general consensus is that on the whole the present experiments seem to rule out the possibility of copper oxide superconductors being anyon superconductors.

1. Laughlin, R. B., *Phys. Rev. Lett.*, 1988, **60**, 2677; *Science*, 1988, **242**, 525; Fetter, A.L., Hanna, C. B. and Laughlin, R. B., *Phys. Rev.*, 1989, **B39**, 9679.
2. Bednorz, J. G. and Muller, K. A., *Z. Phys.*, 1989, **B64**, 189; for many details see special issue of *Physics Today*, June 1991, 22–82.
3. Leinaas, J. M. and Myrheim, J., *Nuovo Cimento*, 1977, **B37**, 1.
4. Khare, Avinash, *Curr. Sci.*, 1991, **61**, 826.
5. Paul, Samir K. and Khare, Avinash, *Phys. Lett.*, 1986, **B174**, 420; **B182**, 415(E); for many details see Khare, Avinash, *Fortschr. de Phys.*, 1990, **38**, 507.
6. Wu, Y.-S., *Phys. Rev. Lett.*, 1984, **53**, 111.
7. Khare, Avinash and McCabe, John, *Phys. Lett.*, 1991, **B269**, 330; Murthy, M. V. N., Law, J., Brack, M. and Bhaduri, R. K., *Phys. Rev. Lett.*, 1991, **67**, 1817; Spoore, M., Verbaarschot, J. J. M. and Zahed, I., *Phys. Rev. Lett.*, 1991, **67**, 1813; Khare, Avinash, McCabe, John and Ouvry, Stephane, *Phys. Rev.*, 1992, **D46**, (in press).
8. Laughlin, R. B., *Phys. Rev. Lett.*, 1983, **50**, 1395; also see Haldane, F. D. M., *Phys. Rev. Lett.*, 1983, **51**, 605; for many details see Prange, R. E. and Girvin, S. M. eds., *The Quantum Hall Effect*, Springer, Berlin, 1987.
9. Bardeen, J., Cooper, L. N. and Schriefer, J. R., *Phys. Rev.*, 1957, **108**, 1175; for many details see de Gennes, P. G., *Superconductivity of Metals and Alloys*, Benjamin, 1966.
10. Wen, X. G., Wilczek, F. and Zee, A., *Phys. Rev.*, 1989, **B39**, 11413; Lee, D.-H. and Fisher, M. P. A., *Phys. Rev. Lett.*, 1989, **63**, 903; Canright, G. S., Girvin, S. M. and Brass, A., *Phys. Rev. Lett.*, 1989, **63**, 2291, 2295; Wen, X. G. and Zee, A., *Phys. Rev. Lett.*, 1989, **62**, 2873; Chen, Y.-H., *et al.*, *Int. J. Mod. Phys.*, 1989, **3**, 1001; Jain, J. K. and Read, N., *Phys. Rev.*, 1989, **B40**, 2723; for a good review and many other references in this field see Wilczek, F., *Fractional Statistics and Anyon Superconductivity*, World Scientific, Singapore, 1991.
11. Frohlich, J. and Marchenti, P., *Comm. Math. Phys.*, 1989, **121**, 177.
12. Jatkar, Dileep P. and Khare, Avinash, *Phys. Lett.*, 1990, **B236**, 283.
13. Lyons, K. B. *et al.*, *Phys. Rev. Lett.*, 1990, **64**, 2949.
14. Spielman, S. *et al.*, *Phys. Rev. Lett.*, 1990, **65**, 123.
15. Weber, H. J. *et al.*, *Solid State Comm.*, 1990, **76**, 511.
16. Kieff, R. F. *et al.*, *Phys. Rev. Lett.*, 1990, **64**, 2082.

ACKNOWLEDGEMENT. It is a pleasure to thank Surjyo Behera for a careful reading of the manuscript.

Endocrine control of fish reproduction

Samir Bhattacharya

Department of Zoology, School of Life Science, Visva-Bharati University, Santiniketan 731 235, India

The majority of fishes breed at a particular time of the year and the seasonal reproductive cycle is precisely maintained by endocrine cycle. Environmental stimuli like photoperiod and temperature are presumably received by the brain which releases a decapeptide hormone, gonadotropin releasing hormone (GnRH). GnRH specifically binds to the receptor in the pituitary gonadotroph cells and stimulates the secretion of gonadotropic hormone (GtH). In fish GtH may be of one or two types. Circulatory level of GtH increases during gonadal development and maturation. GtH surge is highest during the breeding season when ovulation or spermiation occurs. GtH regulates ovarian and testicular function by inducing an exceptional steroid hormone which is 17α,20β-dihydroxy-4-pregnen-3-one. However, there appears to be a shift in GtH function; it induces synthesis and secretion of estradiol-17β during previtellogenic phase which in turn induces vitellogenesis or yolk protein synthesis, while during post-vitellogenic phase GtH triggers the synthesis of 17α,20β-dihydroxy-4-pregnen-3-one which is responsible for final maturation leading to ovulation or spermiation. The hormonal cascade of events is perfectly coordinated with the seasonal reproductive cycle of the fish to ensure spawning at a specific time of the year.

