PHYSIOLOGICAL AND GENETIC CONTROLS OF OVARIAN RESPONSIVENESS TO GONADOTROPINS IN MICE

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SUMMARY

An understanding of the physiological and genetic mechanisms which control ovulation rate should aid in the development of better selection criteria for improving litter size. We have identified six fold genetic variation in the ovarian responsiveness of mice to gonadotropins. The number of eggs ovulated by immature females in response to large doses of PMSG and hCG ranged from 8 $\pm$ 1 in strain A/J to 47 $\pm$ 2 in strain SJL/J and 53 $\pm$ 2 in strain C57Bl/6J. An analysis of inbred strains, and the F1 and F2 offspring of these strains revealed 1) that this trait had a high degree of genetic determination, 2) that while most of the genetic variation was additive, some loci showed dominance and complement one another, and 3) the low strain differed from the high strains by approximately three genetic loci. Genetic differences in the induction of LH receptors by PMSG tended to explain the increased hormone induced ovulation rate of C57Bl/6 over A but not of SJL over that of A. While strains SJL and A had similar numbers of LH receptors, SJL had a much higher hCG stimulated production of cAMP. This implies that at least part of the increased ovarian responsiveness of C57B16 is due to increased number of LH receptors, while that of SJL is due to a higher elevation of hormone-stimulated adenyl cyclase. The applicability of these and other physiological genetic techniques to engineering animal reproduction will be discussed.