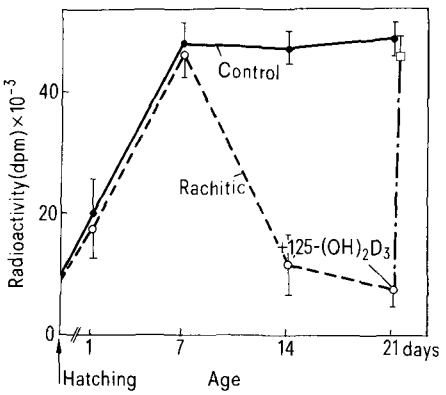
than that found at hatching. The fact that the 2 groups raised either on normal or on rachitogenic diets showed about the same qualitative and quantitative behavior during the 1st week is probably due to the presence in both groups of animals of endogenous $1,25(OH)_2D_3$ sufficient to determine the response observed. In fact, it has been reported that chicks raised on the same rachitogenic diet become vitamin D deficient after 3 weeks of life when endogenous active metabolites of vitamin D_3 are exhausted[8]. In the experiments reported, 3 weeks old chicks showed only 15% residual spermine-binding activity of their corresponding controls. However, in several experiments, the values varied from 11 to 20%, the control values being almost constant in all experiments. The figure also reports the effect of administration of $1,25\text{-}(OH)_2D_3$ to the 3-week-old vitamin D-deficient chicks on duodenal spermine-binding activity. 5 h after a single intracardial injection of the active form of vitamin D, the spermine-binding activity increased by 560% above the values obtained from the rachitic chicks reaching 92% of the control animals.



Duodenal spermine-binding activity during normal and rachitic development. Groups of 8 chicks fed with normal and vitamin D-free diet were killed at the age indicated. Duodenal binding activity of each group purified from the pooled tissues was estimated as described in the Methods section. The data reported are expressed as dpm/150 µg of fractionated cytosol protein. Results are means ± SEM for 3–5 separate experiments.

The almost complete recovery in binding activity shown was a constant and very reproducible phenomenon in all groups of animals tested; however different experiments showed values ranging from 85 to 100%.

Although no evidence is available at the moment supporting a specific physiological role of the binding protein, it is noteworthy that this protein strikingly responds to the $1,25(OH)_2D_3$ status of the animal by modifying its spermine-binding activity as a consequence. This finding strongly suggests that the increase of spermine-binding activity at hatching is hormonally-regulated and reflects changes in vitamin D metabolism and/or action. Further studies on the effects of vitamin D analogs and metabolites on the binding activity from duodenum and from other vitamin D sensitive tissues will help to establish the physiological relevance of this protein.

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Brain dopamine variations in gonadotropin-treated immature rat[1]

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Summary. The cortex, cerebellum, caudate nucleus, and hypothalamus of immature rats treated with pregnant mare's serum gonadotropin (PMS) were isolated and analyzed for dopamine (DA) content at 6-h intervals for 72 h. Results showed that PMS injection caused significant elevation in DA levels in all brain regions studied.

There is substantial evidence that central catecholamines, specifically dopamine (DA), mediate the hypothalamic mechanisms governing the release of pituitary LH and FSH[3]. In the hypothalamus, steroid hormones influence DA content[4]. It has also been shown that DA concentration[5,6] and the rate at which it is synthesized[7] change physiologically during the course of estrus cycle in female rats and mice. Meanwhile, in immature PMS-treated rats, alteration in DA metabolism will prevent ovulation[8,9] while inhibition of the conversion of DA to norepinephrine has no effect[10].

Although many attempts have been made to correlate dopamine with the ovarian activity in mature animals[11,12] few, if any, investigators have observed actual levels of the compound during the period leading up to ovulation. It was of interest, therefore, to examine DA levels in the brain at different intervals during the initial ovulatory period in the immature rats.

Materials and methods. Animals. 36 female rats (21 days of age) of the Sprague-Dawley strain (Southern Animal Farm, Prattville, Alabama), weighing 50–70 g each were housed in groups of 4 per cage and maintained under controlled lighting (from 09.00 to 21.00 h daily) and temperature (23 ± 1 °C) till the time of sacrificing. The animals were provided with standard Purina Lab Chow and water ad libitum.

Induction of ovulation. 25 IU pregnant mare serum gonadotropin (PMS, Sigma Chemical Co.) were injected s.c. in a saline solution on day 25. Simultaneously, control animals were injected with an equal volume of saline. The occur-

rence of ovulation was determined by examination of the oviduct for the ova with no attempt to count the ova.