THE majority of fishes are seasonal breeders. The seasonal reproductive cycle involves recrudescence of the gonad leading to its final maturation, ovulation or spermiation and spawning. Endocrine activity clearly corresponds to the annual reproductive cycle. In order to spawn at a specific time of the year, fishes must use various environmental cues to initiate gonadal recrudescence so that gametes are matured in time for spawning[1]. Among the various possible environmental factors involved in cuing endocrine activity to perform the reproductive event, duration of daily photophase (photoperiod) and temperature have been assumed to be of prime importance in most fishes[2,3]. It appears that external environmental stimuli are received by exteroreceptors which transfer this message to the brain of a fish. The brain releases a humoral factor, gonadotropin releasing hormone (GnRH). GnRH then acts on the pituitary to release gonadotropin which in turn regulates gonadal function. In most cases gonadotropin action is not direct. It acts through the biosynthesis of gonadal steroid hormones which in turn regulate gonadal growth, maturation and ovulation or spermiation.

Nowadays fish flesh is in high demand in various countries not only for its good taste but also for its better nutritional value. Every country is attempting higher production of food fish and to do this,

knowledge about the mechanism involved in the regulation of fish reproduction is essential. Unfortunately, very little is yet known compared to our information on mammalian reproduction. In this review, the current status of the hormonal control of fish reproduction is discussed.

Brain–pituitary axis

The pituitary in fishes is controlled by a certain network of the central nervous system which ensures a link between external factors and endocrine mechanisms. Immunocytochemical studies showed the existence of two groups of neurones in fish containing GnRH, one in the medial basal hypothalamus and the second group in the medial preoptic nuclei. GnRH cell bodies are also observed near the olfactory bulbs, telencephalon and in mid-brain of fish. In goldfish GnRH nerve fibres are present throughout the brain[4,5]. GnRH is released as a neurosecretion from nerve terminals and reaches the pituitary by circulation.

Till date the structure of GnRH has been determined for eleven species of vertebrates using amino acid analysis, and amino acid or DNA sequencing[6]. Among them four are fish forms. Like the mammalian GnRH it is a chain of ten amino acid residues but it varies in amino acid sequence. Since salmon GnRH[7] is of wide use, its chemical structure is given below.

1 2 3 4 5 6 7 8 9 10

pGlu–His–Trp–Ser–Tyr–Gly–Trp–Leu–Pro–Gly–NH_2

In spite of its structural variation from the rest, salmon GnRH induces an increase in GtH level in almost all fishes tested so far and also in all classes of vertebrates. This indicates the likely presence of similar GnRH receptors in different classes of vertebrates[8]. GnRH has also been purified from the brain of other fishes and found to be active in heterologous species[9–12]. Soon after GnRHs were identified in different fishes, synthetic molecules were prepared and tested. Some GnRH analogues were found to be superactive in fish compared to native GnRH[13–16]. However, there are reports also indicating no difference in biopotency between native GnRH and GnRH analogues[17,18]. It has been shown that to release GtH from the pituitary cells of Indian murrel and carp GnRH requires extracellular calcium, incorporation of which into the pituitary cells is an eventual step for GtH release by GnRH[19,20].

It has been reported that in goldfish, GnRH is carried in the circulation by means of a serum binding protein specific for GnRH (GnRH-BP). It is a minor component of serum proteins in goldfish, a single nonglycoprotein of 40 kDa[21]. GnRH-BP possibly transports GnRH to the target site, i.e. pituitary, and difference in the binding affinity between GnRH-BP and gonadotroph cells possibly results in dissociation of GnRH from the binding protein. Occupation of GnRH receptor in the gonadotroph cells thus occurs and GtH is released. A single class of GnRH receptor has been described in the pituitary of catfish[22] and winter flounder[23]. On the other hand, goldfish pituitary contains two classes of receptors, namely high affinity and low affinity sites[24]. It has been shown that binding to high affinity sites by salmon GnRH results in GtH release[25]. It has been reported that mammalian, salmon and chicken GnRH are equally effective in stimulating GtH release in goldfish[16,18] and this indicates that the biologically active site of the GnRH decapeptide is highly conserved.

