ENVIRONMENTAL AND ENDOCRINE CONTROL OF REPRODUCTION IN CULTURED SALMONIDS

by

Penny Swanson

Coastal Zone and Estuarine Studies Division
Northwest Fisheries Science Center
National Marine Fisheries Service
National Oceanic and Atomospheric Administration
2725 Montlake Boulevard East
Seattle, Washington 98 1 lo-2097

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Introduction

Reproduction in salmonid fish involves the growth, development, and release of mature gametes (eggs and sperm), as well as the development of appropriate secondary sex characteristics and spawning behavior. Cultured salmon are artificially spawned; therefore, spawning behavior is not required for successful reproduction of a captive broodstock. The major problems with reproduction of salmonids in captivity involve timely maturation of broodstock, losses due to prespawning mortality, reliable production of high quality gametes, and precocious maturation of male fish. There is an obvious need to develop methods to monitor and control sexual maturation in captive broodstock, to ensure production of high quality gametes and high survival of offspring, and to minimize asynchronous maturation of male and female fish. In this report, literature relevant to these problems will be reviewed as follows: 1) endocrine control of reproduction in salmonids; 2) environmental regulation of reproduction in salmonids; 3) hormonal induction of final oocyte maturation, ovulation, and spermiation salmonids; 4) precocious or early male maturation in salmonids; and 5) factors affecting gamete quality.

Endocrine Control of Reproduction in Salmonids

In temperate-zone fishes like salmonids, the seasonal timing of reproduction is strongly influenced by photoperiod, with temperature having a supplementary role. This environmental information is perceived and processed by the brain, which in mm regulates internal processes through the endocrine system. Major components of the reproductive endocrine system are the brain (hypothalamus), pituitary, and gonads (Fig. 1). The pituitary gland plays a central role in initiating reproductive maturation (puberty), maintaining production of sperm and eggs by the gonads, and inducing final maturation and gamete release (spawning).

In fish gonadotropins are the major pituitary hormones responsible for regulating production of gametes (gametogenesis). Other pituitary hormones such as prolactin, growth hormone, and somatolactin have been shown to stimulate gonadal steroidogenesis in fish or to potentiate the response to gonadotropins, but their potencies are far less than gonadotropins and their specific roles in regulation of gametogenesis are not fully understood. Synthesized by gonadotropes of the pituitary, gonadotropins are secreted into the peripheral circulation and bind to receptors in the gonad with subsequent effects on gametogenesis. In most cases, gonadotropins act through the biosynthesis of steroids, which in turn mediate various stages of gametogenesis: oocyte growth, oocyte maturation, spermatogenesis, and spermiation. The ability of the gonadotropins to modulate gametogenesis depends not only on circulating levels of gonadotropins, but also on expression of the appropriate receptor proteins by potential target cells in the gonad. In this section, the biochemistry and physiology of gonadotropins and how they regulate reproduction in salmon will be briefly reviewed.

Biochemistry of Gonadotropins

Pituitary gonadotropins, follicle stimulating hormone (FSH), and luteinizing hormone (LH), along with placenta-derived chorionic gonadotropin (CG) and a third pituitary hormone, thyroid stimulating hormone (TSH) constitute a family of chemically related hormones (Pierce and Parsons 1981). Each member of this family is a heterodimeric glycoprotein, consisting of the noncovalent association of a common a subunit and a unique β subunit, which confers biological specificity to the hormone. For full expression of biological activity, carbohydrate moieties and association between subunits are necessary (Ryan et al. 1987).

Historically there has been controversy over whether the fish pituitary gland produces one or two types of gonadotropins (Burzawa-Gerard 1982, Idler and Ng 1983, Van Oordt and Peute 1983, Fontaine and Dufour 1987).

Initially it was thought that all phases of gametogenesis in fish were regulated by a single "LH-type" gonadotropin, designated "maturational" gonadotropin (Burzawa-Gerard 1982). Later, two types of gonadotropins were isolated by Idler and colleagues, and were designated carbohydrate rich "maturational" gonadotropin, and carbohydrate poor "vitellogenic" gonadotropin, which does not belong to the LH/FSH hormone family (Idler and Ng 1983). Unfortunately, the biochemical nature of the vitellogenic gonadotropin identified by Idler and colleagues has not been fully characterized, and the original physiological work done with this protein has not been verified in other laboratories. Thus, the controversy of one versus two types of gonadotropins in fish persisted until the late 1980s.

The debate was resolved primarily through the efforts of Kawauchi and colleagues (Suzuki et al. 1988a, b; Itoh et al. 1988, 1990; Sekine et al. 1989; Kawauchi et al. 1989; Swanson et al. 1991) who initially characterized two types of gonadotropins, GTH I and GTH II, in chum salmon (*Oncorhynchus keta*) and coho salmon (0. kisutch). Like tetrapod LH and FSH, both GTH I and GTH II consist of an α and β subunit. The β subunits of salmon GTH I and GTH II have only 31% ammo acid sequence identity to each other, and 30-40% sequence identity to mammalian LH and FSH β subunits (Itoh et al. 1988). In salmon, unlike tetrapods, two types of a subunits, a-l and a-2, have been identified (Itoh et al. 1990). Both of the salmon a subunits are highly conserved, showing approximately 65% sequence identity to mammalian a subunits. The salmon GTH I β subunit associates with either of two a subunits, a-l or a-2. On the other hand, the β subunit of GTH II associates only with the a-2 subunit. GTH II is identical to the previously described chinook salmon (0. *tshawytscha*) "maturational" gonadotropin and is most chemically similar to mammalian LH. Neither GTH I nor GTH II show any biochemical similarity to the vitellogenic gonadotropin identified by Idler and colleagues. Since the identification of the two types of gonadotropins in salmon, considerable progress has been made in understanding their physiological roles.

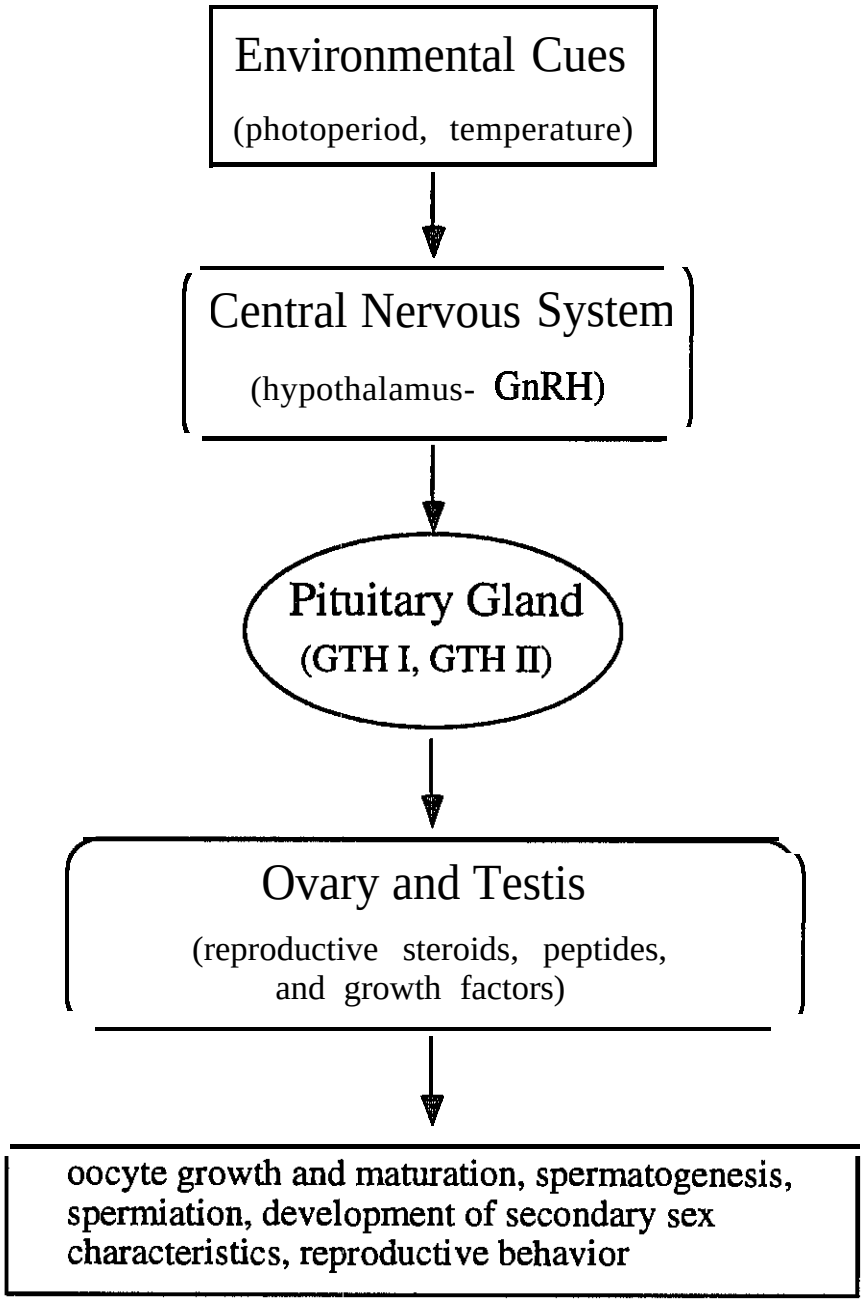

Figure 1. Reproductive endocrine axis in salmonids. Environmental cues are perceived and processed by the central nervous system, which regulates production of hypothalamic peptides such as gonadotropin-releasing hormone (GnRH). GnRH acts on the pituitary gland to regulate synthesis and secretion of gonadotropins (GTH I and GTH II). The gonadotropins are secreted into the peripheral circulation and regulate gametogenesis primarily through effects on the production of reproductive steroids, which also affect the development of secondary sex characteristics and reproductive behavior.

Plasma and Pituitary Levels of Gonadotropins

Plasma levels of GTH II show a similar pattern during the reproductive cycle in a variety of salmonids: levels remain relatively low or nondetectable until the period of final oocyte maturation and ovulation or spermiation, when levels increase in a pattern similar to that of the preovulatory LH surge in tetrapods (Fitzpatrick et al. 1986; Sumpter and Scott 1989; Suzuki et al. 1988c; Swanson 1991; Amano et al. 1992, 1993, 1994; Oppen-Bemsten et al. 1994; Slater et al. 1994). In contrast, GTH I levels increase and remain elevated during the period of vitellogenesis or spermatogenesis and then decline during final maturation (Suzuki et al. 1988c, Swanson et al. 1989, Swanson 1991, Oppen-Bemtsen et al. 1994, Slater et al. 1994). In coho salmon, plasma GTH I levels increase only in maturing fish 6 to 9 months prior to spawning. The lack of GTH II in the peripheral circulation during periods of gonadal growth suggests that GTH II does not play a role in regulating this phase of reproductive development (Pig. 2).

Immunocytochemical studies in salmonids indicate that GTH I and GTH II are produced in distinctly different cell-types (Nozaki et al. 1990a, Naito et al. 1993) and are differentially synthesized during gonadal development (Nozaki et al. 1990b, Naito et al. 1991, Saga et al. 1993). Immunoreactive (ir) GTH I β subunit is first detected about 56 days post-fertilization in the pituitary of the developing embryo (Mal et al. 1988, Mal 1991, Saga et al. 1993), and levels increase during vitellogenesis and spermatogenesis (Nozaki et al. 1990b, Naito et al. 1991). On the other hand, the ir-GTH II β subunit is not detected until late stages of vitellogenesis or spermatogenesis, with the highest pituitary levels at spawning development (Nozaki et al. 1990b, Naito et al. 1991). The a subunits are detected throughout gametogenesis. However, levels of messenger RNA for the a subunit decline in GTH I-producing cells at the time of spawning (Naito et al. 1991).

