

International Meeting
ESTROGENS 2001

May 21-22, 2001

Accademia Militari, Modena (Italy)

ESTROGEN RECEPTORS AND ANDROGENS REGULATE EXPRESSION AND FUNCTIONAL ACTIVITY OF OXYTOCIN RECEPTOR IN EPIDIDYMITIS

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Sperm progression is regulated by androgen and estrogen hormones secreted by Leydig and Sertoli cells, respectively.

Protein expression of oxytocin receptor (OTR) in epididymis of adult mice was analyzed by immunoblotting.

After castration, OTR expression was significantly reduced, whereas administration of testosterone or estradiol restored OTR expression.

These findings suggest that androgen and estrogen hormones regulate OTR expression in epididymis.

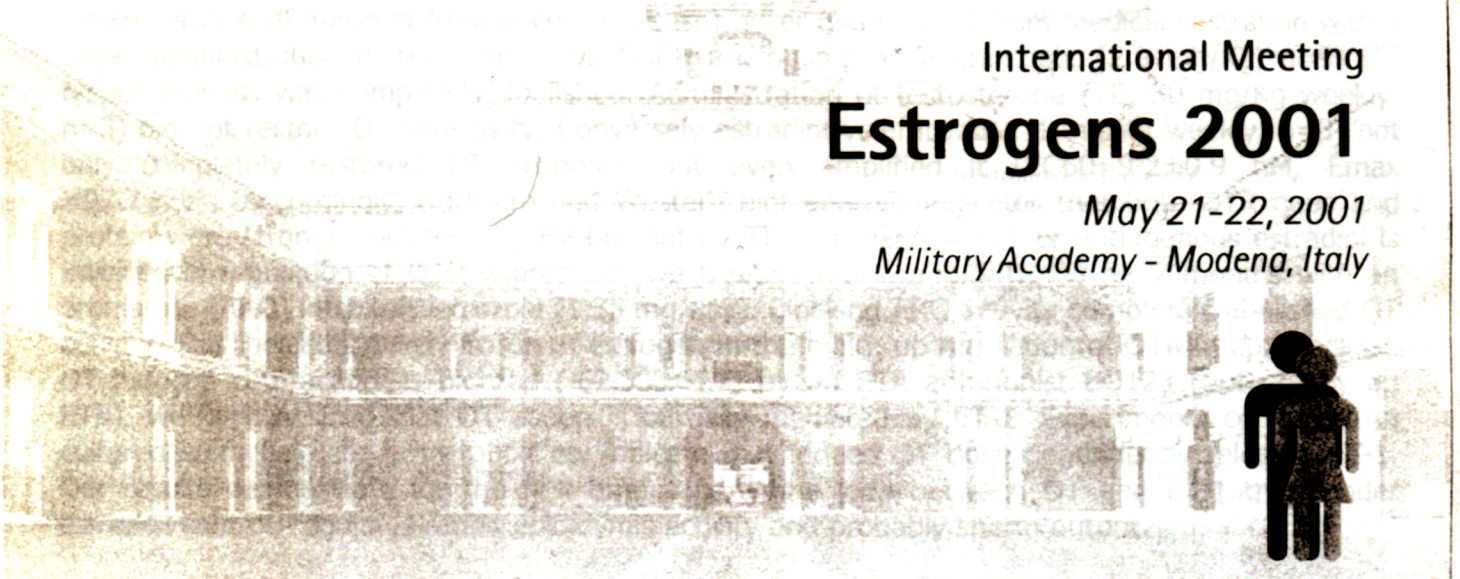
OTR expression is probably involved in the regulation of sperm progression.

Gastroenterology Unit - Endocrinology Unit
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STUDIES ON THE FUNCTIONS OF ER-BETA IN THE PROSTATE

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The finding of the second estrogen receptor (ER), gives us a new insight into understanding estrogen effects that were previously thought to be mediated through the estrogen receptor, ER α . In prostate, both ER α and ER β exist with ER β as the major ER in the stroma. ER β is probably a major component in the human prostate. ER β has been found in normal, benign, and malignant prostate glands and regulates

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2nd Session

ESTROGEN RECEPTORS AND AROMATASE GENES EXPRESSION IN MALES: HUMAN MODELS

Chairman: Ettore Degli Uberti

HUMAN MODELS OF ESTROGEN DEFICIENCY

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Until now, two different conditions have been identified as possible causes of congenital estrogen deficiency in humans: a) *estrogen resistance* caused by disruptive mutations of the estrogen receptor gene, and b) *aromatase deficiency* due to mutations of the gene encoding P450 aromatase, the enzyme which aromatize androgens to estrogens. Clinical findings in male congenital estrogen deficiency are: osteoporosis, tall stature with continuing linear growth into adulthood, unfused epiphyses, bilateral genu valgum, and an eunuchoid proportion of the skeleton. The effect of nine months of estrogen treatment in aromatase-deficient men resulted in both a complete epiphyseal closure and in an increase of bone mineral density, while a previous treatment with high doses of testosterone had no effect on skeletal maturation. Infertility was found only in one of the two patients described with *aromatase deficiency*, while semen analysis revealed a normal sperm density in the only case of *estrogen resistance*.

Already, functions in the male that were previously ascribed to androgens are now known to be the result of estrogen action; this is the case of epiphyseal closure, which stops long bone growth. Estrogen insensitivity has been considered a lethal condition as a consequence of the defective implantation of the blastocyst, until sporadic cases of congenital estrogen deficiency in humans were reported. Again, estrogens in the male may even be essential for fertility; in adulthood, in fact, ERKO male mice are infertile. Further studies will be necessary to assess the possible relationships between congenital estrogen deficiency and male infertility in humans.

Estradiol treatment is able to maintain both a normal bone mass and a physiological serum estradiol in adult men with aromatase deficiency. Thus, in clinical practice estrogen treatment should be continued lifelong in aromatase-deficient men since it could improve both survival and quality of life and LH, testosterone, and estradiol serum levels should be considered useful biochemical indexes of an adequate estrogen supplementation.

GENOMIC AND NON-GENOMIC EFFECTS OF ESTROGEN PRODUCED IN SITU IN BREAST TISSUE.

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Human breast tissue responds to estradiol with an increase in cell proliferation. In pre-menopausal women, the estradiol present in breast is synthesized both by the ovary and by adipose cells present in extra-glandular tissues. The estrogen formed in these locations is then delivered to the breast via the blood stream. In post-menopausal women, estradiol concentrations in breast tissue,

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