Groups of rats were then assigned a behavioral score by giving each behavioral category 1 point in which the animal fulfilled minimal criteria. A score of 5 was considered fully maternal (postpartum rats).

Data were statistically analyzed by the Dunn and Mann Whitney U-test for nonparametric data and a probability of $p < 0.05$ was accepted as statistically significant.

As it can be seen in the figure, maternal behavior is significantly diminished in androgenized as compared to oil treated females. These masculinized rats showed a maternal score which was similar to normal male rats. Interestingly, neonatal androgenization caused a dramatic cannibalism, ($p \ll 0.005$) which was not observed in either of the other groups (table).

These results show that the capacity to manifest maternal behavior towards foster pups can be affected by the process of sexual differentiation induced by androgens. These facts indicate that in the normal rat the maturation of the different mechanisms implicated in the control of maternal behavior take place during a critical period which extends between days 1 and 10 after birth.

The administration of a single dose of testosterone to female rats after birth produces an anovulatory syndrome with persistent oestrus[5], lack of lordosis[6] and stimulation of male components of behavior[7]. From our results, it can be inferred that a true sexual differentiation of the brain has occurred, since the androgenized female behaved in a rather similar way to the normal male rat. These results are in agreement with some recent findings of Döhler and Hancke[7] which proposed that different amounts of oestradiol might bring about functional and structural changes of the brain which would lead either to female or male differentiation.

On the other hand, masculinization caused a cannibalistic behavior which was absent in normal male rats and in oil treated females. There is evidence that neonatal androgenization influences intraspecific aggression in rats[8] and in male and female mice[9-11]. However, further studies on the mechanism involved in the masculinization of the brain are necessary in order to understand the modification of the maternal behavior of androgenized female rats.

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Calcemic responses of Stannius corpuscle extract in parrots *Psittacula psittacula*[1]

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Summary. In parrots *(Psittacula psittacula)*, i.p. injection of Stannius corpuscle extract (10 mg/ml/100 g b.wt) evokes hypocalcemia at 1 h. Thereafter, the values indicate hypercalcemia at 6 h and normocalcemia at 10 h.

The corpuscles of Stannius (CS) are located on, or in, the kidneys of the holostean and teleostean fishes[2]. Stanniectomy in teleosts leads to hypercalcemia[3-5] which can be corrected either by homotransplantation of CS[6,7] or by injections of corpuscular extracts[3,7]. It is now generally accepted that CS produce hypocalcemic principles called hypocalcin[8] and teleocalcin[9].

Conflicting observations exist regarding the effects of CS extract administration in mammals. Both hypocalcemia[10] and hypercalcemia[11] have been reported after CS extract administration. Pang and Copp (both cited by Leung and Fenwick[10]) failed to observe any hypocalcemic action in rats after CS extract administration. So far, there is no record of the effects of CS extract on the serum calcium level of birds. The present work is a first report on such a study in the parrot, *Psittacula psittacula.*

Material and methods. The CS used in this study were surgically removed from both sexes of an adult freshwater mud eel, *Amphipnous cuchia.* These glands were stored in ice and used almost immediately. The glands were weighed and homogenized in ice-cold saline (0.9% sodium chloride solution). The homogenate was centrifuged (5000 rev/min for 10 min) and the supernatant was collected. The final volume of the supernatant was made up so that 1 ml of the solution contained the extract from 10 mg of wet CS. 72 parrots *(Psittacula psittacula)*, weighing from 90 to 110 g, were maintained on paddy under laboratory conditions for 2 weeks prior to use. They were then divided into 2 numerically equal groups a) saline-injected (control); and b) CS extract-injected (experimental).

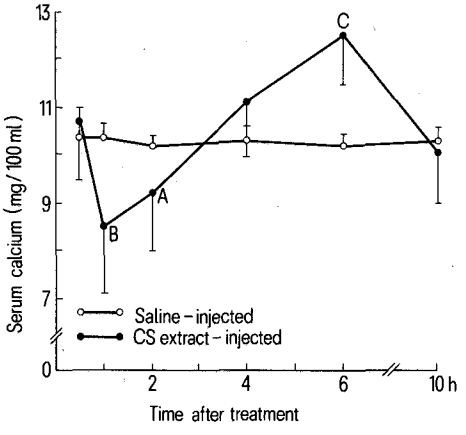
The experimental specimens were injected i.p. with CS extract in a dosage of 10 mg/ml/100 g b.wt. The control specimens were injected i.p. with 1 ml/100 g b.wt of saline. Blood samples from both the groups were collected by cardiac puncture after 0.5, 1, 2, 4, 6 and 10 h following the injection. The concentration of calcium was measured in the serum by Trinder's[12] method. Calcium from a 0.2 ml sample of serum was precipitated as an insoluble orange-red complex by an alkaline solution of naphthalhydroxamic acid. After centrifugation the precipitate was dissolved in alkaline disodium ethylenediamine tetraacetate, then treated with ferric nitrate and the resultant amber color was measured colorimetrically. Calcium concentration has been expressed as mg/100 ml serum.