Regulation of gonadotropin secretion

Secretion of GTH II in fish is regulated by hypothalamic peptides such as gonadotropin-releasing hormone (GnRH) and neuropeptide Y (NPY) as well as by other neuromodulators such as dopamine (DA), serotonin (5-HT), norepinephrine (NE) and gamma-amino butyric acid (GABA) (Peter 1983, Peter et al. 1991). Like LH in other vertebrates, secretion of GTH II in fish is primarily regulated by GnRH (Peter 1983). The endocrine mechanisms involved in regulating GTH I secretion have not been extensively studied; however, GnRH has been shown to stimulate in *vitro* release of GTH I in salmon (Swanson et al. 1987, 1989; Swanson 1992).

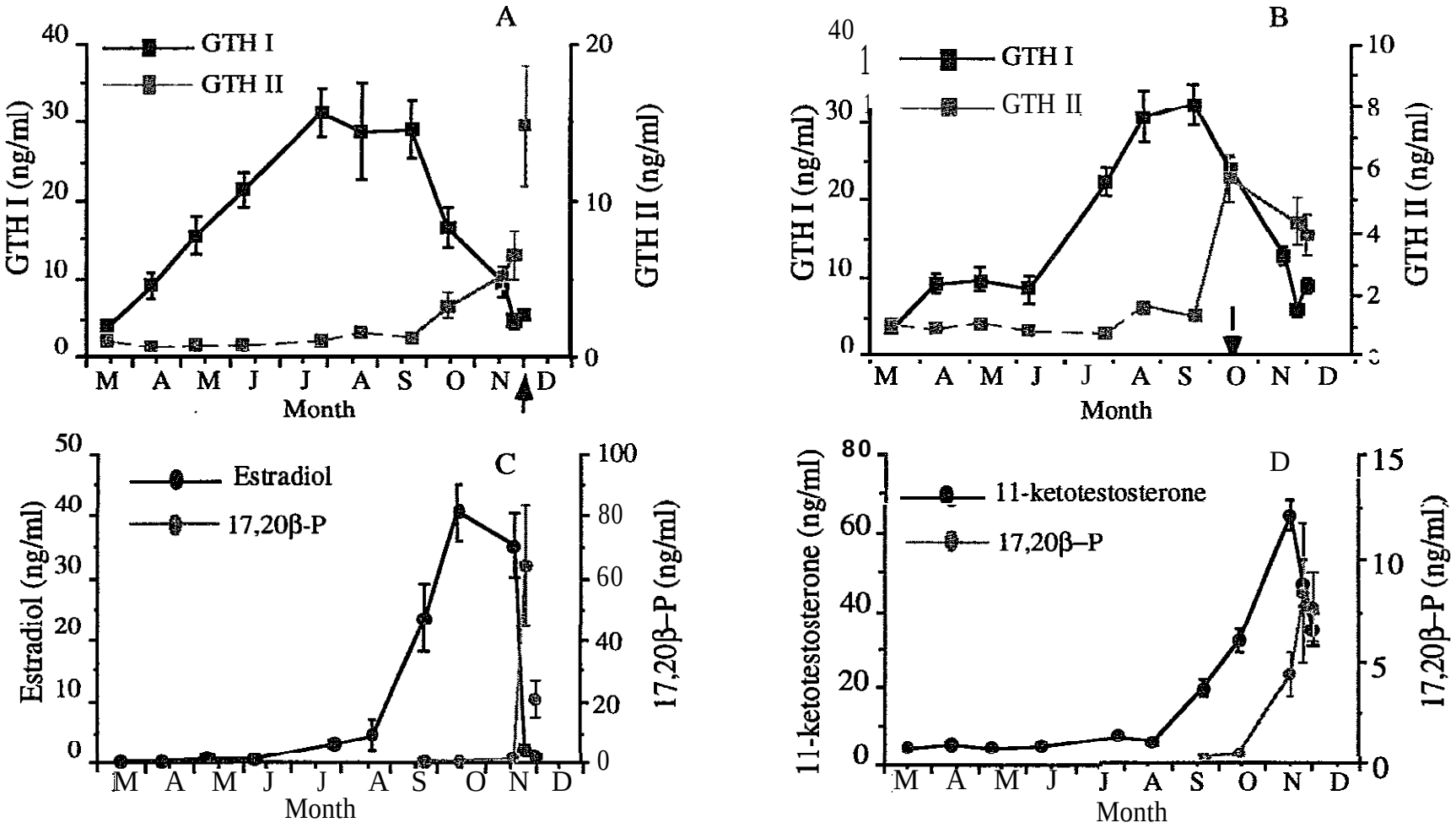

Figure 2. Plasma levels of gonadotropins, GTH I and GTH II, (A,B) and reproductive steroids (C,D) in male (B,D) and female (A,C) coho salmon during sexual maturation. In females, secondary oocyte growth occurred between March and November, and final oocyte maturation and ovulation occurred during late November and December. The last two data points in A and C are from two different groups of ovulated females. The arrow in A indicates when the first ovulated female was observed. In males, spermatogenesis occurred from March through mid-October, and spermiation occurred from October through December. The arrow in B indicates when milt could first be manually stripped from males. Each data point represents the mean ± standard error of 4-8 samples from individual fish. Dashed portion of line indicates levels of GTH II were nondetectable (< 1 ng/ml). 17,20β-P refers to 17α,20β-dihydroxy-4-pregnen-3-one. (from Swanson 1991 and upubl. data, NMFS).

Although the terms GnRH and luteinizing hormone-releasing hormone (LHRH) are frequently used interchangeably, LHRH has been generally used to describe the mammalian form of GnRH. The current and most widely used terminology to describe all members of this hormone family is GnRH. The specific type of GnRH is named according to the species in which it was first discovered.

In salmonids, at least two forms of GnRH have been identified. Salmon (s)GnRH has been purified and sequenced (Sherwood et al. 1983). Like mammalian GnRH, sGnRH is a decapeptide, but it differs from the mammaliam form in amino-acid postions seven and eight. A second form of brain GnRH (chicken (c) GnRH-II) has been found in salmonids (Sherwood et al. 1983, Okuzawa et al. 1990, Amano et al. 1991), which differs from sGnRH in amino-acid positions five and eight. The second form, cGnRH II, is probably not involved in the regulation of gonadotropin secretion in salmonids because cGnRH II was found throughout the brain, but could not be detected in the pituitary (Okuzawa et al. 1990, Amano et al. 1991). Interestingly, both sGnRH and cGnRH II stimulate *in vitro* secretion of GTH I and GTH II in a similar manner (Swanson 1992). However, the response of the pituitary varies according to stage of gametogenesis and the pituitary content of GTH I and GTH II. During vitellogenesis and spermatogenesis when ir-GTH I is elevated and ir-GTH II is low or nondetectable, GnRH stimulates release of GTH I. At the stage of spawning, when ir-GTH II is highest, GnRH stimulates release of GTH II and has either a weak or no effect on GTH I secretion.

In many but not all teleosts, G&I-I-stimulated GTH II release can be blocked by DA through direct effects on gonadotropes and by inhibiting release of GnRH (Peter 1983, Peter et al. 1991). The degree of this inhibition is strong in cyprinids, but either weak or absent in salmonids (Billard et al. 1984).

Gonadotropin Receptors

It is well established that gonadotropins act via binding to specific membrane receptors. Initial studies in fish suggested that there was a single type of GTH receptor (Breton et al. 1986, Kanamori et al. 1987, Kanamori and Nagahama 1988, LeGac et al. 1988). However, recent studies using purified GTH I and GTH II in salmon demonstrated two types of GTH receptors: a type I receptor (GTH-RI), which binds both GTH I and GTH II, and a type II receptor (GTH-RII), which binds GTH II but not GTH I (Yan et al. 1992, Miwa et al. 1994). The GTH-RI was localized in three cell-types by in vitro ligand autoradiography: in the thecal cell-layer and granulosa cells of the vitellogenic follicle, in the thecal cell-layer of the preovulatory follicle, and in presumptive Sertoli cells of the testis at all stages of spermatogenesis (Miwa et al. 1994). In contrast, the GTH-RII was localized in only the granulosa cells of the preovulatory follicle and Leydig cells of the fully mature (sperrniating) testis.

Gonadotropin Regulation of Oocyte Growth

The salmon ovary consists of oogonia, oocytes and surrounding follicle cells, supporting stromal tissue, vascular, and nerve tissue. Oocytes grow while arrested in meiotic prophase. Enormous growth of the salmon oocyte occurs during the phase termed vitellogenesis, which involves the sequestration and packaging of the hepatically derived yolk precursor protein, vitellogenin. The selective uptake of vitellogenin into the oocyte occurs through a receptor-mediated process (Tyler et al. 1987.1988; Kanungo et al. 1990). The process of vitellogenesis is regulated by a two-step mechanism whereby gonadotropin stimulates ovarian synthesis of estradiol-17β (E), which in turn stimulates hepatic vitellogenin synthesis. The specific uptake of vitellogenin by the oocyte is stimulated by GTH I (Tyler et al. 1991) and possibly other growth factors.

During oocyte growth, the eggshell or vitelline envelope between the granulosa cells and oocyte is also formed (Yamagami et al. 1992). The eggshell consists of a thin outer zona pellucida and thick inner zona radiata. The zona radiata (zr) proteins are glycoproteins produced in the liver in response to E (Hamazaki et al. 1989, Oppen-Bemtsen et al. 1992). The mechanism whereby zr-proteins are deposited on the oocyte surface is not understood.

In salmonid fiih, it has been demonstrated that during oocyte growth, plasma levels of E increase and subsequently decline prior to fmal oocyte maturation (Fostier et al. 1978). The production of E by the follicular cells of the salmon ovary requires the involvement of both the special thecal cells and granulosa cells (Kagawa et al. 1982a; Nagahama et al. 1982b; Nagahama 1983, 1987) and is stimulated by gonadotropin (Kagawa et al. 1982b). Testosterone, produced by the thecal cell layer, is converted to E by aromatase in granulosa cells. Both GTH I and GTH II stimulate in vitro production of testosterone and E by intact ovarian follicles (Suzuki et al. 1998d, Swanson et al. 1989, Planas 1993), and production of testosterone by isolated thecal cell-layers (Kanamori et al. 1988, Suzuki et al. 1988d, Planas 1993). The effect of GTH I and GTH II on E production may occur through stimulatory effects on T production because a direct stimulatory effect of gonadotropin on aromatase activity has not been reported in salmon.

The similar ability of GTH I and GTH II to stimulate steroid production at this stage is most likely due to a single type of gonadotropin receptor present in both the thecal and granulosa cells, which binds both gonadotropins (Yan et al. 1992, Miwa et al. 1994). Although both GTH I and GTH II show similar steroidogenic activity in the vitellogenic follicle, it is unlikely that GTH II plays a physiological role at this stage in salmonids because plasma levels are low or nondetectable. Furthermore, levels of GTH I, but not GTH II, are significantly correlated with plasma E and zr-proteins in salmon (Oppen-Bemsten et al. 1994).