Pituitary gonadotropin

As in other vertebrates, GtH in fishes is the major hormone regulating gonadal function. But fish pituitary GtH is indeed very different compared to other vertebrates. There is no FSH and LH and pituitary GtH may be of one or two types which is still a controversial matter. Two types of pituitary GtHs were isolated and purified from the pituitary of salmon[26,27], American plaice[28], African catfish[29] and common carp[30] while single pituitary GtH has been purified from the pituitary of sturgeon[31], rainbow trout[32] and silver carp[33]. Although a large number of different species of fishes are available in India, complete purification of GtH has been achieved only with two species, an Indian major carp and a murrel[34]. It has been shown that in contrast to two types of GtH molecules, Indian murrel and carp contain a single GtH with two distinct subunits[34]. Irrespective of its chemical structure, the function of the pituitary GtH in fishes is to control oocyte growth including vitellogenesis and maturation, spermatogenesis and spermiation. GtH induces steroid hormone synthesis in the gonad which in turn effects maturation of ovary and testis.

The primary event in steroidogenic action of GtH in fishes is believed to depend on its specific binding to its receptor located in the plasma membrane of ovarian follicular cells[35–39]. There is, however, only one report on Indian fishes where a high affinity GtH receptor has been identified in the oocyte plasma membrane of Indian murrel[40]. It has also been shown that binding of GtH to fish ovarian receptor increases steroid hormone synthesis[39,40].

Gonadal steroidogenesis

In fishes GtH level in circulation begins to rise at the initial stage of annual reproductive cycle and GtH surge triggers a cascade of biochemical events which ultimately leads to final gonadal maturation. GtH

induces two biologically important ovarian steroid hormones in fish—estradiol-17β and 17α,20β-dihydroxy-4-pregnen-3-one and testicular steroid hormones, testosterone and 17α,20β-dihydroxy-4-pregnen-3-one (17α,20β-diOHprog). Testosterone in male and estradiol-17β in female are well-known biologically active steroids in other vertebrates also but 17α, 20β-diOHprog is indeed a very special kind of sex steroid in fish which induces spermiation[41] and ovulation[42]. The ovarian follicular layer of fishes, as in other vertebrates, has two major layers—outer thecal layer containing fibroblasts, capillaries, collagen fibres and thecal cells and the inner granulosa layer composed of a single population of granulosa cells. In fishes, ovarian cycle can be broadly divided into previtellogenic, vitellogenic and final maturational stages ending in ovulation. During previtellogenic phase GtH increases estradiol-17β level in the plasma, which remains in peak during vitellogenesis and declines rapidly prior to oocyte maturation[43,44]. Estradiol-17β induces the synthesis of a yolk precursor protein, vitellogenin, in the liver which is transported via circulation to the ovary where it is taken up by the developing ovarian follicles or oocytes through micropinocytosis[45–47]. Nagahama and his associate proposed a two-cell-type model in the production of follicular estradiol-17β in Amago salmon[44,48–50]. According to them, GtH during vitellogenic phase influences the thecal layer to secrete aromatizable androgen, *mainly* testosterone, which then passes to granulosa layer and is converted to estradiol-17β. They found that the thecal layer from the Amago salmon and granulosa layer from rainbow trout could also produce the same effect which implies that there may be little species specificity of these layers among the salmonid group of *fish*[50].

When the vitellogenesis phase ends and the oocytes have grown, estradiol-17β level suddenly declines and 17α,20β-diOHprog production by the oocytes increases in response to GtH[51,52]. Increase of this steroid occurs just prior to the natural maturation period. When postvitellogenic follicular theca and granulosa layers were cultured *in vitro*, addition of GtH did not produce estradiol-17β but only 17α,20β-diOHprog[53]. Here also a two-cell-type model has been proposed. Thecal layer produces large quantities of 17α-hydroxyprogesterone in response to GtH. 17α-Hydroxyprogesterone then enters granulosa cells where GtH activates 20β-hydroxysteroid dehydrogenase, the key enzyme involved in the conversion of 17α-hydroxyprogesterone to 17α,20β-diOHprog[50]. The two-cell-type model of vitellogenic and postvitellogenic oocyte is shown in Figure 1.

