.   He supposed that control as density-dependent mortality is generated by the aggregation of predators on to the initial numbers of eggs and

larvae. Beverton and Holt (1957) arrived at the same curve in formal terms in supposing that fish larvae grow through a critical period at different rates according to the food available, which generates density-dependent mortality, according to the numbers of larvae. Both processes were considered to be active amongst the eggs and larvae in the sea, but there can be no distinction between the eggs produced by one fish and those produced by more than one. If all mortality were density-dependent, the distinction would not matter because only the single process is effective. Then any density-independent mortality is an instantaneous loss which is subsequently modulated in a density-dependent manner during the remaining life-cycle. If we suppose that natural mortality is a density-dependent function of age (because predation is also density-dependent), it can be shown (Cushing, in press) that $N_t = N_0/(1 + M_0 t)$ and that $N_0 = (N_0 - N_t)/N_t \cdot t$, where N_0 is the initial number of eggs or larvae, N_t the number at time t and M_0 the initial mortality rate. The curve of number in time descends sharply and tends to flatten in adult life. Such trends of numbers of larvae, O- and I-group fishes with age have been presented on a number of occasions (e.g., Pearcy, 1962).

In different species of fish, the specific mortality rates $\left(_M{}^X = \ln (N_{t+1}/N_t)\right.$, where N_t and N_{t+1} are numbers in a cohort in successive years calculated from the density-dependent curve on age$\left.\right)$ are practically the same at a given age during adult life. Then the differences in natural mortality between species are really differences in the age of first maturation and the duration of adult life. The growth of adult fishes is not density-dependent as a general rule (an exception is the large Pacific halibut, in which density-dependent growth is evident in the youngest adult age-groups only; Southward, 1967); consequently, the specific growth rate, G, can be readily determined. Then a critical age (corresponding to Ricker's, 1945, critical length) can be defined as that at which $(G-M^X) = O$. Between the age of first maturation and the critical age in the virgin stock, the population fecundity increases, but at greater ages it must decrease and the animals might well then become senescent. Indeed, Greer-Walker (1970) has shown that the white muscle fibres of older fish decrease in size as they grow older—at about the critical age of 16 in cod. In the older fish, the escape speeds and the attack speeds must therefore decrease with age. If senescence is a reality in fishes, the population fecundity is limited by the critical age. Hence, on average, and only on average, the population should replace itself by the critical age. A larger stock would be corrected by density-dependent processes in the subsequent generation and a smaller stock would be augmented by the fecundity of the senescent population. If the population replaces itself by the critical age, two adults should be generated by

n gonads, where n is the number of age-groups between the age of maturation and
the critical age; thus a scale is provided for the equation given above. It can
be located by the observed larval mortality rate of 80%/month in the plaice of
the southern North Sea; then the estimates of 40%/month in the first summer
and of 10%/month in the first winter and of 10%/year from the ages of 5 to
15 (Beverton and Holt, 1957) can be derived and they correspond in fact to the
observed mortalities. For the plaice, at least, the trend of natural mortality with
age is well described by the density-dependent function. At first sight it is an
improbable conclusion, but not on later consideration. A fuller account is given
in Cushing (in press) and in Cushing and Harris (in press, *b*).

Harris (in press), in a study of the equations which relate recruitment to
parent stock, has distinguished stock dependence and density depen-
dence. Ricker (1954, 1958) suggested that fish populations were controlled by
the aggregation by predators on to the initial numbers of eggs or larvae. Such
stock dependence can perhaps be generated in other ways, but it must always
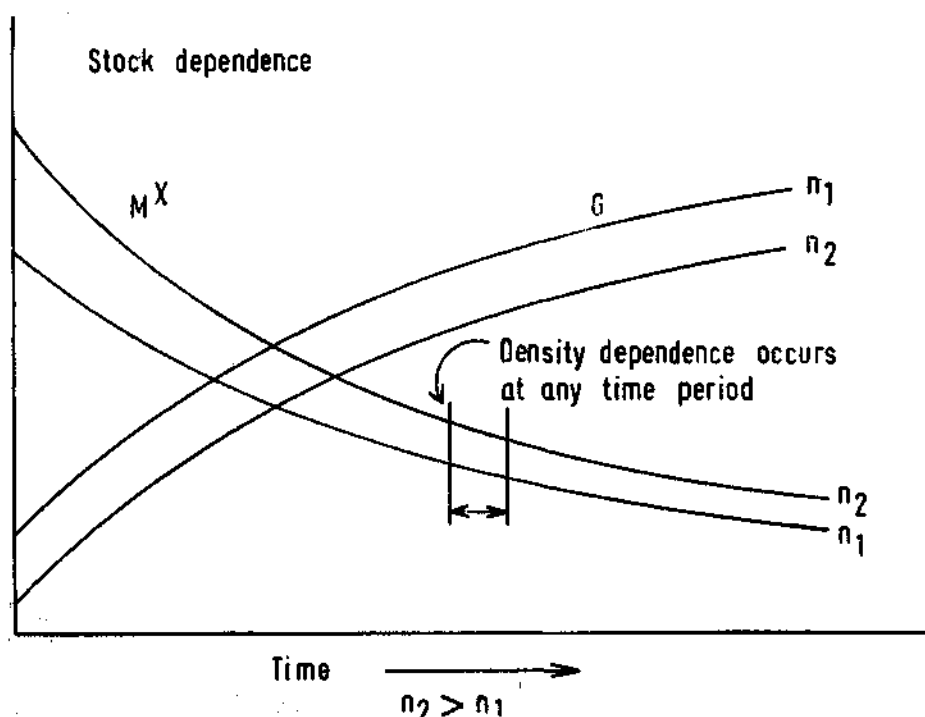play an essential part in control. Density dependence, in the new and narrow