Thus, the production of both vitellogenin and eggshell proteins in salmonids is probably regulated indirectly by GTH I through its effects on E production (Fig. 3). Non-steroid mediated effects of GTH I on oogenesis may also exist, but have not yet been reported.

Gonadotropin Regulation of Final Oocyte Maturation

Oocyte growth is followed by a process called final oocyte maturation (the resumption of meiosis), which must precede ovulation and is required for successful fertilization. It is well established that gonadotropin initiates final oocyte maturation by stimulating production of maturation-inducing hormones (MIH) by ovarian folllcular cells (Nagahama *et* al. 1982b; Nagahama 1983, 1987). A number of C21-steroids induce oocyte maturation *in* vitro, including 17α-hydroxyprogesterone (17-OHP); 17α,20β-dihydroxy-4-pregnen-3-one (17,20β-P), 17α,20β,21-trihydroxy-4-pregnen-3-one (20β-S), cortisol, and deoxycorticosterone. In salmonids 17α,20β-P is the most potent MIH (Nagahama 1983, 1987).

Several studies have shown that gonadotropin stimulates production of 17-OHP in the thecal cells; the 17-OHP is then converted to 17α,20β-P in granulosa cells by the enzyme, 20β-hydroxysteroid dehydrogenase (20β-HSD). In the preovulatory salmon follicle, both GTH I and GTH II stimulate production of 17-OHP by thecal cell layers; however, GTH II is far more potent than GTH I in stimulating conversion of 17-OHP to 17α,20β-P (Suzuki et al. 1988d, Planas 1993). The enhanced potency of GTH II during this period is associated with the appearance of a GTH II-specific receptor in the granulosa cells (Miwa et al. 1994). Additionally, the gonadotropin receptor, which binds both GTH I and GTH II (GTH-RI), is not present in granulosa cells of the preovulatory follicle. The inability of GTH I to stimulate activity is probably due to the loss of GTH-RI in the granulosa cells at this stage.

During the post-vitellogenic period, the capacity of the follicles to produce E declines and is associated with a decline in aromatase activity in granulosa cells, while 20β-HSD activity increases (Kanamori et al. 1988). The capacity of the thecal cells to produce testosterone in response to GTH increases at this time. Just prior to final oocyte maturation, the production of 17-OHP by thecal layers increases. Thus, there is a shift in the steroidogenic pathway in granulosa cells from production of E to 17α,20β-P, and there is enhanced production of 17-OHP by theca cells.

The induction of 20β-HSD and production of 17α,20β-P during final oocyte maturation are most likely controlled by GTH II, because plasma levels of GTH II increase during this period, and a receptor that binds GTH II but not GTH I is present in granulosa cells of the preovulatory follicle (Miwa et al. 1994). The appearance of the GTH II-specific receptor is coordinated with the increase in plasma levels of GTH II that precedes ovulation. Thus, key steps in the process of final oocyte maturation are regulated by GTH II (Fig. 3).

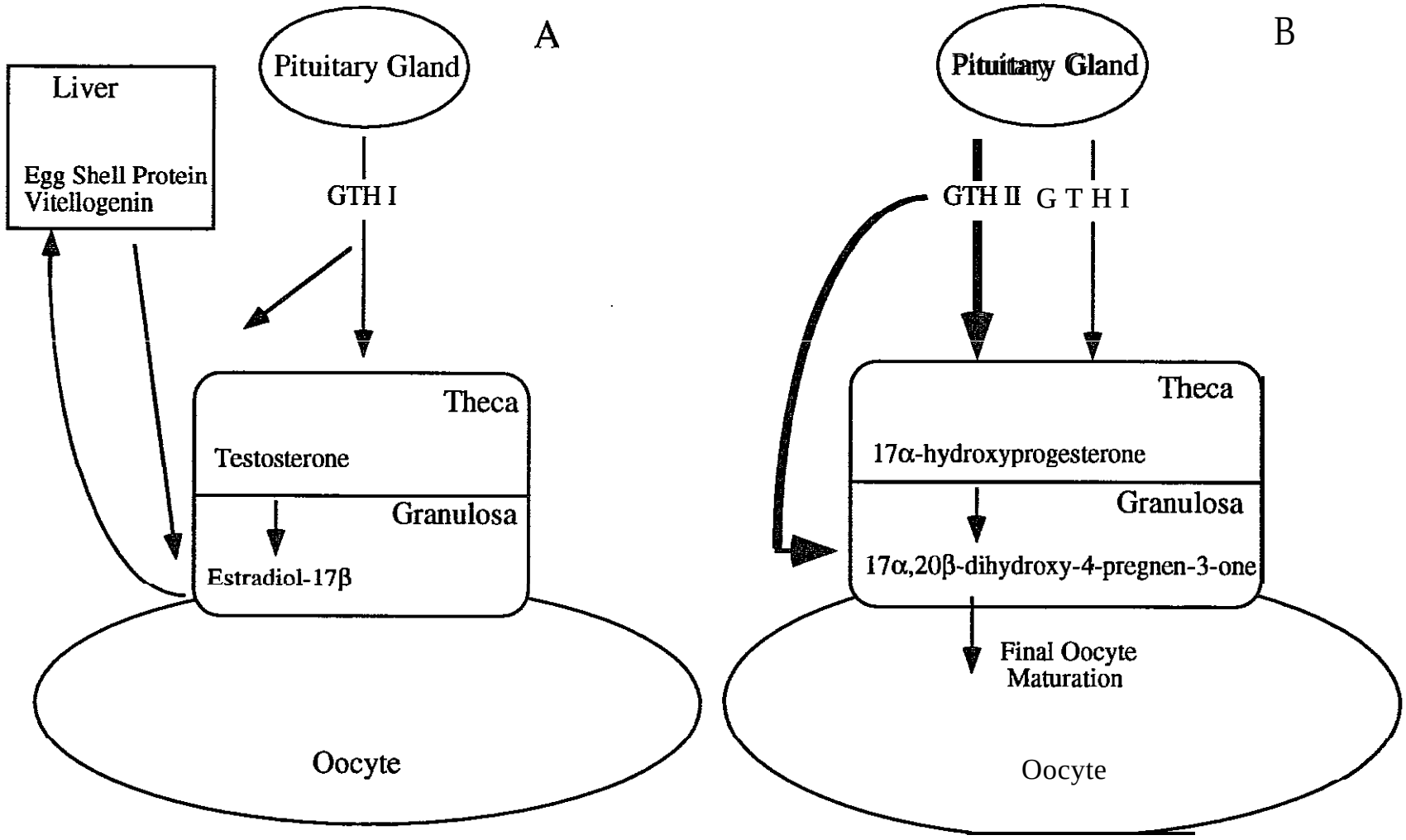

Figure 3. Diagramatic representation of the actions of gonadotropins (GTH I and GTH II) in the salmon ovary during secondary oocyte growth (A) and final oocyte maturation (B). During oocyte growth, plasma levels of GTH I increase. GTH I stimulates production of estradiol-17β, which is secreted into the peripheral ciruculation and stimulates the liver to produce the egg yolk precursor protein (vitellogenin) and the egg shell protein. GTH I also stimulates the uptake of vitellogenin by the oocyte. During final oocyte maturation, plasma levels of GTH I decline, whereas levels of GTH II increase. At this stage, GTH II stimulates production of the maturation-inducing steroid, 17α,20β-dihydroxy-4-pregnen-3-one, which stimulates fmal oocyte maturation. GTH II stimulates production 17α-hydroxyprogesterone by thecal cells and the conversion of this steroid to 17α,20β-dihydroxy-4-pregnen-3-one. GTH I also stimulates production of 17α-hydroxyprogesterone by thecal cells, but is less potent that GTH II. The thickness of the arrows in B reflects the relative difference in plasma levels of GTH I and GTH II.

Gonadotropin Regulation of Spermatogenesis and Spermiation

The salmon testis is composed of interstitial and lobular (tubular) compartments (Billard 1983, 1992; Billard et al. 1986). In the interstitial compartment, Leydig cells are present and are involved in steroid biosynthesis. Within the lobule, two cell types are present: germ cells and Sertoli cells, which line the periphery of the lobule. Sertoli cells in fish have both histcchemical and ultrastructural features, which suggest that they are steroidogenic. However, the role of Sertoli cells in testicular steroidogenesis is unclear (Nagahama 1983). In male salmonids, as well as in other teleost species, plasma levels of testosterone and 11-ketotestosterone (11-KT) increase during later stages of spermatogenesis and decline slightly at the time of spermiation, when plasma levels of 17α,20β-P increase (Hunt et al. 1982, Fostier et al. 1983, Ueda et al. 1984, Baynes and Scott 1985, Billard et al. 1990, Billard 1992).

Spermatogenesis is regulated by gonadotropin indirectly through stimulation of steroid biosynthesis (Billard et al. 1986, 1990; Billard 1992, 1993). Miura and colleagues (Miura et al. 1991) have shown that the entire process of spermatogenesis could be induced ***in vitro*** by 11-KT. However, spermiation and the acquisition of sperm motility are mediated by 17α,20β-P (Miura et al. 1992). Gonadotropin stimulates production of testosterone and 11-KT by somatic cells of the testis (Schulz 1986, Sakai et al. 1989, Schulz and Blum 1990, Planas 1993, Planas et al. 1993, Planas and Swanson 1994), and the capacity of the testis to produce these steroids in response to gonadotropin increases during spermatogenesis. The production of 17OH-P 17α,20β-P increases during late stages of spermatogenesis and spermiation (Depeche and Sire 1982, Sakai et al. 1989, Planas et al. 1993, Planas and Swanson 1994). Planas and Swanson (1994) demonstrated that during early to mid-stages of spermatogenesis, both GTH I and GTH II stimulate production of testosterone, 17α,20β-P, and 11-KT with similar potencies. However, during late stages of spermatogenesis and at spermiation, the steroidogenic potency of GTH II exceeds that of GTH I.

Similar potencies of GTH I and GTH II during the early stages of spermatogenesis could be correlated with the presence of a single type of gonadotropin receptor (GTH-RI) that binds both GTH I and GTH II (Miwa et al. 1994). This receptor was localized in presumptive Sertoli cells of the testis throughout spermatogenesis, but may also be present in Leydig cells. In the spermiating testis, when GTH II has enhanced steroidogenic potency, a second gonadotropin receptor (GTH-RII), which binds GTH II specifically appears in Leydig cells.

It is likely that spenniation and the acquisition of sperm motility in salmonids are regulated by GTH II, not GTH I, because during this period plasma levels of GTH II increase, a GTH II-specific receptor appears in Leydig cells, and the capacity of the testis to produce 17α,20β-P in response to GTH II increases.

In contrast, spermatogenesis is probably regulated by GTH I via stimulation of 11-KT production because plasma levels of GTH I and 11-KT increase during early phases of spermatogenesis and remain elevated throughout spermatogenesis when GTH II levels are non-detectable (Swanson 1991).

Applications of Reproductive Endocrinology to Captive Broodstock Programs for Salmonids and Future Research Needs

Reproductive endocrinology may be applied to captive broodstock programs by using hormones to control and monitor reproduction in broodfish. Later in this report, the use of exogenous hormones to control reproduction in salmonids is discussed. Plasma levels of hormones may be used to determine state of maturity and sex; gonadotropins to distinguish maturing from non-maturing broodfish, and steroids (E and 11-KT) to distinguish male from female fish. In both male and female salmonids, the period of gonadal growth appears to be regulated primarily by GTH I, whereas the period of final oocyte maturation and spermiation is regulated primarily by GTH II. Because plasma levels of GTH I increase 6 to 9 months in advance of the spawning period in maturing coho salmon (Swanson 1991, Fig. 2), it may be possible to use GTH I levels as a nondestructive method to distinguish maturing from non-maturing broodstock well in advance of the spawning period. Plasma GTH I and GTH II levels may also be used as a research tool to monitor the progress of maturation of experimental fish in response to various rearing conditions. Further research on the feasibility and reliability of using GTH I levels as a non-destructive method to distinguish maturing from non-maturing in a variety of salmonids will be required.