Differences between parrots injected with saline and with CS extract were evaluated using Student's t-test.

Results and discussion. The serum calcium level is unaffected at 0.5 h after the injection of CS extract. At 1 h following

the injection, the calcium level exhibits a significant decrease ($p < 0.01$). Thereafter, it gradually increases resulting in a significant hypercalcemia at 6 h ($p < 0.001$). At 10 h, the calcium level has returned to control values (fig.). The present observation clearly indicates that the extract from the CS removed from *A. cuchia* contained a calcium-lowering factor (s). The hypocalcemic effect of CS extract has been reported earlier in eels[3], *Fundulus heteroclitus*[8] and rats[10]. The results reported from rats are surprising as one would not normally expect a hormone, extracted from an organ present only in bony fishes, to be effective in mammals. Leung and Fenwick's[10] report and also the present study lead to the inference that the hypocalcemic factor (s) present in CS is universally effective in vertebrates. This is further strengthened by the hypocalcemic effect of CS extracts in anurans (*Rana cyanophylctis* and *Bufo andersonii*, unpublished data of our laboratory).

In the present study the hypercalcemia at 6 h may be due to the activity of the parathyroid glands in response to the CS extract-induced hypocalcemia.



Changes in the serum calcium level after administration of saline and CS extract. The blood samples were collected at 0.5, 1, 2, 4, 6 and 10 h following the injection. Each point indicates mean ± SD of 6 determinations. A, B and C represent significant responses, $p < 0.05$, $p < 0.01$ and $p < 0.001$, respectively.

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Thyroid gland influences the period of hamster circadian oscillations[1]

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Summary. The locomotor activity of ovariectomized, adult golden hamsters was monitored under constant dim illumination. Thiourea treatment lengthened the period (τ) of locomotor activity of thyroidectomized hamsters in comparison to pre-operative τs and in comparison to the τs of control hamsters. The results confirm hormonal modulation of the circadian system.

The free-running period (τ) of mammalian circadian oscillations is remarkably stable and unresponsive to all but a few chemical manipulations[2]. Removal of various endocrine organs (gonads, pituitary, thyroid, pancreas, pineal and adrenals) of rats does not eliminate free-running activity rhythms, thereby demonstrating that the driving circadian oscillator for this rhythm is not localized in any of these glands[3]. However endocrinological manipulation (castration of male mice[4] and female hamsters[5,6], hypophysectomy[7]) lengthens τ; replacement testosterone[4] and estradiol[5,6] shortens τ. In the canary[8], radiothyroidectomy shortens τ and replacement thyroid hormone lengthens τ. These studies suggest that hormones can modulate the frequency of circadian oscillations.

The present experiment extended this analysis to an assessment of the influence of the thyroid gland on circadian activity rhythms of hamsters. Specifically, we tested the hypothesis that reduction of thyroid activity (accomplished via surgical thyroidectomy plus administration of a thyroid suppressing agent) would lengthen the period of circadian oscillations. Lengthening of circadian activity cycles was predicted on the basis of the general hypometabolic state induced by hypothyroidism.

Methods. 20 adult, ovariectomized golden hamsters bred in our laboratory from stock animals (LVG-LAK) obtained from Lakeview Hamster Colony (Newfield, NJ) were used. Prior to the study, animals were group housed and exposed to 14 h of light per day (LD 14:10; lights on between 08.00 and 22.00 h PST); food and water were available ad libitum. During the experiment all hamsters were exposed to constant dim illumination (average light intensity at cage

Group	n	Period (h) Pre-thiourea		During thiourea
Control	7	24.17 ± 0.03*	vs	24.15 ± 0.03*
Thyroidectomized	9	24.10 ± 0.04	vs	24.30 ± 0.06**

Mean and SEM of the period of the activity rhythm of control and thyroidectomized hamsters before and during thiourea treatment. *t-test nonsignificant; **t-test $p < 0.01$.