Fig. 4. Stock dependence and density dependence in an ideal state.

sense, occurs later, in larval life or in juvenile life and it may well be gene-
rated as in the models described above. An important point, which Dr Gulland
has made to me, is that density dependence must operate during a period of time.

The distinction between the two forms of control is reminiscent of Gulland's (1965) distinction between a coarse control and a fine control. Fig. 4 illustrates the distinction as a trend of numbers with age: stock dependence represents the movement of initial numbers of eggs and larvae on the ordinate, whereas density dependence represents the shift of the curve in age upwards or downwards, that is, between n_1 and n_2 on the figure. For a given initial number of eggs and larvae, the trend of density-dependent natural mortality with age must represent an average condition. The variance about the curve is represented by the variation in recruitment. Another form of variation may be represented by differences in the numbers at the critical age; if it remains at 2.1 for a number of year-classes the population expands rapidly, and vice versa. These considerations show that the trend of natural mortality with age is not quite the rigid structure which was proposed above.

During the first year or so of life, at least, differences in the initial number generate differences in the specific mortality rates at given ages, that is, from month to month. In the models described above, it was shown that density-dependent mortality was a function of the availability of food. Fig. 5 illustrates the trend of specific mortality with age during an early period of life. At a middle period, I have supposed that a marked change in food availability occurred and that it persisted for a period of time, say a month, and that then the change was reversed, that is, the decrement of food later equals the earlier increment. During the period of rich food, the specific mortality rate is less at the new rate than at the old; then when the food availability change is reversed, the trend of mortality must take a different course in age (because it is a little less during the period of time). Thus, recruitment may be partly controlled by variation in the availability of food; Jones and Hall (in press) reached the same conclusion by a quite different route, but with an analogous model. However, it is not the only conclusion. A much more important one is that because the distribution of food is always variable in space, as well as in time, there may be a continuous modulation of the potential recruitment by the variability in the availability of food. It is an active and continuous process of extracting energy from the environment and it should be contrasted with the concept of loss or wastage in the face of an environment which was named as hostile. It should not be implied that there is no such thing as density-independent mortality, but its real effects are instantaneous losses and hence modulations of the form of density dependence. It has long been thought that variability in space should generate temporal variation; indeed Steele (1961) showed how it might occur. A much more important point is that the stabilization mechanism itself should be rooted in the variability of food.
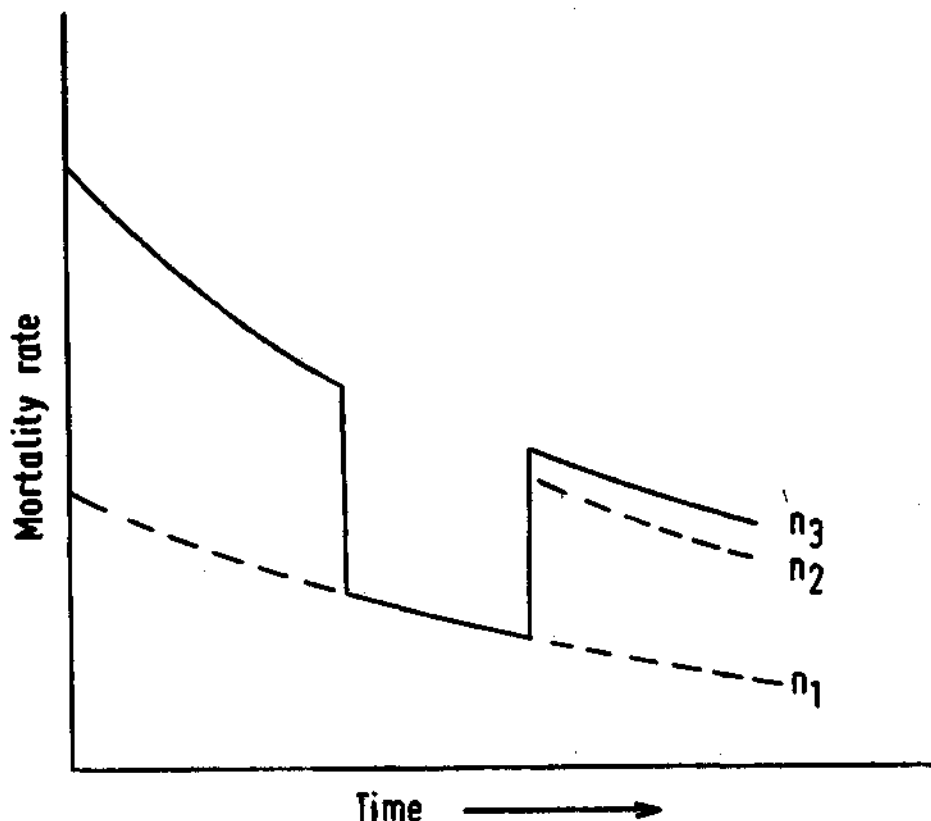
Fig. 5. Trend of specific mortality rate with time and the effect of food upon it.