Environmental Regulation of Reproduction in Salmonids

Fish of the salmon family Salmonidae am native to the oceans, rivers and lakes of the northern temperate zone and are seasonally breeding fish. The majority of sahnonids in the temperate zone spawn during autumn (September to December). However, some stocks of rainbow trout (0. *mykiss*) and steelhead (0. *mykiss*) spawn during winter months (January to March) and a few spawn during the spring and summer. Spawning time has been shown to be a heritable trait in salmonids (Gall 1975, Gardner 1976, Gall et al. 1988). For each strain or stock, spawning occurs at a time which will ensure that the young fish or fry emerge when local climatic conditions am favorable and natural supplies of food are abundant (Brannon 1987, Heggeberget 1988). In addition to genetic factors, the seasonal timing of spawning in salmonids is controlled by photoperiod, temperature, and an endogenous circannual rhythm (reviewed by deVlaming 1972, Lam 1983, Scott 1990, Bromage et al. 1993).

Regulation of Reproduction in Salmonids by Photoperiod

There is substantial evidence that the reproductive cycle in salmonids is driven by an endogenous rhythm (Whitehead et al. 1978a, b; Scott 1979; Duston and Bromage 1986, 1987, 1991; Bromage and Duston 1986). Spawning occurs at approximately annual intervals in fish experimentally maintained on constant photoperiods (LD 12:12). The endogenous rhythm is self-sustaining, has an approximate circannual rhythmicity, and is synchronized or entrained to the annual cycles by environmental cues. Photoperiod is the most important of these environmental cues in sahnonids.

The majority of studies on photoperiod control of reproduction in sahnonids have been conducted in domesticated strains of rainbow trout (Hoover 1937, Bromage et al. 1993) and brook trout (*Salvelinus fontinalis)* (Hazard and Eddy 1951, Henderson 1963, Carlson and Hale 1973), while relatively few studies have been conducted on Pacific salmon (Combs et al. 1959; MacQuarrie et al. 1978, 1979; Johnson 1984) and Atlantic salmon (*Salmo salar*) (Johnston et al. 1987, 1990, 1992; Taranger et al. 1991; Hansen et al. 1992). These studies have demonstrated that advanced or compressed seasonal light cycles induce precocious gonadal development; whereas delayed or extended photoperiods result in later spawnings.

From the early 1960s to the mid-1980s there was considerable debate about whether gonadal development in salmonids is cued by “long” or “short” day lengths. This debate was fueled in part by conflicting definitions of what constituted a “long” or “short” day. In numerous studies, the terms “long” and “short” day were used rather loosely to describe day lengths mom than 16 and less than 8 hours of light per day, respectively.

It was thought that a specific quantity of light (in terms of day length) was required to exert an effect on reproduction.

Now it is clear that the direction of change of photoperiod, rather than the absolute number of hours of light, is the critical factor for entrainment of reproduction by photoperiod (Duston and Bromage 1987, Randall et al. 1987). Therefore, "long" days can be defined as those which are preceded or followed by a photoperiod which is shorter, and "short" days are those which are preceded or followed by a photopetiod which is longer. Based on this definition, Scott (1990) concludes that in all salmonids the reproductive cycle is initiated in springtime, under conditions of increasing or "long-day" photoperiods. Decreasing or "short-day" photoperiods only advance the reproductive cycle in autumn-spawning salmonids.

One of the questions that arose early in the studies on photoperiod control of reproduction was whether seasonally changing light was critical for controlling spawning time. Studies in rainbow trout have demonstrated that the timing of reproduction can be modified by an altered seasonal light cycle; however, seasonally changing light is not essential for maturation (Whitehead et al. 1978a, b; Bromage et al. 1982, 1984; Elliot et al. 1984; Bromage and Duston 1986, 1987; Duston and Bromage 1986, 1987). For example, the rise and decline in day length, which occurs seasonally, can be replaced by constant "long" and "short" photoperlods, respectively (Whitehead and Bromage 1980, Bromage et al. 1982, Bromage and Duston 1986). Because of the endogenous rhythm controlling maturation, the timing of exposure to changes in day length relative to the phase of endogenous rhythm determines whether spawning is advanced or delayed and the degree to which spawning time is altered. This phenomena has been studied extensively in rainbow trout and is reviewed below. Studies conducted on other salmonids are cited when relevant

Spawning time can be advanced if rainbow trout are exposed to "long" days or continuous light during the early part of the reproductive cycle, or from the winter solstice throughout the spring (Whitehead and Bromage 1980; Bromage et al. 1982.1984; Scott et al. 1984; Bromage and Duston 1986, 1987; Duston and Bromage 1987, 1988). The degree of advancement of spawning time appears to be greatest if trout are exposed to "long" days or continuous light just after spawning at the winter solstice (Bromage et al. 1984, Scott et al. 1984). In contrast, the reproductive cycle of salmonids can be extended and spawning time can be delayed by exposing fish to "long" days or continuous light after the summer solstice (Allison 1951; Combs et al. 1959; Henderson 1963; Shirashi and Fukuda 1966; MacQuarrie et al. 1978, 1979; Lundqvist 1980; Whitehead and Bromage 1980; Bromage et al. 1982; Johnson 1984; Bourlier and Billard 1984; Takashima and Yamada 1984). Fish exposed to continuous light after the summer solstice not only show a delay in spawning, but their period of spawning can be extended two- to three-fold (Bourlier and Billard 1984).

However, exposure to continuous light will also cause spawning to be asynchronous within a group of fish and a significant amount of atresia of eggs can occur (Bourlier and Billard 1984).

In general, exposure of trout to constant "short" days during the very early stages of maturation results in a delay in spawning (Whitehead and Bromage 1980, Bromage et al. 1984, Duston and Bromage 1987). The extent of the delay depends on the point at which the photoperiod is applied relative to the stage of reproductive development and natural spawning time of the particular stock of fish. Once maturation has proceeded for approximately 3 to 4 months in rainbow trout, exposure of fish to "short" days advances spawning time (Bromage et al. 1984, Duston and Bromage 1987).

Regulation of Reproduction in Salmonids by Temperature

Although photoperiod is regarded as the most important environmental factor controlling gonadal development and spawning time in sahnonids, it does not act alone in controlling reproductive function.
The extent to which temperature acts in concert with photoperiod to regulate the timing and rate of gametogenesis is poorly understood. Most studies on photoperiod manipulation of reproduction have maintained fish on either constant or ambient temperatures averaging 10 °C Few experimental studies have been conducted on the effects of rearing temperature on reproduction in sabnonids (Henderson 1963; Breton and Billard 1977; Morrison and Smith 1986; Beacham and Murray 1988; Bromage and Cumaranatunga 1988; Nakari et al. 1987, 1988; Johnston et al. 1987, 1990, 1992; Taranger and Hansen 1993).

In rainbow trout, studies have shown that temperatures ranging from 8 to 16 °C have little effect on timing of the reproductive cycle, but higher temperatures adversely affect the quality and quantity of gametes (Billard 1985). The approximate upper limit for successful reproduction of salmonids in the wild is considered to be 13 °C (MacCrimmon 1971, Scott 1990). However, this upper limit may be lower for salmonids which naturally spawn in the northernmost latitudes or high altitudes (Tarauger and Hansen 1993).

While the range of water temperatures for survival and successful reproduction has been studied for a number of salmonids, the question of whether seasonally fluctuating temperatures are important for reproductive performance of broodstock has not been thoroughly investigated. In many hatchery conditions, particularly those using wellwater, fish am reared on relatively constant water temperature. Only one study has compared the effects of constant versus seasonally fluctuating rearing temperature on spawning time. Davies and Bromage (1991) maintained rainbow trout on a river-water supply with seasonal variations in water temperature ranging from 4°C in February to 16.5°C in July. They found that these fish responded similarly to a parallel group of fish maintained on well-water with constant temperature of 8.5°C and exposed to the same stimulatory photoperiod.

Spawning time of both groups was advanced. However, Davies and Bromage did not describe the effects of temperature on the quality of gametes produced by fish reared on the two thermal regimes. Other important factors to consider are that the response of domesticated stocks to rearing temperatures may be very different than that of wild stocks and may vary considerably among species and stocks.

Because fish are poikilothermic animals, temperature directly affects their gonadal physiology by changing the rate of yolk sequestration, steroid biosynthesis, and other general metabolic reactions. In salmonids, low temperatures (< 8°C) reduce the rate of maturation; causing vitellogenesis (Johnston et al. 1987, Korsgaard et al. 1986) and oocyte growth (Crim et al. 1983b) to proceed slowly. At very low temperatures (< 3°C) the final stages of oocyte maturation and ovulation are completely inhibited (Billard 1985). Testicular steroid production is also reduced at low temperatures (Manning and Kime 1985). Spawning time in autumn-spawning trout can be delayed until spring by adversely cold weather conditions, and conversely, spring-spawning trout can be made to spawn in winter or autumn by movement into temperate water conditions (Morrison and Smith 1986, Nakari et al. 1987, Titarev 1975). In Atlantic salmon, high water temperatures (increases from 10 to 13-14°C) during the spawning season inhibit ovulation and have a detrimental effect on gamete quality (Taranger and Hansen 1993). Heggeberget (1988) found that in this species, peak spawning in Norwegian rivers occured when water temperatures were decreasing. This suggests that among sahnonid species and/or strains temperature may affect reproduction differently.

Applications of Environmental Control of Reproduction to Captive Broodstock Programs for Salmonids and Future Research Needs

In commercial farming of salmonids, manipulation of spawning time has been used to spread the availability of juvenile fish over the year. This regulation provides the market with continuous production, and synchronizes or compresses the spawning period. In captive broodstock programs for depleted fish stocks, off-season spawning of fish may not be an appropriate goal, since production of progeny for release into the natural habitat should be timed appropriately for the requirements of the stock. However, photoperiod in captive rearing facilities must be controlled, and inadvertent exposure of fish to light during the dark phases should be avoided. Uncontrolled lighting could have catastrophic consequences if, for example, it artificially extended spawning periods into periods when ambient water temperatures are sufficiently high to impair gamete quality and offspring survival. Furthermore, exposure to continuous light can induce asynchronous maturation and atresia of oocytes (Bourlier and Billard 1984).

MacQuarrie et al. (1978) observed abnormalities in the process of oogenesis and poor fertilization of eggs when coho salmon were exposed to advanced photoperiod and natural thermoperiod.

In addition, the photoperiod history, stage of reproductive development, and natural spawning time of the fish must be considered if alterations in light-cycles are made because the response of the fish to changes in photoperiod depends on all three factors.

The effects of temperature on reproductive performance in salmonids have not been extensively examined. Further studies are necessary to develop better guidelines for rearing temperatures for Pacific salmon broodstock, and to determine whether constant or seasonally fluctuating water temperatures affect reproductive performance. This is particularly important because facilities for captive rearing of broodstock may be practically limited by the ability to control water temperature and the availability of water of constant versus fluctuating temperatures.

