quire a complex neurologic integration involving the nucleus accumbens in order to perform intentional movements, whereas this kind of integration is not necessary when the motivation for movement is not linked to some aversive situation.

Little is known about the exact mechanisms by which the limbic processes gain access to the motor system. The nucleus accumbens receives direct connections from the amygdala, hippocampus and other limbic forebrain structures, as well as indirect connections via the mesolimbic dopaminergic projections from the ventral tegmental area of Tsai. Furthermore, it also has direct and indirect connections to the globus pallidus via the substantia nigra and the nigrostriatal dopaminergic system[2, 11-18]. Therefore, this nucleus appears, on anatomical grounds, to be part of a functional link between the limbic system and the basal ganglia[3]. The fact that the nucleus accumbens has anatomical connections with both the motor and limbic systems, plus the experimental evidence indicating that destruction of this nucleus modifies some complex motor responses to aversive stimuli involving both the emotional status and the motor system of the animal, are clear indications of the importance of this nucleus in the control of locomotor responses. However, motor activity in resting conditions (open field movements) is not modified by destruction of this nucleus, thus indicating that not all motor activities are regulated by this pathway.

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Immunoactive TSH in the amniotic fluid of the rat[1]

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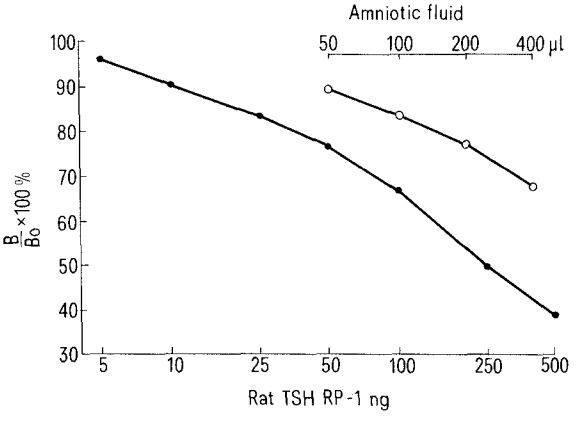
Summary. Amniotic fluid was obtained from 19-day-old rat fetuses by aspiration. Pooled samples measured at 4 different dilutions demonstrated parallelism with standard rat TSH. It is concluded that rat amniotic fluid has TSH immunoactivity.

Pituitary hormones, like prolactin[3] and corticotrophin[4], have been reported in human amniotic fluid. More recently, thyrotrophin has been found in the amniotic fluid of the lamb[5] and human fetuses[6]. Thyrotrophin as well as thyroid hormones have also been measured in the blood of 21-day-old rat fetuses[7]. However, these hormones were undetectable in the amniotic fluid of rat fetuses at term[8]. In this study, we report the estimations of TSH immunoreactivity in the amniotic fluid of 19-day-old rat fetuses.

Materials and methods. Young virgin adult female Sprague-Dawley rats weighing about 200 g were allowed to mate with young adult males. Vaginal smears were made each morning and the day that spermatozoa were found in the smears was taken as day 0 of pregnancy. Altogether 7 pregnant rats were used in 2 separate experiments. On day 19 of pregnancy, the mothers were killed with ether and the fetuses were quickly dissected out and put on ice. Amniotic fluid was obtained by aspiration. Due care was taken to avoid contamination of the sample with blood. Amniotic fluid from 5–10 fetuses was pooled and was stored at −20 °C prior to radio-immunoassay. Aliquots of 0.2 ml were measured in duplicate using a rat TSH RIA kit kindly supplied by the National Institute of Arthritis, Metabolism, and Digestive Diseases. The sensitivity of the assay was about 10 ng, and the intra-assay and inter-assay coefficients of variation were about 3% and 10% respectively. The antibody was highly specific for rat TSH and did not cross-react significantly with any other rat pituitary hormones. In order to establish parallelism between amniotic fluid and



Dilutions of pooled amniotic fluid showed parallelism with standard rat TSH.

standard rat TSH (rat TSH PR-1, specific activity 0.22 USP units/mg), pooled amniotic fluid was measured at 4 dilutions (50 µl, 100 µl, 200 µl and 400 µl) in a total incubation volume of 0.7 ml.

