13. E. B. Lewis, *Proc. Natl. Acad. Sci. U.S.* **45**, 894 (1959).
14. *Federal Radiation Council Staff Rept. No. 2* (Sept. 1961) stipulates (pp. 8–10) 0.5 rem/yr for population groups and 1.5 rem/yr for individuals. For the case of fallout, the 0.5 rem figure applies.
15. Data are taken from Table 1 of a letter, dated 7 June 1962, from Drs. E. Reiss, Barry Commoner, M. Peterson, K. J. Hohenemser, and J. M. Fowler to Dr. Luther L. Terry, U.S. Surgeon General. The letter is quoted with permission of Dr. Commoner.
16. From "Fallout Surveillance and Protection," (U.S. Public Health Service and Food and Drug Administration press memorandum) (26 Oct. 1961).
17. According to a New York *Times* report (2 Aug. 1962), Dr. G. D. Carlyle Thompson, Utah State Health Director, attributes levels of 1600 and 2050 $\mu\mu$c/lit. (on 20 and 25 July, respectively) to Nevada tests of 6 and 12 July. Dr. Robert C. Pendleton of the University of Utah reports iodine-131 concentrations higher than 2500 $\mu\mu$c/lit. on 14 July (personal communication).
18. See, for example, testimony of R. H. Morgan on "Problems of assessment and initiation of control measures" given before the Joint Committee on Atomic Energy in June 1962.
19. See statement of Senator Hubert H. Humphrey, *Congr. Record* (22 Aug. 1962), p. 16195.
20. Statement of Senator William Proxmire, *ibid.* (17 Aug. 1962), pp. 15887–92.

13 August 1962

Anopheles leucosphyrus Identified as a Vector of Monkey Malaria in Malaya

Abstract. *Anopheles leucosphyrus,* an important vector of human malaria in Sarawak, Borneo, was shown to be infected with *Plasmodium inui* in Malaya by the inoculation of sporozoites into an uninfected rhesus monkey. The mosquito was caught while biting a man, thus demonstrating that it would be possible for a monkey infection to be transmitted to man in nature.

The *Anopheles leucosphyrus* group of mosquitoes includes several important vectors of malaria in southeast Asia. Recognition of their significance was largely due to McArthur (*1*) in North Borneo. His observations refer to what is now known as *A. balabacensis,* the type form of which is an important vector of human malaria in North Borneo, and in the monsoon forest regions to the north of Malaya in Thailand, Burma, Laos, Cambodia, and probably Vietnam. *A. leucosphyrus sensu stricto* appears to have a less wide distribution and according to Colless (*2*) is known only from Sumatra, Malaya, Sarawak, and possibly Indonesian Borneo. It was shown by Zulueta (*3*) to be the principal vector of malaria in the interior of northern Sarawak and is probably also a vector of human malaria in Sumatra and eastern Borneo. Another member of this same group *A. hackeri,* has recently been identified as a vector of the monkey parasite *Plasmodium knowlesi* in Malaya (*4*).

Both *A. leucosphyrus* and a subspecies of *A. balabacensis, A. b. introlatus,* occur in central Malaya but neither are common, and they have never been found in close association with man in the numerous entomological surveys that have been undertaken, principally by Hodgkin (*5*). They are however, known to attack man, and were caught on human bait at ground level and in the forest canopy in hill-forest by Macdonald and Traub (*6*). These observations were of particular interest to our studies on the vectors of monkey malaria and we have also found that *A. leucosphyrus* and *A. balabacensis* are attracted to monkeys and to man both in the canopy and at ground level. They are therefore potentially of the greatest importance should monkey malaria prove to be transmissable to man in nature as it is under laboratory conditions (*7*).

Attempts are being made to determine the vectors of monkey malaria in different localities in Malaya by catching and dissecting the mosquitoes attracted to man and to monkeys and inoculating the sporozoite, when it is encountered, into uninfected rhesus monkeys. Observations extending for over a year in uninhabited hill-forest where both *A. leucosphyrus* and *A. balabacensis* are present had failed to incriminate either species, though one oocyst infection was found in *A. leucosphyrus.* Similar observations at an aborigine village at the head of a narrow rice-valley bordered by jungle-covered hills had given abundant evidence that *A. maculatus* is of overwhelming importance as the vector of human malaria. A few *A. leucosphyrus* adults were caught in the same area on monkey bait. This area was chosen for a series of all-night catches on human bait to determine the biting cycle of *A. maculatus* both inside houses and in the open. Included in the outside catches were nine *A. leucosphyrus* (compared with 901 *A. maculatus*). One *A. leucosphyrus* specimen had sporozoites 12 to 14 μ in length in the glands. The sporozoites were inoculated into an uninfected rhesus monkey (*Macaca mulatta*) intravenously, and into man intradermally. The monkey exhibited an infection 17 days later which has been identified as *Plasmodium inui.* No infection developed in the human volunteer.

