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Types and frequency of malformations and mortality in eggs of Arcto-Norwegian cod: A field study

by

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Abstract

Embryological studies on living cod eggs in NUNC-trays showed that spontaneous malformations during embryo development are lethal. They are called natural egg mortality in contrast to external lethal factors, as predation, extreme physical conditions and pollution.

The natural mortality in cod eggs, both coastal and Arcto-Norwegian, is significantly reduced from first to second spawning, from laboratory experiments.

The same embryological method was used during field studies on natural egg mortality on planktonic cod eggs in the spawning area in Lofoten, Northern Norway, in the March - April years 2000 and 2001. The types malformations which was found in a fild also corresponding to certain morphogenetic movements in embryodevelopment and equel discover in the experimental work. Most part of lehal malformations (more than 50 %) is identified after hatching only. The pattern of types malformations differ between spawning grounds and to be consistant during two years. The frequencies of malformations to the end spawning period significantly reduced of 29,7 to 9,8 % for Vestfjord.

To demonstrate the higher natural egg mortality in eggs from first time spawners, two strategies were followed:

1. Comparing natural egg mortality in egg samples from 2000 and 2001, collected during same period and from the same areas.

2.On the basis of the acoustic survey of the spawning population of Arcto-Norwegian cod in Lofoten, areas of high frequency of first time spawners were compared.

Luckily, in the year 2001 the proportion of first time

spawners was specially high. A comparison between the years 2000 and 2001 demonstrated an increase in natural mortality from 23 to 31 %.
In Vesterålen, the area of highest frequency of first time spawners, natural egg mortality increased from 31 to 46 %. Because of two systematic errors the numbers are minimum values.

Introduction

A practical ichthyoplankton problem has been studied using fundamental, modern knowledge of the development of the lost fish (Ballard, 1981; Trinkhaus 1984, 1986). Of special importance, experimental and teratology of fish has been used (Nicholas, Oppenheimer,1942; Oppenheimer, 1947; Laal, 1981; Longwell, 1977). The communication of these groups and the ichthyoplankton scientists have been very limited. Combining methods and using embryological characters and terminology, Makhotin et al. (1984) carried out a study on the White Sea cod, in many ways a model for the present study.

Malformations in egg and larval development are demonstrated both in experimental and field work. (Longwell, Hughes, 1980; Kjørsvik, Stene, Lønning, 1984; Stene, 1987; Solemdal, Makhotin, Fonn, 1998.)
Basic studies in western countries (Nicholas 1942, Oppenheimer, 1947; Laal, 1981, Longwell, 1977), and in the Soviet Union (Svetlov 1960; Vladimirov, 1975) have demonstrated that malformation during embryogenesis occur spontaneously without any external influence.
However, in standard studies of ichthyoplankton, malformations have usually been connected to pollution and extreme physical variations (Camoron et al., 1992; Westernhagen, et al.,1988; Rosenthal, Alderdice, 1976; Mukhina, 1996), though exceptions to this opinion exist (Kjørsvik et al.,1984, Stene, 1987).
Of the large western literature on the different causes of egg mortality, mainly predation, only few authors believe that egg mortality resulting from internal critical developmental stages are of significance in the general egg mortality pattern (Blaxter, Rotchild,1986). The lack of basic knowledge in this field is clear from the following citation by Rotchild (1986): "The possible causes of non predatory egg death are not well understood, but they might be linked to cytological competence or to unfavourable environmental conditions."

Laboratory experiments in unpolluted sea water have shown that eggs from first spawning cod have a significantly higher mortality than for the same individuals as second spawners. This maternal factor (Solemdal, 1997) were similar in two different stocks of cod, coastal cod (Solemdal, Kjesbu, Fonn, 1995) and the Arcto–Norwegian cod (Solemdal, Makhotin, Fonn, 1998). The lethal cause of the eggs were malformations occurring during egg development or the hatching process.

These results are supposed to have some implications on the size of the reproductive potential of the spawning population, caused by natural and human influence on the age/size/condition of the spawning population. Hopefully it will be possible to describe the general reproductive significance in the annually recruitment variations. Until now lacking and inaccurate methods to calculate the reproductive capacity have reduced the reliability of the reproductive capacity calculations.

The extensive development in recruitment mechanism studies, focusing mostly on environmental effects, like predation and the degree of synchrony between fish larvae and its prey, has reduced the significance of the spawning population as a recruitment factor. The present study, together with other recent studies in reproduction, hopefully will give the spawning population her status back as the basic recruitment mechanism (Solemdal, 1997).
Today many spawning populations are heavily exploited and reduced since no clear relation between size of spawning population and recruitment have been demonstrated.
It should be remembered that manipulation of the fishery is the only tool man has to influence upon the future condition of these resources, including general reduction in catches, and regulations in sensitive areas and periods of the year. The majority of commercial fish stocks today are in a state that need fishing restrictions and special regulations to keep away from recruitment overfishing.

Since laboratory experiments, though showing identical results for two stocks of cod, always are looked upon with suspicion, field investigations on the types and frequency of malformation of pelagic eggs of the Arcto-Norwegian cod were carried out in the years 2000 and 2001.
The paper gives the results of these studies and a comparison with the laboratory experiments.

Material and methods

Samples of ichtyoplankton were collected by vertical net hauls 100 – 0 meter depth. Diameter of the net was 80 cm and the mesh size was 375 microns. Speed of net during sampling was 0.5 m/s. The

investigated area, Vesterålen-Røstbank-Røst-Vestfjord, are shown in fig. 1.

The material was sampled in the period 17 March – 9 April 2000 and 21 March – 8 April 2001 with R/V "G.O. Sars". In addition egg samples were collected from the Vestfjord during the periods 9 – 20 March and 9 – 26 April 2001, to study egg mortality and other egg characteristics in the beginning and at the end of the spawning season. Cod eggs were sorted live from the ichthyoplankton samples, and chilled with ice during the sorting procedure. Cod eggs, and also eggs from other species, were counted, the developmental stage and abnormalities recorded. This is the traditional method to describe the frequency of abnormalities in the egg samples (Mukhina et al. 1996). But this method only gives a "snapshot" of the instantaneous situation, and say nothing of the potency of lethal malformation in the eggs. The instantaneous methods are often used to describe defect embryos from external factors, f.inst. pollution (Cameron et al. 1992). For studying the frequency of malformations in pelagic eggs caused by the internal state of the gonad, this method is incomplete.

The present method includes observations of abnormalities on live eggs throughout the egg development from before cleavage or the blastula stage.
Individually eggs in normal development and in early stage were carefully put into NUNC trays, containing 2 ml of 70 % autoclaved seawater. The seawater source was the same for all NUNC-tray experiments, in the laboratory in 1997 and 1998 and in the field 2000 and 2001. Water from the 140 meter inlet to the laboratories at the Institute of Marine Research, Bergen, was used. The seawater contained negligible amounts of aromatic hydrocarbons and PCB (Palmork & Wilhelmsen, 1988).

