portion of the oviduct prevented release of pituitary LH responsible for ovulation, and was suggested that the significance of a neuronal link may serve as a timing device preventing an ovulatory cycle while an egg is in the oviduct. Results of the present study, however, indicate that such a mechanism may not be involved in *C. uniparens* and that control of the ovulatory cycle may be different than in the hen.

[1] For their help in collecting the lizards we are grateful to GLORIA CUELLAR and to our children, CAROLINA, GRACIELA, JAVIER, LETICIA and RUBEN CUELLAR. We also acknowledge IDALIA CUELLAR, GUY MONTOYA and GRADY TOWNS for their assistance in the various aspects of this work. We are grateful to Dr. RICHARD E. JONES for reading this manuscript. This work was supported in part by a predoctoral National Institutes of Medical Sciences Fellowship to O. CUELLAR (No. 1-F1-G-37, 428-01) also by National Institutes of Health Biomedical Science Support Grant No. RR 07092, to H. S. CUELLAR and the University of Utah Research Committee as well as by a National Institutes of General Medical Science Grant No. 1 R01 GM 19533-01 to O. CUELLAR.

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Precocious Puberty in Rats Induced by Hypothalamic Lesions: A Comparison of Platinum and Stainless Steel Electrodes

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Summary. Precocious sexual maturation was induced in immature female rats by 2 types of unilateral hypothalamic lesions. Stainless steel electrodes produced smaller tissue defects but proved more efficient than platinum electrodes.

Certain types of hypothalamic lesions induce precocious sexual maturation in female rats, but their mode of action is obscure[4]. Traditionally, lesions within the central nervous system have been equated with elimination of nervous tissue, and it has been postulated, accordingly, that hypothalamic lesions remove inhibitory brain influences on the pituitary-gonadal axis. In view of the demonstrated presence of luteinizing hormone releasing hormone (LRH) within the basal hypothalamus[5-8], it is possible, however, that lesions placed in these areas act as direct stimuli for the triggering of precocious puberty by disrupting nerve endings which store LRH in the vicinity of the primary capillaries of the hypophysial portal circulation. Since lesions placed through steel electrodes in the medio-basal hypothalamus[9] are particularly efficient in inducing precocious puberty, and appear to do so more rapidly than lesions placed through platinum electrodes in the anterior hypothalamus[10], it was decided to test the effect of both types of electrodes on the same central structure. The region of the arcuate nucleus was chosen for this comparison, because of its demonstrated high concentration of LRH[8].

Female Sprague-Dawley rats weighing > 55 g on day 23 of life were used. Electrodes were made from stainless

[1] Supported by Swiss National Science Foundation grant No. 3.016.73.
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Differential effects of 2 types of hypothalamic lesions on the ovary (mean ± SE)

	N	Ss	Pt	Untreated controls
Ovarian estrogen content after 1 h (pg/pair)	10	126.4 ± 9.2[a]	94.6 ± 9.6[b]	69.8 ± 0.9[c]
Ovarian progesterone content after 1 h (ng/pair)	10	9.8 ± 1.6[d]	6.5 ± 1.7[e]	3.2 ± 0.5[f]
Ovarian weight (mg/pair)	10	18.7 ± 1.0[g]	19.4 ± 1.0[g]	11.7 ± 0.9[h]

[a] vs b) $p < 0.05$; a) vs c) $p < 0.001$. [b] vs [c] $p > 0.05$. [d] vs e) $p > 0.02$; d) vs f) $p < 0.005$. [e] vs [f] $p > 0.05$. [g] vs [h] $p < 0.005$.

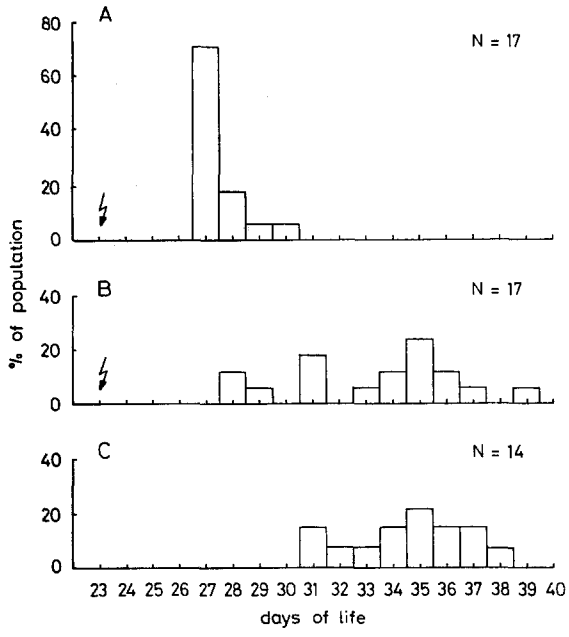

Fig. 1. Occurrence of first ovulation. A) after unilateral hypothalamic lesion placed through steel electrode on day 23 of life; B) after lesion placed through platinum electrode; C) in untreated controls of similar age and body weight.

