

inheritance. A portion of the seedling data is given in the following table:

Cross	Observed No.		Expected No.		$\chi^2$	P value
	R <sup>a</sup>	S <sup>b</sup>	R	S		
W64AxRS	0	20				
(W64AxRS)xRS	262	251	256.5	256.5	0.236	0.50-0.75
(W64AxRS)F <sub>2</sub>	186	522	177.0	531.0	0.610	0.25-0.50

<sup>a</sup>Resistant: small chlorotic lesions with limited fungus sporulation

<sup>b</sup>Susceptible: large tan, oval to rectangular lesions with abundant fungus sporulation

The symbol rhm is proposed for the recessive gene conditioning this chlorotic-lesion resistance to H. maydis.

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1. The Adh<sub>1</sub><sup>FCM</sup> "Operon".

Earlier studies have established that the level of alcohol dehydrogenase activity in the plant is limited by the concentration of a specific factor which is essential for the activity of the Adh gene (Schwartz, 1971). Although various Adh<sub>1</sub> alleles have been shown to differ in their ability to compete for the limited factor, enzyme level in segregating kernels and seedlings is constant and independent of Adh<sub>1</sub> genotype as long as the Adh<sub>2</sub> gene is not active. The Adh<sub>2</sub> gene, which specifies a relatively inactive enzyme, competes with the Adh<sub>1</sub> gene for the limited factor.

The  $\text{Adh}_1^{C^m}$  allele and the  $\text{Adh}_1^{\overline{FC^m}}$  duplication, which consists of two cistrons that specify an F and a  $C^m$  polypeptide, were found segregating with an  $\text{Adh}_1^F$  allele in a line of maize from Colombia, South America (Schwartz, 1966). The  $\text{Adh}_1^{C^m}$  allele produces a stable but relatively inactive enzyme.  $\text{Adh}_1^F/\text{Adh}_1^{C^m}$  heterozygotes exhibit considerably less enzyme activity than their  $\text{Adh}_1^F/\text{Adh}_1^F$  sibs, as expected, since much of the limited factor is used in activation of the  $\text{Adh}_1^{C^m}$  gene which makes inactive enzyme. The two cistrons in the duplication are very tightly linked. No crossovers were detected between the two loci in over 4000 progeny. This analysis involved backcrossing an  $\text{Adh}_1^{\overline{FC^m}}/\text{Adh}_1^S$  heterozygote to  $\text{Adh}_1^S/\text{Adh}_1^S$  and individually scoring the progeny by starch gel electrophoresis to determine if in any cases the pseudoalleles were transmitted separately. Recent studies suggest that the two cistrons may in fact be part of a single "operon". They act as a unit in that they contain only a single activation site which competes for the limited factor. The reasoning behind this conclusion is as follows. If the cistrons in the duplication each had an activation site that competed for the limited factor,  $F_2$   $\text{Adh}_1^{\overline{FC^m}}/\text{Adh}_1^{\overline{FC^m}}$  progeny should show much less enzyme activity than the  $\text{Adh}_1^F/\text{Adh}_1^F$  sibs since in the former the limited factor would be used in the synthesis of inactive  $C^m$  as well as active F polypeptides. This is definitely not the case. Approximately equal enzyme levels are found in both genotypes. Since the  $C^m$  subunit stabilizes the F subunit in a heterodimer (Schwartz and Laughner, 1969), the enzyme levels vary slightly in different tissues as a result of differential enzyme turnover.

The operon hypothesis was tested by comparing the relative concentrations of F and  $C^m$  protomers produced in  $\text{Adh}_1^F/\text{Adh}_1^{C^m}$  vs  $\text{Adh}_1^{\overline{FC^m}}/\text{Adh}_1^{\overline{FC^m}}$  genotypes. Use was made of the method of high resolution immunoelectrophoresis (Schwartz, 1972) which permits comparison, at the protein level, of the relative amount of polypeptides produced by two alleles in a heterozygote.  $\text{Adh}_1^F$  competes better than  $\text{Adh}_1^{C^m}$  in seedlings, and less  $C^m$  than F polypeptides are produced in  $\text{Adh}_1^F/\text{Adh}_1^{C^m}$  genotypes. If the  $\text{Adh}_1^{\overline{FC^m}}$  duplication has a single activation site which binds the limited factor and a bicistronic messenger RNA molecule is produced, one would predict that equal amounts of F and  $C^m$  polypeptides would be synthesized in the duplication homozygotes. This is clearly the condition which is observed (unfortunately the

immuno-electrophoretograms cannot be reproduced in the News Letter).

#### References:

- Schwartz, D. and T. Endo, 1966. Alcohol dehydrogenase polymorphism in maize - simple and compound loci. *Genetics* 53: 709-715.
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- Schwartz, D., 1970. Genetic control of alcohol dehydrogenase - a competition model for regulation of gene action. *Genetics* 67: 411-425.
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#### 2. Regulation of alcohol dehydrogenase (ADH) activity in developing maize endosperm.

In immature seeds, high levels of ADH activity are found in both the embryo and the endosperm. During late stages of maturation, the activity decreases strikingly in the endosperm but remains high in the scutellum. The level of activity of an enzyme depends upon the rate at which it is synthesized, as well as the rate at which it is degraded or inactivated. The Adh<sub>1</sub> gene is probably not active in mature endosperm, but gene repression can not account for the rapid disappearance of ADH activity during the late stages of maturation. The rapid drop in activity must be a result of inactivation of preexisting enzyme. This process has been shown to involve two components, both present in the mature endosperm, which readily inactivate ADH in vitro. One component is a protein and the other is dextrin; neither has any effect by itself.

In the tissues which contain active ADH, only one of the components can be found. The embryo contains only the protein. The immature endosperm contains a high level of dextrin, but the protein component can not be detected until the stage of development at which ADH activity rapidly declines.

In order to be effective in the inactivation process the dextrans must be in a certain size range. Large molecules such as starch, glycogen, and even commercially available dextrans are ineffective unless hydrolyzed; prolonged hydrolysis reduces effectiveness. Furthermore, the dextrin must