activity we observed between the 2 experimental groups might be due to a deficiency in thyroid hormone secretion. Furthermore, purified bovine adrenal DBH is directly inhibited by PTU[8]; the increase we find in the enzymatic activity obviously cannot be produced by a direct effect of PTU.

The evolution of adrenal DBH activity we found in the young control rats is similar to that described in previous reports[9,10]. Male and female rats were used in natural proportions, because no sex-related variations in DBH activity have been found.

The present results show that hypothyroidism accelerates the post-natal evolution of adrenal DBH activity. In the adult rat, some authors studied the influence of a decreased thyroid activity on DBH; thyroidectomy fails to modify DBH activity[11,12]. Comparing these different findings, it appears that adrenal DBH activity is more sensitive to hypothyroidism during development than in the adult. In the young rat, hyperthyroidism has been shown to slacken the evolution of adrenal DBH activity[10]; hypo- and hyperthyroidism appear to have opposite effects on the development of the enzymatic activity.

Finally, the present results concerning DBH activity have to be compared with our previous findings about the adrenal TH activity and CA content[1]; we found that neonatal hypothyroidism accelerates the increase in TH activity and in epinephrine and norepinephrine content. Thus, DBH responds to hypothyroidism just like TH. The consequent increase in CA biosynthesis could explain, at least partially, the increase in the CA content of the glands.

Age (days)	0	10		20		30	
		Cont	PTU	Cont	PTU	Cont	PTU
Body weight (g)	5.4 ±0.1	18.8 ±0.3	11.8 ±0.4 **	37.2 ±0.6	18.0 ±0.3 **	77.8 ±0.9	29.1 ±0.5 **
Adrenal weight (mg/pair)	3.09 ±0.04	3.57 ±0.03	3.19 ±0.12 *	10.54 ±0.62	6.48 ±0.31 **	16.18 ±0.81	8.42 ±0.42 **
Ratio: adrenal weight/body weight (mg/g)	0.57	0.18	0.27 a	0.28	0.36 a	0.21	0.29 a
Number of animals	26	17	22	20	18	18	28

Body weight and adrenal weight of young rats. Cont, control rats; PTU, hypothyroid rats. Means ± SEM. Statistical significance between the 2 groups: *$p \leq 0.05$; **$p \leq 0.01$. a, no statistical analysis.

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Oestrus induction in unisexually grouped mice by multiple short-term exposures to males[1]

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Summary. Oestrus induction and synchronization (the Whitten effect) were achieved in unisexually grouped female mice by short-term (10 and 30 min) exposure to conspecific males.

Female mice housed in large unisexual groups exhibit irregularities in the oestrous cycles[2]. Continuous exposure of such females to conspecific males for at least 48 h results in the synchronization of oestrus 3 days later[3]. An olfactory, androgen-dependent urinary pheromone is involved in this synchronization of oestrus (the Whitten effect[4,5]). In the present study the effect on oestrus induction of multiple short-term exposure of grouped females to males was evaluated.

Regularly cycling, 10–12-week-old virgin females of the Parkes (P) strain were divided into 5 unisexual groups of 30 each and housed in a male-free room for 28 days in cages, 48 × 34 × 11 cm. Females in groups I–III were exposed for varying periods of time (see table for protocol) to 3 confined (in an expanded metal corral) P males on days 29–35. The cages with the females were carried to another room for exposure to males and returned to the male-free room soon after exposure, remaining there until the next exposure. Females in group IV were taken to the room containing the males along with those in group II, but were not exposed to males and kept at a distance of 1.8 m from the cages housing the males. They were returned to the male-free room along with the females in group II. Controls (group V) remained in the male-free room throughout. Vaginal smears were daily examined from all females. The days of oestrus return (vaginal cornification) in the table refer to days 29–35 (7 days) in all groups. Data were analyzed by the χ^2-test.

The results are presented in the table. Unisexual grouping resulted in the incidence of prolonged cycles (10–16 days duration) in most females. Continuous (group I) or intermittent (groups II and III) exposure to males induced acceleration and synchronization of oestrus in females irrespective of the duration of the exposure. No significant difference in the percentage of oestrus return on day 3 between females in the 3 groups was found. Females in groups IV and V did not exhibit synchronized oestrus.

