tive rarity of anuran polyploid species is, therefore, unexpected rather than the reverse. The majority of the currently recognized polyploid frogs have diploid counterparts and, considering the dearth of chromosomal investigations at the population level, additional polyploid populations or "cryptic species" will undoubtedly be uncovered.

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Functional Development of Prefrontal Cortex in Rats Continues into Adolescence

Abstract. *Bilateral removal of the sulcal prefrontal cortex in rats at 60 days of age and older results in aphagia and adipsia, but removal of this area before 60 days of age does not affect food or water regulation. Apparently the development of the role of this neocortical region in feeding and drinking continues well beyond the time of weaning in the rat.*

Studies of food and water regulation have shown that lesions in the lateral hypothalamus, zona compacta, trigeminal system, globus pallidus, dorsomedial amygdala, or sulcal (ventral or orbital) prefrontal cortex in rats (*1*, *2*) produce aphagia and adipsia such that animals will die unless given special care (*3–5*). The involvement of the lateral hypothalamus in feeding and drinking behavior apparently develops quite early in rats since destruction of this area during the first week of life produces aphagia and adipsia even more severe than that produced by comparable lesions in adults (*6*). In contrast, the results of the present study indicate that the sulcal prefrontal cortex of the rat is not critically involved in food and water regulation until nearly 60 days of age despite the fact that rats are normally weaned between the third and fourth week of life and must thereafter forage for food on their own.

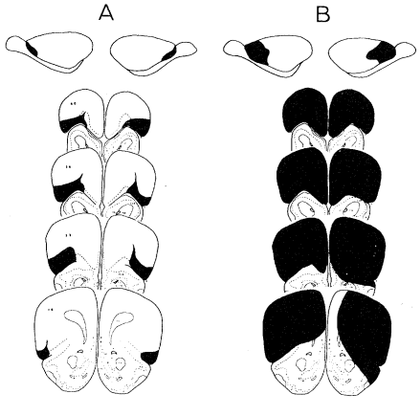

Fig. 1. (A) Coronal sections showing a typical orbital frontal lesion. Most of the lesions in the adults, juveniles, and infants were similar to this one. (B) Coronal sections illustrating the largest lesion. This rat was operated at 6 days of age and failed to exhibit aphagia. It continued to eat and to gain weight normally after surgery.

The subjects were 80 rats, 40 with bilateral lesions of the sulcal prefrontal cortex induced between 2 and 60 days after birth and 40 age-equivalent sham-operated controls. There were eight control rats and eight with lesions (four male and four female) operated at 2 to 6 days and 8 to 10 days after birth, as well as six controls and six with lesions (three male and three female) operated at 25, 35, 40, and 60 days after birth, respectively. Bilateral lesions were made under ether anesthesia in all rats operated at 10 days of age or less while animals operated in the older groups were given intraperitoneal injections of sodium pentobarbital (50 mg per kilogram of body weight). Lesions were made by removal of the sulcal prefrontal cortex by subpial aspiration by procedures outlined elsewhere (*7*) (Fig. 1A). All rats were separated from their mothers at 23 days of age and were then housed in groups of three or four for the duration of the experiment. The animals were weighed daily for the first 21 postoperative days and then at 1- or 2-week intervals until 110 days after surgery. Purina Rat Chow and water were freely available for all subjects throughout the experiment.

Lesions made before 60 days of age failed to alter food and water intake or growth rate (Fig. 2) or to produce the motor dysfunctions observed after similar lesions in older rats (*8*). Indeed, not a single rat operated before 60 days showed aphagia or adipsia, whereas every subject operated at 60 days required gastric intubation for up to 14 days to prevent death from starvation and dehydration. The age effect did not result from smaller lesions in the younger rats since many of those operated 2 to 10 days after birth had large lesions that included up to three times as much tissue as the average adult lesion (Fig. 1). Even those operated early with very large lesions showed only a mild and transient weight loss (Fig. 2).

These results suggest that although rats are normally weaned at about 21 days and must thereafter forage for their own food, the neural mechanisms underlying behavior related to food intake are not fully developed until late adolescence. This suggestion is supported by recent studies of development of ingestive behavior in weanling rats. For example, Epstein (*9*) concludes that although some of the physiological mechanisms controlling feeding and drinking are present at birth, the "developmental process for ingestive behavior continues beyond the time of weaning well into the juvenile period."