Experimental procedures. Animals were sacrificed by decapitation every 6 h beginning at 06.00 h on day 25. The entire brain was removed and rapidly separated into cerebral cortex, cerebellum, caudate nucleus, and hypothalamus. The brain tissues were immediately frozen in a super-histofreeze and hypothalamus. The brain tissues were immediately frozen in a super-histofreeze and stored until needed for the analysis. DA was extracted and assayed using a method adopted from Welch and Welch[13]. DA concentration was determined by means of spectrophotofluorometric analysis. The Aminco-Bowman Spectrophotofluorometer was used at a wavelength of 385 nm for activation peak and 490 nm for emission. Data were subjected to 1-way analysis of variance using F-test.

Results. Hypothalamic concentrations of DA are given in figure A. Peak levels occurred at 18.00 h in the light phase on the 1st and 2nd days of the 3 day study. Peak values for the 3rd day were obtained at 06.00 h in the dark phase. However, significant difference could be determined between peak and trough values on th 3rd day. There was significant ($p < 0.05$) increase in DA values for the treated animals over controls for all 3 days of the cycle.

In the cerebral cortex peak DA levels were observed at 18.00 h in the light phase of the 1st and 2nd days following PMS injection (fig. B). 3rd day values varied from the established pattern of the 1st and 2nd days with peak concentration occurring at 12.00 h in the light phase. Values for PMS-treated rats were again significantly ($p < 0.05$) higher than those values obtained for the control rats.

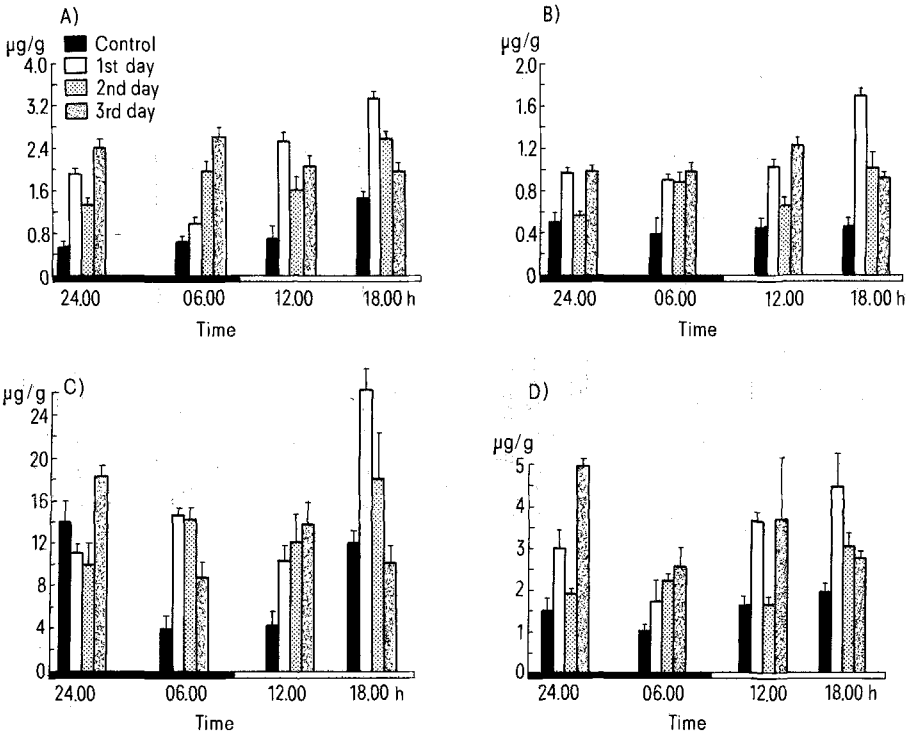
DA concentrations in the caudate nucleus are shown in figure C. The data show a significant increase ($p < 0.05$) in DA in the PMS-treated over the nontreated rats. Peak levels of dopamine were reached at 18.00 h in the light phase of the 1st and 2nd days. On the 3rd day, peak levels occurred at 24.00 h during the dark phase.

Peak levels of DA in the cerebellum occurred at 18.00 h in the light phase on the 1st day as they did in the caudate nucleus (fig. D). A peak in DA concentration also occurred at the same time on the 2nd day. 3rd day determinations showed a significant ($p < 0.05$) increase in level occurring also at 24 h in the dark phase. The differences in overall values between control and treated animals were statistically significant in all 3 days.

Discussion. The data presented here indicate that DA levels in all brain regions studied are affected by PMS treatment which suggests an influence of PMS on DA synthesis, release and/or metabolism. The significant rise of DA after the administration of PMS indicate that this biogenic amine may be an important neurotransmitter in controlling ovulation. The intervention of DA neurons system in the overall regulation of cyclic LH release have been suggested[14]. Moreover, it has been shown using both in vitro and intraventricular infusion techniques, that DA and to lesser extent norepinephrine can induce the discharge of LH releasing hormone and increase plasma LH level[15]. In the gonadotropin-primed immature rat, it was found that depletion of brain monoamines would cause inhibition of ovulation which could be restored by the replacement of DA during the critical period but not with the administration of norepinephrine[8].