Even though the occurrence of all-female groups of *Mus musculus* in nature is doubtful[6,7], our results suggest that brief but frequent male-female encounters in the natural

Oestrus induction and synchronization in unisexually grouped female mice

Group and treatment	Number of females returning to oestrus within 7 days Days 1	2	3*	4	5	6	7	Percent of females returning to oestrus on day 3
I Continuous exposure to males for 7 days	1	6	14	2	2	1	1	46.6
II Intermittent (30 min) exposure to males, thrice daily, for 7 days	4	6	11	3	4	0	0	36.6
III Intermittent (10 min) exposure to males, thrice daily, for 7 days	4	4	14	0	2	0	1	46.6
IV Housed in a room containing males for 30 min, thrice daily, for 7 days	3	3	3	5	4	3	4	10.0
V Controls: housed in a male-free room	3	2	5	3	6	5	0	16.6

*Significance of differences: I vs V, $p < 0.001$; II vs V, $p < 0.001$; III vs V, $p < 0.001$; IV vs V, NS.

habitat may influence the oestrous cycle in mice. Implantation failure (the Bruce effect) in newly inseminated mice can be achieved by topical application of microquantities of male urine[8] or by multiple short-term exposures to males[9]. It appears that brief exposures of female mice to male odour is sufficient to trigger the chain of neuroendocrine events which culminate either in ovulation or implantation failure depending upon the physiological state of the female. These findings suggest that primer pheromonal control on oestrous cycle and implantation in wild populations of rodents cannot be ruled out.

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Radioisotopic determination of cerebrospinal fluid (CSF) folic acid and vitamin B_{12} in neurological disorders

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Summary. In a total of 55 samples of cerebrospinal fluid (CSF) and an equal number of serum samples obtained from 45 patients with neurological disorders and 10 controls, folic acid and vitamin B_{12} were measured. A radioisotopic assay method was used. A significant decrease of CSF folic acid was noted in the group with cerebral tumors.

The determination of cerebrospinal fluid (CSF) levels of folic acid and vitamin B_{12} in neurological disorders has been thoroughly studied. The purpose of this paper is to report our results on the concentrations of folic acid and vitamin B_{12} in the CSF of patients suffering from meningitis of different types, cerebral tumors and demyelinating diseases.

Materials and methods. In a total of 55 CSF samples obtained from the same number of patients after lumbar puncture performed for diagnostic purposes, folic acid and vitamin B_{12} were measured. 19 out of the 55 samples were from patients with meningitis, 10 with a cerebral tumor, 7 with demyelinating disease and 19 were from normal controls. Serum folic acid and vitamin B_{12} were also determined. All samples were stored at $-20\,^{\circ}C$ in small aliquots until analysis. Folic acid in CSF and serum was estimated by a radioisotopic assay[2]. Tritiated methyl-tetrahydrofolic acid as a label, pig plasma as a specific binding agent in a saturation analysis and dextran coated charcoal for separating the free from the bound fractions of folic acid were used. All samples (after separation) were counted in a 'Packard' liquid scintillation counter using 'Insta-Gel' as a scintillation fluid. The counting rate of the samples containing hemolysates was corrected for quenching due to color, by the method of internal standardization. The results are expressed in ng/ml of serum and CSF.

Vitamin B_{12} in CSF and serum was measured by a radioisotopic method based on saturation analysis[3]. Radioactive ^{57}Co-vitamin B_{12} of high specific activity obtained from Amersham was used as label, human serum as a source of vitamin B_{12} binders, and a Sephadex G-25 medium column for separation of the free and the bound portions. The samples were then counted in a 'Nuclear Chicago' well type scintillation counter. The results are expressed in pg/ml of serum and CSF. Estimation of both vitamins was performed in duplicate in each case. The statistical analysis was performed using Student's t-test.

Results. As can be seen in the table, the CSF folic acid levels in the groups with meningitis and demyelinating diseases were above 18.5 ng/ml, as was also observed in the control group. In the group with cerebral tumor the mean value $\pm$SEM was 11.2 ± 2.2 ng/ml with a statistically significant difference ($p < 0.001$) in comparison to the control group. In all the above mentioned groups, serum