The NUNC trays were put into a refrigirator at 4 degrees centigrades. The eggs were inspected regularly, and development of abnormalities and death of eggs and larvae recorded. Different kinds of abnormal development were studied. The experiments was terminated when the yolk sac was 2/3 resorbed. To illustrate the difference in malformation frequency between the two methods some parallels from the two methods are shown in table 1, demonstrating a large difference between the methods. The division of cod embryo development on six stages was given in Fridgeirsson (1978).

Results

Fig. 2 includes photos of the main types of the cod egg malformations. Fig 3 shows the malformed larvae, described from the cruise in 2000.
The following types of malformations which were found in a field study in 2000, corresponding to certain morphogenetic movements in the embryo development:

1. Zygote forming – unfertilized eggs. There were about 5 % unfertilized eggs composed from other malformations. These eggs remain transparent and look as live, keeping buoyancy, and sink after start of epiboly, fig. 4.

2. Cleavage division – irregular, first cleavage and pseudocleavages. There were about 5 % irregular first cleavage, fig. 2A. As known from experimental embryology (Oppenheimer, 1936; Nichols, Oppenheimer, 1942) the first blastomers were not completely equipotenct. About 50 % of the eggs had irregular cleavage and were still able to live to hatching.
There were about 1% pseudocleavage from total material. This type remains transparent till epiboly finished, then dies and sinks, fig. 2B, fig. 4.

3. Blastulation – the first critical stage in development. Embryogenotype starts and embryo development can be continued. If not started there is no morphogenetic competence for further development. The inner cells of the blastodisc are not differentiated to hypoblast and epiblast layers. This is the first peak of mortality in the development, and compose about 2 % of total malformations.

4. Gastrulation – hypoblast cells forming small disorder aggregates in the germ ring, amounting to 1% of the total malformations. The malformation appears later as different faults in the bilateral axial structures, but some of these embryos (about 1%) lived to larvae, fig. 4.

5. Epiboly –

a) Multinuclear cyncytial layer (YSL) can not completely substitute of the yolks cytoplasmical layer. The embryo with this malformation die fast and constitute 2 % of total malformations, fig. 4.

b) Perigerm or cellular envelope layer (EVL) covering together with periblast layer the surface yolk. The cells envelope layer loose contact, periderm is destroyed and embryo will die fast. The embryos who had these malformations die fast and composed 2 % from total malformations. Fig. 4.

6. Organogenesis – start axial convergation of hypoblast cells and embryonic body begin to form

a) abnormal correlation between two relatively independent morphogenetic movements.

1. Of the epiboly provisional structures (periblast with periderm).
2. Of the axial convergation hypoblast cells. This type of malformation forms twisting of the body and blisters before tail node and under germ gut, fig. 2 D,E.

b) the hypoblast cells forming axial embryo structures changing contacts and motility properties. Notochord forming central nervous system have become gray or light brown. It is sign of necroticouse process in this ectodermal cells population, fig. 2, F, G. Part of cells dead, head structure and notochord reduced size. This type of malformation produced many variations abnormalities: microcephalia, cyclopean, micropia, sharpless axial structures, crook tail and body, and other small changes in the embryo organs, fig. 2,F, G, H, I. The extent of abnormality depend on level of changed properties of hypoblast and epiblast cells, fig, 3, A, C, E. 32 % had this type of malformation, 16 % of these were living to larval period, fig. 4.

7. Differentiation of tissue and embryo organs, epiblast cells converge on the body embryo. Late malformations – some epiblast cells are not included in the axial structures, forming different faults in the bilateral symmetrical organs: jaws, gills, pectoral fins, fig. 3,E. Frequency of this type malformation was low.

8. Definite function of embryo organs. Provisional structure of larvae formed, embryo becomes motile. Dysfunction of the hatching glands prevent hatching of embryo, fig. 2, K. dysfunction of chloride cells resulting in loss of water in yolk, or hyperhydration of head hydrosinus, pericardium and protopterygoid folds. Fig. 2, J.

9. Most part of lethal malformations can be identified after hatching only. Many larvae had straight body and normal organ structures, but die after hatching. It occurs because provisional epithelium in the yolk surface or else protopterygoid fold and head hydrosinus lost cell contact, provisional epithelium destroyed, the larvae cavity opening and larvae die, fig. 3, B, D, F. This malformation composed 57 % from malformation after hatching.

In fig. 4 the longevity of different types of malformations are shown, from appearance to death, 2000.

In fig. 5 natural egg mortality from different developmental stages are shown for the years 2000 and 2001. It is clearly demonstrated that the mortality is concentrated at hatching and a few days posthatch, while the mortality at the more sensitive stages, specially the blastula, is relatively low.

Table 2. Frequency of natural egg mortality from four spawning areas and the average numbers, 2000, 2001, including egg diameters from 2001.

The frequency of natural egg mortality from Vesterålen, Røstbank and Vestfjord with abundance of first time spawners, years 2000 and 2001 are shown in fig. 6. Abundance of first time spawners are results from Korsbrekke, 2000 and Salthaug, 2001.

Fig. 7. Average and range of egg diameter from the investigated spawning areas.

Fig. 8. Biological parameters of cod females, caught with danish seine at Henningsværstraumen, Vestfjord 17 March, 10 April and 25 April: Size, type, proportion of first time spawners and maturity stage.

Table 3. Natural egg mortality, Vestfjord, during three periods, 2001.

In fig. 9 egg diameters throughout the spawning period, from the beginning of March to the end of April are demonstrated.

Table 1 shows frequency of malformations according to the direct observation of the ichtyoplankton sample compared to the results from the NUNC-tray method. Eggs from the same samples.

Table 4. Diameter of normal developing eggs and eggs with malformation.

Discussion

The NUNC-tray method, using eggs of the first stages and following them systematically throughout the development until hatching, reveal all types of malformations and "save" all dead eggs from sinking out of the "pelagic" community.

Malformations in pelagic egg have also been studied in NUNC-trays from artificial fertilized eggs with asymmetric cleavage. A large part of these eggs are found to produce normal looking and active larvae (Wallin, Nissling, 1988; van de Braak, K. v. 1994). Pelagic eggs of different stages have also been studied in NUNC-trays (Westernhagen et al. 1988).

The contrast to the NUNC-method, used in the present study, is the direct observation of the ichthyoplankton sample, fresh or conserved. Since malformations in early development stages has only

partly appeared and late stages are already dead and sunk out of the pelagial, the frequency of malformations by this method will always be low. Mukhina et al. (1996) investigated malformations in cod eggs in areas close to the present study, and found annually variation in the frequency of malformations, 9 % being maximum. She indicated an external lethal factor , the pollution from Norwegian oil drilling in the North Sea and off Northern-Norway, to be the cause of the malformations, and did not discuss the possibility of natural egg mortality.
In table 1 a comparison of the two methods are demonstrated on eggs from the same sample collected in 2000, clearly demonstrating an increased natural egg mortality due to the more extensive analysis of the egg material in the NUNC-trays. The advantages of the NUNC-method is clear:
It is a systematic experimental method, starting on very early egg stages, e.g on equal terms. A small number of eggs are needed for the NUNC-tray experiment, in contrast to the "snapshot" of the unsorted eggsample for a direct inspection of malformed eggs. In addition, varying developmental composition of the egg samples, between areas and years, strongly will influence on the results from this method.
On the other hand the direct inspection method has a longer tradition, with better possibilities to perform comparisons with material from earlier studies.

