# DOSAGE EFFECT OF THE SPELTA GENE q OF HEXAPLOID WHEAT[1]

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A dosage effect of chromosome 5A (formerly IX) of common wheat, *Triticum aestivum* L. emend Thell. ssp. *vulgare*, has been well established. With increasing dosage from monosomic to tetrasomic, the phenotype changes from speltoid to normal *vulgare* type (squarehead) to subcompactoid to compactoid (HUSKINS 1946; SEARS 1952, 1954) (Figure 1 and Table 3). This is due to the pleiotropic gene *Q* located near the distal end of the long arm of the chromosome (UNRAU, SMITH and MCGINNIS 1950; MACKEY 1954). The two major effects of *Q*, speltoid suppression and squareheadedness, were thought to be controlled by two different genes, *k* and *q*, until MACKEY (1954) found that the two genes were actually identical and incorporated them into a single dominant gene *Q*. It is the gene *Q* which makes ssp. *vulgare* different from ssp. *spelta*, and spontaneous or induced speltoid mutation (similar to ssp. *spelta*) in ssp. *vulgare* is considered to be a deficiency for a segment including *Q* (SMITH, HUSKINS and SANDER 1949; MACKEY 1954). MACKEY also suggested that the difference between the *spelta* and speltoid effects is simply due to the modifying effect of genes at other loci.

In ssp. *vulgare* there are many varieties which are squarehead. The difference between squareheadedness and non-squareheadedness is also attributed to genes that modify *Q* rather than to any difference in *Q* itself (SEARS 1956). SEARS (unpublished) demonstrated this by substituting chromosome 5A from the non-squarehead strain "Hope" into the squarehead variety Chinese Spring, using a monosomic method (SEARS 1953). The resulting plant was as squarehead as Chinese Spring. Thus Chinese Spring provides a background that leads to squareheadedness in the presence of two *Q* genes. This dependence of squareheadedness on modifiers fits well with the fact mentioned by HUSKINS and SANDER (1949) that there is little difference in glume characters among speltoid mutants, although the parent strains show wide variation.

The dosage series of the *spelta* gene *q* (an allele of *Q*) was also obtained up to four doses (SEARS unpublished). The gene behaved as a null allele. This result tends to favor MACKEY's (1954) suggestion that ssp. *spelta* originated as a speltoid mutation—i.e., as a deficiency for *Q* locus. As SEARS (1954, 1956)

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pointed out, however, other evidence supports the origin of $Q$ from $q$, and in this case $q$ could not be a deficiency. The reason why $q$ behaved as a null allele was thought to be that it is duplicated on one or more other chromosomes. $Q$ is said to be, in the variety Chinese, incompletely dominant and largely ineffective when hemizygous, although MACKEY considered it to be dominant in his materials.

The lack of dosage effect of $q$ up to four doses still leaves two possible explanations other than deficiency: (1) $q$ is an amorph, capable of giving rise to $Q$ but not itself having any function; or (2) $q$ has an effect so feeble that more than four doses may be necessary to cause a detectable phenotypic effect.

KUCKUCK (1959) suggested that ssp. *vulgare* originated through duplication of $q$ to produce $Q$, because of the fact that naked cultivated types appeared in the progeny of a cross between ssp. *macha* and ssp. *spelta* and a cross between two *spelta* strains. Cytological analysis had not yet been completed, but various structural changes of the chromosome concerned were thought to be the cause.

Since the peculiarities of the $Q$ and $q$ genes depend on the polyploid nature of common wheat, an understanding of their nature may provide important information concerning gene action and polyploid genetics. The present study was chiefly concerned with the question of whether or not $q$ is simply a deficiency for $Q$, and if not, what kind of gene it is and whether it is duplicated on the homoeologous chromosomes. The study also sought to locate genes that modify the effects of $Q$ and $q$.

Since the most important effect of $Q$ is to produce the *vulgare* complex of characters, it is appropriate to call $Q$ the *vulgare* gene.

## MATERIALS AND METHODS

The investigations utilized substitution and aneuploid strains which were provided by DR. E. R. SEARS.

"*Spelta*-5A" (2n = 42) (Figure 1f) is a line with chromosome 5A from *T. aestivum* ssp. *spelta* (white spring spelt) substituted into a Chinese Spring background. This substitution line was established by using the methods described by SEARS (1953). Because seven backcrosses were made to Chinese Spring, the line is assumed to be almost entirely Chinese Spring for the background chromosomes. Although the spike superficially resembles *spelta*, it is actually speltoid, for the rachis is almost as tough as Chinese Spring, and the seeds are loosely held by the glumes. This tends to confirm MACKEY's conclusion that the phenotypic difference between *spelta* and speltoid depends on modifiers. In this paper, the phenotype of *spelta*-5A and its derivatives will be designated speltoid.

"Tetra-*spelta*-5A" (2n = 44) (Figure 1h) is a tetrasomic 5A strain carrying the *spelta* gene $q$. This strain was established by crossing ordinary tetrasomic 5A with "*spelta*-5A" and, following two backcrosses to "*spelta*-5A," selecting for a tetrasomic plant from a family homozygous for $q$.

Aneuploid strains of Chinese Spring used were monosomic 5B (V) and monosomic 5D (XVIII). Chromosomes 5B and 5D are homoeologous to 5A. Tetrasomic 5A and nullisomic 5A tetrasomic 5D were grown for observation.

