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}

2 by 2 by 2 mm, in a moist chamber under 220 ft-ca of fluorescent illumination. Diffusion of auxin into the agar blocks took place during a period of 2 hours after which the auxin content was assayed by the usual *Avena* curvature test. Treatment with gibberellin doubles the diffusible auxin in the normal variety and triples the amount in the dwarf variety (Table 1). Corresponding to the increase in diffusible auxin there is an increase in plant height. These data are typical for plants grown under sunny weather. If cloudy days predominated during the growing period the plants were not responsive to treatment. Such dependence on light intensity may be the explanation of other findings in which gibberellin treatment had little effect on the height of the Alaska variety of pea (*3*).

The relationship of gibberellin treatment and diffusible auxin content has been examined also in the sunflower plant, variety Mammoth Russian (*Helianthus annuus* L.). One hundred and twelve embryos were sown in soil in a wooden flat and grown under the same conditions as those used for the peas. Ten days after sowing the first foliage leaves appeared and were treated with 0.1 ml of gibberellin solution in the evening. Two days later a second treatment was given to the same leaves. When the second internode was 0.5 to 2.0 cm long the apical portion with leaves was excised, and diffusible auxin was obtained in the same way as before during a period of 100 minutes under 95 ft-ca. The curvature of *Avena* coleoptiles induced by diffusible auxin from plants treated with gibberellin is shown in Table 1, together with the height of the plant at the time of sampling. Again the increase in plant height brought about by treatment with gibberellin is associated with an increase in the diffusible auxin obtained from the apical portion of the plant. For plants treated with $3 \times 10^{-3} M$ gibberellin the diffusible auxin was 10 times that from untreated plants. From this investigation it must be concluded that as Shibaoka and Yamaki suggested (*6*): "the growth of the stem of the sunflower seedling depends closely on the quantity of auxin supplied from the leaf."

These results correspond to the finding of Nitsch (*7*) that in the shoot tips of sumac treated with gibberellic acid the amount of extractable auxin is greater than it is in untreated plants. Although Nitsch was unable to conclude that a decline in growth rate under short days was the result of a lowering of auxin level since there was also an increase in the level of endogenous inhibitors, his data for plants under long days clearly indicate that as a result of treatment with gibberellin there is a larger increase in endogenous auxin than there is in endogenous inhibitors; this increase is associated with increased growth rate.

In the indoleacetic acid oxidase-inhibitor theory of growth regulation (*2*) the increase in diffusible auxin resulting from gibberellin treatment would be explained by an increase in the inhibitor content which prevents enzymatic destruction. It is just as plausible that gibberellin treatment may directly increase the formation of auxin. Investigation of this mechanism as a possibility is in progress. Studies of the growth effects of gibberellin treatment which include the examination of auxin production may relate all responses to auxin levels (*8*).

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Salmonella Species in Turtles

Abstract. *Salmonella* spp. occur abundantly in the feces of the turtles *Testudo graeca* and *T. hermanni* but rarely in other testudines. These organisms do not seem to produce any kind of infection in the turtles. One possible explanation for the infestation is the coprophagic habit of *T. graeca* and *T. hermanni.*

In the course of inquiries into the origin of a paratyphoid infection in a child in 1952, a chance observation showed that *Testudo graeca* that were imported as household pets contained *Salmonella* spp. in their bowel in large numbers (*1*). This finding has been confirmed in other countries in northern Europe. The conditions under which these reptiles are imported make cross infection likely; however, Hirsch reported similar infestations in wild turtles caught near Haifa, Israel (*2*). Vincent, Neel, and Le Minor found that 96 percent of *Testudo graeca* in the countryside around Tangiers contained one or more serotypes of *Salmonella* (*3*); at several sites in Dalmatia (Yugoslavia) I found infestation of 70 to 80 percent in the closely related *Testudo hermanni* which I examined.

Table 1. Species of turtles of the genus *Testudo* in which *Salmonella* serotypes were found.

Species	Number examined	Number infested
T. graeca (captive)	45	41
T. hermanni (wild)	50	33
T. angulata	1	0
T. chilensis	10	1
T. denticulata	5	0
T. elegans	8	0
T. gigantea	7	0
T. nigrita	3	0
T. pardalis	3	1
T. radiata	4	2
T. sentoria	1	0
T. vicina	1	1
T. sp. (Isla Santa Cruz)	10	0

Other *Testudo* spp. have been less productive (Table 1). Some of the turtles whose feces were examined have been in zoological collections, but the majority of them were taken in the wild. The numbers of each species examined were small, but the general picture suggests that only *T. graeca* and *T. hermanni* (and their subspecies) harbor large numbers of *Salmonella.* I have found that turtles in captivity may continue to harbor these organisms for at least 9 years without any evidence of illness but the number of *Salmonella* in any individual and the number of infected individuals both decrease slowly.

One possible explanation for the infestation of *Salmonella* is that both *Testudo graeca* and *T. hermanni* are coprophagic. Where they occur wild this fact seems to be well known. I have found wild *T. hermanni* chewing horse dung, and in captivity they will eat human, bovine, or their own feces with avidity even when fresh lettuce leaves are available. After eating human feces which were naturally infected with *Salmonella typhimurium*, one tur-