There are two types of uncertainties using the NUNC-tray method for the evaluation of the natural mortality of the eggs. Firstly, the capture of the eggs in the vertical net will damage some of the eggs, selecting the early stages, which are the most susceptible to mechanical stress (Rollefsen, 1932). Since the vertical hauls are standardized according to mesh size, diameter, towing speed, 0.5 m/s, the frequency of damaged eggs should be similar.
The selection of live stronger eggs in early stages from the vertical hauls, the natural egg mortality from the NUNC-tray measurements will be lower than in an ideal situation.

The cumulative mortality is the sum of different lethal malformations from every stage in embryo and early larvae development. Time of appearence and longevity of the different malformations is shown in fig. 4. From this figure it becomes evident that in most eggs, the malformations were observed in Epiboly and Hatching.
In both cases the malformations are connected with abnormal properties in the provisional structures of the embryo - periderm with periblast and the provisional epithelium of larvae. Hatching, constitutes more than 70% of the malformations.

Comparing the curve of natural egg mortality from different developmental stages in 2000, 23%, and in 2001, 31%, fig.5, higher level of natural egg mortality on epiboly and after hatching was found in 2001. In early stages, gastrula and hatching the mortality was the same in the two years.
The general higher frequency in natural egg mortality after hatching in 2001, fig.5, corresponds to the frequency in Vesteraalen,fig.6, in 2000. This indicates that the reduced viability of first time spawner embryos is found just after hatching.
This is caused by weak contacts between cells of the provisional epithelium of the larvae. The same was found in the laboratory experiments.

Fig. 6 and table 2 show the mixing of first and second spawners in different parts of the spawning areas. The highest frequency of first time spawners, 84 %, is found in the northern area in 2001. In the vertical hauls from areas with low frequency of first time spawners a significant contribution of eggs from multiple spawners are found. Since these eggs are undistinguishable from first spawners eggs, the eggs used in the NUNC-tray experiment will be a mixture and the natural mortality will be lower than if using only eggs from first time spawners. The results from our laboratory experiments on first and second spawners can indicate a correction in mixed spawning areas.
The field results of natural egg mortality are significantly lower than the results from the laboratory experiments in 1997 and 1998, using the same individuals as first and second spawners of Arcto–Norwegian cod, 54.9 % and 37.1, respectively. The frequency of egg mortality is highest in Vesterålen 2001, the percentage of first time spawners being as high as 84 %, fig. 6. The reduced natural egg mortality in this area compared to the laboratory results are partly the result in the occurrence of eggs from multiple spawners. Other reasons of difference in natural egg mortality between field and captive cod could be:

1. Some of the captive cod females showed disturbances in the rhythm of spawning, and though the worst cases were excluded from the material, spawning in captivity can be negative compared to natural spawning.

2. The experimental cod were not fed during the spawning period, while wild spawners, specially on the Røstbank, are eating herring to a great extent during spawning.

3. Sampling of pelagic eggs occurred during peak spawning, while eggs from all egg batches were used in the laboratory experiments.

In addition it is logically to assume that natural

spawned eggs in general are of a somewhat better quality.

Kjørsvik, Stene, Lønning, (1984) and Stene (1987) used a genetic technique to describe the spontaneous malformation in cod eggs during peak spawning in the unpolluted Balsfjord in Northern – Norway, and found a high percent of abnormal mitoses of 7% to 70 % in artificially fertilized eggs, and 6 % to 60 % in eggs collected from the plankton, respectively. Also these authors were found that average 20 % of chromosome abnormalities for this spawning ground in start of spawning significant reduced to 0 % in end spawning period. Our investigations early, middle and end spawning periods in Vestfjord spawning ground confirm those results (tab. 3). This study is in principle the same type as the present, and the results are on the same level.

The types of malformations in the pelagic cod eggs from the field, in both years, were identical to those found in the laboratory studies.
The cod eggs from captive cod in Bergen (Solemdal, Makhotin & Fonn, 1998) and the eggs from the Lofoten spawning sites both developed in the same environment, the NUNC-tray. The 70 % autoclaved seawater was in both studies taken from 140 meter depth inlet to the Institute of Marine Research, Bergen. This seawater contained negligible amounts of the most common pollutants, aromatic hydrocarbons and PCB (Palmork, Wilhelmsen, 1988).

From the investigated area in Lofoten, the average natural mortality of the cod eggs was 23,2 and 31 % in the years 2000 and 2001, respectively, table 2. It should be remembered that the samples were collected within the same area and closely during the same periods both years, table 2. In 2001 egg diameter was measured from the different areas. The egg size was very similar, table 2. The egg diameter distribution and average size are shown in fig. 7. The diameter in normal and abnormal developing cod eggs were tested on a limited number, table 4, but the difference was not significantly different.

The sampling period coincide with the peak spawning of Arcto-Norwegian cod (Pedersen, 1984). These investigations were carried out during the 1970 – 80-ies, being very stable from year to year, 50 % of the eggs spawned at 1 April. Possible recent changes in the peak spawning in Lofoten due to changes in the age structure of the spawning population has not been carried out.

Looking at the natural egg mortality from the four areas, the frequencies indicate a general increase for all the areas in 2001 compared to 2000, with a maximum in Vesterålen, 84 % in 2001, and lower values in the other areas with smaller contribution of first time spawners, fig. 6, table 2. Egg diameter, measured only in 2001, are very similar during sampling in the different spawning areas, carried out during the same period the two years. These results are a clear indication that the increased natural egg mortality is connected to age, size and condition of the spawning cod females.

From earlier studies (Hiemstra, 1962; Sundby, Solemdal, 1981; Kjesbu, 1994) a significant reduction in egg diameter throughout the spawning season have been demonstrated. In 2001 egg diameter measurements were carried out throughout the spawning period, 9 March – 26 April in the Vestfjord area, fig. 9. The egg diameter at the end of spawning is significant reduced compared to the diameter in the start of spawning, while the diameter in the cruise period is on a more equal level, fig. 7. In table 3 the natural mortality of the eggs from Vestfjord, during start, peak and end of spawning season. The natural egg mortality is reduced at the end of spawning. This is surprising as egg quality of a batch spawner is supposed to be lower low at the end of spawning.
Samples of female cod spawners were caught from the periods shown in table 3 and analysed according to the following characteristics: length, maturity, type and frequency of first time spawners. Data of the spawners are given in fig. 8 (a-f).
Spawning cod were caught at Henningsværstraumen, Vestfjord, with Danish seine, and the females analysed. The size distribution of the first time spawners, fig. 8 a, indicates an increased size throughout the spawning season. The smallest spawners, the 50 cm group, were caught in March. Cod larger than 90 cm were only caught in last half of April. Fig. 10 c includes the total material with a similar trend. Frequencies of first time spawners during spawning period are shown in fig. 8 b.
According to earlier observations on spawning cod in Lofoten (Sund, 1937), older spawners arrive earlier at the spawning sites. Our small material did not verify these results. Similar behaviour is observed on the Icelandic cod (Marteinsdottir, Petursdottir, 1995), and other species.
Kjesbu (1994) found no special size trend for the spawning start of Loften cod, using a biopsy method to calculate time to spawning of first batch on the basis of size of the ocytes, It must be emphasized that this study also are based on a small material.
Of more interest for the present problem is the distribution of cod type: Arcto-Norwegian, coastal, etc. (Rollefsen, 1932) and the distribution of the maturity

stage throughout the spawning season. Fig. 8 d, indicates that the proportion of running females are at the maximum at the end of the study period. Analysing maturity distribution separately from Arcto-Norwegian and coastal cod shows clearly the delayed spawning of the Arcto-Norwegian cod, fig. 8 e, in conflict with other authors. But also in the present study the material is small.

