## A sex related difference in gonadotrophin response to castration in the rat

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Summary. Castration at birth, or at 25 days of age, increased plasma gonadotrophin levels in adult male and female rats. In the females, the effects of castration at 25 days of age or at birth were essentially the same. In the males castrated at 25 days of age, plasma gonadotrophin levels were less than for those castrated at birth. These results indicate a sex-related difference in the gonadotrophin response to castration.

Castration of male or female rats at birth<sup>1,2</sup>, at prepubertal ages<sup>3-5</sup> or as adults<sup>4,6</sup> raises LH and FSH plasma levels. Together, such results indicate a negative feedback control of testicular or ovarian steroids on gonadotrophin secretion, from the neonatal period onwards. Although such negative feedback effects have been extensively studied, no data are available to compare the effects of castration at different phases of development on the gonadotrophin function. The aim of the present study was to compare gonadotrophin responses to castration, either at birth or at 25 days of age, for a given sex.

Material and methods. Experimental subjects were male and female Wistar rats aged 60 days, born in the laboratory.



Figure 1. Plasma gonadotrophins in the 60-day-old female rat; in diestrus (D) or castrated, either at birth (CN) or at 25 days of age (C 25); (n).  $\Box$  LH;  $\boxtimes$  FSH.



Figure 2. Plasma gonadotrophins in the 60-day-old male rat; normal (N) or castrated, either at birth (CN) or at 25 days of age (C 25); (n).  $\Box$  LH;  $\boxtimes$  FSH.

They were housed under a schedule of 12 h light 12 h darkness (lights on 07.00 h), in a temperature-controlled room (23 °C) with food pellets and water available ad libitum. Litters were reduced to 8 pups at birth. Day of birth was defined as the 1st day of life; weaning was carried out at 21 days of age. Castration was performed at birth or at 25 days; controls were sham-operated. Subjects were isolated for 24 h in individual cages. They were decapitated between 09.00 and 10.00 h, under rearing conditions and in an adjacent room. Trunk blood was collected on heparin at 0 °C. Plasma was separated by centrifugation and stored at 20 °C until assayed for gonadotrophins. Plasma LH and FSH concentrations were measured by radioimmunoassay with NIAMDD rat LH and FSH kits; gonadotrophins were idodinated'. Duplicate plasma samples were measured for LH and FSH levels. Plasma levels were expressed in terms of ng equivalents of NIAMDD-LH-RP1 or NIAMDD-FSH-RP<sub>1</sub>/ml. Inter and intra-assay variations were 11.5 and 9.0% respectively for the LH assay and 8.0 and 9.0% respectively for FSH assay. The sensitivities of the assays were 2 ng LH/tube and 10 ng FSH/tube. The data were analyzed using Student's t-test.

Results and discussion. The results are given in figures 1 and 2. Castration at birth, or at 25 days of age, significantly increased plasma gonadotrophin levels in male and female rats at 60 days of age (p < 0.001). In the females, the effects of castration at 25 days of age or at birth were essentially the same (LH: 968±109 vs 1019±166 ng/ml, p > 0.05, n=8; FSH: 1146±73 vs 1124±89 ng/ml, p > 0.05, n=8). On the other hand, plasma gonadotrophin levels in male rats castrated at 25 days of age were significantly lower than those of animals castrated at birth (LH: 411±51 vs 852±79 ng/ml, p < 0.001, n=8; FSH: 861±42 vs 1088±61 ng/ml, p < 0.05, n=8).

The results indicate sex-related difference in the gonadotrophin response to castration. The presence of testicles (until 25 days of age), unlike that of ovaries, appears to prevent a high secretory level of LH and FSH.

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  Goldman, B.D., Grazia, Y.R., Kamberi, I.A., and Porter, J.C.,
- 2 Goldman, B.D., Grazia, Y.R., Kamberi, I.A., and Porter, J.C., Endocrinology 88 (1971) 771.
- 3 Meijs-Roelofš, H.M.A., and Kramer, P., J. Endocr. 81 (1979) 199.
- 4 Moger, W. H., J. Endocr. 67 (1975) 135.
- 5 Moger, W. H., Biol. Reprod. 14 (1976) 665.
- 6 Rabii, J., and Ganong, W.F., Neuroendocrinology 20 (1976) 270.
- 7 Campbell, C.S., Schwartz, N.B., and Gorski-Firlit, M., Endocrinology 101 (1977) 162.
- 8 Greenwood, F.C., Hunter, W.M., and Glover, J.S., Biochem. J. 89 (1963) 114.

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