Results and discussion. Dilutions of pooled amniotic fluid exhibited parallelism with the standard curve of rat TSH (fig.), demonstrating the immunological identity of amniotic fluid and standard rat TSH. Moreover we found TSH immunoactivity in all 12 samples obtained from 2 different experiments with a mean concentration of 305.4 ± 24.1 (SE) ng/ml, which was about 25% of the level found in adult rat serum.

We have demonstrated unequivocally the presence of TSH immunoactivity in rat amniotic fluid, although El-Znheri and his coworkers failed to detect TSH in the amniotic fluid of rat fetuses at term[8]. The discrepancy between the results may be due to a difference in the assay methods and/or sample volumes used, as Chopra and his co-workers[9] also failed to detect TSH in human amniotic fluid in earlier studies. The amniotic TSH levels found in this study were similar to those found in rat fetal blood[7,8,10]. In view of this, and the previous finding that hypothyroidism resulted in elevated TSH in the amniotic fluid of lamb fetuses[5], we infer that this amniotic TSH is probably of fetal origin. This aspect of the problem is currently under investigation in our laboratory.

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Cadmium-induced inhibition of brain monoamine oxidase in the freshwater catfish[1]

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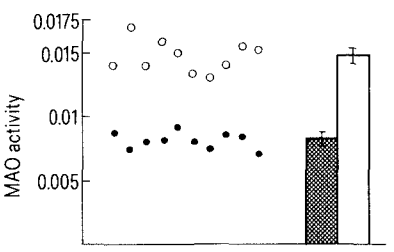
Summary. The freshwater catfish *Clarias batrachus* (L.), exposed to 50 ppm of cadmium (Cd) chloride for a period of 135 days exhibited marked inhibition of brain monoamine oxidase (MAO) ($p < 0.001$). The retarded gonadal growth observed in the present study suggests that Cd may be capable of creating imbalance in the aminergic activity of the hypothalamus, which modulates the secretion and release of gonadotropins.

Cadmium is one of the apparently nonessential metals which can enter the bodies of animals, including man, through food, water and air. It activates or inhibits several enzymes, and causes developmental abnormalities, anemia, bone mineral loss, hypertension, cardiac enlargement, enteropathy, gonadal atrophy and kidney damage[2-5]. Although it has been suggested that the toxic effects of Cd are due to induced deficiencies of essential nutrients which are accompanied by increased Cd absorption, little is known about the biochemical basis of its chronic toxicity[4,6]. In mature rats, Cd exerts its neurotoxic effects on the sensory ganglia[7,8] whereas in immature ones it causes damage to the central nervous system accompanied by hemorrhagic lesions and dysfunction of neurons.

Fifteen *C. batrachus* were exposed to 50 mg/l of Cd chloride for 135 days, changing the medium every alternate day after feeding. An equal number was kept as control. The experiment was started in February 1982 when the gonads were in the resting phase and terminated in June 1982, when the control exhibited spawning phase stage IV gonads. All the fish were sacrificed by decapitation and the whole brain MAO was estimated adopting the enzyme isotopic technique of Parvez and Parvez[9]. 50 µl of 0.44 nM ^{14}C-tryptamine bisuccinate (specific activity 49.6 mCi/mmole; obtained from New England Nuclear, USA) was used as the enzyme substrate. MAO is a mitochondrial enzyme responsible for oxidative deamination of monoamines. Hence quantitative variations in MAO are an indirect indication of changes in monoamine activity. In Cd-exposed rats, Magour et al.[10] reported inhibition of MAO in the mitochondrial extract of brain and liver. In the rat, intraocular injection of lead was accompanied by hyperinnervation but Cd did not show any change in nerve fiber density whereas mercury caused degeneration of adrenergic fibers[11]. *C. batrachus* exhibited a significant ($p < 0.001$) (44%) depletion of whole brain MAO in response to Cd chloride treatment (fig.). It is well established that the monoaminergic system of brain is not only involved in neurotransmission but also modulates the various tropic hormones secreted by the pituitary. In Cd chloride exposed *C. batrachus* the gonads remained in the resting phase whereas in the controls they were fully matured. Apart from its probable direct effect on the gonads, Cd may impair the aminergic system responsible for the modulation of gonadotropic function.



Brain MAO activity (nM/mg tissue/h) in cadmium-exposed and control groups (n = 10); statistical analysis was ratified through Student's t-test ($p < 0.001$). □, control; ○, control individual value; ▨, Cd-treated; ●, Cd-treated individual value.