In the case of male fishes, GtH increases the circulatory level of testosterone, 11-ketotestosterone with glucuronide ester and also 17α,20β-diOHprog[54–58]. However, 17α,20β-diOHprog appears to be the most significant steroid involved in spermiation in salmonids.

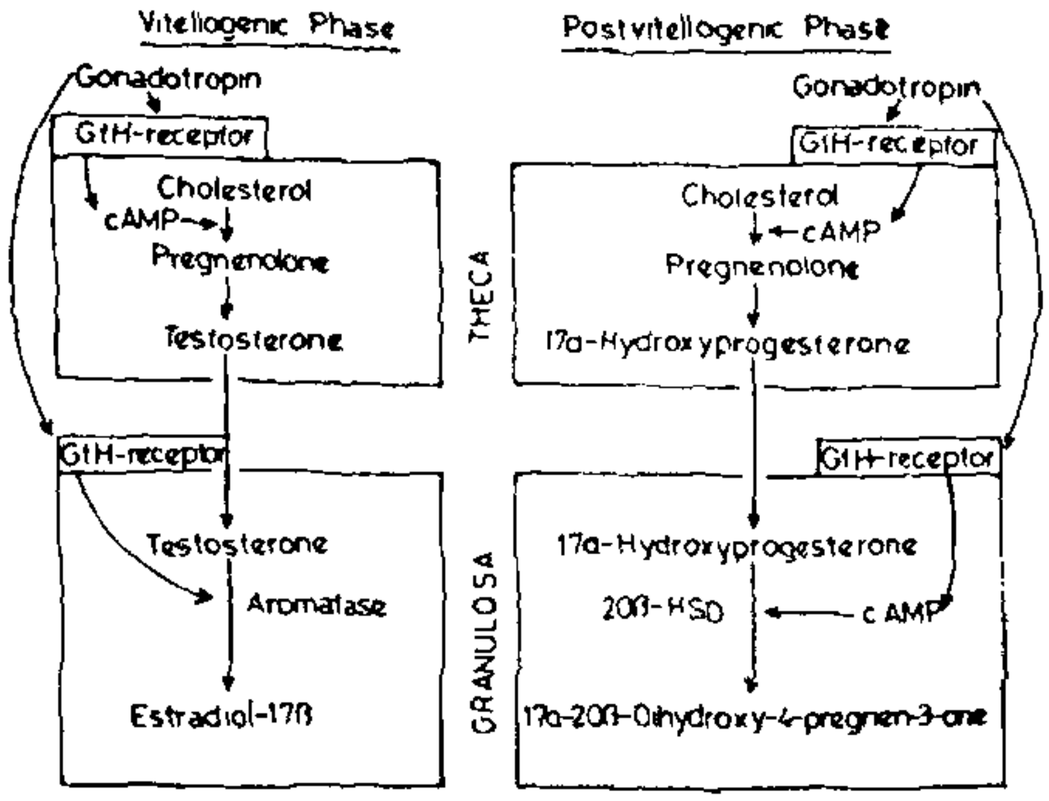

Figure 1. *Two-cell-type model for the production of* estradiol-17β during vitellogenic phase and 17α,20β-diOHprog during postvitellogenic phase in the ovarian follicle of salmonid fishes (according to Y. Nagahama, 1987, ref. 50). In the oocytes of vitellogenic phase thecal layer secretes testosterone which then passes to granulosa layer where it is converted to estradiol-17β under the influence of GtH. In contrast to this, GtH binds to the theca cells of the layer of postvitellogenic oocytes to produce 17α-hydroxyprogesterone which traverses to the granulosa layer and is converted to 17α,20β-diOHprog where GtH acts to increase the activity of 20β-HSD.

It has been found that GtH surge during spermiation is accompanied by dramatic increase in circulatory level of 17α,20β-diOHprog[41]. A similar observation was also made in the case of goldfish[59].

Despite the accumulation of considerable information regarding the influence of GtH on gonadal steroidogenesis certain central questions concerning its mechanism of action on gonadal steroidogenesis still remain unanswered. In fishes, as well as in mammals, GtH increases the level of cAMP in gonadal cells by stimulating adenylate cyclase. Search for the mediator of GtH action in mammalian system leads to the interesting observation that GtH stimulates ovarian follicular mitochondria to synthesize pregnenolone and progesterone from cholesterol precursor. It has also been shown that this increased ability of mitochondria is due to the increased synthesis of specific mitochondrial proteins[60,61]. Unlike the mechanism suggested in mammals, GtH in fishes, induces the generation of a proteinaceous factor. Its addition along with cAMP greatly stimulates mitochondrial steroidogenesis[62]. However, our knowledge is rather limited regarding the precise mechanism of action of GtH in inducing gonadal steroidogenesis. A schematic outline of the principal events involved in hormonal regulation of fish reproduction is presented in Figure 2.

