after reinnervation which had not been seen before. It is not certain, however, whether this corpuscle had arisen anew; the corpuscle lay in fat and may have escaped our notice earlier.

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$$\frac{d\text{Velocity}}{d\text{Diameter}} = 6 \times 10^{6}\ \text{sec}^{-1}\ (37°\text{C})$$

[J. B. Hursch, *Amer. J. Physiol.* **127**, 131 (1939)]. Velocity data were multiplied by 1.8 to correct to 37°C [J. Tasaki and M. Fujita, *J. Neurophysiol.* **11**, 311 (1948)].
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14. The experiments with hypogastric fibers were done after those with IM fibers, when we had become better at joining severed nerves. This probably accounts for the higher incidence of hypogastric corpuscle innervation.
15. C. Dijkstra's [*Z. Mikrosk. Anat. Forsch.* **34**, 75 (1933)] elegant histological studies on transplanted Grandry corpuscles of the duck skin suggest that these receptors may receive foreign axons too. However, these studies, purely histological, do not show whether the reinnervated receptors were functioning [see also M. Jacobson, *Developmental Neurobiology* (Holt, Rinehart & Winston, New York, 1971), p. 288].
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19. We thank Drs. M. Jacobson and S. J. Socolar for valuable discussion. Supported by research grants from the National Institute of Neurological Diseases and Stroke and from the National Science Foundation. Part of this work was done at the Cell Physics Laboratory, Physiology Department, Columbia University, College of Physicians and Surgeons.

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Field and Vegetable Crop Mutants with Increased Resistance to Herbicides

Abstract. *Wheat mutants with increased seedling resistance to terbutryn (2-*tert*-butylamino-4-ethylamino-6-methylthio-*s*-triazine) and tomato mutants with increased resistance to diphenamid (*N,N*-dimethyl-2,2-diphenylacetamide) were selected by the experimenters out of populations grown from seeds treated with ethyl methanesulfonate. Induced mutations may thus provide a tool for breeding crop cultivars with increased resistance to certain herbicides.*

There is extensive use of selective herbicides, that is, weed killers which control the weed population within a certain agricultural crop without causing economic injury to the crop. Their selectivity may be due to differences between the crop and the weeds in one or more of the following processes: uptake, translocation, inactivation, or breakdown of the herbicide. However, these differences often are small and dependent on climatic and soil factors. Consequently, slight changes in dosages, as well as effects of herbicide-environment interaction may result in damage to the crop or insufficient weed control. Moreover, no appropriate herbicides have yet been found for the control of the important weed species of some crops.

Intercultivar differences in resistance to herbicides have been reported (*1*). We investigated the possibilities of breeding crop cultivars with improved resistance to herbicides by means of induced mutations.

Mutations were induced in the spring wheat cultivar 'Alpha' and in the tomato cultivar 'VF 145-B 7879' by soaking the seeds at room temperature in 8 m*M* ethyl methanesulfonate (EMS) for 15 hours, and in 64 m*M* EMS for 24 hours, respectively. The M_2 populations contained plants with variegated leaves or other morphologic abnormalities. Seedling resistance of wheat to terbutryn (2-*tert*-butylamino-4-ethylamino-6-methylthio-*s*-triazine) and of tomato to diphenamid (*N,N*-dimethyl-2,2-diphenylacetamide) was tested in the M_3 through M_5 and M_2 through M_4 generations, respectively (*2*). Seedlings were grown in loamy sand soil in undrained square plastic pots (17 by 17 by 7 cm). Terbutryn [1 part per million (ppm), by weight], or diphenamid (40 ppm) were applied by mixing them thoroughly with the soil prior to potting, on the day before sowing the seeds. In the replicated trials, six to ten seedlings of each mutant line, and an equal number of seedlings of the original cultivar, were grown in two parallel rows in each pot.

Screening of 50,000 M_3 plants, descending from 2,800 M_2 families, yielded 588 relatively resistant wheat seedlings. No seedlings resistant to the herbicide were found in a parallel screening of 10,000 plants of the original cultivar. Seven lines from two families were selected in M_4 by the experimenters (Fig. 1) and were then tested in a trial with ten replications in the M_5 generation. Every day, throughout the period of increasing seedling mortality, the percentage of surviving seedlings was significantly higher for

Table 1. Effect of diphenamid, added to soil prior to seed sowing, on the fresh weight of 25-day-old tomato seedlings.

