

day of life revealed very similar dissociation constants (K_D) of the receptor-hormone complex in both groups, namely ranging between 4.95 and $5.12 \times 10^{-10} \text{ M}^{-1}$ in control rats and between 5.15 and $5.38 \times 10^{-10} \text{ M}^{-1}$ in adrenalectomized rats.

The decrease in ovarian gonadotrophin sensitivity after adrenalectomy can be restored by glucocorticoids^{10,11}. The question arises in which way glucocorticoids may influence ovarian gonadotrophin sensitivity; glucocorticoids have been found to modulate the action of various hormones onto their target cells^{21,22}. Recently Engel and Frowein²³ and Schmidtke et al.²⁴ presented evidence that in the rat testis this 'permissive effect' of the glucocorticoids is possibly due to an inhibitory effect onto the cyclic-AMP phosphodiesterase, which is engaged in the hydrolysis of cyclic-AMP. If glucocorticoids act in a similar way in the ovarian cells, the lack of glucocorticoids in adrenalectomized prepubertal rats may result in a lowered intracellular cyclic-AMP level and, in consequence of this, in a reduced gonadal steroidogenesis.

The interplay of the adrenals and the ovaries during female rat development is rather short⁹. Sexual maturation

is only impaired if the adrenalectomy is performed before the 25th day of age, but even in these rats vaginal opening and ovulation occur spontaneously, although delayed. Furthermore, adult adrenalectomized rats maintain ovulatory cycles and can reproduce³. If the glucocorticoids act in the ovarian cells by inhibition of cyclic-AMP phosphodiesterase, as suggested above, their action should be restricted with respect to developmental time. Cyclic-AMP phosphodiesterases have been found to exist in the pre- and postpubertal ovary in 2 molecular forms exhibiting distinct kinetic properties (J. Schmidtke, personal communication). The question is now under study of whether these molecular forms (isozymes) differ from each other in their inhibition by glucocorticoids before and after the 25th day of age.

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Behavioural consequences of vasectomy in the mouse

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Summary. Vasectomy was found to have no influence on the sexual activity of male mice. Testis and seminal vesicle weights were similarly not influenced by this operation although a significant increase in epididymus weight was observed.

In order to demonstrate the acceptability of vasectomy as a means of sterilization, the influence of this operation on long term sequelae such as auto-immune disease²⁻⁴ and granuloma development⁵ as well as sexual activity and libido must be established. While there has been no shortage of research on the physiological status of vasectomized animals^{6,6}, the behavioural changes induced by this treatment have been largely neglected despite their importance. Rodgers and Ziegler⁷ observed a slight increase in the frequency of human intercourse following vasectomy, although the interview and questionnaire procedure used in this, and similar clinical studies, may have been influenced by motivated distortions of recall and reporting by the subjects. Unfortunately, few attempts have been made to obtain more objective data with laboratory species. McGlynn and Erpino⁸ compared various aspects of coital behaviour in intact and vasectomized rats, but did not investigate coital frequency in these animals. In a clinical context, however, it is the frequency rather than

the normality of copulation which would seem the more valid criterion of sexual activity. A study was therefore designed to assess the influence of vasectomy on coital frequency in the mouse. The mouse was chosen for this experiment because the presence of a vaginal copulation plug in this species permits the ready identification of mated females.

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Table 1. Coital frequencies (No. of females covered in each 10-day-period) observed in sham-operated and vasectomized mice

Treatment	Duration of pre-operative test period (days)	Pre-operative coital frequency (\pm SE)	Duration of post-operative test period (days)	Post-operative coital frequency (\pm SE)	Number of mice
Experiment 1 Sham	23	5.61 ± 0.52	78	5.55 ± 0.31	10
Vasectomized	23	4.78 ± 0.52	78	5.61 ± 0.35	11
Experiment 2 Sham	51	6.58 ± 0.64	155	5.04 ± 0.73	9
Vasectomized	51	7.09 ± 0.62	155	5.11 ± 0.76	12

Table 2. Mean testis, epididymus and seminal vesicle weights in sham-operated and vasectomized mice

Treatment		Mean testis weight \pm SE (mg)	Mean seminal vesicle + coagulating gland weight \pm SE (mg)	Mean epididymus weight \pm SE (mg)
Experiment 1	Sham	103.44 \pm 5.25	371.33 \pm 13.82	44.02 \pm 1.49
	Vasectomized	94.55 \pm 3.78	340.80 \pm 11.81	*54.84 \pm 3.48
Experiment 2	Sham	102.39 \pm 4.31	314.09 \pm 28.22	38.67 \pm 1.17
	Vasectomized	98.16 \pm 4.79	313.75 \pm 31.26	*65.59 \pm 3.97

* $p < 0.01$.

Materials and methods. This study was performed using adult male mice of the random bred Q strain. Bilateral vasectomy was carried out under Nembutal anaesthesia using a ventral approach. The vas deferens was cut between single proximal and distal ligatures placed approximately 5 mm apart. In the sham operation the vas deferens was mobilized with a pair of forceps but not interrupted. The animals were rested for 2 weeks following the operation before the recommencement of mating trials. In order to record coital frequency the males were placed in separate cages together with 4 normal adult females of the same strain. These females were subsequently checked daily for the presence of vaginal plugs. As soon as an animal had been mated she was removed from the cage and replaced by another female. At the end of the experiment the males were killed by cervical dislocation and their testes, epididymes and seminal vesicles removed, cleaned and weighed. 2 experiments were performed which differed only in the duration of the pre- and post-operative periods. The significance of the results was assessed by the analysis of variance.

Results and discussion. The coital frequencies (number of females mated in each 10-day-period) observed in experiments 1 and 2 are presented in table 1. The pre- and post-operatives scores observed for the vasectomized animals and the post-operative scores recorded for the sham-operated and vasectomized groups were not significantly different from each other. Testis and seminal vesicle weights (table 2) were similarly not influenced by vasectomy, and no significant correlation was observed between testis weight and coital frequency. In contrast, epididymus weight showed a highly significant increase ($p < 0.01$) following vasectomy (table 2), apparently as the result of fluid accumulation.

The results obtained in these experiments indicate that male sexual activity is not influenced by vasectomy, thereby confirming the clinical data. The measurement of coital frequency would seem a particularly useful test of sexual activity in a number of other contexts, such as assessing the influence of anti-androgens, oestrogens or progesterones on male sexual function, and may provide more meaningful information than the detailed analysis of coital behaviour.

[Ovarian HCG-binding in hemicastrated immature female rats*

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Summary. Hemigonadectomy in 9-day-old female rats results in a drastic increase in the weight and the number of HCG-binding sites of the remaining ovary during further development. However, on a per mg basis of ovarian weight, the number of HCG-receptors is identical in hemicastrated and control rats.

In the adult rat after unilateral ovariectomy, the other ovary enlarges ('compensatory hypertrophy')³⁻⁵. Even after removal of one ovary and a half the remaining ovarian fragment has been found to hypertrophy to the weight of one ovary in control rats⁶. The most favoured hypothesis to explain compensatory hypertrophy is that the transient decrease in blood steroid levels from removal of one ovary reduces the negative feedback effect of the steroids and thereby triggers an increase in gonadotrophin secretion of the pituitary⁷⁻¹¹. This increase causes the remaining ovary to increase in size and secretory activity. Accordingly, oestradiol or oestriol treatment of the hemiovariectomized rat prevents the compensatory ovarian hypertrophy^{9,12}.

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