GENETIC REGULATION OF HEPATIC STEROID 16α-HYDROXYLASE ACTIVITIES IN INBRED STRAINS OF MICE.

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

1. The sexual differentiation of 16α-hydroxylase takes place during puberty in all species but the female mice display higher enzymatic activities than the males, which is contrary to results obtained from rat livers.

2. The steroid 16α-hydroxylase present in the female mouse liver has a higher affinity for progesterone and testosterone, and a lower affinity for dehydroepiandrosterone and pregnanetriol. 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 16α-hydroxylase for the various steroid substrates.

3. In 129/J mice, the female steroid 16α-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 pregnanetriol and DHEA. The low level of steroid 16α-hydroxylase observed in these female mice is inherited as an autosomal, dominant trait. These results partially contradict those published by H.C. Ford et al. (Endocrinology 104, 857, 1979).