Hormonal Induction of Final Oocyte Maturation, Ovulation, and Spermiation in Salmonids

Reproduction in captive broodfish can artificially controlled at two levels: through manipulation of environmental cues and through alterations in the reproductive endocrine system. As mentioned in the previous section on environmental control of reproduction in sahnonids, spawning time can be delayed or advanced through alterations in environmental factors such as photoperiod. Photoperiodic influences on reproduction, after perception and integration by the central nervous system, alter release of hormones by the hypothalamus (primarily GnRH), which in turn regulate secretion of pituitary gonadotropins (Fig. 1). Techniques for the control of reproduction through exogenous administration of hormones have been developed which intervene at each level of the brain (GnRH)-pituitary (gonadotropin)-gonad (steroid) axis.

Although manipulation of gametogenesis (gonadal growth) has been achieved in some species through chronic treatment of fish with various preparations of gonadotropins and/or steroids (Billard et al. 1986, 1990; Billard 1992), this technology is not presently used on cultured salmonids. However, hormonal induction of final oocyte maturation, ovulation, and spermiation is widely used on salmon broodstock to prevent losses due to prespawning mortality and to advance or synchronize spawning time (Donaldson and Hunter 1983; Donaldson 1986; Zohar 1988.1989).

Hormones Used to Induce Final Oocyte Maturation, Ovulation, and Spermiation

The hormonal induction of spawning is achieved by producing an increase in plasma levels of gonadotropin (GTH II or homologous proteins) in fish that have completed vitellogenesis and spermatogenesis. The gonadotropins act on the ovary to induce final oocyte maturation and ovulation or on the testis to induce spermiation (as previously described). Plasma levels of gonadotropins can be elevated either by stimulating secretion of endogenous gonadotropins, or by administration of pituitary extracts, human chorionic gonadotropin (a human hormone which is chemically similar to fish GTH II), or purified preparations of fish gonadotropins.

Induction of spawning with pituitary extracts or partially purified salmon gonadotropin has been demonstrated widely in salmonids (Jalabert et al. 1978; Hunter et al. 1978, 1979, 1981; Sower et al. 1982; Donaldson et al. 1985; Van Der Kraak et al. 1985). However, this technique has some major limitations. There is a high degree of variability in the purity and quality of the gonadotropin preparations, and highly purified gonadotropins are expensive and difficult to obtain.

Furthermore, there is a high degree of species specificity in fish gonadotropin: a preparation from one species may not be effective in another. Finally, the content of biologically active gonadotropin in pituitary extracts is variable, and the presence of other hormones in the extracts can lead to undesirable effects.

A more reliable and economical alternative to administration of gonadotropin preparations has been the use of GnRH or superactive analogues of GnRH (GnRHa) for induction of spawning. This appears to be the most efficient therapy because GnRH stimulates release of endogenous gonadotropin, thus avoiding problems with species specificity and quality of the gonadotropin preparations. GnRH and GnRHa are non-immunogenic decapeptides, which can be synthesized and obtained in pure form and are readily available from a variety of chemical companies. They also can be administered in very low doses (few micrograms/kilogram body weight), which is more economical than gonadotropin preparations.

Induction of Spawning with Analogues of GnRH in Salmonids

Induction of ovulation and spermiation in fully mature fish using GnRH or GnRHa has been achieved in a wide variety of salmonids (Donaldson et al. 1981, 1984; Sower et al. 1982, 1984; Crim et al. 1983a, b, 1986, 1987, 1988a, b; Donaldson and Hunter 1983; Weil and Crim 1983; Crim and Glebe 1984; Fitzpatrick et al. 1984, 1987; Van Der Kraak et al. 1985; Zohar et al. 1990a, b; Breton et al., 1990; Taranger et al. 1992; Mylonas et al. 1993; Haraldsson et al. 1993; Slater et al. 1995). In initial studies of GnRH or GnRHa-induced spawning in salmonids, GnRH or GnRHa were tested in conjunction with gonadotropin preparations (Donaldson et al. 1981, 1984, 1985; Sower et al. 1982, 1984; Van Der Kraak et al. 1983, 1984, 1985). However, treatment with GnRHa alone has been shown to be equally effective to that of GnRHa combined with gonadotropin as long as two injections of GnRHa were given.

Superactive analogues of GnRH are generally more potent *in* vivo than native forms of GnRH (Crim et al. 1987, 1988b; Donaldson et al, 1981) because of their prolonged biological half lives (Goren et al. 1987, Zohar et al. 1990a) and higher affinity to GnRH receptors (Habibi et al. 1987). The analogues of GnRH most commonly used for induction of spawning are as follows: [D-Arg^{6}-Pro^{9}NET] salmon GnRH, [D-Ala^{6}-Pro^{9}-NET] mammalian GnRH, and des-Gly^{10}[D-Ala^{6}]-mammalian GnRH ethylamide. These analogues differ somewhat in their ability to induce spawning in goldfish and in their affinity to goldfish GnRH receptors (Habibi et al. 1987, Habibi and Peter 1991); however, no significant differences have been observed among these analogues when used in salmonid fish or seabream (*Sparus aurata*) for spawning induction (Zohar et al. 1989, 1990a; Haraldsson et al. 1993).

For successful induction of ovulation in salmonid fish, GnRHa dissolved in saline has been administered by two intramuscular or intraperiotoneal injections spaced at an interval of 3 days (Donaldson 1986; Crim et al. 1987; Van Der Kraak et al. 1984, 1985; Fitzpatrick et al. 1984, 1987; Zohar et al. 1989, 1990a; Slater et al. 1995). In general, G&Ha-induced ovulation was observed within 10- 14 days after the initial injection. However, the rate of response to the GnRHa depended on the timing of treatment relative to the natural period of ovulation and the dosage of GnRHa. If GnRHa was administered within 3 to 4 weeks prior to the normal spawning time, ovulation was synchronized and advanced by approximately 2 weeks (Taranger et al. 1992). Ovulation was synchronized, but not significantly advanced when fish were. treated within 2 weeks of the normal spawning time. Dosages ranging from 10 to 150 μg GnRHa per kg body weight have been used to advance or synchronize ovulation in female salmonids. However, several studies (Crhn and Glebe 1984, Taranger et al. 1992) have reported reduced fertilization rates and survival of offspring to the eyed-stage when females were treated with high dosages of GnRHa (100-150 μg/kg body weight). Dosages of 10-20 μg GnRHa/kg body weight have been shown adequate for advancing or synchronizing ovulation without impairing offspring survival (VanDerKraak et al. 1985, Taranger et al. 1992).

Technology for controlled-release GnRHa delivery systems has been developed for induction of ovulation and spermiation in sahnonids (Crim et al. 1983b, 1988a; Crim and Glebe 1984; Zohar et al. 1990b; Breton et al., 1990). The advantages of this technique are that 1) the quantity of hormone administered and labor required for the treatment can be reduced, making the treatment more cost-effective, and 2) stress to the broodfish associated with protocols requiring mutiple injections can be reduced. Several types of delivery systems for GnRHa have been developed and tested in salmonids.

Crim and colleagues (Crim et al. 1983b, 1987, 1988a; Crim and Glebe 1984) advanced and synchronized ovulation with a pelleted form of GnRHa in a cholesterol matrix. The minimum dose of GnRHa has not been determined for this technique; however, a single treatment with 20-25 μg GnRHa/kg body weight was effective in advancing ovulation (Crim and Glebe 1984). Zohar and colleagues (Zohar et al. 1990b) have developed two types of GnRHa delivery systems: nonbiodegradable and biodegradable. The nonbiodegradable delivery system is a pellet (approximately 0.5 mm diameter) composed of an ethylene vinyl acetate copolymer (EVAC). Implants containing dosages of GnRHa as low as 25 μg per fish (body weight ranging from approximately 0.8 to 2.5 kg) effectively induced ovulation in trout (Breton et al. 1990) and coho salmon (Fig. 4) without impairing offspring survival. These implants were administered by intramuscular injection and induced ovulation or spermiation approximately 8-12 days after treatment.

A second type of delivery system developed by Zohar and colleages (Zohar et al. 1990b) is a preparation of biodegradable microspheres consisting of a polyglycolic acid copolymer.

The microspheres containing GnRHa (75 µg GnRHa/kg body weight) and administered by intramuscular injection have been used to induce ovulation and spermiation 2 to 4 weeks in advance of the normal spawning time in coho salmon (Pig. 5). In farmed coho salmon, a combination of compressed photoperiod and treatment with GnRHa-microspheres was used to advance spawning by 4 to 8 weeks (Greg Hudson, Domsea Broodstock, Inc., Rochester, Washington, Pers. commun. June, 1991). Similar results have been obtained for advancement of maturation in sockeye (0. *nerka)* salmon without impairing gamete quality using microspheres containing GnRHa (Fig. 6). Presently, the minimum dosage of GnRHa-microspheres required for induction of ovulation and spermiation has not been established in salmonids.

Timing of Hormone Treatments for Spawning Induction

One of the major considerations in developing technology for induction of ovulation and spermiation is the timing of hormone injection. Presently, females are selected for treatment on the basis of overall appearance (coloration, shape of abdomen, etc.) or stage of oocyte development, which is determined after ovarian biopsy in fish with sufficiently small eggs. Unfortunately, morphological indicators are not always reliable indicators of responsiveness, and fish with large eggs like sahnonids am not easily biopsied. Determination of the stage of maturity can be particularly difficult in some wild salmon stocks that have extended spawning periods.

Fitzpatrick et al. (1987) have suggested that plasma testosterone levels, which are elevated preceding ovulation in sahnonids, may be used as a hormonal indicator to predict the sensitivity of the female to GnRHa. This technique is somewhat limited because it is most reliable if two plasma samples can be taken at an interval of several days, so that a relative change in testosterone levels can be observed to gauge more precisely the stage of maturation. In addition, it currently requires sophisticated laboratory facilities to analyze the testosterone levels by radioimmunoassay, and several days or weeks before the information is available to hatchery managers. The reliability of testosterone levels as indicators of responsiveness to GnRHa will require further testing to reach a practical level of use in management of captive broodstock.

Another method to determine the timing of hormone-induced spawning is simply use of the calendar. Hormone-induced spawning has been successful when treatments were administered within 2 to 4 weeks in advance of the normal spawning period, depending on the mode of administration of GnRHa. This technique is practical when the normal spawning period for the broodstock is well characterized and occurs within a 4- to 6-week period, as in the case of domesticated stocks of farmed salmon. Obviously, when secondary sex characteristics develop near the time of spawning, GnRHa can be used reliably to synchronize spawning among broodstock.