Anopheles leucosphyrus has therefore been added to *A. hackeri* as a vector of monkey malaria in Malaya. The finding is of considerable significance since the mosquito was caught in the act of biting a man, showing that it is possible in nature for the same mosquito to feed both on monkey and on man. Though this may not be a common occurrence, a single bite of a mosquito infected with a strain of monkey malaria transmissible to man would be sufficient to reintroduce malaria to a human population from which malaria parasites had been previously eliminated. Many factors are involved in determining whether or not this malaria would persist in the human population.

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9 July 1962

Male Sexual Behavior Induced by Intracranial Electrical Stimulation

Abstract. Electrical brain stimulation in the anterior dorsolateral hypothalamus produced a marked increase in sexual capacity in some male rats. Several measures of sexual behavior, including the length of the postejaculatory refractory period, were significantly affected.

The importance of the role of certain areas of the anterior hypothalamus in the mediation of male sexual behavior has been indicated by studies of ablation, chemical stimulation, and intracranial self-stimulation (*1*). The purpose of our investigation was to determine whether changes in the sexual behavior of male rats could be produced by electrical stimulation of the hypothalamus. Electrodes were permanently implanted

in 30 adult male rats. After recovery the animals were tested with estrous female rats. Mounts without intromission, with intromission, and with ejaculation, with and without stimulation, were recorded. A short-pulse stimulator (Grass) delivered alternating positive and negative square waves.

Three of the subjects showed the following behavior, to varying degrees, during electrical stimulation in the 8- to 10-ma range. Within seconds after the onset of stimulation, mounting began and continued at a high rate; it stopped immediately on termination of stimulation. The grooming behavior which ordinarily follows an intromission was notably absent in all three subjects. After intromission the male's behavior remained oriented toward the female, even if no mounting occurred for some time. The postejaculatory period was significantly shortened in two of the subjects and not affected in the third. Penile erection was virtually constant during stimulation. Normal sexual behavior in the absence of stimulation was sometimes observed, but it disappeared after 5 to 10 minutes of stimulation.

The major effects were especially marked in one animal, rat C. Its median postejaculatory period was 27 seconds, as compared with the normal average of more than 5 minutes. This rat had as many as four ejaculations during one 5-minute stimulation period. We observed that the ejaculations were not merely "behavior" but were physiological ones: four sperm plugs were found on the cage floor after the first 5-minute stimulation period on one of the test days.

After preliminary studies to determine the general characteristics of the effect, two of the subjects, rats B and C, were subjected to further tests of specific variables. These included the duration of and progressive changes in sexual behavior under stimulation, the effect of variation of the stimulus animal, and changes in sexual responses as a function of variation in the intensity of electrical stimulation.

To determine changes in the effect over time, rat C was tested with an estrous female for 7½ hours, during which time the stimulation was turned alternately on and off every 5 minutes. The results are shown in Fig. 1. During the 220 minutes of stimulation, the subject had 174 mounts without intromission, 81 mounts with intromission, and 45 ejaculations. The following changes

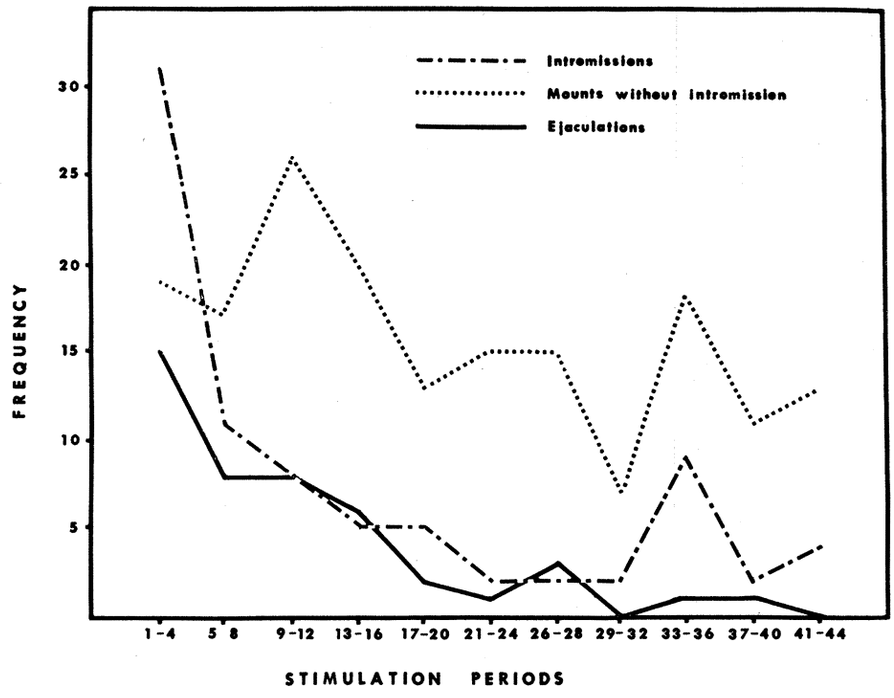

Fig. 1. Number of mounts without intromission, intromissions, and ejaculations of rat C in 44 successive 5-minute stimulation periods.

