M. Feldman

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THE ORIGIN AND FUTURE OF WHEAT*

E. R. Sears

Agricultural Research Service
U. S. Department of Agriculture
and
Agronomy Department
University of Missouri
Columbia, Missouri 65201

The sources of two of the three genomes of common wheat, *Triticum aestivum* L. em. Thell., have been determined beyond reasonable doubt. Genome A came from wild diploid wheat, *T. monococcum* L., which was one parent of a cross that gave rise to wild tetraploid wheat, *T. turgidum* L. *T. turgidum* (genome formula AABB) was taken into cultivation in the Near East about 10,000 years ago and was soon converted into a cultivated (non-fragile) form (1,2). Eventually the cultivated tetraploid hybridized with the weed *T. tauschii* (Coss.) Schmal. (= *Aegilops squarrosa* L. = DD) to produce hexaploid wheat, AABBDD (3-5). This happened about 8,000 years ago (1,2).

The origin of the B genome is presently unknown. From about 1958 through the 1960's, it was believed to have come from *T. speltoides* (Tausch) Gren. ex Richter (= *Ae. speltoides* Tausch), whose morphological characteristics were suitable and which grows in the proper area (6,7). However, *T. speltoides* has the wrong chromosomes (8,9) and the wrong cytoplasm (10).

Where did the B genome come from: There are at least four possibilities:

1. From a species that is now extinct.
2. From a species that has not yet been discovered.
3. From a diploid wheat; that is, the B is a modified A genome (11). However, the chromosomes of Johnson's assumed B-

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genome donor pair with those of the A genome, not the B (12, 13).

4. From two or more species. This is the view currently held by most students of the wheat group. By means of introgression the B genome of the original tetraploid wheat could have become greatly modified. Such introgression would most likely have occurred through hybridization of two or more tetraploids that both had the A genome but that had different second genomes (14). The common or "pivotal" A genome would have assured fertility of the hybrids, enabling segregates to arise having mixtures of chromosomes or chromosome segments from the different second genomes.

Although no breakthrough seems imminent concerning the origin of the B genome, there is ground for optimism that the problem will eventually be solved. One promising angle is to identify the donor of the B through the characteristics of its cytoplasm. It seems clear that the donor of the B genome also provided the cytoplasm of tetraploid wheat, for the tetraploids are male sterile when when put in the cytoplasm of diploid wheat. If cytoplasms are as slow to change as most people seem to think, then determination of which diploid species has the some cytoplasm as tetraploid wheat may reveal the B-genome donor. At least, this approach should eliminate all but a few of the potential donors. Several methods are now available for deciding whether two cytoplasms are identical. The traditional way, determining whether they interact in the same way with various sets of chromosomes, is still fruitful. A promising new way is to compare the fraction-1 protein of the respective chloroplasts (15). Another, probably more discriminating technique is to compare extracted, digested DNA of mitochondria (16) or chloroplasts.

Meanwhile, cytogenetic methods can presumably show the nature and degree of homology of B-genome chromosomes with those of various diploid species. The chromosomes of almost any diploid relative can be added, one at a time, to hexaploid wheat and studied for pairing with their B-genome homoeologues. It may be desirable in some cases to force pairing (by deleting or suppressing Ph, the inhibitor of homoeologous pairing) and to recover recombined chromosomes for analysis.

Whether or not the donor of the B genome is ever identified, the relatives of wheat are of potentially great importance to the future of wheat. It is to these species, mostly wild inhabitants of western Asia and the Mediterranean area, that we must increasingly turn for new genes for resistance to diseases and insects, for tolerance of drought and poor soil, and eventually even for increased productivity. Making these interspecific and intergeneric transfers of genes is not easy, but it can be done, and we can always hope to develop better methods. For example, a reliable technique for producing plants from microspores would be very useful.

