echolamines II, N. Weiner and U. Trendelenburg, Eds. (Springer-Verlag, Berlin, in press); I. W. Chubb, W. P. DePotter, A. F. DeSchaepdryer, *Naunyn-Schmiedebergs Arch. Pharmacol.* **274**, 281 (1972); J. P. M. Finberg and M. Tenne, *Br. J. Pharmacol.* **77**, 13 (1982); L. Simpson, *J. Pharmacol. Exp. Ther.* **205**, 392 (1978); B. Blackwell, *Lancet* **1963-II**, 849 (1963); C. Braestrup, H. Anderson, A. Randrup, *Eur. J. Pharmacol.* **34**, 181 (1975); G. B. Baker, M. Raiteri, A. Bertollini, R. Del Carmine, P. E. Keane, I. L. Martin, *J. Pharm. Pharmacol.* **38**, 456 (1976); S. R. Philips, in *Non-catecholic Phenylethylamines*, A. D. Mosnaim and M. E. Wolf, Eds. (Dekker, New York, 1978), p. 113. R. Ashkenazi, in *Enzymes and Neurotransmitters in Mental Disease*, E. Usdin, T. L. Sourkes, M. B. H. Youdim, Eds. (Wiley, Chichester, 1980), p. 445; L. F. Tseng, M. K. Menon, H. H. Loh, *J. Pharmacol. Exp. Ther.* **197**, 263 (1976).

23. D. K. Banerjee, M. B. H. Youdim, H. B. Pollard, abstract of paper presented at the 9th International Congress of Pharmacology, London (1984); M. B. H. Youdim, D. K. Banerjee, H. B. Pollard, E. Heldman, abstract of paper presented at the international symposium on Molecular Biology of Peripheral Catecholamine-Storing Tissues, Colmar, France (1984).
24. A. Pletscher, in *Essays in Neurochemistry and Neuropharmacology*, M. B. H. Youdim, W. Lovenberg, D. F. Sharman, J. Lagnado, Eds. (Wiley, Chichester, 1978), vol. 3, p. 49.
25. M. B. H. Youdim, H. F. Woods, B. Mitchell, D. G. Grahame-Smith, S. Callender, *Clin. Sci. Mol. Med.* **48**, 289 (1975).
26. N. A. Garrick and D. L. Murphy, *Biochem. Pharmacol.* **24**, 4061 (1982).
27. C. Goridis and N. H. Neff, *Neuropharmacology* **10**, 557 (1971).
28. R. Ashkenazi, J. P. M. Finberg, M. B. H. Youdim, *Br. J. Pharmacol.* **79**, 765 (1983); M. B. H. Youdim and J. P. M. Finberg, in *Handbook of Neurochemistry*, A. Lajath, Ed. (Plenum, New York, 1983), vol. 4, p. 293.
29. M. B. H. Youdim, *Br. J. Pharmacol.* **79**, 477 (1983).

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Reduction of a Larval Herring Population by Jellyfish Predator

Abstract. *The scyphomedusa* Aurelia aurita *consumes large amounts of yolk-sac herring larvae in Kiel Fjord. The decline of the larval herring population in late spring coincides with a major population growth of the jellyfish. The size of the larval herring population seems to be more significantly affected by the size of the predator stock than by the size of the parental herring stock.*

The mass occurrence of jellyfish is a nuisance in various coastal areas, causing clogged fishing nets and power plant intake systems and injury to bathers and fishermen. *Aurelia aurita* causes such problems in Japanese waters and in the Baltic Sea, *Cyanea capillata* in the Belt Sea, *Chrysaora plocamia* in Peruvian waters, and *Pelagia noctiluca* in the Mediterranean Sea (*1, 2*).

The mass occurrence of ctenophores and scyphomedusae has been found to reduce local stocks of copepods (*3–6*). These organisms therefore are severe competitors for fish larvae and planktivorous adult species. Five species of ctenophores and 34 species of medusae have been identified that feed on fish larvae in the field or under laboratory conditions (*7*).

In Kiel Fjord, young *Aurelia aurita* prey heavily on herring larvae but nearly exclusively on herring yolk-sac stages (5 to 7 mm long). During three periods of investigation in May 1979 and May 1980, the stomach contents of 5800 young medusae (6 to 50 mm in bell diameter) were examined. On the average, 0.4, 0.2, and 4.4 larvae were found per medusa. The number of larvae per *Aurelia* increased with increasing medusa size: the smallest *Aurelia* that had caught a herring larva measured 6 mm in bell diameter; as many as ten larvae were found in a medusa 12 mm in bell diameter; and up to 68 larvae were discovered in a medusa 42 mm in bell diameter (*2*).

Kiel Fjord is one of the spawning grounds for herring (*Clupea harengus*) in the Western Baltic Sea. Large shoals invade the fjord in March and April and deposit their eggs on gravel beds, mussel banks, and algae along the western shore of the fjord. The first hatch of yolk-sac larvae (5 mm in length) can be observed between mid-April and mid-May, depending on water temperature. Upon hatching, herring larvae are exposed to swarms of jellyfish because Kiel Fjord is also a major area of *Aurelia aurita* production. Most of the ephyra-stage jellyfish are released from the polyps in April, and an increase in their biomass takes place in May and June (*8*).

