measured by a decayed, missing, and filled teeth rating. This finding demonstrates that dental caries, as well as other somatic complaints, should be considered in any appraisal that attaches a general term, such as good, fair, or poor, to a person's condition of health.

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14 July 1954.

## Chemical Induction of Male Sterility in Cucurbits\*

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Male sterility in crop plants is receiving increased attention for its usefulness in the production of hybrid seed. A chemical method of inducing male sterility would have great merit in certain cucurbits where daily hand removal of staminate flowers on a large scale is economically prohibitive and the maintenance of naturally occurring sterile lines is extremely difficult. This primarily monoecious family presents a special challenge in that flower sex expression has been reportedly altered by chemical treatment (1-3). The chemical suppression of all male flowers in cucurbits would be a means of producing purely female plants.

Preliminary studies were conducted in the greenhouse with cucumbers (Cucumis sativus, vars., National Pickling and Burpee Hybrid) and squash (Cucurbita pepo, var. Table Queen). By spraying young cucumber plants after 2 to 3 true leaves had formed with 100 ppm of a-naphthaleneacetic acid (NA) or 25 ppm of 2,3,5-triiodobenzoic acid, the ratios of staminate to pistillate flowers were reduced from approximately 23:1 to 8:1 in National Pickling and from 14:1 to 2:1 in Burpee Hybrid. Results were comparable to those reported by Laibach and Kribben (1), in which the decreases in ratios were accompanied by both an increase in the number of pistillate flowers and a decrease in the number of staminate flowers. NA (100 ppm) applied to Table Queen squash when the first true leaf had fully expanded resulted in an average decrease in staminate-pistillate flower ratio from 1.47:1 to 0.4:1, with occasional plants producing no staminate flowers.

Subsequent field studies (1953) with Table Queen squash revealed that NA (100 ppm) applied as a spray when the seedlings had 1 to 2 true leaves and the treatment repeated 10 days later (4 to 5 true leaves) delayed the appearance of staminate flowers, and treated plants produced exclusively pistillate flowers for 8 days prior to the opening of any staminate flowers. During the fall, winter, and spring months (1953– 54), additional chemicals, including maleic hydrazide (4), were evaluated. Table Queen squash (Stock No. D0421, Ferry-Morse Seed Co.) was used as the test plant (2) and was grown in the greenhouse at  $68^{\circ}$ F night temperature and a photoperiod of 16 hr. Such an environment normally favored the early production of staminate flowers followed by the presence of both staminate and pistillate types, as described by Nitsch et al. (2).

A remarkable suppression of staminate flower buds was obtained with maleic hydrazide (MH); the selective inhibition was comparable to that reported by Moore (5) and Navlor (6) for maize. Several spray patterns successfully induced male sterility during the 3- to 4-wk interval that each crop was allowed to flower in the greenhouse. Dipping or spraying the plants in a solution containing 250 or 350 ppm of MH when the first true leaf was expanding and followed by a second treatment when 4 to 5 true leaves had developed resulted in plants that produced the usual number (8 to 10) of pistillate flowers in normal spatial arrangement, with no staminate flowers. Characteristic flowering patterns following treatment showed only rudimentary staminate flower buds or bud initials at the nodes in which they normally occurred. In other instances nonfunctional staminate male flower buds developed in which the sepals were abnormally enlarged (Fig. 1B) and some flowers eventually opened, but the androecia failed to develop (Fig. 1D), having aborted in a manner similar to that of the male-sterile mutant in winter squash described by Scott and Riner (7). It was further found that a single application of MH (250, 350, or 500 ppm), applied when 1 to 2 true leaves had developed, resulted in a high percentage of plants that produced only pistillate flowers.

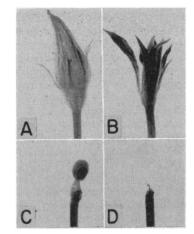


Fig. 1. Effect of maleic hydrazide (MH) on staminate flower bud development in squash (var. Table Queen). ( $\mathcal{A}$ ) and ( $\mathcal{C}$ ) Normal buds intact and with the sepals and petals removed to show the fully developed androecia. ( $\mathcal{B}$ ) and ( $\mathcal{D}$ ) Buds from plants previously treated with MH (350 ppm), intact and with the sepals and petals removed. Such buds were typical of treated plants, aborting prior to anthesis and containing no viable pollen.