All of the experimental materials were grown in greenhouses. Single-anther fixation was used for meiotic observations. A 1:3 mixture of acetic acid and ethyl alcohol was used for fixation. For making preparations, the aceto-carmine squash method was used. The best preparations were obtained when fixed anthers were stained about eight hours at room temperature after treatment in four percent iron-alum solution for about 30 minutes.

It is considered that in many cases duplicates of a gene are located on the homoeologous chromosomes, making six doses for each (SEARS 1954, 1956). Thus for a particular chromosome the aneuploid series from nullisomic to tetrasomic makes a dosage series of from four to eight for most of the genes carried by the chromosome. Genes not duplicated on other chromosomes have a dosage series of from zero to four. In either case a natural test for gene dosages is available without any structural change in the chromosome being required. In diploid organisms such a test with intact chromosomes is almost an impossibility.

It is also easily possible in wheat to obtain telocentrics and isochromosomes (SEARS 1952, 1954). These have been used in the present study for varying the dosage of one arm of a chromosome independently of the other.

Although squareheadedness, the character on which the symbol *Q* was originally based (PHILIPTSCHENKO 1930, 1934; MACKEY 1954), is not found in all varieties of ssp. *vulgare*, it is the most conspicuous effect of *Q* when it does occur and permits the most precise distinction of *vulgare* type from non-*vulgare*. Since the variety used in the present study, Chinese Spring, is squareheaded, the determinations of dosage were largely based on the degree of squareheadedness.

## RESULTS

*The dosage effect of the* q *locus on chromosome 5A:* Plants with higher doses of *q* were discovered in the process of combining monosome 5B with tetrasome 5A carrying *qqqq*. To obtain this combination, first mono-5B was crossed by tetra-*spelta*-5A. The $F_1$ generation was checked at meiosis to be sure that each of the three plants had $19^{II}+1^{III}+1^{I}$. The trivalent in the $F_1$ plants should consist of one chromosome 5A which carries the *Q* gene from the mono-5B parent and two *spelta*-gene-carrying chromosome 5A's from tetra-*spelta*-5A. Therefore, the genotype of these three plants may be designated *Qqq*.

One plant was backcrossed by tetra-*spelta*-5A. Of 11 $B_1$ plants checked for chromosome pairing one proved to be tetrasomic-monosomic and four were trisomic-monosomic. All four plants that were tri-5A mono-5B showed speltoid expression, while the one that was tetra-5A mono-5B (59–2238.1–4) had a squarehead spike. The latter case could be explained by the occurrence of chromatid segregation. Since *Q* is located near the distal end of the chromosome arm concerned (MACKEY 1954), gametes with two chromosomes 5A carrying *Q* would be obtainable from *Qqq* through chromatid segregation. However, another explanation for squareheadedness is possible and will be presented later (see discussion).

In the following season 25 seeds were sown from each plant selected. None

of the progeny from tri-5A mono-5B plants appeared to have the *Q*, indicating that all of the parents were homozygous for *q*. The tetra-5A mono-5B plant (59–2238.1–4) showed that it was, as suspected, heterozygous for *Q*. It segregated seven plants either compactoid or subcompactoid, nine squareheads, and eight speltoids.

In the next season 30 plants were grown from one of the tri-5A mono-5B plants (59–2238.1–1) (Figure 3g) which had been shown to be homozygous for *q*. All the progeny again showed only speltoid expression. Thus altogether 53 offspring from this tri-5A mono-5B plant were tested in two seasons, and none showed the squarehead phenotype.

From the second population grown, 19 of the 30 plants were checked at meiosis (Table 1). One tri-5A plant monosomic for a telocentric 5B chromosome was obtained. From this plant, 16 offspring were grown. Fifteen of them showed the expected speltoid expression, but one was squarehead (Figure 1i). It had short spikes with softer glumes, and the tips of the spikes were compact and infertile. These characters agreed with those of typical *vulgare*, particularly with Chinese Spring. Its culms were shorter than those of Chinese, and its nodes were more intensely pubescent. That this plant was not a contaminant was quite evident from cytological analysis. It had 44 chromosomes, of which one pair was telocentric. Such a telocentric pair was to be expected following selfing of the monotelocentric parent plant, and could not have resulted from outcrossing with a squareheaded plant, since there were none carrying telocentric 5B. The fact that none of the sib plants showed squareheadedness confirms that the parent plant was homozygous for *q*.

One of the 44 chromosomes was an isochromosome which frequently paired with what was evidently chromosome 5A. In this case, the plant must have had three normal chromosomes 5A and one iso-5A. The isochromosome was undoubtedly the long arm, and thus the plant must have had five doses of the *spelta* gene *q*.

