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Abstracts

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GROWTH HORMONE (GH) AND REPRODUCTION : a short review.

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Interaction between growth and reproduction occurs in many vertebrates and is particularly obvious at certain stages of the life cycle in fish. However the endocrine mechanisms involved in these phenomena have not been investigated to a great extent.

Gonadotropic axis interference with GH secretion: In mammals data support the modulation of GH secretion by sex steroids at the pituitary and at the hypothalamus levels. In fish, treatments with exogenous androgens have the most potent effect on overall growth and estradiol can stimulate the activity of somatotrophs. However the relationship between the natural changes in steroid levels and growth rate or GH secretion remains unclear.

In goldfish and probably in trout as well, gonadotropin releasing hormone also functions *in vivo* and *in vitro* as a growth hormone releasing factor, possibly through binding to specific sites on both gonadotrophs and somatotrophs.

In turn, elements of the somatotrophic axis may influence various events leading to gamete production: in this review the potential role of GH will be emphasised.

It has been known for many years that in mammals natural or experimentally induced GH deficiency during prepubertal development delays the onset of puberty and, in the adult, GH deficiency has been associated with infertility. These effects are linked to decreased gonadal receptivity to gonadotropins and they may be overcome by GH supplementation. In the last 2 to 3 years GH therapy has been studied as an adjuvant to gonadotropin treatment for ovulation induction. In hypophysectomised fish, *in vivo* administration of GH was able to augment spermatogonial proliferation and to enhance the testis maturation process obtained with gonadotropin treatment. In several species, GH plasma levels increase towards the end of the gametogenic cycle and in male salmonids high GH levels are associated with an elevated production of $17\alpha 20\beta\text{OHP}$, a progestin necessary for effective spawning.

Several studies have provided evidence for direct gonadal site of action for GH: In mammals GH enhances gonadotropin induced differentiation of cultured granulosa cells as measured by progesterone production and aromatase activity. It increases LH receptor content and IGF production of these cells. Few direct effects of GH on testicular tissue have yet been demonstrated. In fish GH is able to bind to specific high affinity receptors in the testis as well as in the ovary. GH affects *in vitro* androgen and estrogen secretion by testis or ovary fragments from hypophysectomised or intact fish, either directly or by potentiating the steroidogenic action of GTH. Using testicular cells or granulosa cells in culture we found that GH stimulates $17\alpha 20\beta\text{OHP}$ production during long term treatments.

GH action may directly affect the cells being regulated or may be mediated through the modification of IGF1 production and/or IGF1 binding in gonads. In mammals IGF1 influences steroidogenesis and gonadal somatic cell differentiation and division. In fish, hIGF1 and hIGF2 stimulate the proliferation rate of cultured trout spermatogonia, and hIGF1 increases the incorporation of ^3H -thymidine in premeiotic cysts from the dogfish testis. Human IGF1 binds to specific sites in carp ovary and stimulates $17\alpha 20\beta\text{OHP}$ production by trout granulosa cells in culture.