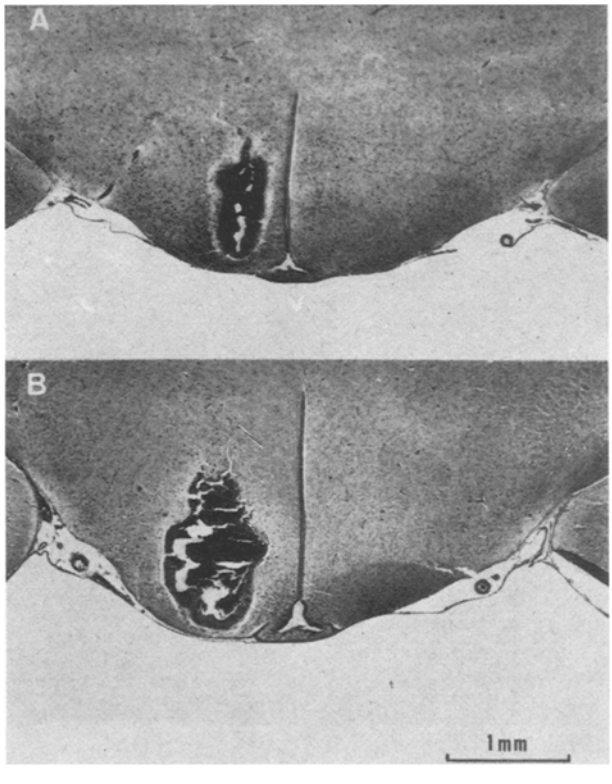

Fig. 2. Comparison of lesion size on typical frontal brain sections fixed 1 h after the passage of current. A, steel electrode. B, platinum electrode.

steel (Ss) tubing (outside diameter 0.254 mm), or from platinum (Pt) wire (outside diameter 0.457 mm); Pt electrodes made from smaller gauge wire lacked the rigidity necessary for accurate stereotaxic work. Both types of electrode were coated with epoxylite resin, except for a tip of about 1 mm, and were stereotaxically inserted into the brain (under short ether anaesthesia) according to the coordinates determined by Sherwood and Timiras[11]. Unilateral electrolytic lesions were placed by passing a direct current of 0.5 mA during 15 sec (7.5 milliCoulombs)[12]. Brains were perfused with 10% formalin/saline, and the extent of the lesion was determined on serial coronal paraffin sections (10 μm). Some animals were monitored for the occurrence of the first (pubertal) ovulation following brain surgery, others were killed after 1 h for the determination of ovarian steroid content as assessed by radioimmunoassays[13].

As in earlier experiments[12], unilateral hypothalamic lesions placed by Ss electrodes were highly efficient in advancing puberty as judged by the occurrence of first ovulation (Figure 1 A). In contrast, lesions placed through Pt electrodes in the same area induced precocious ovulation only in a minority of experimental animals (Figure 1 B) as compared with untreated controls (Figure 1 C). Histological analysis of 2 groups ($N = 5$) of randomly chosen brains fixed 1 h after the lesion revealed marked differences in the degree of tissue damage resulting from the 2 types of electrodes (Figure 2). Lesions placed through Ss electrodes had a vertical diameter of 1.02 ± 0.08 mm (mean $\pm$ SE) and a horizontal diameter of 0.56 ± 0.02 mm in their centre-most part, resulting in a mean area of 0.46 ± 0.05 mm^2. Lesions placed by the same amount of current through Pt electrodes were of significantly larger dimensions (two-tailed Student's t-test; $p < 0.05$). The corresponding measurements were 1.69 ± 0.23 mm, 1.08 ± 0.10 mm, 1.50 ± 0.34 mm^2. In contrast, the rostro-caudal extension of lesion sites was not significantly different (0.61 ± 0.03 mm for Ss, and 0.66 ± 0.02 mm for Pt). In further confirmation of earlier results[12], it was found that Ss lesions significantly increase the ovarian content of immunoreactive estrogen and progesterone within 1 h (Table), whereas the elevation observed after Pt lesions did not reach statistical significance. Both types of lesions increased ovarian weight as compared to untreated controls. This stimulatory effect of hypothalamic lesions on the ovary is probably mediated by luteinizing hormone (LH)[14,15].