TABLE 1

*Chromosome constitution of plants grown from selfed seeds of a tri-5A mono-5B plant* (59–2238.1–1)

| Chromosome constitution | | 2n | No. of plants | Phenotype |
|---|---|---|---|---|
| Tri-5A mono-5B tri-5D | $18^{II}+2^{III}+1^{I}$ | 43 | 1 | speltoid |
| Tetra-5A mono-5B | $19^{II}+1^{IV}+1^{I}$ | 43 | 2 | speltoid |
| Tri-5A | $20^{II}+1^{III}$ | 43 | 4 | speltoid |
| Tri-5A mono-5B | $19^{II}+1^{III}+1^{I}$ | 42 | 6 | speltoid |
| Tri-5A monotelo-5B | $19^{II}+1^{III}+t^{I}$* | 42 | 1 | speltoid |
| Tri-5A nulli-5B | $19^{II}+1^{III}$ | 41 | 2 | speltoid |
| Disomic | $21^{II}$ | 42 | 1 | speltoid |
| Mono-5B | $20^{II}+1^{I}$ | 41 | 1 | speltoid |
| Monotelo-5B | $20^{II}+t^{I}$ | 41 | 1 | speltoid |
| Total | | | 19 | |

* $t^{I}$ = telocentric monosome.

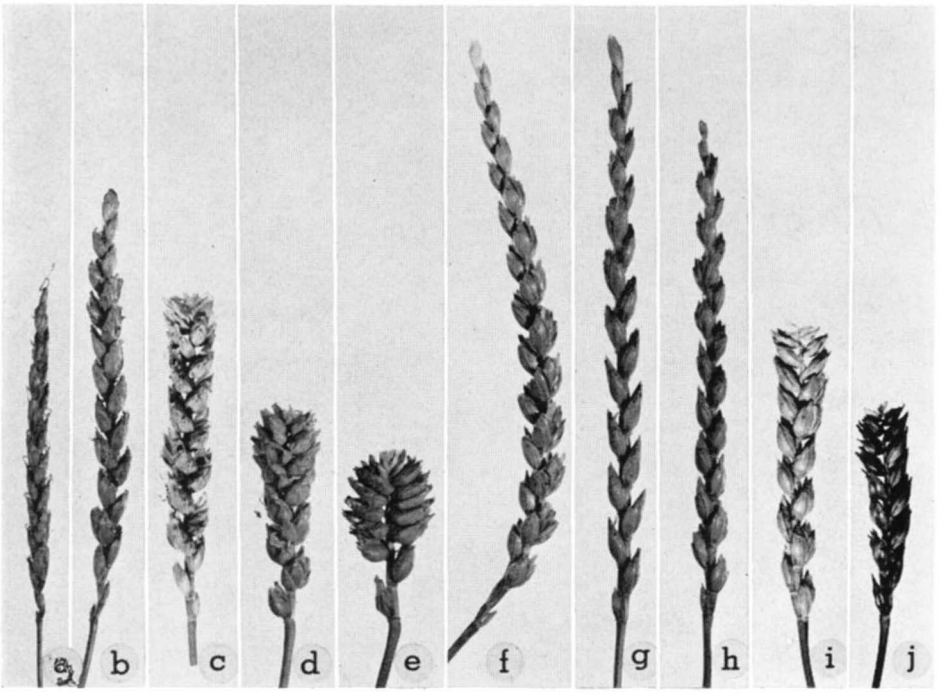


FIGURE 1.—The dosage effect of the *vulgare* gene *Q* (a-e) and the *spelta* gene *q* (f-j): (a) nullisomic 5A; (b) monosomic 5A; (c) disomic (Chinese Spring pure); (d) trisomic 5A; (e) tetrasomic 5A; (f) *spelta*-5A; (g) tri-*spelta*-5A; (h) tetra-*spelta*-5A; (i) spike with five doses of *q*; and (j) spike with six doses of *q*. *ca.* 5/8 ×.

To check the constitution of this plant, 29 seeds were sown. All germinated, but two seedlings died at the coleoptile stage. Three plants were lost by worm injury. Of the remaining 24 plants, 23 were analyzed cytologically (Table 2). The chromosome constitution of these varied from $20^{II}$+telo$^{II}$ to $19^{II}$+$1^{V(incl.\ one\ iso)}$+telo$^{II}$ (Figures 2d and 1j). Four plants had $19^{II}$+$1^{IV(incl.\ one\ iso)}$+telo$^{II}$, the parental constitution. The phenotype of these four plants was the same as that of the parent (Figure 2c). The plant with $19^{II}$+$1^{V(incl.\ one\ iso)}$+telo$^{II}$, which had six doses of *q*, bore spikes still more compact than those with five doses, going beyond squarehead toward subcompactoid expression (Figure 1j). It had shorter culms and set no seed, although some anthers shed pollen.

There was a parallel relationship between dosage of chromosome 5A and phenotype (see Table 2 and Figure 2). However, when only two short arms of 5A were present in plants with four long arms (two entire 5A's and one iso-5A), the plants were shorter and had spikes a little more nearly like squarehead (Figure 3b) than did plants with four entire chromosomes 5A. Evidently the short arm also affects speltoidy slightly. This will be dealt with further in the next report.

