Relationships between GTH1 and GTH2 pituitary responsiveness to GnRH stimulation, and GTH1 and GTH2 blood plasma levels at different stages of the reproductive cycle in the female rainbow trout

Bernard Breton, Marina Govoroun, Florence Le Gac, Jean-Marc Gomez, - Yusnaini

Hokkaido, JAPAN

3rd International Symposium on Fish Endocrinology

Abstracts

Hakodate Citizen's Hall

Hakodate, Hokkaido, JAPAN

May 27 - 31, 1996

RELATIONSHIPS BETWEEN GTH1 AND GTH2 PITUITARY RESPONSIVENESS TO GnRH STIMULATION, AND GTH1 AND GTH2 BLOOD PLASMA LEVELS AT DIFFERENT STAGES OF THE REPRODUCTIVE CYCLE IN THE FEMALE RAINBOW TROUT

B. Breton, M. Govoroun, F. Le Gac, J. M. Gomez and Yusnaini
INRA Laboratoire de Physiologie des Poissons, Campus de Beaulieu, 35042 Rennes Cedex, France

GTH1 and GTH2 have been purified from rainbow trout pituitary glands, using immobilized metal ion chromatography. After separation of their sub-units specific RIA have been developed using antibodies against thier β sub-units. GTH1 and GTH2 blood plasma levels have been measured during the gametogenesis, and every 2 days around the ovulatory period, after determination of the stage of maturation by egg stripping. Pituitary responsiveness had been determined after injection of 20 µg/kg of a salmon GnRH analogue alone, or in combination with pimozide : at the initiation of vitellogenesis (mid-April), the full vitellogenesis (end of July), the end of vitellogenesis, just prior to maturation and 15 days after ovulation. During gametogenesis GTH2 levels were undetectable, whereas GTH1 increased just at the initiation of vitellogenesis in March-April, reaching its maximum values at the begining of the phase of exponential growth of the oocyte. Then it decreased until the end of vitellogenesis. During the periovulatory period GTH2 increased just on the day of maturation, peaking at 20 to 30ng/ml. This GTH2 peak was preceded by a progressive elevation of GTH1 blood plasma levels, starting 8 days before maturation and peaking at the same time as GTH2. The frequency of sampling dose not allow to be sure that GTH1 and GTH2 peaks were synchronous. After ovulation there was a new increase of GTH1 blood plasma levels starting 8 days after and being maximum at day + 15. GTH2 levels increased as well, but later than GTH1. The weak cross-reactivity of GTH2 in the GTH1 assay (less than 6-8%) cannot explain the GTH1 levels reached after ovulation. The significance of these increases is not elucidated.

During the gametogenesis, the pituitary responsiveness for GTH1 secretion was maximum at the initiation of the gametogenesis, it remained high during the vitellogenesis, then it decreased until maturation, when it was not significant. The pituitary responsiveness to GnRHa for GTH1 secretion was partially inhibited by pimozide (50-70 %), only during the initiation of the vitellogenesis and at mid-July, may be indicating that dopamine could be necessary for the action of GnRH on the stimulation of GTH1 secretion, and to maintain high levels of GTH1. At the initiation of the vitellogenesis, GnRHa did not stimulate GTH2 secretion alone or in combination with pimozide. The responsiveness began to appear in July, being maximum just prior to maturation. Dopamine could be one of the main factor determining the differential secretion of GTH1 and GTH2 during the active gametogenesis and at maturation. After ovulation, although it decreased, the pituitary responsiveness for GTH2 secretion still existed, in agreement with the elevation of GTH2 plasma levels; whereas GTH1 cannot be stimulated by GnRHa, although there was an increase of its blood plasma levels. This could indicated that the control of GTH1 and GTH2 secretion would depend on different mechanisms and that other factors than GnRH would be involved, especially for the control of GTH1 secretion.