We may return to the distinction between stock dependence and density dependence. Whereas density dependence is a continuous process of the exploratory extraction of energy from the food available, stock dependence has a conservative character. That is, in each generation, stock summarizes the exploratory activity characteristic of recruitment. Each cohort extracts the most energy from the food available during its life, but stock represents the average of a number of cohorts. The average represents the energy conserved, or in another idiom, information stored. It has long been recognized that the variability of recruitment is damped by the number of year-classes, but it is averaged in time rather like a moving mean—for the same reason, because the variability in a time series is conserved by such a procedure. Perhaps the variability in recruitment should not be regarded strictly as a random variable, but as one which is generated in climatic trends (Ottestad, 1942).

If we contrast stocks of high and low abundance, the difference in specific mortality rate at a given low age is much less in low stock than in high. Not only is the dependence of mortality on density during a time period less, but the

growth is less if growth and mortality are linked. Less food is extracted, the animals are perhaps smaller and the variance of recruitment is probably less. To put the argument the other way round, the abundance of stocks depends on the availability of food.

Differences in number between species are the results of competition, and from cohort to cohort they will be expressed in the proportion of recruitment to parent stock, or in another terminology, in differences in the net rate of increase. During any time period in the juvenile life-histories, such differences between species would be expressed in the ratio (G/M^X) which would be high in abundant stocks $(G/M^X)_1$ and low in less abundant ones $(G/M^X)_2$. Competition is then high between abundant populations and low between the less abundant ones. Hence there is a greater chance of a species being eliminated under conditions of abundance than under conditions of scarcity. The number of species is less where food is abundant in the sea than where it is scarce. The argument assumes that the competitive advantage or disadvantage expressed in the two ratios $(G/M^X)_1$ and $(G/M^X)_2$ for short periods in larval life can be maintained from generation to generation. Diversity is then explicable as the result of processes within the two stabilization mechanisms.

The processes of production in the sea may be classed in two groups, the continuous cycle of the open ocean in tropical and subtropical seas and the discontinuous one of high latitude and upwelling areas. The difference between the two in primary production may be of an order of magnitude or more. Cushing (1972) suggested that the transfer from primary to secondary production was three times as efficient in the continuous cycle as in the discontinuous one, so in secondary production the difference is probably reduced to a factor of 3 to 10. Efficient transfer occurs where competition may be less intense. Where (G/M^X) is high, feeding is intense and so is predation; that is, the competition for food is great in the face of predatory mortality. The continuous production cycle with efficient transfer is perhaps one of muted competition, whereas in the discontinuous one transfer is inefficient because competition is more severe. In the long term of evolution more species would survive in the continuous cycle, where food is not abundant, than in the discontinuous one, where it is. The inverse relationship between diversity and abundance is thus seen to be one of the degree of competition, itself a function of the part played by food availability in the stabilization mechanism.