Gametogenesis

Under the influence of pituitary GtH ovarian follicles in

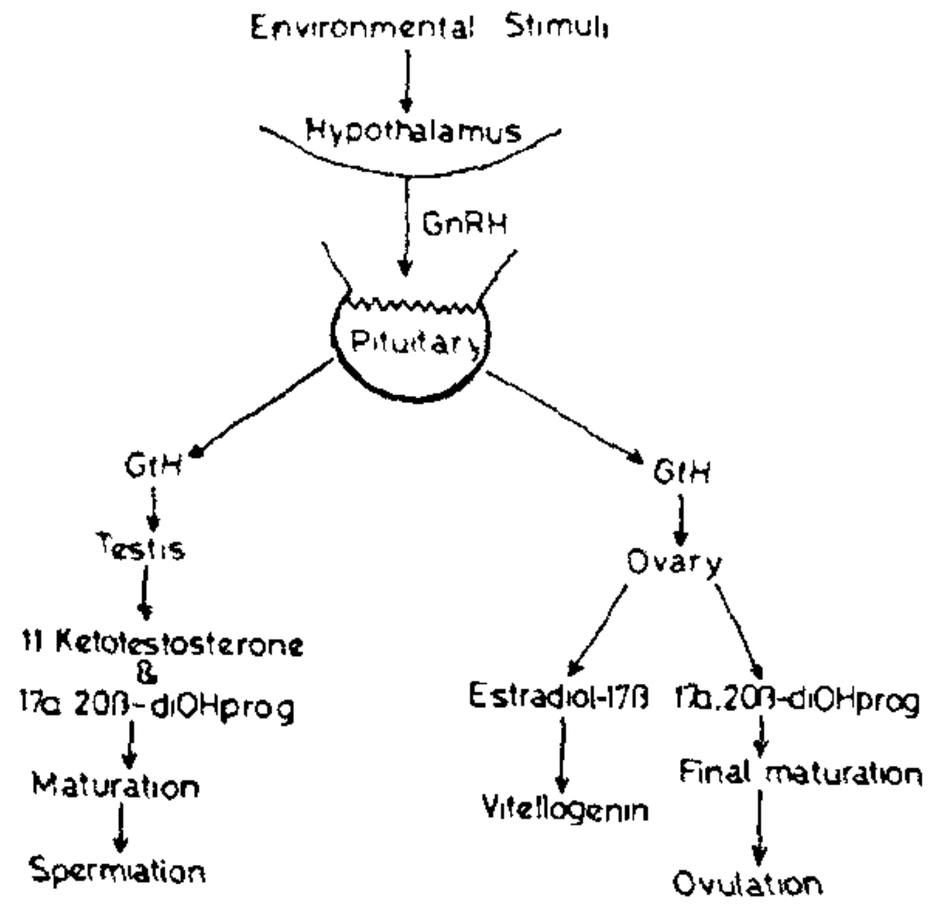

Figure 2. Hormonal control of reproduction in fishes. Environmental stimuli like photoperiod, temperature, etc. are received by exteroreceptor and reach the brain (hypothalamus). Environmental cues result in secretion of GnRH from the brain which in turn causes the release of pituitary GtH. GtH acts on the testis or ovary and stimulates the production of steroid hormones. An exceptional steroid hormone, 17α,20β-diOHprog, released from the testis and ovary induces the final maturation of germ cells leading to spermiation and ovulation (spawning).