occurred over time: (i) the number of intromissions and ejaculations declined rapidly and the number of mounts without intromission declined more slowly; (ii) the latency of the first response and first ejaculation for each stimulation interval increased throughout; (iii) postejaculatory refractory periods increased in length; (iv) the number of intromissions for each ejaculatory sequence decreased; and (v) the ratio of mounts without intromission to mounts with intromission increased throughout. Very similar results were obtained in a test with rat B, in which the frequency of stimulation was somewhat different, but the total stimulation time was the same. Neither of these animals had ceased responding when the tests were terminated.

The effect of a change in the stimulus animal was investigated by testing rat C for 60 minutes (stimulation on 5 minutes, off 5 minutes) with an ovariectomized female. The subject had 15 mounts without intromission and no other sexual responses. This may be compared with the same animal's score of 28 mounts without intromission, 36 with intromission, and 19 ejaculations during the first 60 minutes of the previously described test. On the other hand, the subject without stimulation did not respond at all to the ovariectomized female. Thus, the stimulation produced some increase in sexual behavior toward a nonestrous female, but far less than toward an estrous one.

How important is the intensity of the electrical current? An intensity of 5 ma in rat B produced somewhat heightened activity and caused no increase in sexual behavior, 8-ma intensity produced a marked increase. In rat C, three intensities—4, 6, and 8 ma—were systematically varied (8 ma produced the optimal effect in this animal). The amount of sexual behavior was significantly affected, an increase in all three categories of sexual response occurring with an increase in the level of stimulation. The results were almost entirely accounted for by behavioral differences occurring at the 8-ma level; the difference under 4 and 6 ma was negligible. Furthermore, at both 4 and 6 ma, the subject showed grooming and resting responses, while at 8 ma such activity was absent. Ten milliamperes produced disorganized leaping or seizures in both animals.

An attempt was made to assess the "drive value" of the stimulation in rat C by requiring it to learn to run to the right side of a T-maze, while under electrical stimulation, for the opportunity to copulate with an estrous female. Its rate of learning was compared with that of two control subjects (without stimulation) under the same conditions. When the number of trials required to learn was computed the control rats were significantly superior.

We doubt that these results could be attributed to a low drive level under electrical stimulation. Other factors, such as aversive side effects of high-intensity stimulation or motor effects which interfere with performance could be involved. Even more probable, an unusually intense state of drive, indicated in these animals by both objective and subjective assessment, may in and of itself include aversive components which would preclude clear cut results in a test of motivated learning.

Histologies of rats B and C showed that the electrode tip was in the lateral anterior hypothalamus. In one case the tip impinged on the upper boundaries of the lateral preoptic area. More exact localization, as well as specific differentiation between positive and negative electrode placements, was not possible, thus indicating that the area involved may be quite circumscribed.

The extremely short latency of the first sexual response after onset of stimulation and the immediate termination of sexual behavior with the termination of stimulation indicate that the effect is mediated neurally rather than humorally. Apparently the electrode tip rested in an excitatory area for male sexual behavior. The stimulation of this area not only increases sexual response level but also eliminates or reduces the effects of certain inhibitory factors—evidenced by the lack of postintromission grooming and the marked curtailment of the post-ejaculatory refractory period. Exactly how the mediation of the effect takes place cannot be determined from this study, but several possibilities may be noted. Stimulation of the excitatory area may: (i) raise its firing level to a point where it overcomes normal inhibitory influences; (ii) block the action of an inhibitory mechanism directly; (iii) lower the firing threshold of the excitatory area; or (iv) produce a combination of two or more of these effects.

The results of our experiment supplement and extend the experimental data obtained with different techniques (*1*) and provide additional evidence for an anterior hypothalamic integrating system within the sexual behavior circuit (*2*).

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2. Hormone preparations used in this study to bring the females into estrus were generously supplied by Dr. Edward Henderson of the Schering Corp., Bloomfield, N.J. Acknowledgment is due Harry LeWinter for aid in implanting many of the animals used in this study; the research was supported by a grant (M1951) from the National Institute of Mental Health.

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Increase in Diffusible Auxin after Treatment with Gibberellin

Abstract. When dwarf pea plants, normal pea plants, and sunflower plants were treated with gibberellin, they yielded 3, 2, and 10 times more auxin, respectively, than untreated plants.