Fishes grow through a number of trophic levels during their lives : their initial food may be algae and then they move on to copepod nauplii, copepods, worms or fish. Except for sharks, they are rarely the top predators, but they

are usually at a high position in a food chain. During the whole of their lives they exploit a considerable part of the chain. Consequently, they compete not only with analogous fishes, but with animals in the plankton or in the benthos. The competition will be most severe in the juvenile stages. If a density-dependent form of natural mortality in age were characteristic of fishes, perhaps it is also so of other animals in the chain, if only because of the important part played by fishes in the food chain structure. More to the point, the processes described briefly in the models above are of general application and could perhaps be used in invertebrate population studies.

If such an extension is permissible, there are two general conclusions :

1   that high diversity in evolutionary time periods is linked to low abundance of food because the intensity of competition is less;

2   that the structure of food chains depends upon the stabilization mechanism of the component populations.

REFERENCES

BEVERTON, R. J. H AND S. J. HOLT. 1957. On the dynamics of exploited fish populations. *Fishery Invest.*, Lond., Ser. 2, **19**: 533 pp.

CUSHING, D. H. 1971. The dependence of recruitment on parent stock in different groups of fishes. *J. Cons. perm. int. Explor. Mer.*, **33**: 340–362.

CUSHING, D. H. 1972. Production in the Indian Ocean and the transfer from the primary to the secondary level. *Ecol. Stud.*, (3): 231-239.

CUSHING, D. H. (In press). Stock and recruitment studies. Lectures given in University of Washington.

CUSHING, D. H. (In press). The natural mortality of the plaice.

CUSHING, D. H. AND J. G. K. HARRIS (In press, *a*). Stock and recruitment and the problem of density dependence. *Rapp. P.-v. Reun. Cons. perm. int. Explor. Mer.* **164**.

CUSHING, D. H. AND J. G. K. HARRIS, (In press, *b*). Senescent mortality in plaice.

GREER-WALKER, M. 1970. Growth and development of the skeletal muscle fibres of the cod *(Gadus morhus* L.) *J. Cons. perm. int. Explor. Mer.*, **33** : 228-244.

GULLAND, J. A. 1965. Survival of the youngest stages of fish, and its relation to year-class strength. *Spec. Publ. Int. Comm. NW. Atlant. Fish.*, (6) : 363-371.

HARDING, D. W. AND J. W. TALBOT (In press). Recent studies on the eggs and larvae of the plaice *(Pleuronectes platessa L.)* in the Southern Bight. *Rapp. p.-v. Reun. Cons. perm. int. Explor. Mer.*, **164**.

HARRIS, J. G. K. (In press). Studies on stock/recruitment problems.

JONES, R. (in press). Stock and recruitment with special reference to cod and haddock. *Rapp. P.-v. Reun. Cons. perm. int. Explor. Mer.*, **164**.

JONES, R. AND W. B. HALL. (In press). A simulation model for studying the population dynamics of some fish species. In : "*Proceedings of the Symposium on Mathematical studies of Population Processes*", Oxford, September 1972.

LACK, D. 1954. *The natural regulation of animal numbers.* Oxford, Clarendon Press, 343 pp.

OTTESTAD, P. 1942. On periodical variations in the yield of the great sea fisheries and the possibility of establishing yield prognoses. *FiskDir. Skr. (Ser. Havunders.),* **7** (5) : 1-11.

PEARCY, W. G. 1962. Ecology of an estuarine population of winter flounder, *Pseudopleuronectes americanus* (Walbaum). Parts I-IV. *Bull. Bingham oceanogr. Coll.,* **18** (1) : 78 pp.

RICKER, W. E. 1945. A method of estimating minimum size limits for obtaining maximum yield. *Copeia,* 1945, (2) : 84-94.

RICKER. W. E. 1954. Stock and recruitment. *J. Fish Res. Bd. Can.,* **11** : 559-623.

RICKER, W. E. 1958. Handbook of computations for biological statistics of fish populations. *Bull. Fish. Res. Bd. Can.,* (119) : 300 pp.

RICKER, W. E. AND FOERSTER, R. E. 1948. Computation of fish production. In : "A Symposium on Fish Populations." *Bull. Bingham oceanogr. Coll.,* **11** (4) : 173-211.

SOUTHWARD, G. M. 1967. Growth of Pacific halibut. *Rep. int. Pacif. Halibut Comm.,* (43) : 40 pp.

STEELE, J. H. 1961. Primary production. *Oceanography. Publs Am. Ass. Adv. Sci.,* (67) : 519-538.