in Fig. 1B gave birth to a litter of five pups and, although somewhat clumsy in retrieval, reared all of them.

Four hamsters in which there was an additional loss of the midline limbic structures (cingulate convolution and the underlying dorsal part of the hippocampus) (Fig. 1C) retained most of the listed hamster-typical forms of behavior, with the notable exceptions that they displayed no play-fighting during development and exhibited severe deficits in maternal behavior. The female whose brain is shown in Fig. 1C gave birth to a litter of eight pups and nursed them, but failed completely in pup retrieval. Like the other female in this category, she performed an excessive number of scent marks and sexually presented to a male separated by a screen. The animals without neocortex were generally more difficult to handle than the controls; but those with additional loss of the midline limbic structures exhibited a ferocity that led their handlers to refer to them as "wild." In addition, they engaged in stereotyped pacing suggestive of caged animals in a zoo; video analysis revealed that an average of 6.5 hours per day was spent in such activity as opposed to 0.5 hours for controls.

The deficits in play-fighting and parental care are of particular interest because two cardinal developments in the evolution from reptiles to mammals were the origination of nursing and maternal care of offspring and play behavior (*2*). The midline limbic cingulate cortex has been implicated in maternal behavior (*7*), but there has been no previous evidence that the limbic cortex of the medial wall of the hemisphere is involved in play.

In related investigations of brain mechanisms underlying species-typical communication, it was found that in animals as diverse as lizards and monkeys lesions of certain parts of the striatal complex interfere with social display behavior (*8*). Our present study adds new dimensions to these observations by demonstrating in a rodent that the striatal complex and limbic system, with the remaining neuraxis, are sufficient for giving expression to a wide range of unlearned forms of species-typical behavior and that the midline limbic structures are required for the expression of play behavior and integrated performance of maternal behavior.

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References and Notes

1. The mammalian striatal complex consists of the olfactostriatum (olfactory tubercle and nucleus accumbens), corpus striatum (caudate-putamen), globus pallidus, and satellite collections of gray matter (*2*). The limbic system includes the cortex of the limbic lobe and structures of the brainstem with which it has primary connections [P. D. MacLean, *Electroencephalogr. Clin. Neurophysiol. J.* **4**, 407 (1952)].
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9. We thank G. E. Schneider for help with experiment 1, T. K. Bussard for the histological preparations, and E. Lowe for assistance with the observations.

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Parasitism and Behavioral Dominance Among Male Mice

Abstract. *Infestations by the nematode* Heligmosomoides polygyrus *can prevent adult male mice from becoming behaviorally dominant. The effect is dose-dependent and is more likely to influence the development of dominance than to disrupt existing dominance relationships. Doses capable of exerting this effect are not lethal and do not affect weight.*

Hausfater and Watson (*1*) observed an inverse relation between dominance status in male baboons and fecal output of helminth eggs and protozoan cysts, but did not imply that parasitic infestation affects the development of dominance in males. I investigated whether and under what circumstances a helminth (*Heligmosomoides polygyrus*) can influence dominance relationships among male laboratory mice.

Two experiments were carried out. The first was designed to determine whether various doses of the helminth could prevent the development of behavioral dominance among males that previously had not encountered one another. The second experiment was designed to determine whether equivalent doses of the helminth could disrupt established dominance relationships.

In the first experiment, 21-day-old male CF1 mice were housed individually and kept isolated for 5 weeks. They were then marked, weighed, and housed in groups of three. Two mice in each group received a dose of *H. polygyrus* (50, 150, or 250 larvae, orally) (*2*). After 14 days the dominance status of each mouse was determined (*3*), and it was killed and weighed. In the second experiment, 21-day-old male CF1 mice were housed individually and kept isolated for 3 weeks. Then they were marked and housed in groups of three. After 2 weeks the dominant mouse in each group was identified and inoculated (50, 150, or 250 larvae), and all the mice were weighed. After 2 weeks dominance status was again determined and the mice were killed and weighed. Nineteen groups of control mice were treated in the same

Table 1. Results of the first experiment.

Larvae administered	Groups	Groups in which uninfected mouse became dominant	χ^2	P
50	21	7	0.0536	>.05
150	30	15	3.0375	>.05
250	20	14	11.5931	<.005

Table 2. Results of the second experiment.

Larvae administered	Groups	Groups in which dominant mouse became subordinate	χ^2	P
0	19	3		
50	20	1	1.2323	>.05
150	21	1	1.3478	>.05
250	19	5	0.6333	>.05

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way as mice in the second experiment except that none received larvae.

Among the groups in which two individuals were dosed with 50 or 150 larvae, the number of groups with dominant uninfected mice was not significantly different from the number for which chance alone would account (Table 1). Uninfected mice became dominant significantly more often in groups in which two individuals received a dose of 250 larvae (Table 1). The heaviest mouse in each group became dominant no more often than expected by chance [χ^2 (1) = 0.4171, $P > .05$]. Analysis of variance indicates that neither dosage nor dominance status influenced weight gain by individuals during the experiments [50 larvae $F(51) = 0.52, P > .67$; 150 larvae, $F(84) = 1.13$, $P > .32$; 250 larvae, $F(58) = 0.79$, $P > .51$]. None of the doses used in the second experiment resulted in dominant mice becoming subordinate significantly more often than occurred among the control groups (Table 2).

