Gene Evolution in Polyploid Wheat. E. R. SEARS (Columbia, U.S.A.).

Where genes are duplicated, as in polyploids, mutations to a different level of activity–even to the zero level—may not be detectable, because of the masking action of the duplicates at other loci. Harland [1] suggested that a duplicated gene could escape this masking effect by mutating to an allele with a divergent function. An example of this kind of mutant may be Neatby's virescent (*v*) on chromosome 3B (III) of common wheat, a hexaploid.

Increase in dosage of *v* from 2 to 3 results in greater abnormality (less chlorophyll), whereas increased dosage of the normal allele *V* or either of the homœologous (related) chromosomes 3A (XII) and 3D (XVI) shifts the phenotype toward normal. This suggests that *v* is antimorphic to *V* and that *V* (or *V1*) has duplicates, *V2* and *V3*, on chromosomes 3A and 3D.

That *V3*, and therefore presumably *V1* and *V2*, are involved in chlorophyll production was shown by inducing a deficiency for *v1* and combining this with nullisomes 3A and 3D, respectively, thus reducing the dosage of *V* from the normal 6 to only 2. Nullisomic-3D plants (*V2V2*) had normal chlorophyll, whereas nullisomic-3A plants (*V3V3*) were of reduced chlorophyll content. Thus *V2* is more potent than *V3* in promoting chlorophyll development. On the other hand, *V3* is more effective than *V2* in reducing the expression of *v*. Although *v* may be a mutant with a divergent function, another explanation is possible; namely, that it is less efficient than *V* in producing chlorophyll but more efficient in competing for substrate.

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1. *Biol. Rev.* **11,** 1936.

Proc. XI Int. Congr. Genet. 1: 123-124. 1963.