While increased dopamine level observed in the caudate nucleus, cerebellum and cerebral cortex may not be directly related to the control of ovulation, studies have suggested possible roles for these brain regions in regulating neuroendocrine events[16]. Clearly the changes in brain DA cannot be entirely related to ovulation, as the changes were observed on days when pituitary LH had been released. Moreover, the observed changes occur in parts of the brain which as far as we know are not directly involved in gonadotropin release.

In the present study, ovulation is induced by exogenous administration of the PMS gonadotropin; therefore, the



Effect of PMS treatment on dopamine levels in: hypothalamus (A); cerebral cortex (B); caudate nucleus (C); and cerebellum (D). Each bar represents the average of 6 animals ± SEM. Dopamine concentration was based on wet weight of each brain region.

observed changes in dopamine levels are more likely to be consequence, rather than a cause, of the ovulatory process. The increase in dopamine levels may be caused by PMS-induced increase of estrogen secretion[17], since estrogen in ovariectomized female rats has been shown to increase dopamine levels in the median eminance and the olfactory tubercle[18].

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The aggressive behavior repertoire of an anophthalmic phreatic fish from Somalia[1]

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Summary. The authors complete the aggressive behavior repertoire of *Uegitglanis*, and refine the description of some patterns. The temporal sequence of various patterns is also shown. The biological significance of some patterns and of the persistence of a complex aggressive behavior in this highly regressed hypogean species is briefly discussed.

Until recently, aggressive behavior in blind fish dwelling in subterranean habitats (water-bearing layers and karstic systems) or in coastal anfractuosities, was known only in *Amblyopsis spelaea* De Kay[2], *Typhlogobius californiensis* Steindachner[3] and *Caecobarbus geertsi* Boulenger[4,5]. Then similar behavior was recorded both in the laboratory and in the field in *Uegitglanis zammaranoi* Gianferrari (Clariidae, Siluriformes), an anophthalmic phreatic fish from Somalia[6]. In further laboratory experiments, 10 specimens of *Uegitglanis* raised for a long period in complete isolation were tested in pairs in a neutral aquarium (i.e., new to both fish) and then in the home aquarium of each partner. These tests revealed not only the complexity of their aggressive behavior, but also that it leads to the rapid - and evident - establishment of a dominance hierarchy between the 2 components of each pair[7]. Further analysis of our edited and unedited data (totalling 632 min of observation) has enabled us to refine and complete our catalogue of the aggressive behavior of *Uegitglanis*, and to pinpoint the behavioral sequences leading up to each aggressive display. The present paper reports these latest findings.

Behavioral repertoire. Listed below are the behavioral patterns observed with varying frequency in all the fish pairs.

1. Bottom dive: swift dive at an angle of 45° to the bottom of the aquarium, instantly after release in the test aquarium.
2. Jerking: repeated short forward-and-backward jerks along the antero-posterior axis.
3. Zig-zagging: swift right-left oscillations on a horizontal plane of the anterior part of the body.
4. Pitching: swift forward movement along a sinusoidal path on a vertical plane.
5. Speed increase: abrupt increase in swimming speed prior to chases and attacks, or as part of exploratory activity.
6. Patrolling: tenacious inspection of the aquarium floor, usually along the perimeter, sometimes crossing the floor diagonally.
7. Bottom brushing: swift lateral movements of the anterior

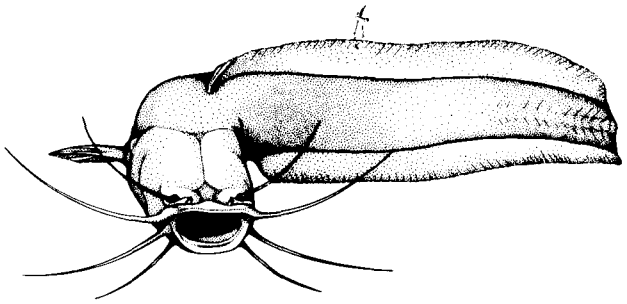

Figure 1. Gaping. Redrawn from a video-tape sequence.

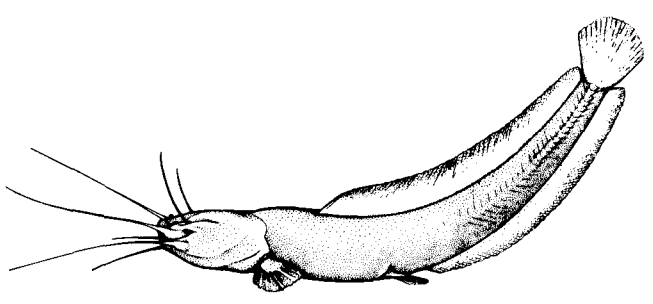

Figure 2. Back-bending. Redrawn from a video-tape sequence.