These results indicate that the degree of induced sexual maturity is not simply a function of lesions size, as could be inferred from the pattern of hypothalamic LRH distribution[8]. The passage of current through Ss electrodes causes electrolytic iron deposits which are considered to constitute foci of chronic tissue irritation[16]. In contrast, deposits from Pt electrodes are minimal, and the biological effects of such lesions appear to reflect the acute consequences of current flow per se. Therefore, it is conceivable that Ss lesions induce a prolonged discharge

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of LRH whereas the effect of Pt lesions is confined to the phase of acute disruption of LRH-containing neurons. In view of the short biological half-life of circulating LRH[17], differences in the degree of induced ovarian steroidogenesis are likely. In related studies[18], it has been found that estrogen released in response to the brain stimulus is able to prime the reproductive axis for the subsequent preovulatory release of gonadotrophins.

In assessing the mode of action of various types of brain lesions on neuroendocrine tissues, it appears useful to distinguish between sites of hormone production and sites of origin of neuronal connections with hormone producing neurons. In the amygdala, opposite effects of Ss and Pt lesions on gonadotrophin release have been described[19], and such qualitative differences are to be expected in a brain structure in which lesions may either irritate or destroy synaptic inputs to hypothalamic peptidergic neurons. In the basal hypothalamus, on the other hand, different methods of brain stimulation (electrical stimulation, passage of direct current, high-frequency lesions) yield merely quantitative differences in the degree of activation of the reproductive axis[14]. Our own results confirm that lesions of any type placed in areas containing LRH are likely to stimulate the pituitary-gonadal axis to varying degrees.

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THEORIA

Man's Strategy in Domestication - a Synthesis of New Research Trends

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Summary. The minimum brain size possible in the relevant wild species and certain colour types which, because of alterations in the neurotransmitter system caused by the respective colour genes, are related to behavioural traits diverging from the wild animal's norm appear to be first-rate bases for domestication either separately or in combination.

Domestication is not only an historical, but also a contemporary process. This is shown by the efforts made to use the moose (*Alces alces*) and the eland (*Taurotragus oryx*) as domestic animals[1,2], as well as by the new breeding of fur-bearing animals, for example. The knowledge of how domestication starts to succeed, beyond pure description of the phenomena of domestication, has therefore not only academic but also high economic value. So one of the most urgent tasks of research in this field is to acquire a conception, practically applicable in strategies for new domestications, on the principles at work at the very starting point of former domestications, whether these occurred intentionally or by chance, and to verify this conception experimentally.

Domestication of any animal species requires that the species in question has some potential in this respect and also direct interest or at least some readiness in man to keep this animal. Compared to their previous state, there must be some changes in the man-animal relations which require changes in the wild animal's behaviour. Surely it is not accidental to find alterations usually in only one direction mainly in the sphere of the central nervous system and behaviour when observing the multiple and mostly unspecified widening of variability in domestic animals: spontaneous activity, shyness and aggressiveness towards man are commonly reduced.

These and other changes of behaviour were interpreted by Herre et al. (e.g.[3]) as being related to brain changes taking place at the transition from wild to domestic animals, the most conspicuous of which was supposed to be a considerable reduction of relative brain size, said to amount to between 19 and 34% in carnivores and artiodactyles[3]. Revisions made for two species[4,5] have shown that this view is not always true, being based on methodical assumptions which cannot be maintained.

Case 1: The domestic cat. Hardly anyone believed that the domestic cat derived from the European Wildcat, but the belief in a reduction in relative brain size of about 23% from wild to domestic cat arose by comparing this form of the widespread species *Felis silvestris* with domestic cats[3,6]. The picture changes considerably on taking into account the desert-steppe cats of the northeast African and southwest Asian regions[4], where the domestic cat originated, as is well known from comparative morphological studies as well as from the prehistoric documents (e.g.[3,7]). The index $\frac{\text{skull length}}{\text{braincase capacity}}$ as brought into discussion by Schauenberg[8] provides good information here (Figure). (This index is justified because the exponent of the length/capacity allometry does not considerably differ from isometry). Mummified cats from ancient Egypt of the first prechristian millenium[12] scatter, on the whole, in the range of variability of the

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