fishes at the initial stage of annual reproductive cycle synthesize and release an estrogen, estradiol-17β, which reaches the liver by circulation and stimulates the hepatic synthesis and secretion of vitellogenin, a yolk precursor protein. Vitellogenin is released into the circulation and is selectively taken up by the developing oocytes. This is the major factor responsible for oocyte growth. Vitellogenin has been isolated and characterized in a variety of fish and is a high molecular weight lipophosphoprotein which is sequestered after its uptake in the oocyte[46,47,63–66]. In rainbow trout estradiol-17β has been shown to express the vitellogenin gene[67]. When the vitellogenesis is completed oocytes are termed postvitellogenic but they are still physiologically immature as they cannot be fertilized. To make them suitable for fertilization, oocytes have to undergo final maturation process which consists of the breakdown of germinal vesicle (GVBD), chromosome condensation and extrusion of the first polar body. For this, in fishes, three factors have so far been found to be responsible: GtH, maturation inducing hormone (MIH) which is 17α,20β-diOHprog and the maturation promoting factor (MIF)[50,51,68]. MIF is generated by the binding of MIH to the ovarian follicle and surprisingly MIH being a steroid binds to the plasma membrane and not to the nuclear receptor which is indeed an exception compared to other vertebrates including mammals[69]. Sex steroid in mammals usually binds to the nuclear or cytosol receptor. Very little work, however, has been done with male fishes. The association between high circulatory level of 17α,20β-diOHprog and spermiation has been reported in the salmonid fishes[70–72]. These reports indicate that 17α,20β-diOHprog is involved in the final maturation of spermatozoa and spermiation in salmonids. GtH has been found to stimulate 17α,20β-diOHprog production by intact testicular fragments but not by sperm free preparations. This is due to the fact that the key enzyme, the 20β-HSD which converts 17α-hydroxyprogesterone to 17α,20β-diOHprog, is possibly localized in the sperm[50].

Concluding remarks

This overview about the hormonal control of fish reproduction shows that although the basic plan of hormonal regulation is similar to that occurring in other vertebrates some interesting and significant differences can be noted in the finer details. Brain GnRH which releases GtH from the pituitary is chemically different from mammalian GnRH and pituitary GtH may be of one or two types and not LH or FSH as in other vertebrates. In fishes, GtH binds to gonadal receptor to produce sex steroids which in turn are responsible for growth and maturation of the gonad. GtH induces the production of two very unusual sex steroids, 17α,20β-diOHprog in both testis and ovary and 11-ketotestosterone in testis. Again there is a remarkable shift of ovarian steroidogenesis influenced by the same GtH. During vitellogenic phase it is the estradiol-17β and during postvitellogenic phase it is 17α,20β-diOHprog in female fishes. It is still not clear how such a shift of steroidogenic pattern is regulated and how such a change in the receptor quality and quantity occur at a specific time of gonadal maturation.

1. Peter, R. E. and Hontela, A., in *Environmental Endocrinology* (eds. Assanmacher, I. and Farner, D. S), Springer, 1978, pp. 20–25.
2. Sundararaj, B. I. and Vasal, S., *J. Fish. Res. Bd. Can*, 1976, **33**, 959.
3. Vlaming, V. L. De, *Biol. Bull.*, 1975, **148**, 402.
4. Kyle, A. L., Stacey, N. E. and Peter, R. E., *Behav. Neural. Biol.*, 1982, **36**, 229.
5. Kah, O., Breton, B., Dulka, J. G., Numez-Rodriguez, J., Peter, R. E., Corrigan, A., Rivier, J. and Vale, W. W., *Cell Tissue Res.*, *1986*, **244**, 327.
6. Sherwood, N. M., Doroshov, S. and Lance, V., *Gen. Comp. Endocrinol*, 1991, **84**, 44.
7. Sherwood, N. M., Eiden, L., Brownstein, M., Spiess, J., Rivier, J. and Vale, W., *Proc. Natl. Acad. Sci. USA*, 1983, **80**, 2794.
8. Peter, R. E., *Am. Zool.*, 1983, **23**, 685.
9. Wu, P., Ackland, J. F, Ling, N. and Jackson, I. M. D., *Reg. Pept.*, 1986, **15**, 311.
10. Lovejoy, D. A., Sherwood, N. M., Fisher, W., Jackson, B. C., Rivier, J. E. and Lee, T., *Gen. Comp. Endocrinol.*, 1991, **82**, 152.
11. Halder, S., Sen, S., Bhattacharya, S., Ray, A. K., Ghosh, A. and Jhingran, A. G., *Aquaculture*, 1991, **97**, 373.

