Self-Regulation of Japan Chum Salmon Abundance

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Japanese chum hatcheries have been releasing a large number of fry during the past 10–15 years. The biomass of Japanese chum reached 200,000 t by the early 1990s, and remained at that level until 1996. The emergence of such amounts of artificially bred fish could not but change the ecological environment in the North Pacific. We became witness of an experiment on a global scale. We were challenged by the need to comprehend the mechanism and the results of that experiment. We conducted our study between 1994 and 2001 from drift-netters which operated in the exclusive economic zone of Russia between Navarin Cape and the South Kurils from May till October.

Considerable changes have taken place in the North Pacific during these years. Catches of chum per net at the outset of our study were rather large which agreed with the high abundance of the Japanese stock. The number of fish in the Russian wild stocks of chum had concurrently gone down nearly everywhere. Alongside their rise in numbers, the average length and weight of the Japanese adult chum declined while their mean age increased (Ishida et al. 1993; Kaeriyama 1996). However, the increasing amount of culturing has had greater effects on the ecosystem than a simple arithmetic increment in the number of juveniles released. Both the greater volumes of culturing, together with increasing mean age, resulted in increased feeding times at sea. Our estimates indicate that, given the increased mean age of chum from 3 to 3.75 years, the number of fish found in the ocean at one time rises by 34%. VNIRO scientist A. Jarzhombek`s calculations indicate that the non-productive loss of energy in 1995 compared to 1970 was 34% higher as well, i.e. the functioning of the North Pacific epipelagic ecosystem became less effective. Low calorie organisms (coelenterates and tunicates) prevailed in the diet of chum. At the same time, a softening of muscle tissue in chum salmon was recorded by us in 1994. The average percentage of chum with this condition was, at that time, 35%. The seasonal dynamics of chum having softened muscles (flabby chum) followed the migration pattern features of the Japanese chum. It allowed us to assume that it was expressly Japanese chum that were exposed initially to the myopathy.

The situation began to change gradually. Catch rates dropped, chum began to consume food of high energy content: fish, squids and crustaceans. The percentage of flabby fish in catches decreased from 40% in 1996 to 7.4% in 2000 (Klovatch 2000). By 2000, the abundance of the East Kamchatka stocks of chum which had been especially subdued by Japanese chum abundance, had gone up. Total returns of the Japanese chum declined notably. Hence, it was 20% lower in 1998, and 30% lower in 2000 than in 1996 (Watanabe 2000). The age composition of catches had changed. The fish became younger. In 1996–1997 catches at sea consisted basically of five-year-olds, whereas the catches taken after 1998 began to consist mostly of four- year-olds (Gritsenko et al. 2000).

The factors causing that change were cooling in the North Pacific which began in mid-1990s, and the mechanisms of density dependence of chum in the ocean. It is impossible to quantify the role of those factors. That is why on the first factor we shall reduce ourselves to simply stating the actual condition. We shall examine the second group of factors in greater detail. The most pronounced phenomenon that occurred in the first half of the 1990s was mass myopathy in chum. We examined two hypotheses (parasitological and trophic) in order to track down the immediate causes of myopathy in chum. In 1997 Dr. P. Golovin conducted a clinical, pathalogoanatomic and microscopic studies of the muscles and the inner organs in chum, as well as helmintological examination of the intestine in order to detect organisms that might be causing softened muscles. No pathogenic organisms causing the softening of tissue in chum were found. The parasitological hypothesis did not prove to be correct. The second trophic hypothesis was corroborated by histological studies of the muscles of healthy and flabby chum. The muscles taken from all parts of body of healthy individuals of chum had normal structure. The fish with abnormal muscle structure had their destructive changes more pronounced in the groups of muscles localized immediately behind the head and under the dorsal fin.