*Variation of speltoid phenotypes due to altered dosage of* q *and chromosome 5D*; 1. *Detailed comparison of tetra-*spelta-5*A and* spelta-5*A:* Plants which have

TABLE 2

*Chromosome constitution and phenotype of progeny of a squarehead plant with five doses of the long arm of chromosme 5A carrying the* spelta *gene* q

| Chromosome constitution | | 2n | No. of plants | Dosage of *q* | Phenotype |
|---|---|---|---|---|---|
| Disomic 5A ditelo-5B | $20^{II}+t^{II}$* | 42 | 2 | 2 | speltoid |
| Tri-5A ditelo-5B | $19^{II}+1^{III}+t^{II}$ | 43 | 5 | 3 | speltoid |
| Tetra-5A monotelo-5B | $19^{II}+1^{IV}+t^{I}$ | 43 | 2 | 4 | speltoid |
| Disomic plus iso-5A ditelo-5B | $19^{II}+1^{III(incl.\ 1\ iso)}+t^{II}$ | 43 | 2 | 4 | speltoid |
| Tetra-5A ditelo-5B | $19^{II}+1^{IV}+t^{II}$ | 44 | 3 | 4 | speltoid |
| Tri-5A plus iso-5A ditelo-5B | $19^{II}+1^{IV(incl.\ 1\ iso)}+t^{II}$ | 44 | 4 | 5 | squarehead |
| Tetra-5A plus iso-5A ditelo-5B | $19^{II}+1^{V(incl.\ 1\ iso)}+t^{II}$ | 45 | 1 | 6 | transitional type, squarehead-subcompactoid |
| Others | | | 4 | | |
| Total | | | 23 | | |

* $t^{II}$ = telocentric disome.

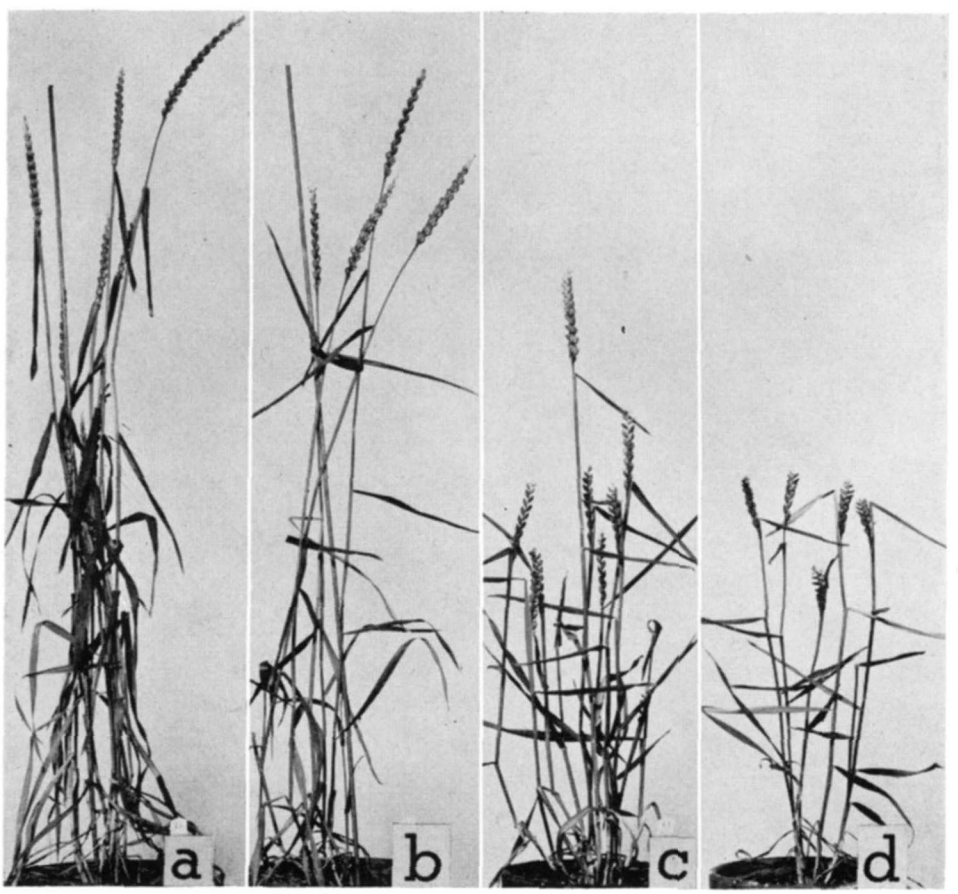


FIGURE 2.—Progeny of a squarehead plant which had five doses of the long arm of chromosome 5A carrying the *spelta* gene *q:* (a) disomic; (b) tetrasomic; (c) plant having three normal plus one isochromosome 5A, parental type; and (d) plant with six doses of *q*. *ca.* 1/6.6 ×.

four doses of *q*, tetra-*spelta*-5A (2n = 44), are essentially of speltoid expression. However, when the spikes of these plants are compared with *spelta*-5A (2n = 42, disomic), certain characters in tetra-*spelta*-5A show a tendency toward *vulgare* type, namely rounder, less strongly keeled and softer glumes with fairly yellow coloration, and more spikelets at the tip of the spike showing infertility; but the spikes are not squareheaded. As a whole the spike of tetra-*spelta*-5A is shorter and is somewhat laxer than that of *spelta*-5A (Figures 1f and 1h). This is due to a reduction of the number of spikelets and increase of the length of the rachis segments. In laxity, then, tetra-*spelta*-5A is more speltoid than *spelta*-5A, while in glume characters the reverse is true.