Plant	Weight per seedling (mg)	
	Untreated	Treated
Original cultivar	728	433
M_4 lines	674	503
Standard error	26.7	26.7

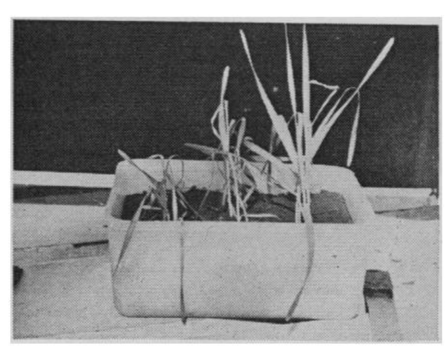

Fig. 1. Wheat seedlings grown in soil treated with terbutryn. Right, resistant M_4 line; center, original cultivar; left, susceptible M_4 line.

the M_5 lines than for the original cultivar. The daily seedling mortality, as measured by the slope of the probit regression line (*3*), was lower for these mutant lines than for the original cultivar.

A screening of 20,000 M_2 tomato seedlings revealed 120 resistant individuals. In 23 M_3 progenies, 117 resistant seedlings were found, from which 12 M_4 lines were selected. A trial, with ten replications, of four pooled M_4 lines showed a 25 percent reduction in seedling weight of these lines, grown in soil treated with diphenamid, whereas the reduction in seedling weight of the original cultivar was 40 percent (Table 1).

These results indicate that increased resistance of crops to herbicides may be obtained by selection from mutagenically treated populations. This could eventually improve the efficiency of herbicides, and also restrict their applications to those which are satisfactory from the public health viewpoint. The resistant mutants will also provide suitable plant material for studies of the inheritance and the mechanisms of selectivity. A better understanding of selectivity could assist in deliberate synthesis of new herbicides with specific characters.

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Genetic Determination of Phenotypic Variation in Sickle Cell Trait

Abstract. *Two genetic sources of variation influence the percentage of sickle cell hemoglobin found in heterozygotes. One factor is strongly related to the percentage of hemoglobin S in the carrier parent and appears to be determined by sickle hemoglobin isoalleles, whereas the other is related to racial background and may well be polygenic.*

In heterozygous carriers of sickle cell trait, the proportion of abnormal hemoglobin varies from person to person but is usually less than 0.5 (*1*). To investigate the causes of individual variation, replicate measurements were made of the proportion of sickle hemoglobin (HbS) in 272 heterozygous parents and children in 67 families that were segregating for sickle cell trait. The subjects were members of a migrant population from northeastern Brazil that was of triracial origins (*2*). A single blood sample was drawn from each family member. The red cells were washed three times with saline and then frozen in a glycerol citrate solution and stored at below −60°C until hemoglobin typing and quantitation were performed. The proportions of hemoglobins A, S, and A_2 were estimated by the starch gel electrophoresis method of Sunderman (*3*). Duplicate determinations of each hemolysate were made on separate gels, and yielded a standard error for the percentage of HbS of ± 0.9 percent.

When the percentage of HbS was expressed as the proportion of β-chain-containing hemoglobin, that is, HbS/(HbA + HbS), a negatively skewed frequency distribution was observed with a range of 27 to 50 percent and a major mode at 44 percent (Fig. 1). Analysis of the results from 184 children in the sample showed that the variance among families was more than three times larger than the variance within families ($F = 3.42$; d.f. 66,117) while the within-family variance was, in turn, more than eight times as great as the variation between replicate determinations ($F = 8.72$; d.f. 117,184). Both of these differences were significant at the 0.001 confidence level, and suggest that a major component of the observed variation is genetically determined.

Linear regression analysis showed that there was a highly significant relationship between the weighted mean value of the percentage of HbS in children and the percentage observed in their carrier parents ($b = 0.44 \pm 0.10$, 65 d.f., $P < .001$). This parent-offspring regression leads to a heritability estimate of 0.88, which implies that 88 percent of the variation in the proportion of HbS is additive. The intraclass correlation coefficient among siblings was 0.47, giving a heritability estimate of 0.94. The close agreement of these two estimates provides little evidence that the observed genetic influence includes a major dominance component.

The effects of age, sex, and a racial-admixture index on the percentage of HbS were also investigated by multiple regression analysis, and only in the case of race was a highly significant effect observed ($b' = 0.282 \pm 0.079$, 268 d.f., $P < .001$) (*4*). Each individual in the study had been classified into one of seven racial admixture groups by a single trained observer who had no knowledge of the hemoglobin typing results. The classification was based on a subjective evaluation of abdominal skin pigmentation, hair color and type, and physiognomy. Analysis of the blood typing results showed that the categories did, in fact, correspond to increasing degrees of Negro ancestry (*5*). The overall effect of the racial variable was to reduce the average percentage of HbS in carriers classified as

Table 1. Effect of racial class on the percentage of HbS in sickle cell heterozygotes.

Statistical test	Regression coefficient ± S.E.	Degrees of freedom	P
Among-sibships regression of racial class on percent of HbS	− 0.43 ± 0.14	65	< .01
Within-sibship regression of racial class on percent of HbS	− 0.51 ± 0.23	119	< .05
Regression of parental racial class on percent of HbS	− 0.05 ± 0.13	75	> .05