

GENETIC REGULATION OF HEPATIC STEROID 16 $\alpha$ -HYDROXYLASE ACTIVITIES IN INBRED STRAINS OF MICE.

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By comparing the hepatic steroid 16 $\alpha$ -hydroxylase activity in rats and in various strains of mice (C57Bl/6J, DBA/2N, BALB/cAmN, C3H/Han, 129/J, AKR/J), we determined that:

1. The sexual differentiation of 16 $\alpha$ -hydroxylase takes place during puberty in all species but the female mice display higher enzymic activities than the males, which is contrary to results obtained from rat livers.
2. The steroid 16 $\alpha$ -hydroxylase present in the female mouse liver has a higher affinity for progesterone and testosterone, and a lower affinity for dehydroepiandrosterone and pregnenolone. Similar properties are observed for the female rat enzyme (Fr. Pasleau *et al*, Eur. J. Biochem. 120, 213, 1981). It is not possible to discriminate between the affinities of the male mouse 16 $\alpha$ -hydroxylase for the various steroid substrates.
3. In 129/J mice, the female steroid 16 $\alpha$ -hydroxylase activity is much lower than in the other strains and displays biochemical properties which are similar to those of the male enzymes; for example, the female 129/J enzyme has a higher affinity for pregnenolone and DHEA. The low level of steroid 16 $\alpha$ -hydroxylase observed in these female mice is inherited as an autosomal, codominant trait. These results partially contradict those published by H.C. Ford *et al* (Endocrinology 104, 857, 1979).