2. *Morphology of spike of tetra*-spelta-*5A when nullisomic or monosomic 5D:* This combination was obtained by crossing monosomic 5D with *spelta*-5A and then to tetra-*spelta*-5A. The $F_1$ plant presumably was heterozygous for *Q* and *q*. Of eight plants analyzed cytologically following the cross to tetra-*spelta*-5A, five were found to be tri-5A mono-5D, with chromosome pairing of $19^{II}+1^{III}+1^{I}$. Two of the remaining three were tri-5A plants and one was mono-5D. Progeny tests of the tri-5A mono-5D plants showed that one was homozygous for *q*, showing no segregation of squarehead among 23 offspring observed. From this homozygous family, 12 late plants, which were expected to be monosomic for chromosome 5D, were selected, and seven of them were checked at meiosis. One tri-5A mono-5D plant was found.

In the next season 30 offspring were obtained from this tri-5A mono-5D plant, and all showed speltoid expression. Ten plants were checked at meiosis. One (Figure 3c) was found to be tetra-*spelta*-5A mono-5D ($19^{II}+1^{IV}+1^{I}$), and one (Figure 3d), an extremely slow-growing plant, was tetra-*spelta*-5A nulli-5D ($19^{II}+1^{IV}$).

These two plants differed somewhat in phenotype, and both differed from tetra-*spelta*-5A. With reduced dosage of chromosome 5D, spikes became denser. Both mono- and nulli-5D showed low fertility in the upper part of the spike, but as with tetra-*spelta*-5A, did not show any squareheadedness (Figure 3).

Evidently the presence of chromosome 5D affects spike length, at least to lengthen rachis segments. This is proven by comparing *nullisomic* 5A with nulli-5A tetra-5D. The latter has a longer rachis (Figure 3e). Since tetra-*spelta*-5A nulli-5D clearly has a shorter spike than *spelta*-5A, extra dosage of chromosome 5A carrying *q* does not substitute for chromosome 5D in bringing spike length to that of normal *spelta*-5A. Besides, chromosome 5D is probably more essential for fertility of the tip of the spike, for in *spelta*-5A very few spikelets are infertile but in tetra-*spelta*-5A nulli-5D many show infertility. In other words, extra dosage of 5A carrying *q* is unable to take the place of 5D in bringing about fertility of the upper spikelets.

Besides its effect on spike length, the absence of chromosome 5D makes glumes smaller, tougher and more strongly keeled. This suggests that chromosome 5D resembles 5A carrying *q* in its effect on glume characters.

3. *Tri*-spelta-*5A tri-5D with monosome 5B:* One plant with chromosome pairing of $18^{II}+2^{III}+1^{I}$ was obtained (Table 1). From the parentage of this plant, one trisome must have involved chromosome 5A. Careful cytological study of

the other trisome indicated that it involved chromosome 5D, which is karyomorphologically distinct at meiosis. Also the plant was the earliest in the family, as expected with increased dosage of 5D. Therefore, the plant presumably was tri-*spelta*-5A mono-5B tri-5D. Spikes of this plant were speltoid and more lax than those of tri-*spelta*-5A mono-5B, thus confirming that chromosome 5D makes spikes less dense (Figure 3f).

*Nullisomic 5B trisomic 5A plants homozygous for* q: In the progeny of tri-*spelta*-5A mono-5B (59–2238.1–1) nullisomic 5B was obtained (Table 1). Nulli-5B was expected in much higher frequency from this progeny than from ordinary mono-5B, for the extra chromosome 5A compensates for the absence of chromosome 5B to cause higher transmission of pollen not carrying chromosome 5B. Seventeen plants out of 19 checked at meiosis in the progeny were clearly either mono- or disomic 5B with various numbers of chromosome 5A. The other two plants showed some inconsistency in chromosome pairing. Both had 41 chromosomes. One was clearly tri-*spelta*-5A nulli-5B, because it frequently showed $19^{II}+1^{III}$. The chromosome constitution of the other plant was not so clear; there was inconsistency of pairing including $20^{II}+1^{I}$, $18^{II}+1^{V}$, $16^{II}+1^{III}+6^{I}$, $18^{II}+5^{I}$, $17^{II}+2^{III}+1^{I}$ and so on. Although no $19^{II}+1^{III}$ was observed, the plant was deduced to be tri-*spelta*-5A nulli-5B, because a trivalent