Since most known indices of neo-

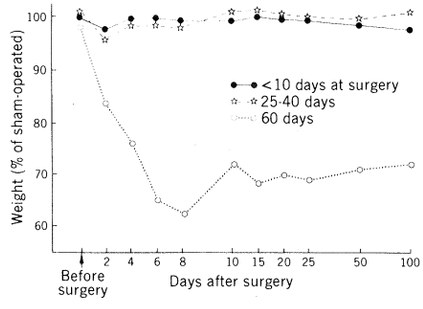

Fig. 2. The effects of orbital frontal lesions on body weight in the first 100 days after surgery. Each point represents the ratio (as a percentage) of the weight of rats with lesions to the weight of sham-operated rats in the respective groups.

cortical development in the rat reach adult values within the first month after birth, the maturational event leading to the apparent development of sulcal cortex regulation of ingestive behavior between 40 and 60 days is not apparent. However, one possibility is that the onset of puberty during this interval might trigger this development. This possibility is speculative at present. However, Kennedy (*10*) has argued that the ontogeny of ingestive behavior is never complete until puberty, and it is conceivable that gonadal hormones at puberty might alter the neural circuitry controlling ingestive behavior in much the same way that they alter the neural substrate of sexual behavior during the perinatal period (*11*). Gonadal hormones are involved in weight regulation of adult rats (*12*), and there is some evidence that medial hypothalamic control of feeding depends upon the attainment of puberty. Neither parasagittal hypothalamic knife cuts nor estradiol injections in female rats affect food intake before 40 to 50 days after birth (*13*), the age at which females attain sexual maturity.

Another possibility is that the present results depend on the prolonged development of the catecholaminergic "feeding system" in rats (*5, 14*). Although the cell bodies for this system are formed before or just after birth, the outgrowth of axons and the formation of terminals in the rostral diencephalon and telencephalon continues for several weeks after birth. Complete maturation of the cortical component of this system does not occur until 60 days or later (*15*). The sulcal cortex is ideally situated to modulate the activity of this system since it not only receives afferents from the dorsal noradrenergic pathway but also sends efferents which seem to overlap with many of the terminal projections of the dorsal and ventral adrenergic and the dopaminergic pathways (*2, 5*). Whether the projections from the sulcal cortex and from the catecholaminergic "feeding system" actually end on the same cells within the hypothalamus and caudoputamen is unknown.

Finally, the fact that our animals given early lesions showed no feeding impairment at any age indicates that sparing of function occurs in rats even after large frontal cortex lesions. This result conforms fairly well with those of Goldman (*16*) on sparing of function after early destruction of the frontal cortex. Goldman's data on rhesus monkeys show that substantial sparing occurs after early removal of the orbital region but that little or none is obtained when the dorsolateral region is removed at the same age. Since the behavioral effects of prefrontal lesions in rats and monkeys appear to be remarkably similar (*1, 2*), our results raise the possibility that the prefrontal cortex of the rat may provide a model for the study of the developmental phenomena that Goldman has described in the rhesus monkey (*16*).

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Visual Discrimination Impaired by Cutting Temporal Lobe Connections

Abstract. *An attempt was made to transect the white matter that connects the anterior temporal lobe with dorsal and medial brain areas. Eight monkeys were trained preoperatively on a visual discrimination and tested postoperatively for retention and relearning of the task. They were also tested for Klüver-Bucy symptoms. The two animals that had complete lesions were unable to relearn the visual discrimination. It is suggested that human medial temporal lesions may produce their effects on learning and retention by damage to temporal white matter rather than by destruction of hippocampus.*

A thin band of white matter at the dorsal and medial aspect of the primate temporal lobe forms a bridge between the medullary core of the temporal lobe and the capsules of the brainstem. Within this white matter, the albal stalk of the temporal lobe, run nerve fibers that interconnect temporal lobe structures with subcortex and frontal cortex, and fibers of the temporal limb of the anterior commissure (*1*). In a previous experiment we cut the albal stalk of the temporal lobe by a surgical approach through the lateral sulcus (*2*). A severe loss in retention and relearning of a visual habit and Klüver-Bucy symptoms resulted. Unfortunately, in this procedure there was partial damage to the geniculostriate system which complicated our interpretation of the data. In the present experiment we cut the albal stalk and avoided the geniculostriate system by entering the anterior end of the temporal lobe in an approach much like that used for uncotomy and hippocampectomy (*3*). Our results suggest a new interpretation for the deficits produced by medial temporal lobe lesions in man.

Eight female adolescent *Macaca mulatta* weighing 3.7 to 6.3 kg were studied.