Repeated (4 to 5) applications at 5- to 7-day intervals of 100 ppm, beginning at the time of cotyledon expansion and continuing until 4 to 5 true leaves had developed, effectively suppressed staminate flower bud development in many plants. The pistillate flowers on maleic hydrazide-induced male-sterile plants in almost all instances appeared normal and were fertile. When pollinated the fruit developed normally and produced abundant quantities of viable seed. Investigations now in progress suggest that the results reported here for Table Queen squash can be reproduced in many varieties of C. pepo and in other cucurbitaceous species and may have widespread utility in making the production of hybrid seed an economic reality.

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9 July 1954.

# Recovery from the Failure to Eat Produced by Hypothalamic Lesions

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In two recent papers, Anand and Brobeck (1) have reported that bilateral lesions of the lateral hypothalamus can cause rats and cats to refuse food and thus starve to death. The effective lesions in these cases were 1 mm above the floor of the brain and 2 mm off the midline on each side, at the same anterior-posterior setting as the ventromedial lesions that produce overeating (hyperphagia). Two of the cats used in these experiments were kept alive by tube-feeding postoperatively. One cat began eating after 7 days of refusing food, but its intake was always below normal. The second cat refused food for 6 wk of tubing and at this time was sacrificed for histological purposes.

The experiment reported here (2) first of all verifies Anand and Brobeck's findings that lateral hypothalamic lesions can cause rats to refuse to eat and starve to death. Second, the present experiment shows that a recovery of eating behavior can be brought about in such animals.

Bilateral lesions in the lateral hypothalamus were made with the aid of a stereotaxic instrument (3) so as to produce animals that refused laboratory food (Purina Laboratory Chow Meal) and water for at least 5 days postoperatively. These starving animals were then divided into two groups, one of which was maintained with only laboratory food and water and

was thus allowed to starve to death, while the second group was maintained with a nutritive, fluid diet (4)administered by stomach-tube. Within a few days after tubing was begun, these animals were offered a number of special foods in an effort to induce them to eat.

All 40 operated rats showed some failure to eat following the lateral hypothalamic lesions: 9 rats, used in preliminary work, refused to eat for a period of 6 to 9 days postoperatively but then recovered eating behavior spontaneously; 17 rats that were not tubed or offered special food refused to eat laboratory food and to drink for 6 to 15 days and thus starved themselves to death in this postoperative testing period; 14 rats, maintained with tubing and special foods, recovered eating and drinking behavior within 6 to 65 days.

The course of recovery of the eating behavior is llustrated in Fig. 1 by data on one animal. All 14 recovered animals showed the same general course of recovery of eating behavior following lateral hypothalamic lesions. There is an initial period of complete refusal to eat. Following this, animals will accept only evaporated milk or, somewhat less readily, milk chocolate. Only later will they accept water; and only after they have been drinking water will they eat the regular laboratory food. Individual differences in the time spent in each of these stages of recovery are very great, but the sequence of stages is almost invariable.

Tubing the animals may retard the recovery of eating somewhat; yet in a number of cases where tubing was stopped as soon as the animals began to eat the special foods, the final course of recovery was also prolonged. Starvation itself is never a sufficient inducement to eat, for even after animals had been eating evaporated milk and chocolate, they still refused to eat laboratory food when deprived of these special foods, even to the point of great weight loss.

Foods other than evaporated milk or chocolate, of

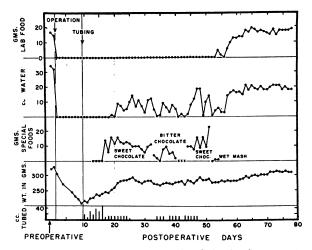


Fig. 1. Course of recovery of eating behavior in one rat, following complete refusal to eat produced by hypothalamic lesions. The x's show when 25 g of evaporated milk was offered and eaten.