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► To cite this version:

Patrick Prunet, Pascale Le Goff, G. Salbert, J.F. Gonnard, Claudine Weil, et al.. Use of primary culture to study the control of prolactin secretion in rainbow trout: inhibitory effects of dopamine and GABA. 2. International Symposium on Fish Endocrinology, Jun 1992, Saint-Malo, France. 116 p. hal-02778388

HAL Id: hal-02778388

<https://hal.inrae.fr/hal-02778388v1>

Submitted on 4 Jun 2020

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**INTERNATIONAL
SYMPOSIUM
on FISH
ENDOCRINOLOGY**

Abstracts

PALAIS DU GRAND LARGE

SAINT-MALO

JUNE 1 - 4 1992



MINISTÈRE
DE LA RECHERCHE
ET DE LA TECHNOLOGIE



VILLE
DE
SAINT-MALO



USE OF PRIMARY CULTURE TO STUDY THE CONTROL OF PROLACTIN SECRETION IN RAINBOW TROUT: INHIBITORY EFFECTS OF DOPAMINE AND GABA.

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To study the control of Prolactin (PRL) secretion in rainbow trout, an in vitro technique using a monolayer cell culture system of pituitary glands was developed (WEIL et al. 1986). Study of the effects of dopamine (DA) on PRL cells indicated that a maximal inhibitory effect (52%) was observed after 36h treatment. Significant inhibition was obtained with doses between 10^{-5} and 10^{-6} M. Shorter period of incubation (<24h) did not induce significant change in PRL release. DA had also inhibitory effect on total PRL content. Specificity of this effect was confirmed using DA analogs (apomorphine and ergocryptine). Moreover, DA inhibition was reversed by a DA antagonist, sulpiride. Involvement of DA receptors of D2 type was confirmed by using D2 agonist (RU 24926 and SKF).

In the same system, effects of GABA was also studied: a significant inhibition of PRL release was observed when incubating 10^{-5} M GABA during 40h. Baclofen, a GABA agonist specific of type B receptors, induced also a significant decrease of PRL release when tested at 10^{-5} or 10^{-6} M. Short term inhibitory effect of GABA tested at 10^{-6} or 10^{-5} M could be also observed using perfused pituitary fragments.

The above results suggest that both DA and GABA are potent inhibitory factors acting directly on PRL cells. In order to test the possible modulation of these effects by estradiol-17 β , localization of estradiol receptor mRNA in the pituitary was carried out by in situ hybridization. No labelling was discernable over PRL cells whereas other pituitary parts were labelled. Grains counting corroborated this localization.

This indicate absence of possible direct modulation of GABA and DA effects by estradiol-17 β at the level of PRL cells.

Recent studies in rainbow trout indicated that PRL cells are under a dominant stimulatory control of hypothalamus (GONNET et al. 1989; YADA et al. 1991). DA and GABA would then appear to be potential PRL-inhibiting factors modulating a dominant stimulatory control by other hypothalamic factors.

GONNET et al. (1989), Fish Physiol. Biochem. 7, 301-308

WEIL et al. (1986), Gen. Comp. Endocrinol. 62, 202-209.

YADA et al. (1991), Endocrinology 129, 1183-1192.