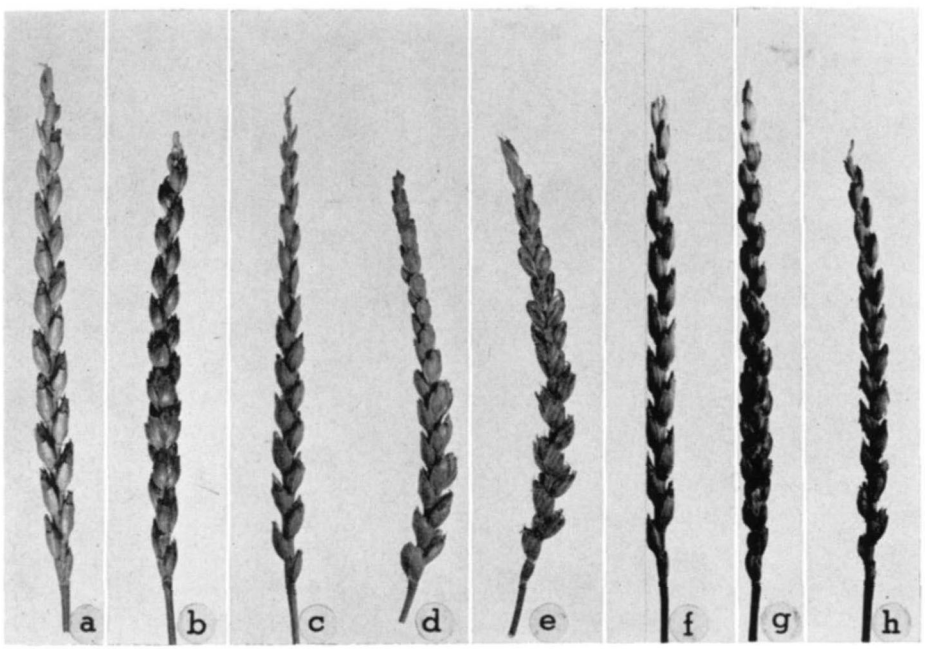


FIGURE 3.—Effect of homoeologous chromosomes on speltoidy: (a) Tetra-*spelta*-5A ditelo-5B; (b) Spike from a plant having two normal 5A plus one isochromosome 5A and ditelo-5B. It has four doses of *q* and shows tendency toward squareheadedness; (c) Tetra-*spelta*-5A mono-5D; (d) Tetra-*spelta*-5A nulli-5D; (e) Nulli-5A tetra-5D; (f) Tri-*spelta*-5A mono-5B tri-5D; (g) Tri-*spelta*-5A mono-5B; (h) Tri-*spelta*-5A nulli-5B. *ca.* 5/8 ×.

was seen rather frequently and the number of cells observed was small. The occurrence in these two plants of higher associations of chromosomes than expected may be attributed to the increased synapsis brought about by the absence of chromosome 5B (OKAMOTO 1957; RILEY 1958; SEARS 1958). One plant had only slight fertility and the other was sterile. The spikes were of speltoid type and rather small (Figure 3h). Glumes were also small and tended toward more speltoidy. Thus chromosome 5B appears to be similar to 5D and to 5A carrying *q* in its effect on glume characters.

## DISCUSSION

In general, in diploid organisms the dominant-recessive relationship is explained by the dominant allele's supplying a sufficient amount of product that one dose brings about the full effect. This allele is thus dominant over hypomorph, amorph and deficiency. In the aneuploids of hexaploid wheat this requires special consideration. A gene is subject to modification by its duplicates on the homoeologous chromosomes.

Certain genes in common wheat are not expressed in hemizygous condition. According to SEARS (1954), the *vulgare* gene *Q* is one of these. He also found that the wild allele *V* of the virescent gene *v*, a hemizygous-ineffective gene on chromosome 3B (III), acts as a null allele from zero to four doses. However, *V* is evidently not a deficiency, for *v* arose from *V* by mutation. It was shown (SEARS 1959, and unpublished) that *V* is a gene which is duplicated on other chromosomes.

The fact that *Q* is ineffective when hemizygous resembles the case of the virescent gene *v*. The gene *v* manifests virescence only in two doses; in three doses, it causes albinism. However, the relationship between *v* and its allele *V* differs from that between *Q* and its wild allele *q*, the *spelta* gene, in that *V* does not have an effect like that of *v*, but instead has an effect opposite to *v*. This was proved by varying the dosage of the duplicates of *V* on the homoeologous chromosomes (SEARS 1959). The indications were that *v* is an antimorphic gene (SEARS unpublished).

Evidence that *Q* is not an antimorph comes from nulli-5B and nulli-5D. Assuming that chromosomes 5B and 5D carry duplicates of *q*, squareheadedness should be more extreme in these nullisomics if *Q* were an antimorphic gene. In reality, however, these two nullisomics are less, rather than more, squareheaded (see SEARS 1954, 1956).

The fact that five doses of the long arm of chromosome 5A of ssp. *spelta* causes squareheadedness shows that *q* is not a deficiency nor an amorph but is an allele which has an effect similar to that of *Q* but of lesser degree. In this sense, *q* is hypomorphic to *Q*. Relative to *q*, *Q* is a hypermorph, and five doses of *q* corresponds approximately to two doses of *Q*.

In Table 3 are summarized the genotypes and phenotypes of plants with various dosages of chromosome 5A carrying the genes *Q* or *q*. *Q* was calculated as 2.5 *q*'s, according to the fact that both *qqqqq* and *QQ* show the squarehead

TABLE 3

*Dosage effect of the* vulgare *gene* Q *and the* spelta *gene* q *in Chinese Spring background*

| Status of chromosome 5A | 2n | Genotype | Dosage of $q$ ($Q=2.5\ q$) | Phenotype |
|---|---|---|---|---|
| Nullisomic | 40 | —* — | 0 | speltoid |
| Monosomic | 41 | *Q* — | 2.5 | speltoid |
| Disomic | 42 | *Q q* | 3.5 | speltoid |
| Disomic | 42 | *Q Q* | 5 | squarehead |
| Trisomic | 43 | *Q Q Q* | 7.5 | subcompactoid |
| Tetrasomic | 44 | *Q Q Q Q* | 10 | compactoid |
| Mono-*spelta*-5A | 41 | *q* — | 1 | speltoid, (SEARS unpublished) |
| *spelta*-5A | 42 | *q q* | 2 | speltoid |
| Tri-*spelta*-5A | 43 | *q q q* | 3 | speltoid |
| Tetra-*spelta*-5A | 44 | *q q q q* | 4 | speltoid |
| Tri-5A*(spelta)* plus iso-5A*(spelta)* | 44 | *q q q q q* | 5 | squarehead |
| Tetra-5A*(spelta)* plus iso-5A*(spelta)* | 45 | *q q q q q q* | 6 | transitional type, squarehead-subcompactoid |

* — indicates absence of one whole chromosome 5A.

phenotypes. When a plant has a dosage of $q$ below five, the phenotypic expression is always speltoid. Therefore, the threshold level for squareheadedness must lie somewhere between four $q$'s and five $q$'s.