The range of pathological changes found can be divided into three major groups. First, there were dystrophic changes observed in the muscle fibres which were expressed in damaging of the regularities in the cross-wise line pattern. In some parts the structure of fibres was totally broken and turned into a shapeless mass. The number of atypical muscular fibres reached 60%. The second group of pathological changes was notable for broad zones of necrosis resulting from complete disintegration of muscular fibres. The other part of fibres is subjected to partial

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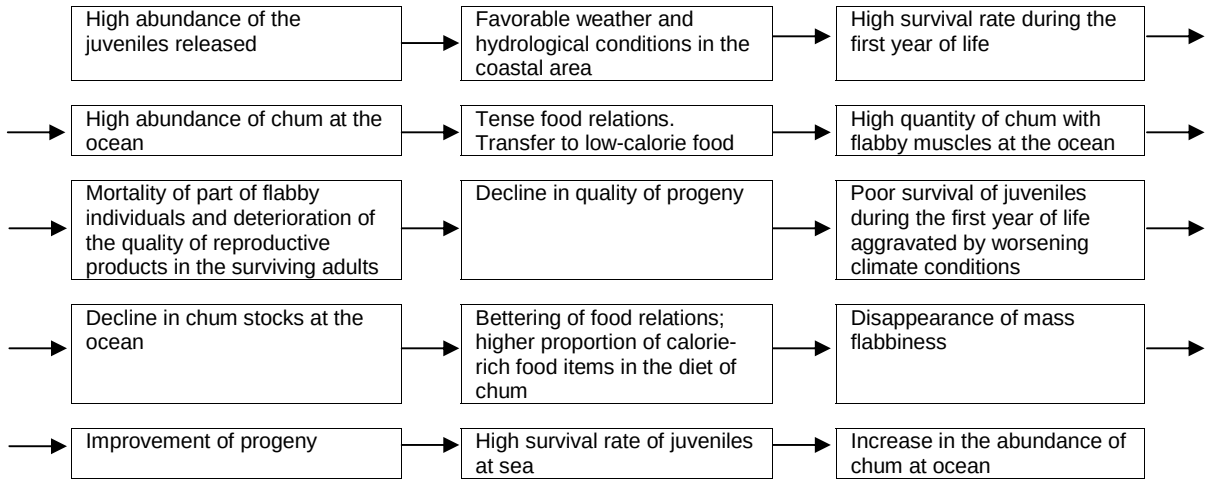
disintegration where a part of the fibrillar structure, sarcoplasm and nuclei remain. This group of muscular fibres is a potential source of rehabilitation processes. The third type of change was detected in the largest individuals where clear signs of muscular fibre regeneration were found.

We suggested that when chum salmon feed on jelly-like organisms, the expenditure of lipids and proteins is not compensated by ingestion, which causes destructive modifications and takes the form of muscle softening and changes in body shape. When feeding resumes the structure of muscular fibres is restored by regeneration, which was indeed observed in the largest fish. The process of destruction of the myofibrillar structure of the muscle fibres in chum salmon can, under favorable conditions, be replaced with their restoration. Therefore, worse feeding leads to the mobilization of reserve muscle proteins, and subsequently structural proteins. When protein products are severely limited during the growth of the fish, the destructive changes in the muscles will undoubtedly be so considerable that it will inevitably cause death. The mortality among a part of the abnormal specimens is essentially the realization of the regulatory mechanism in the period of great abundance of population (Lange and Klovatch in press).

The situation described is an example of population abundance control on the tissue level. In order to understand the mechanism of regulation we carried out a biochemical analysis of muscles, liver and gonads of normal and flabby female chum. We detected considerable differences between them in a number of characteristics. There were differing volumes of water, protein, lipids, carbohydrates and nucleic acids which points to a changed metabolism in organs and muscles. The metabolic changes included greater muscle energy reserve losses, and exhaustion of lipid reserves in the liver, offset by the carbohydrate component. The shift in metabolism allowed flabby females to form relatively valuable oocytes, in terms of their protein and lipid composition, at the expense of higher energy loss. However the RNA content, a matrix for synthesis of proteins, is lower in flabby fish oocytes than in healthy fish gonads, 0.209±0.047 and 0.441±0.0809% of dry weight, respectively. This causes low quality progeny from flabby chum females, and the lower survival rate is responsible for lowering the population abundance.

This is exactly what we observed in Japanese chum salmon. Although releases of juveniles from Japanese hatcheries have remained constant (about 2 billion) throughout the recent decades, the return of brood stock in recent years has been invariably going down. This picture indicates a higher rate of mortality in Japanese chum during the sea period of life as compared to the previous years. The recent decline in aggregations of large-sized chum in the ocean is another proof of the death of a significant part of fish occurring at the initial stage of the sea period of life. The self-regulation of the Japanese chum abundance is shown in Fig. 1.

Fig. 1. Scheme of Self-Regulation of Japan chum salmon abundance.



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