A 4-year survey was carried out at 26 stations in the fjord to determine the impact of jellyfish predation on the development of the local larval herring population. Double vertical hauls were taken weekly with a CalCOFi plankton net 1 m in diameter and 0.5 mm in mesh size. Herring larvae were counted, and the abundance of jellyfish was expressed as volume (milliliters) per 100 m^3. The recorded fluctuations in both populations included only those surveys in which more than one herring larva per 100 m^3 was caught (Fig. 1).

The impact of jellyfish predation should be demonstrable in three contexts. Relatively low numbers of herring larvae should be found (i) at stations with a relatively large *Aurelia* biomass, (ii) during days when the *Aurelia* biomass averaged from 26 stations was relatively large, and (iii) during years when the number of jellyfish averaged from all surveys was relatively large. Negative rank correlations were calculated on comparing the abundances of both species at stations where single surveys were conducted. Confidence levels

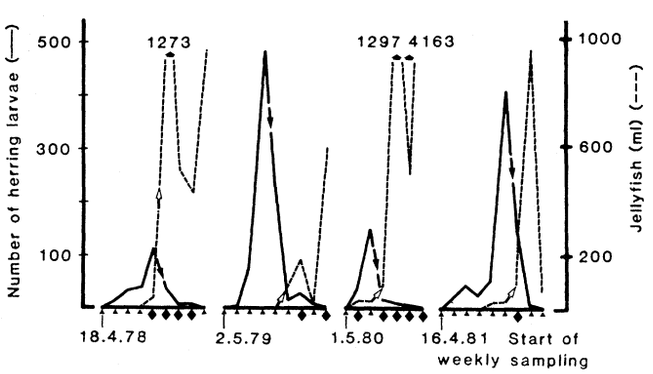
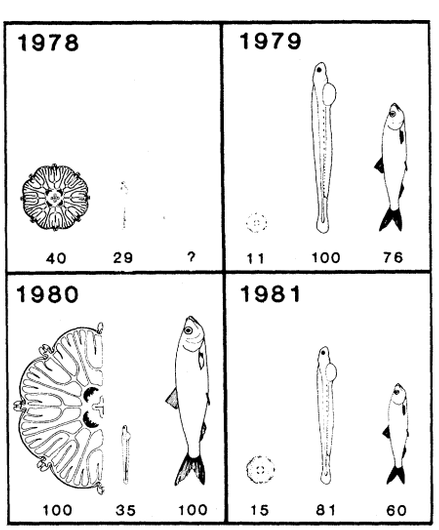


Fig. 1 (left). Average abundance of yolk-sac herring larvae (number per 100 m^3) and of *Aurelia aurita* (milliliters per 100 m^3) from 26 stations in Kiel Fjord sampled at weekly intervals (▲). The start of a major population growth of the jellyfish (↑) coincides with the rapid decline (↓) of the larval herring population. Negative correlations between the sizes of both populations were found during 12 surveys (◆). Fig. 2 (right). Drawings symbolize the relative abundances (100 = value in the year of the maximum abundance) of *Aurelia aurita*, yolk-sac herring larvae, and adult spawning herring during 4 years in Kiel Fjord. *Aurelia* and herring larvae: average abundance during eight weekly surveys after the first emergence of herring larvae; adult herring: catches of spawners from Kiel Fjord in April of the year indicated.



above 90 percent were found on 12 occasions when, on averaging a station series, more than six herring larvae and more than 90 ml of *Aurelia* were caught (Fig. 1). This can be interpreted as an effect of predation since the distribution of jellyfish in the fjord is independent of the localization of the herring spawning grounds (*1*).

During the 4 years of investigation, the first emergence of herring larvae in Kiel Fjord each year occurred between 20 April and 6 May (±4 days). An increase in the *Aurelia* biomass was observed about 3 weeks later, except in 1979. In 1978, 1980, and 1981 a decline of 50 percent or more in the larval yolk-sac herring population took place during those weeks when the biomass of *Aurelia* exceeded 75 ml per 100 m^3 for the first time. This decline was observed during 4 weeks in 1978, during 2 weeks in 1980, and during 5 weeks in 1981 after the first emergence of the larvae. This supports the hypothesis that the yolk-sac period of the larvae present in the plankton might be influenced by the growth of the jellyfish population.

The greatest number of herring larvae was caught in 1979, when the biomass of jellyfish was the smallest of the 4 years of the investigation (Fig. 2). Only about one-third of this larval quantity was caught in 1978 and 1980, the same 2 years during which the greatest biomass of *Aurelia* was present. The negative relation between the sizes of the larval herring and jellyfish populations seems to be strong, whereas larval abundance and abundance of the spawning parents (symbolized by an adult herring in Fig. 2) for the years indicated do not seem correlated.