The squarehead plant (59–2238.1–4) that appeared in the progeny of the cross *Qqq* × tetra-*spelta*-5A could be explained on this basis. The plant could have been *Qqqq* (with 5.5 $q$ equivalents) and therefore squareheaded.

In tetra-*spelta*-5A, glumes are more round, softer and less greenish, and the tip of the spike shows higher infertility than *spelta*-5A. These are obviously tendencies toward *vulgare* type (squareheadedness). However, this seems to contradict the fact that tetra-*spelta*-5A has more lax spikes than *spelta*-5A, since one distinction between *vulgare* and *spelta* is the latter's lax spikes. This could be attributed to the dosage effects of other genes located on the same chromosome, probably on the short arm. Reduced plant height and a tendency toward squareheadedness which were seen in the plant with two normal chromosomes 5A plus a long-arm isochromosome 5A could be explained by an effect of the missing short arm of 5A. As reported by SMITH, HUSKINS and SANDER (1950) the short arm "apparently influences development in the opposite direction to that determined by the long arm." A substantial effect of the short arm of chromosome 5A on plant height was demonstrated by THRELKELD (1962) in the variety Thatcher.

It was observed that chromosome 5D has a stronger tendency to make spikes lax than 5A has, since tetra-*spelta*-5A nulli-5D is denser than tetra-*spelta*-5A, and nulli-5A tetra-5D is laxer than nulli-5A. This is confirmed by the fact that a tri-*spelta*-5A mono-5B tri-5D plant had a laxer spike than tri-*spelta*-5A mono-5B (see Figures 3f and 3g).

MACKEY (1954) thinks that there is no primary difference between *spelta* and speltoid, both depending on lack of *Q*. The difference depends on the rest of the genotype. Historically, the term speltoid has usually been used for mutants or segregants with touch rachis and relatively loose glumes, and *spelta* for types with brittle rachis and strongly held seeds. That the substitution line "*spelta*-5A" has a touch rachis means that Chinese Spring provides a nonspelt background, and *spelta*-5A can be classified phenotypically as speltoid.

Although *spelta*-5A is phenotypically speltoid, it has an important genetic difference. The gene *q* carried by *spelta*-5A can, in higher dosage, modify the phenotype in the direction of *vulgare*, since *q* acts like *Q* but to a lesser degree. Speltoids, on the other hand, involve deficiency for the *Q* locus, and therefore show no dosage response. In other words, the *q* gene in *spelta*-5A is potentially capable of bringing the phenotype toward *vulgare*, while speltoid mutations have no such potency.

Against a ssp. *spelta* background a speltoid mutation would presumably give rise to an extreme *spelta* phenotype. In other words, ssp. *spelta* differs from most *vulgare* varieties more than the speltoid mutants do, not because of the situation at the *Q* locus, but in spite of it. It is the rest of the genotype that makes ssp. *spelta* more extremely spelted than speltoid. In reality, the *spelta* gene itself has less of a spelting effect than does deficiency for the locus.

Thus, with the same background, *Q* has the greatest effectiveness counterbalancing the spelting effect of the rest of genotype, *q* has less effectiveness, and speltoid mutations have none. Deficiencies for the *Q* locus have only been recovered in ssp. *vulgare*, because deficiency for the *q* of ssp. *spelta* results in a relatively inconspicuous alteration of the phenotype. Against the *vulgare* background, deficiencies for *Q* result in a speltoid phenotype, intermediate between *vulgare* and *spelta* type, with tough rachis and more loosely held seeds than in *spelta*.

To avoid confusion the term speltoid should be restricted to designation of a phenotype, not to identification of an allele at the *Q* locus. Deficiencies for *Q*, which have usually been referred to as speltoid mutations, had better be called *Q*-locus deficiencies. They produce the speltoid phenotype in most *vulgare* varieties, but so does the *spelta* gene *q*; and in ssp. *spelta* they would give a *spelta* phenotype.

In order to make clear that the mutations in ssp. *vulgare* that result in speltoidy are actually *Q*-locus deficiencies, different from the *spelta* gene *q*, it is suggested that the symbol $q^-$ be used to designate them.

It is now clear that the ambiguities of dominance in the $F_1$ hybrids between ssp. *vulgare* and either speltoid mutants or ssp. *spelta* are due to the pleiotropic nature of the *Q* alleles and the modifying effect of the genetic background. The dosage of *Q* or *q* which is required to bring the phenotype above the *vulgare* threshold level depends on the intensity of the background action and on which of the several pleiotropic effects of *Q* is being considered. For example, in Chinese Spring a relatively low dosage of *Q* is required to produce squareheadedness, whereas in non-squarehead *vulgare* varieties a rather high dosage is necessary

for squareheadedness. Thus *QQ* is squareheaded in Chinese Spring and non-squareheaded in the other varieties. On the other hand, with respect to the glume characters, nervation (or color) and prominence of keel, the situation is reversed, and Chinese Spring is more like *spelta* than are many non-squarehead *vulgare* varieties.