12. Sherwood, N. M., Sower, S. A. Marshak, D. R., Fraser, B. A. and Brownstein, M. J., *J. Biol. Chem.*, 1986, **261**, 4812.
13. McLean, E., Parker, D. B. Warby, C. M., Sherwood, N. M. and Donaldson, E. M., *J. Fish Biol.*, 1991, **38**, 851.
14. Lin, H. R., Van Der Kraak, G., Zhou, X. J., Liang, J. Y., Peter, R. E., Rivier, J. and Vale, W. W., *Gen. Comp. Endocrinol*, 1988, **69**, 31.
15. Weil, C. and Crim, L. W., *Aquaculture*, 1983, **35**, 103.
16. Peter, R. E., Nahorniak, C. S., Sokolowska, M., Chang, J. P., River, J. E., Vale, W. W., King, J. A. and Millar, R. P., *Gen. Comp. Endocrinal.*, 1985, **58**, 231.
17. Crim, L. W., Evans, D. M., Coy, D. H. and Schally, A. V., *Life Sci.*, 1981, **28**, 129.
18. Mackenzie, D. S., Gould, D. R., Peter, R. E., Rivier, J. and Vale, W. W., *Life Sci.*, 1984, **35**, 2019.
19. Jamaluddin, M., Banerjee, P. P., Manna, P. R. and Bhattacharya, S., *Gen. Comp. Endocrinol.*, 1989, **74**, 190.
20. Bhattacharya, S., Manna, P. R., Haldar, S. and Jamaluddin, Md., in *Progress in Comparative Endocrinology* (eds. Epple, A., Scanes, C. G. and Stetson, M. H.), Wiley-Liss, New York, 1990, p. 572.
21. Huang, Y. P., Guo, D. C. and Peter, R. E., *Gen. Comp. endocrinol.*, 1991, **84**, 58.
22. De Leeuw, R., Conn, P. M., Van't Veer, C., Goos, H. J. Th. and Van Oordt, P. G. W. J., *Fish Physiol. Biochem.*, 1988, **5**, 99.
23. Crim, L. W., St Arnaud, R., Lavoie, M. and Labrie, F., *Gen. Comp. Endocrinol.*, 1988, **69**, 372.
24. Habibi, H. R., Peter, R. E., Sokolowska, M., Rivier, J. E. and Vale, W. W., *Biol. Reprod.*, 1987, **36**, 844.
25. Habibi, H. R., Marchant, T. A., Nahorniak, C. S., Van der Loo, H., Peter, R. E., Rivier, J. E. and Vale, W. W., *Biol. Reprod.*, 1989, **40**, 1152.
26. Swanson, P., Suzuki, K., Kawauchi, H. and Dickhoff, W. W., *Biol. Reprod.*, 1991, **44**, 29.
27. Kawauchi, H., Suzuki, K., Itoh, H., Swanson, P., Natio, N., Nagahama, Y., Nozaki, M., Nakai, Y. and Itoh, S., *Fish Physiol. Biochem.*, 1989, **7**, 29.
28. Ng, T. B. and Idler, D. R., *Gen. Comp. Endocrinol.*, 1979, **38**, 410.
29. Goos, H. J. Th., de Leeuw, R., Burzawa-Gerard, E., Terlou, M. and Richter, C. J. J., *Gen. Comp. Endocrinol.*, 1986, **63**, 162.
30. Van Der Kraak, G., Suzuki, K., Peter, R. E., Itoh, H. and Kawauchi, H., *Gen. Comp. Endocrinol.*, 1992, **85**, 217.
31. Burzawa-Gerard, E., Goncharov, B. F. and Fontaine, Y. A., *Gen. Comp. Endocrinol.*, 1975, **27**, 289.
32. Breton, B., Jalabert, B. and Reinaud, P., *Ann. Biol. Anim. Biochem. Biophys.*, 1976, **16**, 25.
33. Kobayashi, M., Aida, K., Hanyu, I. and Ishii, S., *Bull. Jpn. Soc. Sci. Fish.*, 1985, **51**, 405.
34. Banerjee, P. P., Bhattacharya, S. and Nath, P., *Gen. Comp. Endocrinol.*, 1989, **73**, 118.
35. Schlaghecke, R., *Gen. Comp. Endocrinol.*, 1983, **49**, 261.
36. Van der Kraak, G., in *Fish Physiology* (eds. Hoar, W. S., Randall, D. J. and Donaldson, E. M.), Academic Press, New York, 1983, vol. IX(A), p. 405.
37. Aida, K. and Ishii, S., in *Current Trends in Comparative Endocrinology* (eds. Lofts, B. and Holmes, W. N.), Hong Kong University Press, Hong Kong, 1985, p. 745.
38. Kanamori, A., Kagawa, H. and Nagahama, Y., *Gen. Comp. Endocrinol.*, 1987, **66**, 210.
