Regulation of GtH secretion by GnRH and steroid hormones in male and female rainbow trout. An in vitro study

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REGULATION OF GtH SECRETION BY GnRH AND STEROID HORMONES IN MALE AND FEMALE RAINBOW TROUT. AN IN VITRO STUDY

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Male and female rainbow trout exhibit an annual sexual cycle characterized by variations of pituitary gonadotropin hormone (GtH) levels, of plasma GtH and steroid hormones levels and of pituitary responsivness to gonadotropin-releasing hormone (GnRH). For this latter parameter maximal values were recorded at the time of maturation - ovulation or prespermiation - spermiation (Weil, 1981).

In the present work, we test in vitro the change in gonadotroph sensitivity to GnRH and its modulation by steroid hormones during the reproductive cycle.

Primary cultures of whole pituitary maintained in standardized conditions (Weil et al., 1986) were used. At definite stages of gametogenesis, cells dispersed with collagenase were preincubated for 3 days in control medium or in medium containing the main steroids involved in oocyte maturation 17 αhydroxy 20ßdihydroprogesterone (17, 20-P) and spermiation 11-Ketotestosterone (11K-T) and 17,20-P. Cultures were then incubated with sGnRH during 24 hrs after which GtH released in the medium was measured.

Pituitary responsivness to sGnRH was studied in female at the beginning of vitellogenesis (BV), at the SPGV stage - corresponding to oocytes with subperipheral germinal vesicle-and the day of ovulation. Two doses of 17,20-P were tested : the first one corresponding to circulating levels at maturation (400 ng/ml), the second one to levels just prior maturation (20 ng/ml) when the germinal vesicle is in a peripheral position (PGV stage). In control cultures, pituitary responsivness to GnRH is maximal at the time of ovulation. This might partly be due to high in vivo circulating levels of 17,20-P since a pretreatment with this steroid (maturation dose) increases the GnRH-induced GtH release of pituitary cultures from females at the BV stage. At the SPGV stage, only the PGV dose induces an increase likely to explain SPGV to PGV stages rise in plasma GtH levels (Weil, 1981). At the time of ovulation, both doses of 17,20-P induce a decrease in pituitary sensitivity to GnRH. This might account for the decline in plasma GtH levels following the injection of 17,20-P (Jalabert et al., 1976).

Males were studied at the beginning of spermatogenesis, at spermiation and presper miation. 11K-T and 17,20-P were used at doses corresponding to spermiation circulating levels, 50 and 20 ng/ml respectively. In control cultures, pituitary sensitivity to GnRH increase from beginning of spermatogenesis to spermiation. This could be related to the in vivo rising levels of 17,20-P and 11K-T since a pretreatment with these hormones increases the GnRH-induced GtH release of beginning of spermatogenesis cultures. At spermiation, no effect of steroid pretreatment is recorded whereas during prespermiation a slight decrease is linked with the presence of 17,20-P. This latter observation might explain the slight decline in plasma GtH observed at onset of spermiation (Sanchez-Rodriguez et al., 1978).

In conclusion, we demonstrate that the variation of circulating GtH levels may partly be due to a direct action of gonadal steroid hormones on pituitary gonadotrophs by modulating their responsivness to GnRH.

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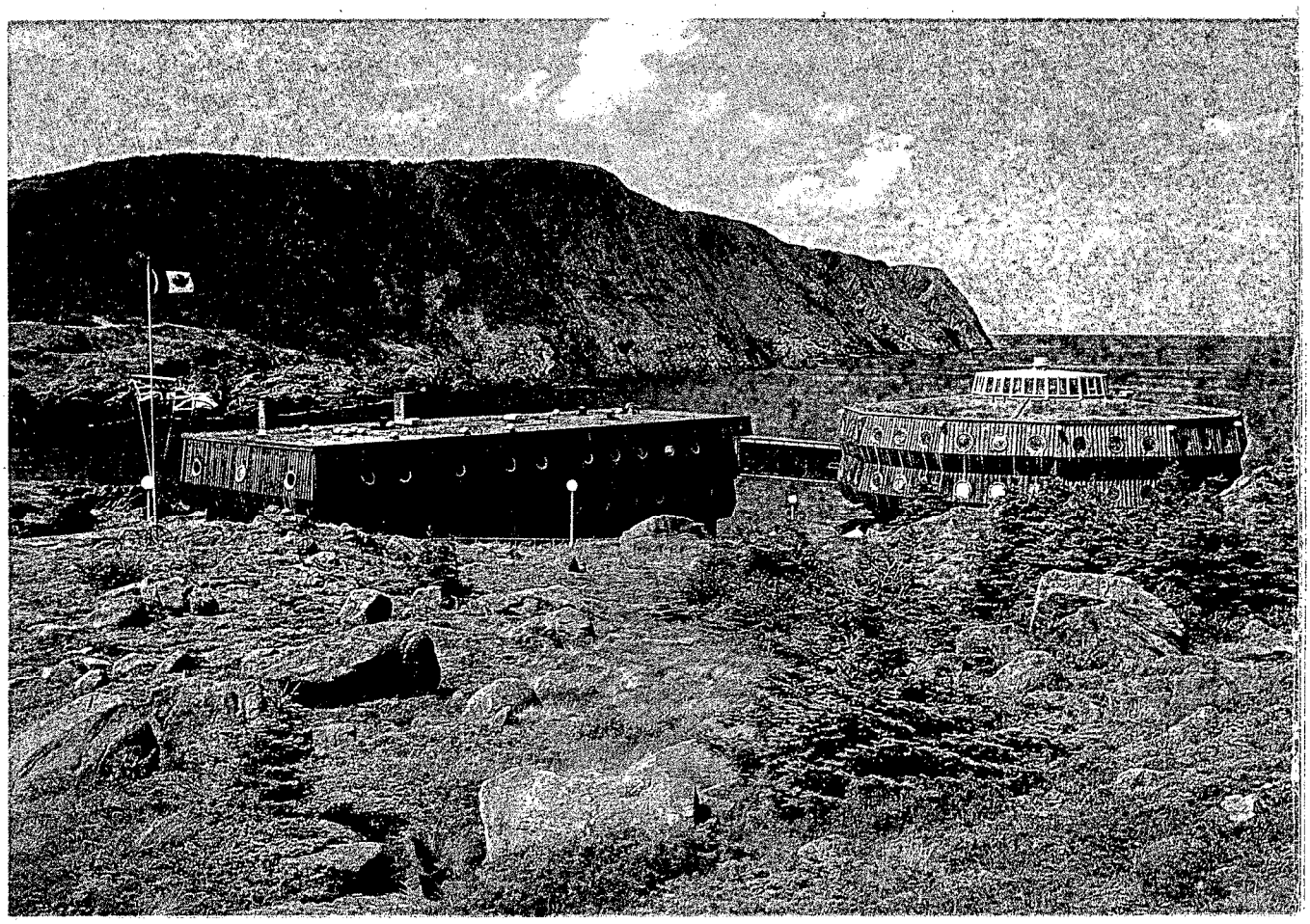

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