Since the fraction of first time spawners are on the same level during the three periods in the Vestfjord, fig. 8 b, other factors must be responsible for the variation in the natural mortality in the extreme points of the spawning period. Kjørsvik et al. (1984) using a genetic method found a similar reduction in the frequency of genetic abberations at the end of the spawning period in the unpolluted Balsfjord, Northern Norway. In Verrabotn, a narrow part of the Trondheimsfjord, with stable physical environment, extremely low frequency of egg malformation was observed (Kjørsvik, pers.com). Observations on low frequeency of malformations in the last batches are observed, but not as a general phenomenon (Makhotin, unpublished).

A comparison of diameter from normally and abnormal developing eggs did not show any significant difference in diameter, table 4. This test was performed with eggs from the last part of spawning in Vestfjord, 28 March – 12 April.

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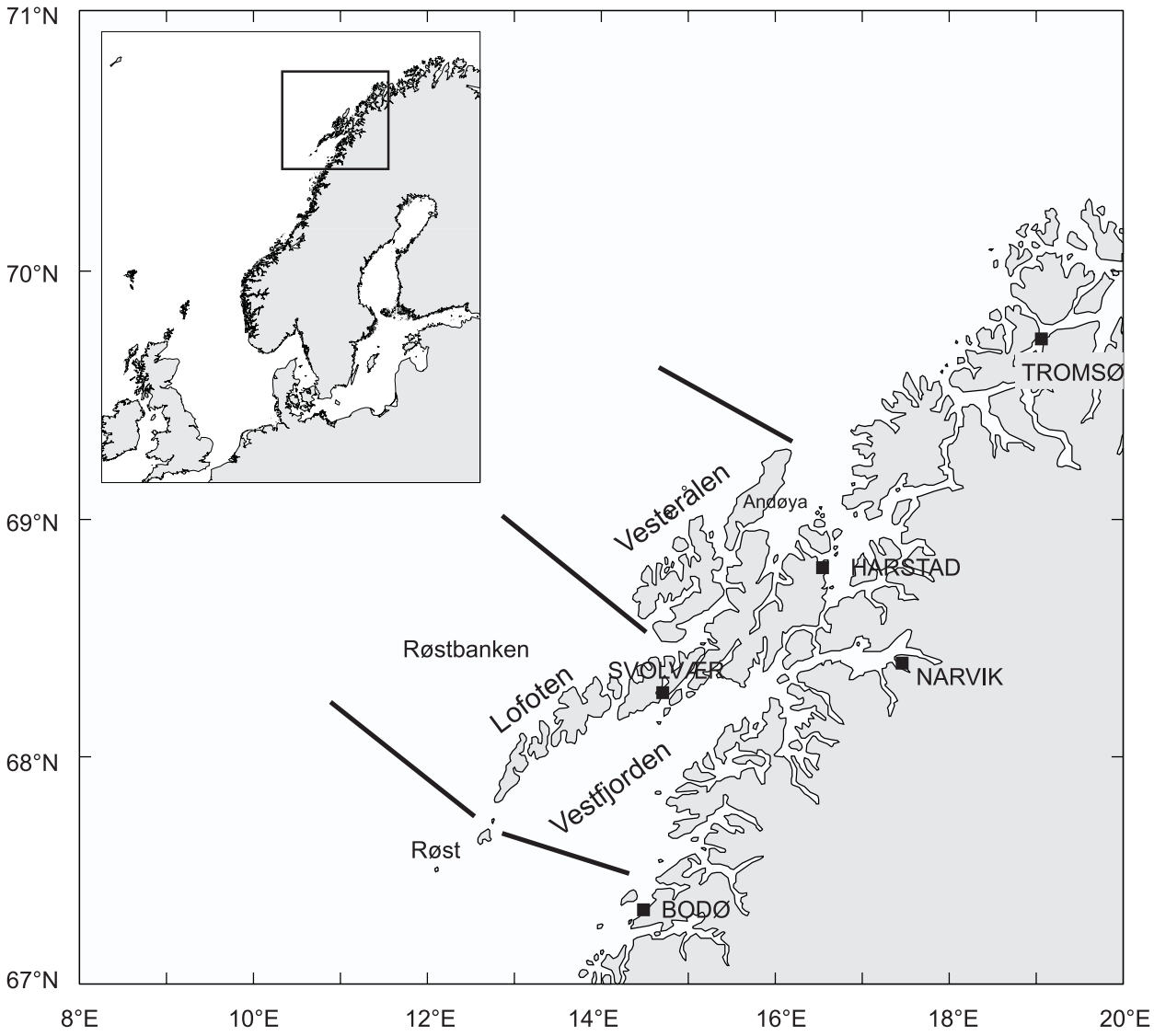