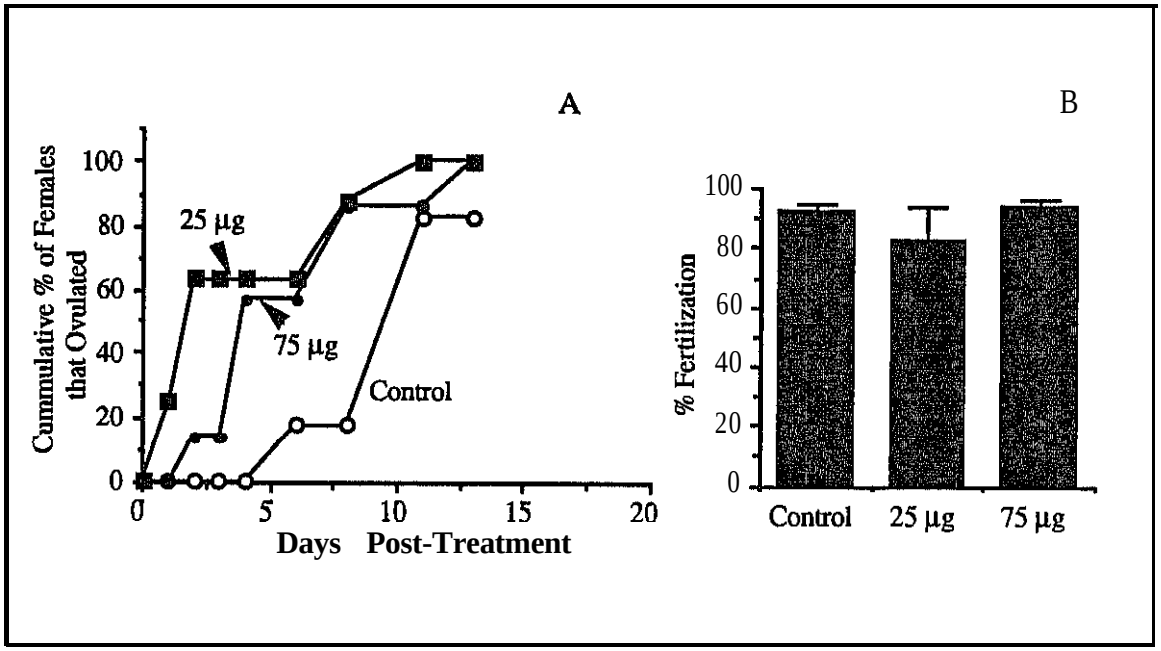

Figure 4. Induction of ovulation in female coho salmon using gonadotropin-releasing hormone analogue (GnRHa) administered via ethylene vinyl acetate copolymer (EVAC) implants. Fish (N = 10 per treatment) were injected intramuscularly with EVAC implants containing either 25 or 75 micrograms GnRHa. Control fish received blank implants. Data on ovulation are expressed as cummulative percent of females that ovulated during the course of study. Both doses of GnRHa effectively advanced ovulation (A) without impairing egg quality (B). Data on fertilization are mean ± standard error. (Swanson, Dickhoff, Larsen, and Zohar, unpubl. data, NMFS).

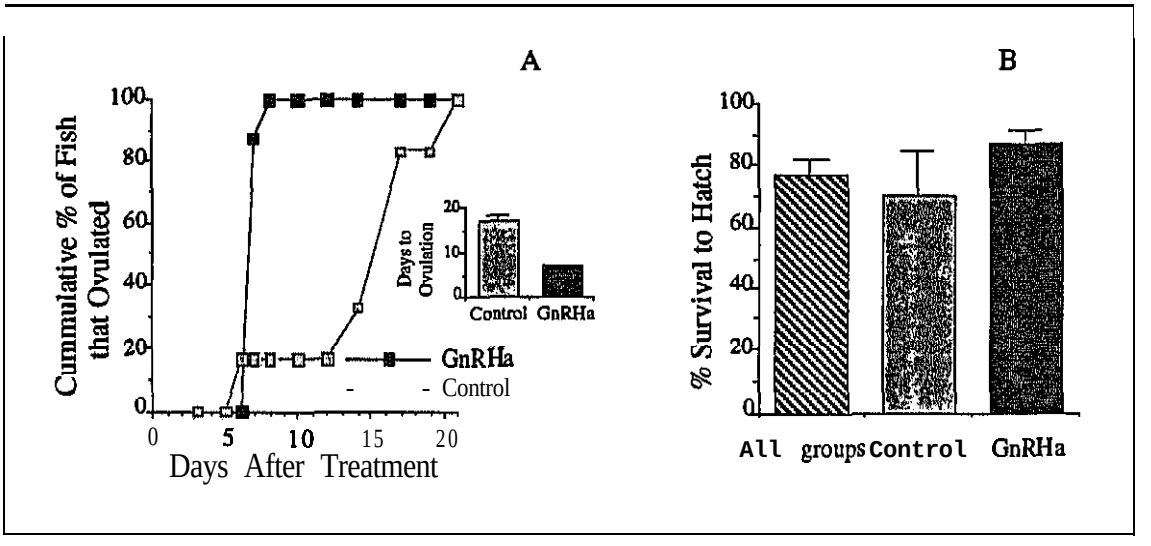

Figure 5. Induction of ovulation in adult female coho salmon using microspheres containing GnRHa (75 micrograms& body weight). Fish (N = 6 per treatment) were treated approximately 3 weeks in advance of the historical spawning date for the stock. Treatment with GnRHa-microspheres advanced ovulation by about 2 weeks without affecting survival of embryos to hatching. (Swanson, Dickhoff, Larsen, Mylonas and Zohar, unpubl. data, NMFS)

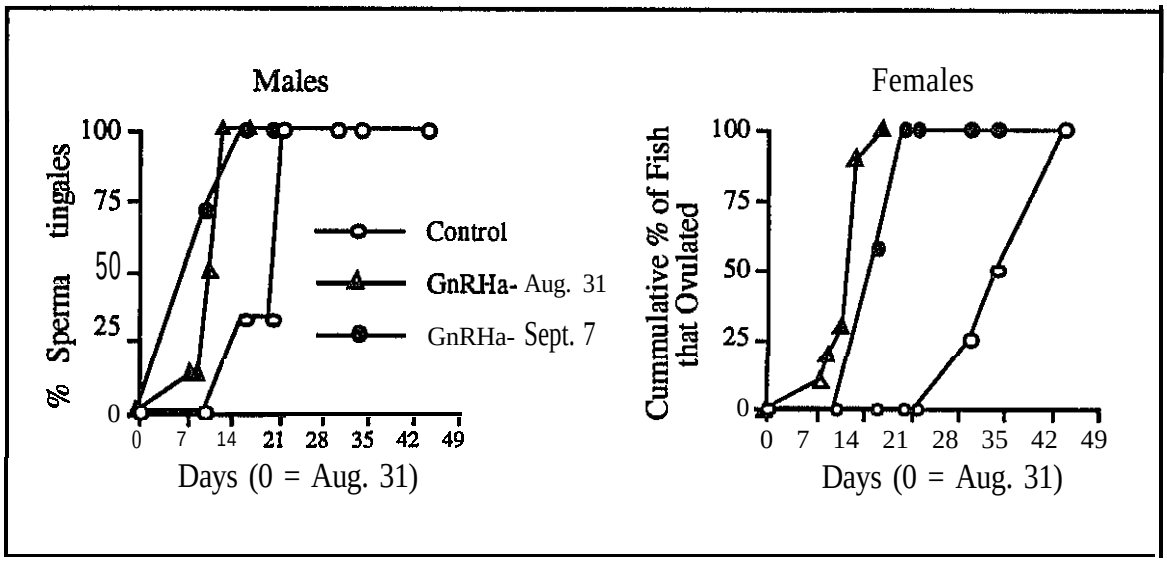

Figure 6. Induction of spermiation and ovulation in Lake Wenatchee sockeye salmon. Fish were injected intramuscularly with microspheres containing GnRHa (75 micrograms/kg body weight) either on day 0 (Aug. 31) or day 7 (Sept. 7). Control fish received blank microspheres on day 0. N = 10 fish of each sex per group. Data are expressed as cummulative percent of either spermiating males or ovulated females. The GnRHa treatment successfully advanced and synchronized maturation in both male and female fish without affecting the rate of fertilization or survival of fertilized eggs to the eyed stage (Swanson, Yan, Dickey, and Zohar unpubl. data, NMFS).

Applications of Hormone-Induced Maturation to Captive Broodstock Programs for Salmonids and Future Research

Technology for hormonal induction of ovulation and spermiation has several important applications to captive broodstock programs for endangered or threatened salmonid fish. This technology can be used to 1) prevent loss of gametes due to prespawning mortality, 2) synchronize spawning in wild and captive fish, 3) extend the period of spermiation and yield of sperm, and 4) to synchronize and/or advance spawning in male and female broodfish. Hatchery managers may not need to routinely induce spawning in broodstock, but this technology is a tool that should be available to managers who may need to produce a predictable spawning period or advance spawning if there is a risk of prespawning mortality. However, artificial induction of spawning using GnRHa should be further refined for general use in control of reproduction in Pacific salmon broodstock.

Further research is necessary to develop a reliable index of when GnRHa can be administered to both male and female fiih for successful induction of spawning. This will be particularly important for species with extended spawning seasons. The minimum effective dosages and optimal modes of administration of GnRHa must be established in several species and stocks for both males and females.

Precocious or Early Maturation in Male Salmonids

In both captive and wild populations of anadromous salmonids, sexual maturation may occur at an earlier age for males than for females in the breeding population. Early, or precocious maturation in male salmonids may occur in fresh water prior to smoltification and seawater migration or after a period of seawater residence. The term "precocious parr" has been used to describe male salmonids that mature without smolting and migrating to sea, whereas the term "jack" has been used to describe males that mature after a period of seawater residence, usually at least 1 year prior to the first maturation of females. The period of seawater residence for jacks depends on the species, and is less than 1 year for coho salmon and up to a year for chinook salmon.

Precocious maturation of male parr is common in Atlantic salmon (Thorpe 1986), but also has been documented in a wide variety of anadromous ***Oncorhynchus*** sp. including chinook salmon (Robertson 1957, Gebhards 1960, Taylor 1989, Foote et al. 1991), sockeye salmon (Ricker 1938, 1959; Burgner 1991), coho salmon (Silverstein and Hershberger 1992), steelhead (0. *mykiss*) (Schmidt and House 1979), amago (0. ***rhodurus)*** (Nagahama et al. 1982a), and masu salmon (0. *masu*) (Aida et al. 1984, Kato 1991). Early maturation of males after seawater residence has been documented for chinook salmon (Hard et al. 1985, Healy 1991, Bocking and Nass 1992), coho salmon (Bilton 1980, Bilton et al. 1981, Sandercock 1991) and sockeye salmon (Burgner 1991).

The phenomena of precocious sexual maturation of male salmonids is an evolutionarily viable, alternative life-history strategy. Smaller, younger males gain access to females for reproduction by utilizing "sneaking" behavior, while the larger, older males guard females and fight each other to defend their territory (Gross 1985). Thus in nature, precocious male maturation is not necessarily an undesirable characteristic. However, in captive breeding programs for depleted salmon stocks, maturation of males in the absence of matnre females could be catastrophic.

In commercial aquaculture, early maturing male fish have reduced market value and poor survival, thus representing serious financial loss to the farmer. In addition, precocious male maturation in hatchery-reared Atlantic salmon causes a significant decrease in the rate of adult recaptures from hatchery releases (Lundqvist et al. 1988) and a loss of adult chinook salmon to the fishery (Foote et al. 1991, Mullan et al. 1992). Precocious male parr may reabsorb gonadal tissue and smolt in the year following maturation (Robertson 1957, Gebhard 1960, Lundqvist and Fridberg 1982, Bemier et al. 1992).

However, the maturation process impairs seawater adaptability and smolting during the subsequent spring in precocious Atlantic salmon (Saunders et al. 1982, Langdon and Thorpe 1985, Lundqvist et al. 1988, Thorpe 1987), sea trout (S. *trutta*) (Dellefors and Faremo 1988), masu salmon (Aida et al. 1984), amago salmon (Nagahama et al. 1982a) and chinook salmon (Foote et al. 1991). It has also been shown that the mortality rate of mature male parr is higher than that of immature parr (Myers 1984, Dempson et al. 1986). Therefore, a better understanding of the control of early male maturation is needed to develop rearing methods that will maximize the synchronous maturation of both sexes and reduce the proportion of precocious males while not adversely affecting the survival of sexually immature smolts.