39. Kanamori, A. and Nagahama, Y., *Gen. Comp. Endocrinol.*, 1988, **72**, 25.
40. Jamaluddin, M. and Bhattacharya, S., *J. Endocrinol.*, 1986, **111**, 407.
41. Ueda, H., Kambegawa, A. and Nagahama, Y., *Gen. Comp. Endocrinol.*, 1985, **59**, 24.
42. Goetz, F. W. in *Fish Physiology* (eds. Hoar, W. S., Randall, D. J. and Donaldson, E. M.), Academic Press, New York, 1983, vol. IXB, p. 117.
43. Fostier, A., Jalabert, B., Billard, R., Breton, B. and Zohar, Y., in *Fish Physiology* (eds. Hoar, W. S., Randall, D. J. and Donaldson, E. M.), Academic Press, New York, 1983, vol. IXA, p. 277.
44. Kagawa, H., Young, G. and Nagahama, Y., in *Salmonid Reproduction* (eds. Iwamoto, R. N. and Sower, S.), Washington Sea Grant Program, University of Washington, Seattle, 1985, p. 20.
45. Wallace, R. A. in *Developmental Biology* (ed. Browder, L. W.), Plenum Press, New York, 1985, vol. 1, p. 127.
46. Sundararaj, B. I. and Nath, P., *Gen. Comp. Endocrinol.*, 1981, **43**, 201.
47. Nath, P. and Sundararaj, B. I., *Gen. Comp. Endocrinol.*, 1981, **43**, 184.
48. Nagahama, Y., in *Fish Physiology* (eds. Hoar, W. S., Randall, D. J. and Donaldson, E. M.), Academic Press, New York, 1983, vol. IXA, p. 223.
49. Nagahama, Y., *Gunma Symp. Endocrinol.*, 1984, **21**, 167.
50. Nagahama, Y., *Zool. Sci.*, 1987, **4**, 209.
51. Nagahama, Y., Goetz, F. W. and Tan, J. D., *Dev. Growth Differ.*, 1986, **28**, 555.
52. Young, G., Ueda, H. and Nagahama, Y., *Gen. Comp. Endocrinol.*, 1983, **52**, 329.
53. Young, G., Adachi, S. and Nagahama, Y., *Dev. Biol.*, 1986, **118**, 1.
54. Scott, A. P., Bye, V. J., Baynes, S. M. and Springate, J. R. C., *J. Fish. Biol.*, 1980, **17**, 495.
55. Kime, D. E., *J. Steroid Biochem.*, 1978, **9**, 891.
56. Kime, D. E., *Gen. Comp. Endocrinol.*, 1979, **39**, 290.
57. Ueda, H., Young, G., Crim, L. W., Kambegawa, A. and Nagahama, Y., *Gen. Comp. Endocrinol.*, 1983, **51**, 106.
58. Ueda, H., Nagahama, Y. and Takahasi, H., *Gunma Symp. Endocrinol.*, 1984, **21**, 21.
59. Kobayashi, M., Aida, K. and Hanyu, I., *Bull. Jpn. Soc. Fish.*, 1986, **52**, 755.
60. Dimino, M. J. and Berman, S. K., *Endocrinology*, 1979, **104**, 780.
61. Rowe, M. J., Beiszczard, R. R., Neymark, M. S. and Dimino, M. J., *Endocrinology*, 1981, **108**, 127.
62. Deb, S. and Bhattacharya, S., *Indian J. Biochem. Biophys.*, 1988, **25**, 344.
63. Wallace, R. A., *Can. J. Biochem.*, 1985, **44**, 1647.
64. Selman, K. and Wallace, R. A., *J. Exp. Zool.*, 1983, **226**, 441.
65. Hamazaki, T. S., Iuchi, I. and Yamagami, K., *J. Exp. Zool.*, 1987, **242**, 333.
66. Bradley, T. J. and Grizzle, M. J., *Gen. Comp. Endocrinol.*, 1989, **73**, 28.
67. Vaillant, C., Guellec, C., Pakdel, F. and Valotoire, Y., *Gen. Comp. Endocrinol.*, 1988, **70**, 284.
68. Venkatesh, B., Tan, C. H. and Lam, T. J., *Gen. Comp. Endocrinol.*, 1992, **85**, 450.
69. Nagahama, Y. and Yamashita, M., *Fish Physiol. Biochem.*, 1989, **7**, 192.
70. Ueda, H., Kambegawa, H. and Nagahama, Y., *J. Exp. Zool.*, 1984, **231**, 435.
71. Fitzpatrick, M. S., Van Der Kraak, G. and Schreck, C. B., *Gen. Comp. Endocrinol.*, 1986, **62**, 437.
72. Truscott, B., Idler, D. R., So, Y. P. and Walsh, J. M., *Gen. Comp. Endocrinol.*, 1986, **62**, 99.