Fig. 1: *Area of investigation.*

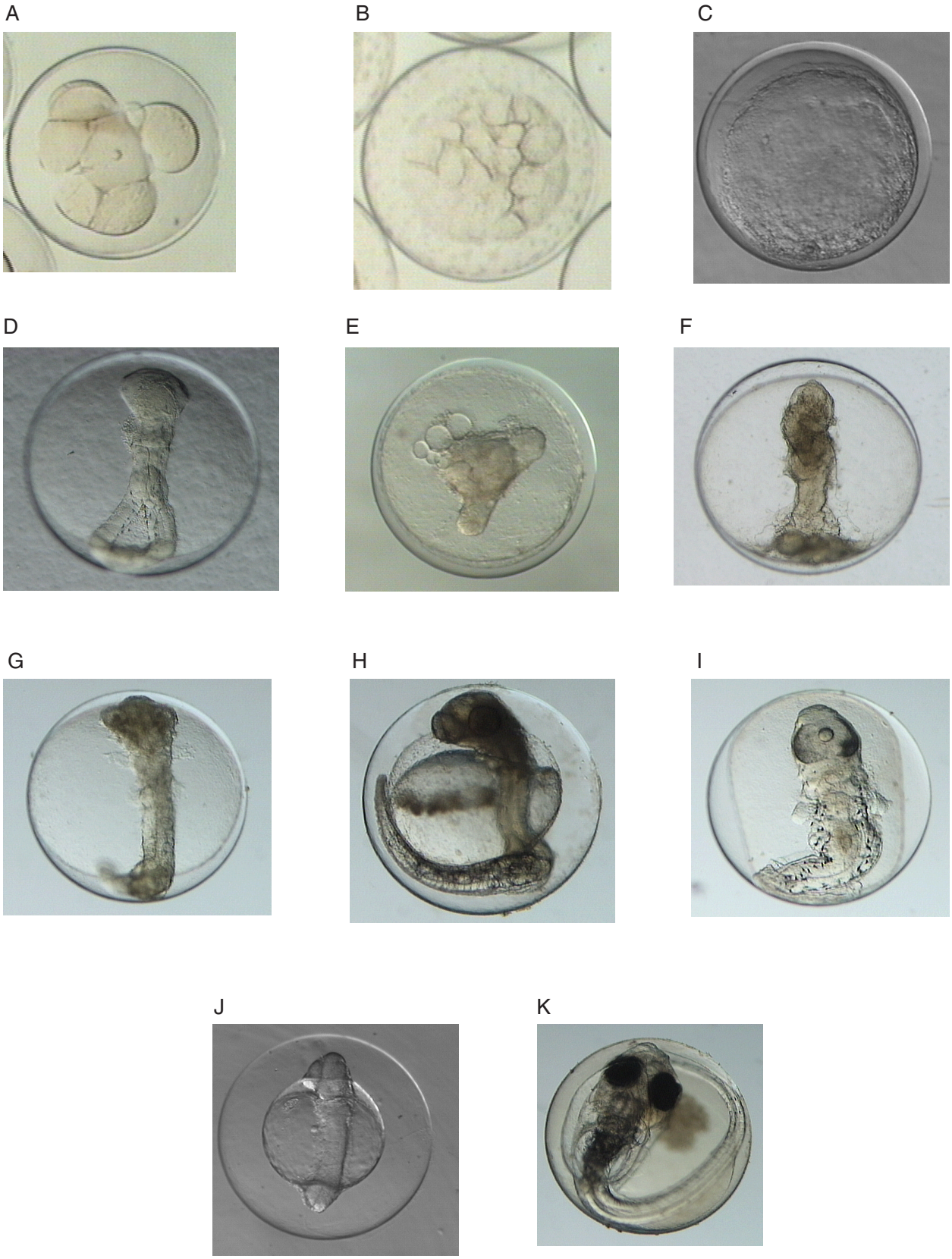


Fig. 2: *Examples of malformations during egg development.*

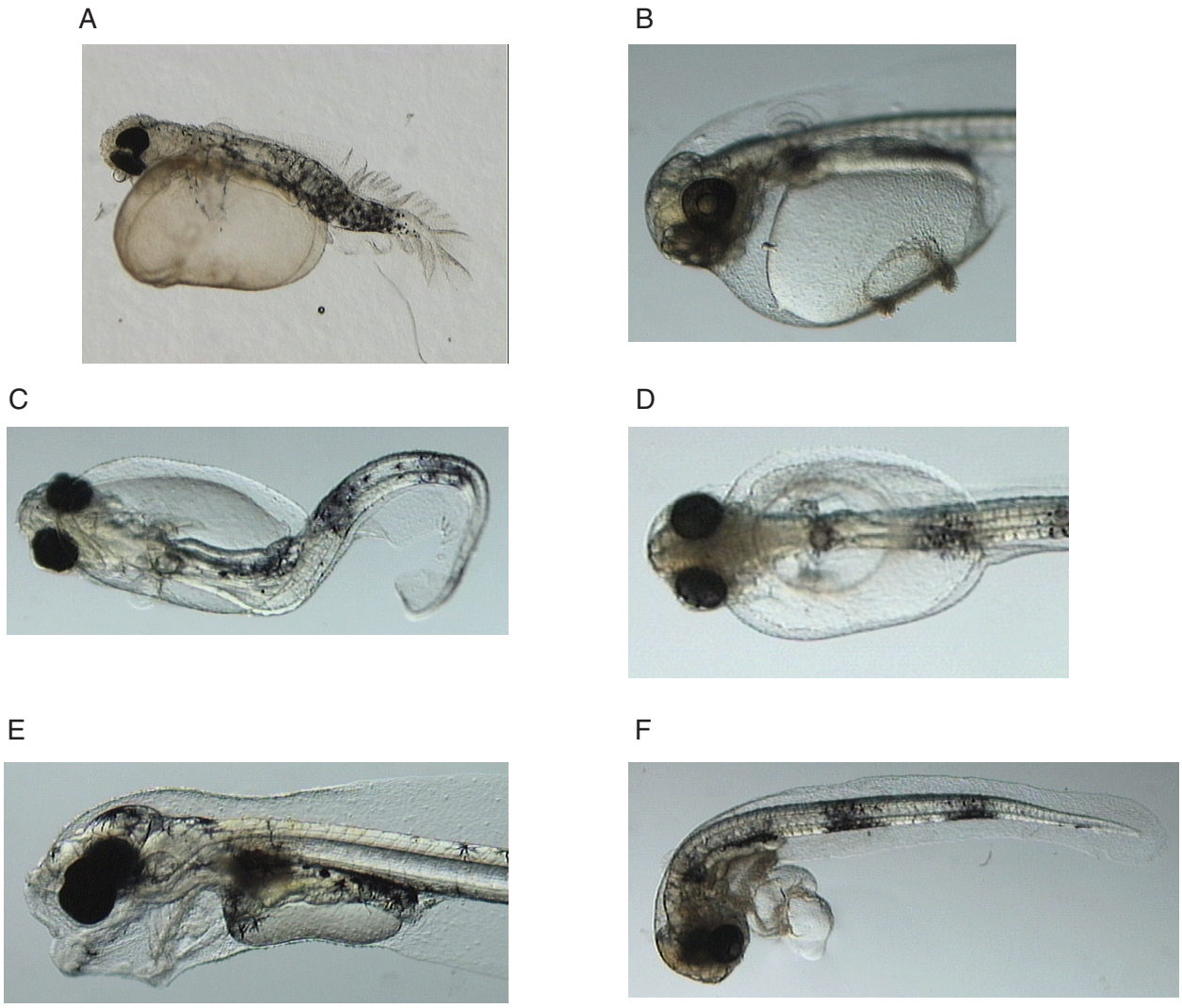


Fig. 3: *Examples of early larvae malformations.*