There is strong evidence that the age of sexual maturity in salmonids is regulated by both genetic and environmental factors (Purdom 1979, Randall et al. 1986, Gjerde 1984a). Environmental factors include both abiotic factors such as photoperiod and temperature, which affect the seasonal timing of maturation, and biotic factors such as food availability and diet composition, which affect growth and energy status. Although effects of both genotype and environment on precocious male maturation have been found, clarifying the relative roles of these factors has been difficult because of their interrelatedness. In the following sections, biological factors that affect the rate of early maturation in male sahnonids are discussed.

Genetic Basis of Precocious Maturation

There is strong evidence that genetic factors play a role in determing the age of sexual maturation in several species of salmonids (Purdom 1979, Gjedrem 1985, Naevdal 1983). Most genetic evidence for age of sexual maturity has come from work on Altantic salmon (Gardner 1976; Naevdal et al. 1978a; Thorpe and Morgan 1978.1980; Thorpe et al. 1983; Gjerde 1984a, b; Myers et al. 1986; Glebe and Saunders 1986) and rainbow trout (Gall 1975; Moller et al. 1976; Naevdal et al. 1979; Gjedrem 1985; Gall et al. 1988; Crandell and Gall 1993a, b). However, there is also evidence in coho salmon (Iwamoto et al. 1984, Silverstein and Hershberger 1992), chinook salmon (Hard et al. 1985, Heath 1992), and ***Arctic*** charr, ***Salvelinus alpinus*** (Nilsson 1992).

One difficulty in genetic analyses of precocious maturation in salmonids is sorting out clear genetic from non-genetic maternal effects. For example, egg sire can affect early growth of offspring, which in turn may play a role in precocious maturation (Silverstein and Hershberger 1992). A maternal effect on early growth could have an impact on estimates of the dam component of observed variance in maturation timing and must be considered in the analyses (Bradford and Peterman 1987).

Breeding experiments have identified both a maternal and paternal genetic component to precocious male maturation. An effect of sire age has been found in chinook salmon (Heath 1992), coho salmon (Iwamoto et al. 1984, Silverstein and Hershberger 1992), and Atlantic salmon (Thorpe and Morgan 1980; Glebe and Saunders 1986; Thorpe et al. 1983; Gjerde 1984a, b). Maternal genetic effects on precocious maturation have also been found (Heath 1992, Silverstein and Hershberger 1992).
In addition, significant genotype-by-environment interactions for temperature and growth have been found in a number of salmonids (McKay et al. 1984, Iwamoto et al. 1984, Heath 1992, Nilsson 1992). In other words, the maturation response to growth acceleration in early life is not equivalent in all genetic groups or genotypes.

Clearly, genetic effects on maturation may be linked to genetic effects on growth (Thorpeand Morgan 1978; Purdom 1979; Thorpe et al. 1983, 1984; Crandell and Gall 1993a, b). Thorpe et al. (1983, 1984) have shown that fast growth rate and early maturation were genetically linked to developmental rates. In a later study, Thorpe (1986, 1994) proposed that maturation depended on a genetically determined rate of development that must be exceeded during a specific time of year.

Growth and Age at Maturity

In fish, different populations of the same species may show a wide range in mean age of maturity. There is also variation within the same population that suggests that age at first maturation in fish is probably linked to environmental effects on individual growth rates. Each species may need to reach a certain size or body condition before successful sexual maturation and breeding can take place (Policansky 1983), and these thresholds may be partially determined by environmental conditions.

Within populations of salmonids, there is a high degree of variation in age of maturity, ranging from 1 to 7 years. It is generally thought that males are able to mature at an earlier age than females because the energetic "cost" of producing sperm is very low compared with that of producing viable eggs (Wootton 1985). Thorpe (1994) suggested that the high degree of phenotypic plasticity in age of maturity in salmonids is most likely an adaptation to the variable productivity of the freshwater environment, which the fish occupy during early life-history stages. Furthermore, he has proposed that timing of maturation is influenced by growth opportunity at critical life-history stages.

It is well established that age at fist maturity in salmonids is strongly dependent on size and growth. Ahn (1959) was the first to show that in a population of brown trout (*Salmo trutta*), fish which grew fastest matured earliest. Similarly, Lamont (1990) used individually tagged rainbow trout and coho salmon to show that the fastest growing individuals were early maturing males. Several studies have shown that maturing male parr are usually larger than nonmaturing siblings (Naevdal et al. 1978b, Bailey et al. 1980, Thorpe et al. 1983, Rowe and Thorpe, 1990a, Heath 1992).

Furthermore, age at first maturity in salmonids is negatively correlated with growth rate, or in other words, early maturation is positively correlated with growth rate (Kato 1975, 1978; Gardner 1976; Hagar and Noble 1976; Glebe et al. 1978; Naevdal et al. 1978b, 1979; Bilton et al. 1981; Lundqvist 1980; Hunt et al. 1982; Thorpe et al. 1983; McCormick and Naiman 1984; Gjedrem 1985; Thorpe 1986; Rowe and Thorpe 1990a; Bergland 1992; Clarke and Blackburn 1994).

However, the correlations of early male maturation to growth rate are not maintained throughout gonadal development. Several studies have shown that during later stages of gonadal growth the specific growth of maturing males decreases (Thorpe and Morgan 1980; Saunders et al. 1982; Thorpe et al. 1983; Glebe and Saunders 1986; Rowe and Thorpe 1990a, b, Foote et al. 1991). This is consistent with the generally accepted idea that as fish mature, a reduction in somatic growth occurs due to allocation of energy to gonadal development (Ware 1980, Roff 1983). In contrast to this model, a reduction in somatic growth was not observed in in one study of early maturing male chinook salmon (Heath 1992).

A number of studies have indicated that the rate of early male maturation can be modified by the hatchery rearing environment, primarily by factors that affect growth opportunity (Saunders and Henderson 1965, Schmidt and House 1979, Bilton et al. 1981, Saunders 1986, Saunders et al. 1982, Taylor 1990, Rowe and Thorpe 1990b, Thorpe et al. 1990, Bergland 1991, Herbinger and Friars 1992, Clark and Blackburn 1994).
Bilton et al. (1981) demonstrated that acceleration of growth in chinook salmon fry with elevated water temperature also increased the incidence of early male maturity. In Atlantic salmon, Bailey et al. (1980). Saunders et al. (1982) increased the number of mates maturing at 0+ age by increasing growth during the winter months with elevated water temperatures. Similar results of elevated termperamre on precocious maturation in Atlantic salmon were found by Bergland et al. (1991) and Herbinger and Friars (1992).

Rowe and Thorpe (1990b) found that in Atlantic salmon, reduced feeding opportunity during the spring prior to maturation suppressed early male maturation, whereas increased feeding at this time increased the incidence of male maturation. A similar effect of spring feeding opportunity on grilsing in Atlantic salmon held in seawater cages has been reported (Thorpe et al. 1990). In chinook salmon, Clarke and Blackbum (1994) increased the proportion of sexually maturing yearling fish by increasing ration from November through June. They also noted that males destined to mature as yearlings grew more rapidly as underyearlings than their immature cohorts, and thus concluded that sexual maturation is conditional to and facilitated by rapid growth.

These relationships between maturation and size have led to several hypotheses to explain the role of growth in the initiation of sexual maturation. Early work led to the proposal that there was a minimum threshold of size for maturation of male parr (Elson 1957, Refstie et al. 1977, Bailey et al. 1980, Myers et al. 1986) and that this size threshold' was higher for maturation than for smoltification (Thorpe et al. 1980, Saunders et al. 1982, Thorpe et al. 1987). Myers (1984) and Myers et al. (1986) found that the proportion of mature male parr over a 5-year period was correlated with growth to a size threshold of 70-72 mm in Atlantic salmon.

Later, it was recognized that growth opportunity at critical seasonal periods was important for initiation of maturation. Thorpe (1986) proposed that maturation is initiated if a set-point in growth rate is exceeded at a particular time of year. Because maturation in salmon is initiated under increasing day lengths (Scott 1990, Adams and Thorpe 1989), Thorpe proposed that the initiation of maturation depended on growth performance during the winter or spring. Rowe and Thorpe (1990b) could reduce the proportion of maturing male parr by reducing feeding and growth during the spring months. Although maturing male parr tended to be larger than nonmaturing siblings in the winter, no relationship between monthly specific growth rates and maturation could be established in this study. However, maturing parr showed greater increases in condition factor than nonmaturing fish during the spring.

This led to another hypothesis: that levels of stored energy reserves in the spring, rather than body size, were involved in the physiological initiation of maturation (Herbinger and Newkirk 1990, Rowe and Thorpe 1990b, Rowe et al. 1991, Simpson 1992). In salmonids, mesenteric fat is the major energy store (Henderson and Sargent 1981). A study by Rowe et al. (1991) demonstrated that levels of mesenteric fat in May, prior to maturation, are higher in precocious Atlantic salmon male parr than in their nonmaturing siblings. These investigators proposed a model describing a potential mechanism for the effect of fat on maturation.

In this model, mesenteric fat stores act as sites for aromatization of androgens to estrogens, which in turn trigger maturation through stimulation of pituitary gonadotropin synthesis. However, no further endocrine studies have been reported to support this model. It is likely that the endocrine mechanism involved in initiation of maturation is far more complex than proposed in this model and probably involves metabolic hormones such as insulin.

More recently, Thorpe (1994) has suggested that during the life-cycle of salmon, there is an annual opportunity for sexual maturity and that during critical periods of this annual cycle, growth opportunity acts as a gate permitting maturation to be initiated. In Thorpe's model, the maturation process is initiated by the time of fist feeding, and takes priority over somatic growth: completion of maturation is environmentally dependent and can be arrested annually.

Whether or not maturation is arrested depends on the status of energy stores of the individual at particular critical times of year. Thii model and data on the relationship between growth and maturation has several implications for the impact of feeding/growth regimes in captive culture on maturation timing.

In captive culture, the feeding opportunity for fish far exceeds that normally experienced in the wild. Food availablity is constant, not seasonally variable as in nature. Frequently hatchery managers rear juvenile fish to a specific size for release, but when during the season that size is achieved varies according to management practices. The ration is relatively constant and is generally adjusted according to rearing temperature and desired growth pattern. In fish, the rate of growth also affects the rate of development. Because growth in culture conditions frequently exceeds that in the wild, the developmental cycle is also accelerated. As a result, life-history transitions during seasonal periods that are abnormal for the stocks often occur. Thus, it is not surprising to find that hatchery-reared fish are substantially fatter than wild fish (Ludwig 1977, Shearer and Swanson, 1994) and that precocious male maturation rates as high as 80% occur in captive culture.

Future Research Needs on Early Male Maturation

In a captive broodstock program for depleted stocks of salmon, it is undesirable to produce mature males at a time when females of the same stock are not mature. In addition, selective mortality of precocious males could reduce the effective breeding population size of a captive broodstock. Thus, there is a critical need to develop methods to minimize precocious male maturation in captive broodstock programs for depleted salmon stocks. From the above review of literature, it is clear that the time of sexual maturation is controlled by genetic, abiotic (e.g., photoperiod, temperature, salinity) and biotic (e.g., diet, growth rate, energy stores) factors. The relative importance of these factors and how they interact are poorly understood. Because artificial genetic selection should be minimized in a captive broodstock program for depleted stocks, rearing strategies that minimize expression of precocious male maturation should be developed.