The dominance status of a male mouse is of prime importance in its gaining access to females (*4*). The same is true for other mammalian species (*5*). If parasites impair the development of dominance in males of other host species, this would support Freeland's (*6*) suggestion that female sexual characteristics (olfactory, visual, and so forth) evolved in part to induce competition by males so as to reveal their disease states. Males with high parasite loads are thus eliminated from the sexual arena, reducing the probability that a female will contract a pathogen capable of diminishing or eliminating her reproductive output. Also, healthier males may provide genes or gene complexes associated with resistance to pathogens, thereby increasing the probability that the offspring will survive and reproduce.

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3. Mice do not establish dominance hierarchies. When male mice are grouped, one usually becomes dominant over the others. Dominance status among the nondominant mice is difficult or impossible to determine reliably. The criterion used most frequently and reliably to determine which individual is dominant is the relative absence of tail and rump wounds [F. H. Bronson, *Physiol. Behav.* **10**, 947 (1973); J. C. DeFries and G. E. McClearn, *Am. Nat.* **104**, 408 (1970); C. Desjardins, J. A. Maruniak, F. H. Bronson, *Science* **182**, 939 (1973); J. A. Lloyd, *Proc. Soc. Exp. Biol. Med.* **137**, 19 (1971); J. G. Vandenberg, *Anim. Behav.* **8**, 13 (1960); J. P. Scott, *Am. Zool.* **6**, 683 (1966)].
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Epileptogenic Agents Enhance Transmission at an Identified Weak Electrical Synapse in *Aplysia*

Abstract. *To examine the possibility that alterations in the effectiveness of electrical synapses might participate in epileptogenesis, the effects of several convulsants on an identified weak electrical synapse in* Aplysia *were examined. Application of pentylenetetrazole, strychnine, or tetraethylammonium led to a dramatic increase in the size of the electrical postsynaptic potential mediated by the synapse; penicillin was considerably less effective. In a number of animals, the increased electrical synaptic effectiveness led to the abnormal conduction of spikes across the synapse. If convulsants have a similar action in mammalian cortex, enhanced transmission at weak electrical synapses may provide abnormal pathways for the flow of seizure activity and contribute in part to the synchronous firing of neurons characteristic of epileptic activity.*

Recent studies suggest that a surprising number of cells in the mammalian cerebral cortex are interconnected by weak electrical synapses (*1*, *2*). For example, MacVicar and Dudek (*3*) have recently found evidence for electrical coupling between hippocampal pyramidal cells by measurements of electrical coupling, observations of dye passage, and detection of gap junctions by freeze-fracture electron microscopy. In this instance and others, the electrical coupling is relatively weak in that action potentials in one cell do not lead to firing of the connected cell, but only to small postsynaptic potentials (PSP's). However, in

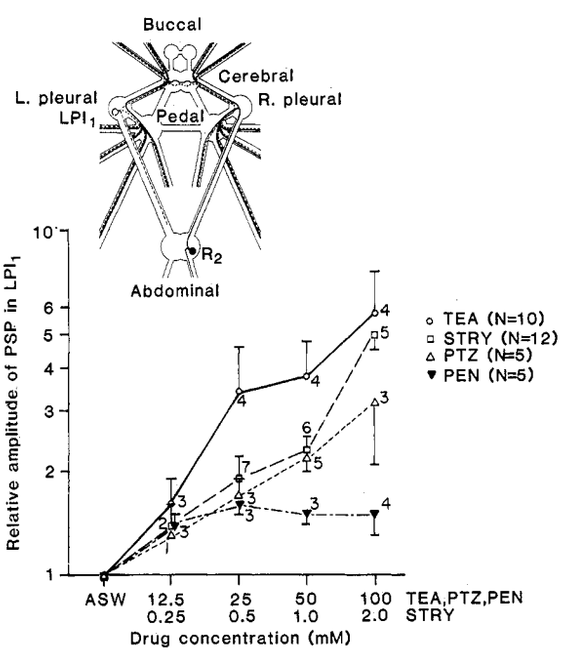

Fig. 1. Enhancement of electrical transmission at the R_2-LPl_1 synapse by convulsants. The PSP in the LPl_1 soma resulting from R_2 spikes provides a measure of synaptic effectiveness. Dose-response curves plotted on a log-log basis are shown for the four drugs. Abbreviations: *TEA*, tetraethylammonium; *STRY*, strychnine; *PTZ*, pentylenetetrazole; *PEN*, penicillin; *ASW*, artificial seawater; *N*, number of animals. Numerals on graph indicate number of observations; data are shown as mean ± standard error of the mean indicated in one direction. Drugs were applied in increasing concentration (Fig. 2A shows data from a typical trial). For comparison, the PSP's have been normalized by taking the initial control PSP in ASW as 1.0. (Inset) Schematic of the nervous system of *Aplysia* with the axonal trees of R_2 (solid line) and LPl_1 (dashed line) indicated (*5*, *15*). The cells are coupled by way of an electrical synapse in the cerebral ganglion. By virtue of the asymmetry in the axonal trees of the cells, a reciprocally functioning synapse appears to rectify: spikes in either cell produce 10-mV PSP's in the postsynaptic axon of the other cell in the cerebral ganglion. However, because the R_2 soma is much farther from the synapse than that of LPl_1, PSP's are recorded in the LPl_1 soma, while none are seen in the R_2 soma.

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