The Mutation Process in Hexaploid Wheat

E. R. Sears

U. S. Department of Agriculture and University of Missouri

As pointed out in 1932 by J. B. S. Haldane in his book, "The Causes of Evolution", and again in 1934 by S. C. Harland (J. Genet. 29), there is available to polyploids a possibility for evolution through mutation that is largely denied to diploids. This is the change of an allele to one having a different function. Such a change cannot occur in a strict diploid, because in taking on a new function a locus presumably relinquishes its old function, and this, assuming each gene to be essential, is lethal. In a polyploid, however, the duplicate locus carries out the original function, leaving the mutating gene free to change in any way whatever, so long as the new form does not upset the vital processes of the plant and cause lethality. In assuming a new function the gene escapes from the masking action of its duplicate and so is able to bring about a change in phenotype. If it is effective in one dose, the mutant is classified as dominant ; if it requires two doses to reach its threshold of activity, it is recessive.

Evidence is accumulating that hexaploid wheat is genetically largely auto- rather than allopolyploid : that is, most of its genes are present at triplicate loci. This means there is virtually no possibility for wheat to evolve through quantitative changes in genes, for there are relatively few triplicate series which do not reach a plateau of effectiveness at a dosage level of four or less. In other words almost any locus may be completely deleted without a detectable phenotypic change resulting. Almost the only avenue open for the evolution of wheat appears to be mutation to alleles of different function.

An example of this type of mutation is thought to be the spontaneous mutant, Neatby's virescent, v_1, on chromosome 3B. Its normal allele, V_1, has near-duplicates V_2 and V_3 on chromosomes 3A and 3D, respectively. The V genes are involved in chlorophyll production, as evidenced by the fact that V_3V_3 (deficient for V_1 and V_2) is virescent. The mutant allele v_1 competes with the V genes in such fashion that one dose of v_1 neutralizes approximately one dose of V. The various possible explanations for the nature of v_1 action seem to involve some sort of altered function.

It seems clear that hexaploid wheat must have a great reservoir of triplicated genes, all of which are available for mutation. Some of these mutations could well be of practical value. The mutation process, however, cannot be one that merely involves loss of genes, but must consist of intragenic changes. It appears that chemical mutagens can induce such changes, for Shama Rao and Sears obtained a recessive *chlorina* mutant following treatment with ethyl methane sulfonate, as well as numerous presumed dominant mutations that resulted in chlorophyll-defective sectors in M_1 ; and Konzak and others have had similar success with chemicals. These chlorophyll mutations cannot be mere deficiencies or duplications, for all possible deficiencies and duplications are already at hand in the 21 nullisomics and tetrasomics that have been obtained in the common-wheat variety Chinese Spring, and none of these is defective with respect to chlorophyll.

Ionizing radiations have been notably ineffective in inducing simply inherited chlorophyll mutations in hexaploid wheat. This and other evidence indicates that radiation produces gross changes almost exclusively.

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