Research to date, primarily from work on Atlantic salmon, indicates that growth rate, size, and levels of stored energy at specific times of year or critical periods of the life cycle, are important factors affecting the incidence of precocious maturation. It may be possible to reduce levels of precocious male maturation through alterations in rearing conditions, growth rates, and diet. However, it is not known whether all of the results from studies of Atlantic salmon are applicable to Pacific salmon species such as chinook salmon.

Before methods that minimize the rate of precocious male maturation can be developed, research is necessary to determine how stored energy levels (body fat content), growth rates, or rates of energy deposition at critical developmental stages either permit or prevent the onset of maturation.

It will be necessary to determine the relative roles of growth rate and stored energy levels, as well as interactive effects of these factors, on precocious maturation. Diets and growth regimes that sustain somatic growth and provide sufficient stored energy for appropriate life-cycle transitions must be developed: in other words, rearing methods that minimize precocious maturation in a population but do not affect the quality of smolts are needed.

Factors Affecting Gamete Quality

Successful fertilization of eggs and subsequent development of offspring depend greatly on the quality of gametes produced by the parent fish. Various biological and nonbiological factors have been implicated as determinants of gamete quality and subsequent survival of progeny. These include: composition and size of the egg, quality of the sperm, genetic makeup and nutritional status of the parents, husbandry procedures, and quality of the water supply (Bromage and Cumaranatunga 1988; Bromage et al. 1992; Springate and Bromage 1983, 1984a, b, c, 1985a, b; Springate et al. 1984; Springate 1985).

In many instances it is difficult to separate the relative effects of these parameters because they are frequently interrelated. For example, the chemical composition of the egg is affected by nutrition of the female (diet composition and quantity, Forster and Hardy 1995), genetics of the fish, and water quality. Environmental factors and husbandry practices that affect egg size, fecundity, and egg composition frequently also affect intake of food (see discussion below on stress), therefore is it difficult to determine cause-effect relationships among these variables.

Composition and Size of Eggs

The composition, size, and number of eggs produced by an individual female are influenced strongly by nutrition and are reviewed by Forster and Hardy (1995). There have been a considerable number of studies conducted on the effects of dietary constituents on egg quality, and the relationship between chemical composition of the egg and egg qualtiy in rainbow trout. However, Bromage and Cumaranatunga (1988) concluded that 1) there is no clear relationship between any single component of the egg and egg quality, and 2) profound effects of dietary constituents on egg quality are observed only when specific vitamins or minerals are absent or present at very low levels. Much of the data suggesting that egg size affects egg quality are inconclusive because experiments were not controlled for ripeness of the egg. For example, when eggs were collected within 1 week of ovulation and fry were given proper nutrition and water quality, no relationship between egg quality and egg size was observed (Springate and Bromage 1985a).

Timing of Egg Collection and Husbandry Practices

Fish husbandry practices can have dramatic effects on egg quality. Probably the most profound effects are those that result from the timing of egg collection (stripping) and handling of the gametes (Craik and Harvey 1984, Springate et al. 1984, Springate 1985, Springate and Bromage 1985a, Bromage and Cumaranatunga 1988, Bromage et al. 1992).

The time of stripping relative to ovulation can have marked effects on fertilization rates and survival of developing embryos (Nomura et al. 1974, Sakai et al. 1975, Escaffre and Billard 1979, Springate et al 1984). In eggs stripped too soon after ovulation, a modest reduction in fertilization has been observed (Sakai et al. 1975, Springate et al. 1984, Springate and Bromage 1984b), and this may be due to excessive force used during stripping or incomplete ripening of the eggs. In addition, overripe eggs have very low rates of fertilization and subsequent survival (Sakai et al. 1975, Springate et al. 1984). Eggs collected more than 10 days after ovulation in rainbow trout exhibited reduced viability. Viabilities of over 70% were observed when eggs were stripped within 10 days of ovulation, whereas when eggs were collected 30 days after ovulation 0% viability was observed (Nomura et al. 1974, Sakai et al. 1975).

Overripeness of the egg is characterized by the aggregation and fusion of oil droplets and migration of the cortical alveoli (Nomura et al. 1974). It is not known exactly how these morphological changes are related to specific chemical changes in the egg and reduction in viability of the egg and fry. Whatever the cause of reduced viability when eggs remain in the body cavity for an extended period after ovulation, overripeness of the egg remains a major cause of egg loss in fiih culture (Lam et al. 1978, Bromage et al. 1992).

The timing of egg collection has been most precisely determined for rainbow trout reared at 10 °C (Springate and Bromage 1984a, b; Springate et al., 1984; Bromage and Cumaranatunga 1988; Bromage et al. 1992). In studies conducted under these conditions, eggs that were collected between 4 and 10 days after ovulation exhibited high rates of fertilization. Thus, checking female rainbow trout for "ripeness" every 7 to 10 days was recommended by Bromage et al. (1992). They also recommended that sorting and stripping of ripe fish should be carried out under anesthesia to avoid stressing broodstock and damaging eggs, and that recovery of fish that are not spawned should be done with good water flow or auxilliary aeration. When administered properly, anesthetics have no effect on the quality of gametes (Billard 1981). Unfortunately the rate of ripening may vary with rearing temperature, and differences among salmonid species as well as stocks within a species may exist.

Methods of collection and handling of gametes also affect gamete quality. General handling procedures have been described in several texts on fish farming (Leitritz and Lewis 1976, Piper et al. 1982, Springate and Bromage, 1985b). Batches of broken or overripe eggs, as well as eggs that have poor fertilization or survival 24 hours post-fertilization, should be discarded so that contamination of good eggs is avoided. Wilcox et al. (1984) demonstrated that broken eggs caused poor survival of embryos in coho salmon. Generally, fertilization rate and survival during the first few days of incubation is a good predictor of subsequent survival to hatch (Springate and Bromage 1983, 1984 a, b, c; Craik and Harvey 1984).

Effects of Stress on the Quality of Gametes

Studies in rainbow trout have shown that acute and chronic stress have suppressive effects on the reproductive endocrine system. Both types of stress reduced plasma testosterone and gonadotropin levels in males, and reduced plasma sex steroids, vitellogenin, and gonadotropin in females (Billard and Gillet 1981, Pickering et al. 1987). During both acute and chronic stress, plasma levels of cortisol increased to a point that impaired reproductive function in trout (Carragher et al. 1989, Carragher and Sumpter 1990). A thorough study by Campbell et al. (1992) evaluated the effects of repeated acute stress on gamete quality and quantity in rainbow trout. The stress consisted of exposure to a brief period of emersion approximately once per week at random intervals during a 9-month period. This stress delayed ovulation, reduced egg size and sperm count, and lowered survival rates of progeny of stressed fish. However, it had no effect on fertilization rates or somatic growth in the adults. Mortality of offspring was highest from fertilization up to hatch, but also persisted through 28 days post-hatch.

Other studies have examined the effects of stress on oogenesis and spermatogenesis using fish exposed to sublethal levels of pollutants or low pH. Exposure of female fiih to acid stress has been shown to delay ovulation and reduce fecundity, egg quality, and survival of progeny (Tam and Payson 1986, Mount et al. 1988, Weiner et al. 1986). Negative effects of low environmental pH on the reproductive system of male fish have also been observed @aye and Glebe 1984). However, one of the problems in interpreting results of these studies is that acid stress also reduces food intake and growth (Tam et al. 1987, 1990). This would explain the reduction in egg and body size in acid-stressed fish, since reduction in food intake suppresses gametogenesis and reduces fecundity (Love 1980, Billard and Gillet 1981). Thus when stress impairs growth, it is not possible to distinguish between the direct effects of stress on reproduction and the indirect effects, which are due to nutritional factors.

The effects of stress on reproduction, which have been described primarily in trout, may not be generalized to all sahnonids. Several investigators have demonstrated that there is considerable variation in the sensitivity of different strains and species of trout to stress (Pickering et al. 1982, Refsie 1982). In addition, the stage of development and history of exposure to stress also affect the response. Mature or maturing trout show a substantially reduced response to stress compared to juvenile fish (Sumpter et al. 1987). It is also possible to acclimate trout to regular periods of mild stress (Pickering and Pottinger 1985, Barton et al. 1987).

Most work on stress and reproduction in fish has been conducted on females, with little work carried out on the effects of stress on sperm quality. It has been shown that frequent stripping decreases sperm density and total sperm number, and reduces sperm motility (Billard 1992, Buyukhatipolglu and Holtz, 1984).

Unfortunately, it was not possible to determine whether the effects of stripping on sperm quality were due to the frequent handling stress or to a change in quality of the sperm released by the testis during the period of spermiation. One of the problems encountered in these studies has been the difficulty in assessing the quality of sperm.

Assessing the Quality of Sperm

At present there is no truly dependable criteria for estimating sperm quality. In fish, the length of time and intensity of spermatozoon motility (Temer 1986, Moccia and Munkittrick 1987, Billard and Cosson 1992), the percentage of motile spermatozoa (Levanduski and Cloud 1988), sperm density (Moccacia and Munkittrick 1987, Scott and Baynes 1987), and the chemical composition of seminal plasma (Hwang and Idler 1969, Morisawa 1988) are all factors that have been measured in an attempt to assess sperm quality. However, there is little strong evidence directly linking any of these factors with fertility.

In salmonids, greater length of time and intensity of motility are not consistently correlated with higher fertilizing ability (Billard 1988). Moccia and Munkittrick (1987) concluded that the critical factor for fertilization was the number of motile sperm, not the motility per se. It is widely agreed that functional tests of sperm quality, such as fertilizability, probably provide a more valuable means of assessing quality of the sperm. However, until more techniques are developed that allow evaluation of sperm quality in the field, hatchery managers will probably have to rely on counts of motile sperm.

Future Research Needs on Gamete Quality

Most studies on gamete quality in sahnonids have been conducted on domesticated stocks of rainbow trout. Little is known about the factors affecting egg and sperm quality in wild stocks of Pacific salmon captive broodfish. It is likely that wild stocks of fish may be more stressed by the rearing environment and handling, thus the impact of stress on the quality of gametes may be more dramatic. Procedures which have been established for domesticated stocks of salmonids may not be applicable to wild stocks of fish. Thus, research is needed on the effects of rearing environment (water quality and rearing density) and on developing handling procedures that minimize stress in wild stocks of Pacific salmon. The effects of rearing temperature on maturation timing and gamete quality in captive broodstocks needs to be evaluated in a variety of Pacific salmon species, as does the impact of diet on the quality of gametes (Forster and Hardy 1995). In addition, better methods to accurately determine the quality of sperm in the field are needed.

Conclusion

The major problems with reproduction of Pacific salmon species in captivity include: inappropriate tuning of sexual maturation of broodstock, losses due to prespawning mortality, unreliable production of high quality gametes, and precocious maturation of male fish. Most studies to date on factors affecting gamete quality and the age and seasonal timing of sexual maturation have been conducted on domesticated stocks of rainbow trout and Atlantic salmon. Although some of the information generated from these species is applicable to Pacific salmon, further research is necessary to solve problems with poor reproductive performance of wild Pacific salmon stocks reared in captivity. There is an obvious need to develop methods to monitor and control sexual maturation in captive broodstock, to ensure production of high quality gametes and high survival of offspring, and to minimize asynchronous maturation of male and female fish.

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