Before expending much energy on exploitation of the relatives of wheat, most breeders will want to take advantage of the considerable genetic variability still available in wheat itself. Here they will be aided by a cytogenetic project being conducted by the European Wheat Aneuploids Cooperative (Coordinator: Dr. C. N. Law, Plant Breeding Institute, Cambridge, England). This project has as its goal the cataloguing of all the genes in wheat that have quantitative effects--cataloguing them not only as to their individual effects and their interactions, but also as to where they are located on the chromosomes. The successful completion of this program could result in the conversion of wheat breeding from an art to a science.

With the current concern for protein and amino acid levels, it is interesting to note that deficiency for the short arm of chromosome 2A results in about a 50% increase in percentage of protein, with no apparent decrease in seed weight (17). This of course suggests that high-protein mutations may be easily obtained in wheat.

What the future of Triticale and hybrid wheat may be is difficult to predict. Triticale (the amphiploid of tetraploid wheat and diploid rye) is reportedly grown on several hundred thousand acres, and many knowledgeable people are optimistic about its possibilities. Enthusiasm for hybrid wheat, on the other hand, seems to have largely subsided. One of the problems with hybrid wheat is that full seed sets are almost never obtained on the male-sterile lines, even under the best of circumstances, and sets may fall to disastrously low levels if weather conditions are unfavorable. This is surely due in large part to the small anthers of wheat and the extremely short lifetime of its pollen. Adapted as wheat is to self-pollination, it is presumably uniform with respect to these characters; but rye has very large anthers and long-lived pollen. Why not, then, take full advantage of the rye component of Triticale and concentrate on hybrid Triticale rather than hybrid wheat? It may be argued that since Triticale is already a hybrid, no additional vigor can be expected from crosses between Triticale cultivars. But wheat itself is a hybrid in the same sense, and some wheat hybrids are exceedingly vigorous. Actually, since rye is self-sterile and therefore an obligate out-pollinator, which shows pronounced depression upon inbreeding, there is particular reason to expect better performance when the rye chromosomes are heterozygous. Also, since it is now clear that vigorous lines can be obtained that have one or more D-genome chromosome pairs and only six or fewer pairs from rye, perhaps combinations can be found that have mostly the wheat D genome and only those rye chromosomes that are required for large anthers and long-lived pollen.

I. REFERENCES

1. Helbaek, H., *Econ. Bot.* 21, 350 (1966).
2. Helbaek, H., *Mem. Mus. Anthrop. Univ. Michigan* 1, 383 (1969).
3. McFadden, E. S., and Sears, E. R., *Rec. Genet. Soc. Amer.* 13, 26 (1944).
4. McFadden, E. S., and Sears, E. R., *J. Hered.* 37, 81 and 107 (1946).
5. Kihara, H., *Agri. and Hort.* 19, 889 (1944).
6. Sarkar, P., and Stebbins, G. L., *Amer. J. Bot.* 43, 297 (1956).
7. Riley, R., Unrau, J., and Chapman, V., *J. Hered.* 49, 91 (1958).
8. Kimber, G., and Athwal, R. S., *Proc. Nat. Acad. Sci. USA* 69, 912 (1972).
9. Gill, B. S., and Kimber, G., *Proc. Nat. Acad. Sci. USA* 71, 4086 (1974).
10. Maan, S. S., and Lucken, K. A., *J. Hered.* 62, 149 (1971).
11. Johnson, B. L., *Can. J. Genet. Cytol.* 17, 21 (1975).
12. Dvořák, J., *Can. J. Genet. Cytol.* 18, 371 (1976).
13. Chapman, V., Miller, T. E., and Riley, R., *Genet. Res.* 27, (1976).
14. Zohary, D., and Feldman, M., *Evol.* 16, 44 (1962).
15. Chen, K., Gray, J. C., and Wildman, S. G., *Science* 190, 1304 (1975).
16. Levings, C. S., and Pring, D. R., *Science* 193, 158 (1976).
17. Bozzini, A., and Giacomelli, M., *Genetics* 74, s29 (1973).