Quantification and explanation of the high natural mortality among early larval stages of marine fish is one of the key problems in fisheries biology. Starvation after metamorphosis from the yolk-sac stage to an active feeder has been considered as the main contributive factor to this mortality. However, evidence has accumulated that shows that the effects of cannibalism (*9*) and predation by other planktivorous fish species (*10*) and invertebrates (*7*) might have been underestimated. The results of this study indicate that, at least in cases where fish spawn in small specific areas and where they are unable to avoid jellyfish blooms, predation might be a major regulating factor of the size of the surviving larval population.

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References and Notes

1. H. Möller, *Meeresforschung* **28**, 61 (1980).
2. Z. Maretić, paper presented at the Workshop on Jellyfish Blooms in the Mediterranean (United Nations Environment Programme, Athens, Greece, November 1983).
3. V. G. Burrell and W. A. von Engel, *Estuarine Coastal Mar. Sci.* **4**, 235 (1976).
4. E. E. Hulsizer, *Chesapeake Sci.* **17**, 260 (1976).
5. V. Kamshilov, cited by J. H. Fraser, *Rapp. P. V. Réun. Cons. Int. Explor. Mer* **153**, 121 (1962).
6. H. Möller, *Meeresforschung* **17**, 1 (1978).
7. J. R. Hunter, *Flødevigen Rapportser.*, in press.
8. H. Möller, *Mar. Biol.* **60**, 123 (1980).
9. J. R. Hunter and C. A. Kimbrell, *Fish. Res. Bull.* **78**, 811 (1980).
10. V. Øiestad, paper No. L:39 presented at the Council Meeting of the International Council for the Exploration of the Sea, Göteborg, Sweden, October 1983.

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A Potential Second Messenger Role for Unsaturated Fatty Acids: Activation of Ca^{2+}-Dependent Protein Kinase

Abstract. *Arachidonate and other unsaturated long-chain fatty acids were found to activate protein kinase C from human neutrophils. Kinase activation by arachidonate required calcium and was enhanced by diolein but did not require exogenous phosphatidylserine. Submaximal levels of arachidonate also enhanced the affinity of the kinase for calcium during activation by phosphatidylserine. Thus the release of arachidonate, which is triggered in many cell types by ligand-receptor interactions, could play a second messenger role in the regulation of cellular function by activation of protein kinase C.*

A calcium- and phospholipid-dependent protein kinase (protein kinase C) may have an important role in the regulation of cellular function (*1*). Activation of protein kinase C in cells can be accomplished by an increase in intracellular calcium concentration or the release of diacylglycerol by stimulus-induced phosphatidylinositol turnover or both (*2*). The tumor promotor phorbol myristate acetate (PMA) also activates protein kinase C from rodent brain extracts (*3, 4*). PMA is a potent inducer of the respiratory burst in human neutrophils and other phagocytes (*5*), a response that is critical for the microbicidal, tumoricidal, and inflammatory functions of these cells. Physiological stimuli such as chemoattractants and opsonized particles also trigger the respiratory burst in phagocytes (*6*), as well as the remodeling of membrane phospholipids (*7*) and the release of arachidonate in leukocytes (*8*). Interestingly, arachidonate and other unsaturated fatty acids stimulate the respiratory burst in phagocytic cells (*9*). Moreover, arachidonate has been reported to directly activate the respiratory burst enzyme in disrupted cell preparations from macrophages (*10*). Taken together, these findings suggest that arachidonate or a metabolite could serve a second messenger function. We investigated the possibility that arachidonate could also affect the activity of protein kinase C and now report that unsaturated fatty acids, including arachidonate, directly activate human neutrophil protein kinase C in a dose- and calcium-dependent fashion.

Protein kinase C activity was measured in detergent extracts of isolated human neutrophils (*11*). Cells at a concentration of 5×10^7 per milliliter in 0.1 percent Triton X-100 extraction buffer (*12*) were sonicated (*11*) and stirred for 1 hour at 4°C, and insoluble debris was sedimented by centrifugation for 1 hour at 100,000*g*. Activity was assayed as the amount of incorporation of ^{32}P from [γ-^{32}P]ATP (New England Nuclear) into histone H1 (Sigma) for 10 minutes at 30°C (*1, 13*) and was expressed as picomoles of ^{32}P incorporated per minute per milligram of protein (*13*). Without addition of exogenous lipid, no calcium-dependent protein kinase activity was measurable (Fig. 1A), but the addition of arachidonate (100 μM) stimulated activity. At 2 mM added calcium, protein kinase C activity was 189 pmole of ^{32}P incorporated per minute per milligram of protein. When the arachidonate concentration was increased to 200 μM, higher protein kinase C activity was obtained (314 pmole of ^{32}P incorporated per minute per milligram of protein at 2 mM Ca^{2+}). Diolein alone did not activate neutrophil protein kinase C, similar to observations in other cell types (Fig. 1B) (*2*). However, 100 μM arachidonate plus diolein resulted in 1.6-fold more kinase activation than arachidonate alone (293 pmole of ^{32}P incorporated per minute per milligram of protein). This arachidonate concentration plus a normally optimal dose of phosphatidylserine also resulted in protein kinase C activity that was higher than that observed with either alone (Fig. 1C). Arachidonate had no further effect on kinase activation in the presence of optimal amounts of both phosphatidylserine and diolein, suggesting that arachidonate and phosphatidyl-