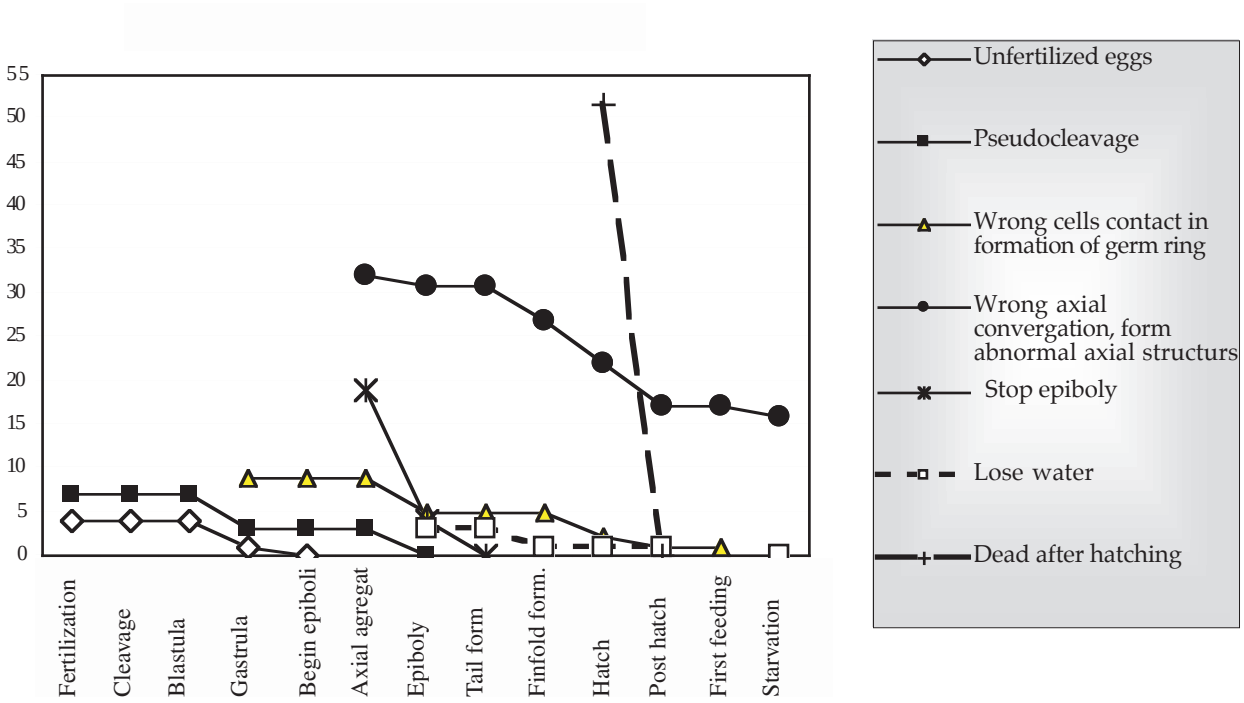


Fig. 4: *Living period for different types of malformations.*

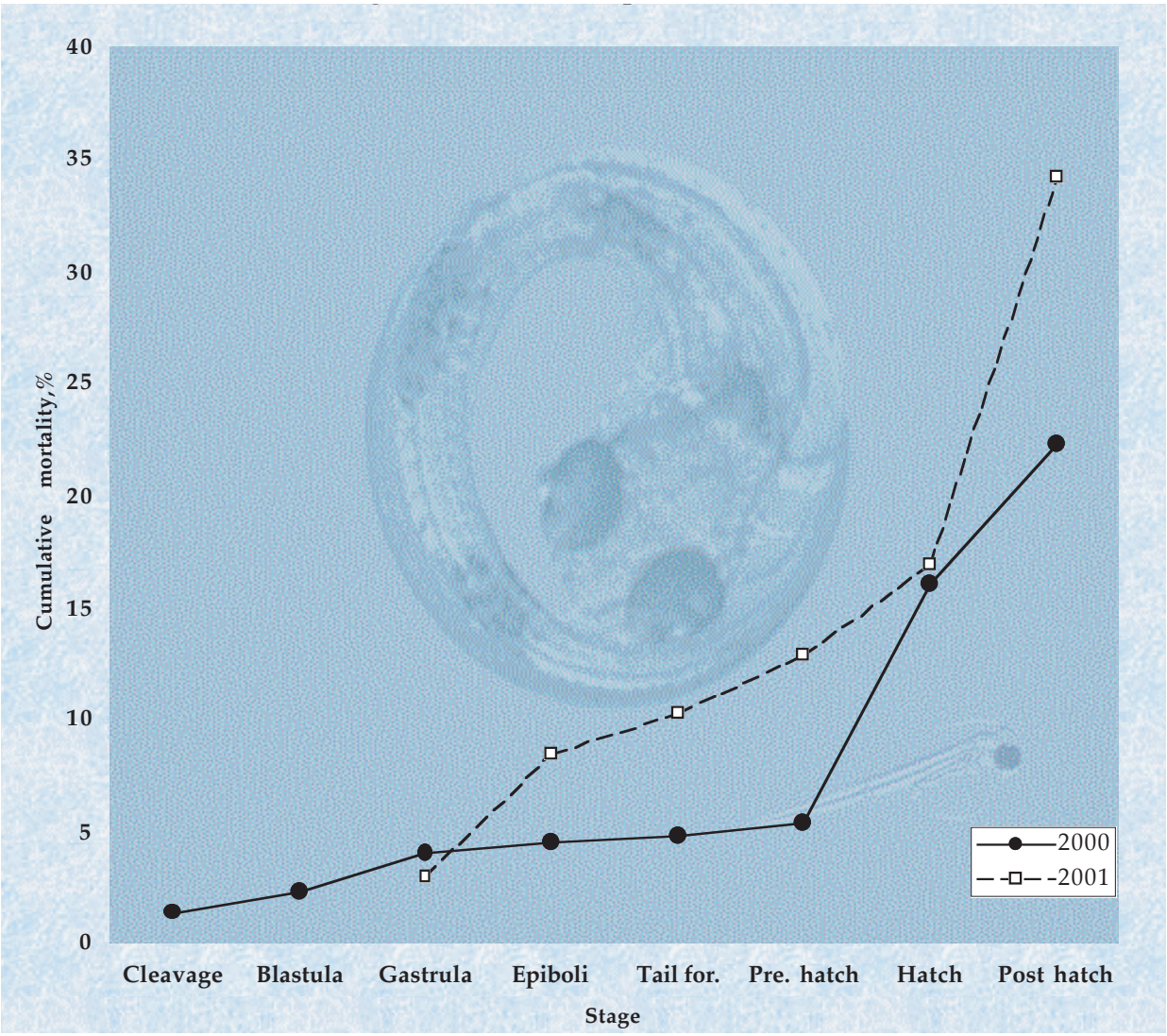
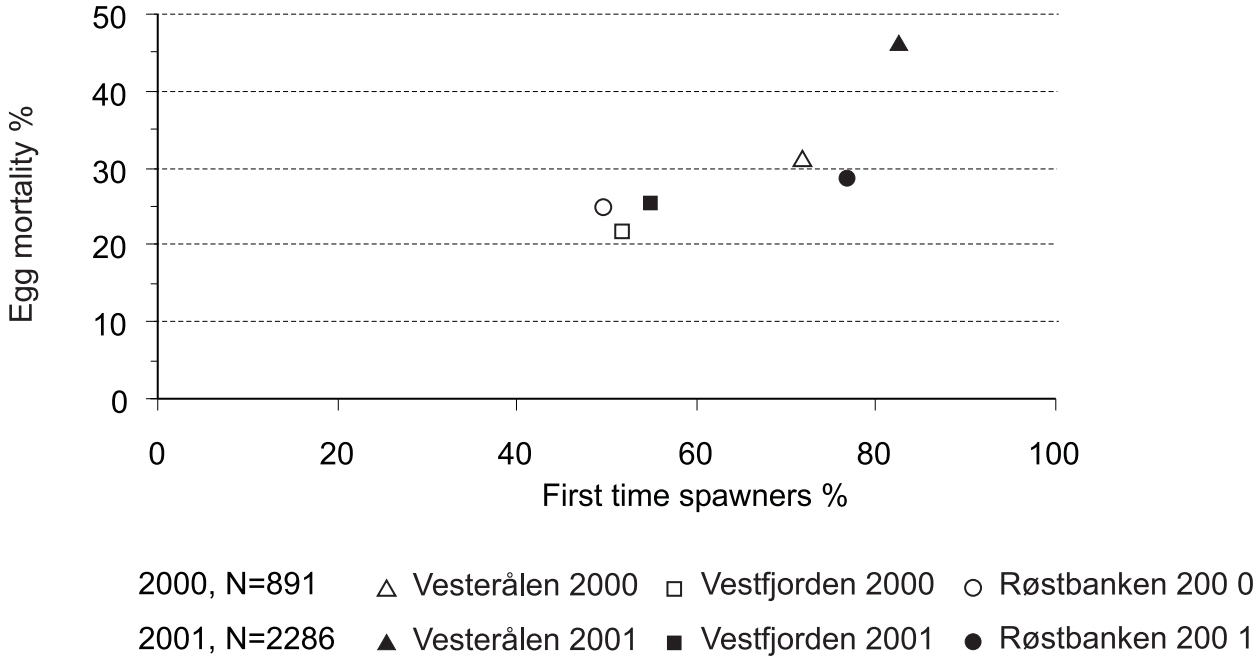