The effects of gibberellin on plant growth have been interpreted generally in terms of a dependency on the presence of auxin (*1*) or in terms of an inhibition of indoleacetic acid oxidase (*2–4*). The possibility that gibberellin may accelerate growth by increasing the amount of auxin produced in the tissue has not been examined thoroughly. Further investigation of this possibility was suggested by the observation that in artificially dwarf pea plants (dwarfness was brought about by treatment with the growth retarding chemical, 2-chloroethyl trimethylammonium chloride) the diffusible auxin from the stem apex is only 1/6 that of normal pea plants (*5*). Thus it was of interest to compare the diffusible auxin in stem apices of genetically dwarf and tall pea plants and sunflower plants, and to measure the effect of gibberellin treatment on the amount of diffusible auxin.

The seeds of tall pea, variety Alaska, and dwarf pea, variety Little Marvel (*Pisum sativum* L.), were soaked in distilled water with aeration for 12 hours. After sowing, they were kept in the greenhouse at 70°F during the day and 60°F at night. When the leaf at the third node was half expanded (about 10 days after sowing) the plants were selected for uniformity and treated with gibberellin (gibberellic acid, 85 percent, Eastman Organic Chemicals). Treatment consisted of application of 0.02 ml of a 0.003*M* or 0.03*M* solution to the surface of the leaf in the evening; a second treatment was given after 2 days.

When the fifth internodes were 0.5 to 1.5 cm long, the sixth node with leaf and stem apex was excised and placed on a block of 1.5 percent agar,

Table 1. Diffusible auxin obtained from the internodes of untreated pea and sunflower plants and from plants treated with gibberellin. Each figure is the average for 10 to 12 plants with its standard error. In experiments 1 and 2, 1.7×10^{-7}, 6.0×10^{-7}, and $1.7 \times 10^{-6}M$ IAA gave 3.0 ± 0.64, 9.9 ± 1.2, and 19.6 ± 0.94 degrees of curvature, respectively. In experiment 3, 1.5×10^{-7} and $5.0 \times 10^{-7}M$ IAA gave 3.6 ± 0.68 and 17.5 ± 0.71 degrees, respectively. In experiments 4 and 5, 2.0×10^{-7}, 6.0×10^{-7}, and $2.0 \times 10^{-6}M$ IAA gave 3.3 ± 1.1, 12.2 ± 1.1, and 21.7 ± 1.1 degrees, respectively. In experiment 6, 2.0×10^{-7}, 6.0×10^{-7} and $2 \times 10^{-6}M$ IAA gave 3.2 ± 0.45, 8.8 ± 1.2, and 16.1 ± 1.0 degrees, respectively. A duplication of experiment 6 gave nearly identical results.

Expt.	Gibberellin (molar)	Plant height (cm)	Curvature (degree)	Equivalent IAA (molar)
		Alaska pea		
1	0.0	7.51 ± 0.09	7.0 ± 1.4	3.5×10^{-7}
1	3×10^{-2}	14.90 ± 0.15	14.1 ± 1.1	9.2×10^{-7}
2	0.0	9.34 ± 0.18	9.8 ± 1.1	6.0×10^{-7}
2	3×10^{-2}	14.96 ± 0.15	17.7 ± 1.8	1.3×10^{-6}
3	0.0	8.70 ± 0.14	9.2 ± 1.1	2.4×10^{-7}
3	3×10^{-3}	15.30 ± 0.30	17.4 ± 1.7	5.0×10^{-7}
		Little Marvel pea		
4	0.0	5.23 ± 0.12	9.7 ± 1.8	4.5×10^{-7}
4	3×10^{-3}	11.70 ± 0.34	18.1 ± 1.6	1.3×10^{-6}
5	0.0	4.74 ± 0.12	9.2 ± 2.0	4.3×10^{-7}
5	3×10^{-3}	7.78 ± 0.38	17.4 ± 2.6	1.2×10^{-6}
		Sunflower		
6	0.0	21.94 ± 0.62	10.9 ± 1.4	8.4×10^{-7}
6	3×10^{-8}	22.38 ± 0.46	11.3 ± 1.3	9.1×10^{-7}
6	3×10^{-7}	23.52 ± 0.53	11.7 ± 1.1	9.7×10^{-7}
6	3×10^{-6}	26.10 ± 0.73	15.5 ± 1.6	1.9×10^{-6}
6	3×10^{-5}	27.92 ± 1.1	17.1 ± 1.2	2.6×10^{-6}
6	3×10^{-4}	30.77 ± 0.51	22.9 ± 1.0	7.8×10^{-6}
6	3×10^{-3}	29.88 ± 0.56	24.2 ± 0.8	9.6×10^{-6}