Fig. 5: *Malformation rate of cod development in the Lofoten spawning ground. March-April 2000/2001.*



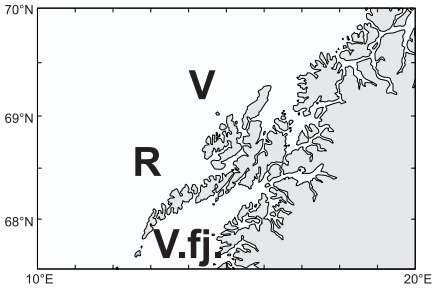


Fig. 6: *First time spawners and natural egg mortality %.*

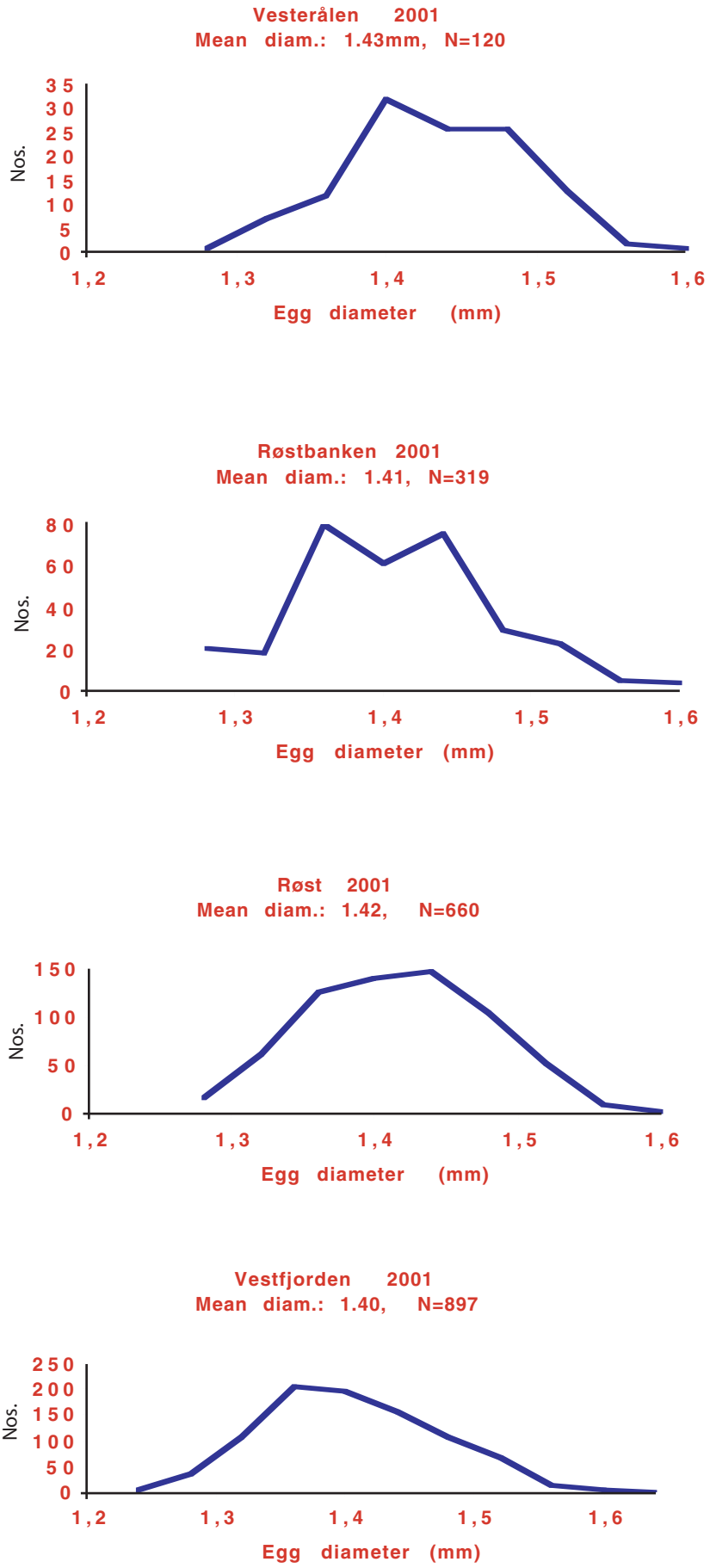


Fig. 7: *Egg diameter distributions from four spawning sites, 2001.*

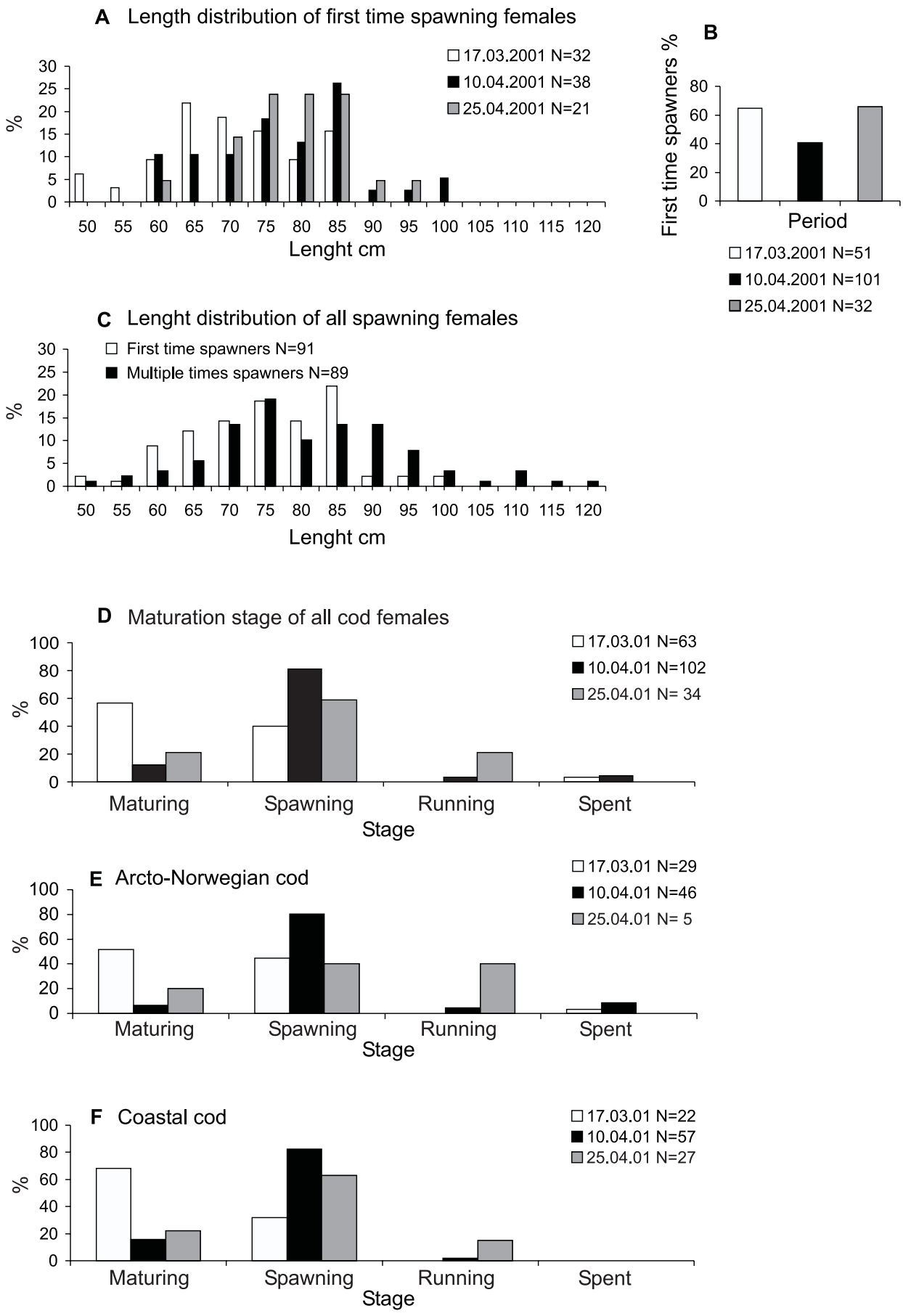


Fig. 8: *Characteristics of cod caught by Danish seine in Vestfjorden, 2001.*

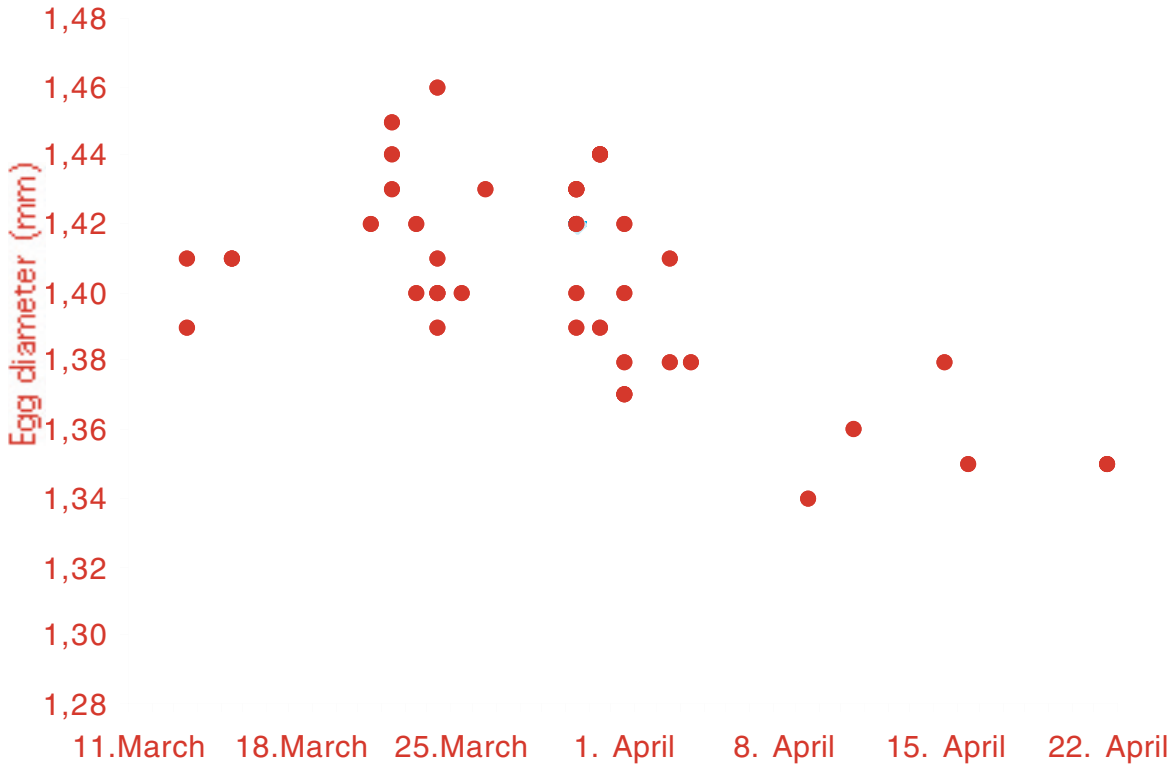


Fig. 9: *Mean diameter from all egg samples, 2001.*

Table 1: *Comparison of two methods describing the frequency of lethal malformations in pelagic cod eggs:*
Method 1: Inspection of ichthyoplankton samples.
Method 2: Description of lethal malformation from the blastula stage to post hatching. The egg develop in NUNC-trays.

Method I Inspection of Ichthyoplankton sample		**Method II** NUNC-tray
Nos. Cod eggs	Lethal malformations, %	Lethal malformations, %
43	2.3	33
99	1.0	24
473	0.8	39
159	1.9	37
34	2.9	20
52	3.8	27
114	7.0	46
200	1.0	49
147	2.6	34.4

Table 2: *Localities and periods of sampling and natural mortality of cod eggs, 2000 and 2001.*

Locality	2000		2001		Diameter	No. of eggs
	Period	Nat. mortality	Period	Nat. mortality		
Vesterålen	March 25-29	30.7	March 22-23	45.9	1.434	120
Røstbanken	March 30-April 3	25.0	March 24-26	28.7	1.409	319
Røst	April 4	18.0	March 27-31	26.4	1.416	660
Vestfjorden	April 5-6	21.5	April 2	25.3	1.403	897
Average		23.8		31.0		

Table 3: *Frequency of egg malformations and egg diameter from pelagic eggs in Vestfjord in three periods.*

Period	Nat.egg mort. %
10.-16. March	29,7 (235)
2. April	25,7 (378)
10.-24. April	9,8 (567)
Average	18,6 (1176)

Table 4: *Diameter of normal and abnormal eggs.*

P: 0,5870	Egg Diameter	
	Normal	Abnormal
Average	1.386	1.38
Standard devation	0.066	0.063
Nos. eggs	164	44