In any given background, with sufficiently high dosage of *Q* or *q*, all of the assemblage of characters controlled by the *Q* locus are of *vulgare* type, while at lower dosage all of them are of *spelta* or speltoid type.

At intermediate dosages some characters may be of *vulgare* type while others are non-*vulgare* (see Figure 4). Since the heterozygotes *Qq* and $Qq^-$ are intermediate in dosage, a particular $F_1$ may be like *vulgare* in one respect and more like *spelta* in others. This makes it difficult and somewhat arbitrary to decide whether *Q* or *q* is dominant. With a Chinese Spring background, it is evident, as shown in Table 3 and in Figure 4 that in *Qq* squareheadedness is fully recessive and the characters of glume and rachis are incompletely dominant. In this particular instance, *q* might well be considered dominant over *Q*. With a different background, however, the average degree of dominance may clearly favor *Q*.

The demonstration that *Q* has the same effect as *q*, but stronger, constitutes


FIGURE 4.—Spikes showing ambiguity of dominance in a cross between ssp. *vulgare* and ssp. *spelta*: (a) and (b) Chinese Spring; (e) and (f) ssp. *spelta Duhamelianum* and (c) and (d) $F_1$. Head-type is evidently like ssp. *spelta* in $F_1$, but other characters, like shattering, glume coloration and brittleness are only partly dominant or intermediate. *ca.* 5/8 ×.

evidence for the suggestion of KUCKUCK (1959) that the relation between *q* and *Q* might be like non-Bar and Bar in Drosophila. In this case, as KUCKUCK points out, *Q* might arise from *q* through unequal crossing over. If *Q* did arise from *q*, this must have occurred in cultivated wheat, for no wild wheat is known to have *Q*.

The question which of the hexaploid wheats came first, ssp. *spelta* or ssp. *vulgare*, is complicated by the fact that one of the tetraploid wheats, *T. carthlicum*, is *QQ*. It is not at all clear whether *Q* arose at the tetraploid or the hexaploid level. However, it can at least be said that ssp. *spelta* did not necessarily originate by speltoid mutation from ssp. *vulgare*. Ssp. *vulgare* may just as well have originated as a mutant from ssp. *spelta*.

## SUMMARY

The *spelta* gene *q* (an allele of the *vulgare* gene on the long arm of chromosome 5A) behaves much as a null allele up to four doses. Five doses of *q*, however, results in squareheadedness, and six causes more compact spikes. Evidently *q* is not a deficiency nor an amorph but is an allele which has an effect similar to that of *Q* but to a lesser degree. Relative to *Q*, *q* is a hypomorph, and five doses of *q* corresponds to two doses of *Q*. The threshold level for squareheadedness lies between four and five *q*'s. Below that level the phenotype is always speltoid. Other genes on the same chromosome, particularly those on the short arm, have an effect opposite to that of *Q* and *q*. Chromosome 5D tends to make spikes less dense.

Contrary to previous belief, the *spelta* gene *q* has less spelting effect than does deficiency for the *Q*-locus. *Q*-locus deficiency results in speltoidy in ssp. *vulgare*, while in ssp. *spelta* it presumably gives rise to a more extreme *spelta* phenotype. The allele *q* can cause either speltoid or *spelta* depending on the background.

The ambiguities of dominance in the $F_1$ between ssp. *vulgare* and ssp. *spelta* are due to both the pleiotropic nature of the *Q* alleles and the intensity of genetic background action on which the several pleiotropic effects are being considered. The average degree of dominance may favor *Q*.

Since *q* is an active gene, not merely a deficiency, ssp. *spelta* can not have originated from ssp. *vulgare* in the way that speltoids arise now.

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LITERATURE CITED

HUSKINS, C. L., 1946 Fatuoid, speltoid and related mutations of oats and wheat. Botan. Rev. **12**: 457–514.

HUSKINS, C. L., and G. F. SANDER, 1949 Mutations in polyploid cereals. I. Introductory outline. Can. J. Res. C. **27**: 332–347.

KUCKUCK, H., 1959 Neuere Arbeiten zur Entstehung der hexaploiden Kulturweizen. Z. Pflanzenzücht. **41**: 205–226.

MACKEY, J., 1954 Neutron and X-ray experiments in wheat and a revision of the speltoid problem. Hereditas **40**: 65–180.

OKAMOTO, M., 1957 Asynaptic effect of chromosome V. Wheat Inform. Serv. **5**: 6.

PHILIPTSCHENKO, J., 1930 Again on the question of genes and the development of the form of ear in wheat. Bull. Bur. Genet. **8**: 1–18.

1934 *Genetics of Soft Wheats*. 262 pp. States Press Lit. State Coll. Farms, Moscow-Leningrad. (Cited by J. MACKEY 1954.)

RILEY, R., 1958 Chromosome pairing and haploids in wheat. Proc. 10th Intern. Congr. Genet. **2**: 234–235.

SEARS, E. R., 1952 Misdivision of univalents in common wheat. Chromosoma **4**: 535–550.

1953 Nullisomic analysis in common wheat. Am. Naturalist **87**: 245–252.

1954 The aneuploids of common wheat. Missouri Agr. Exp. Sta. Res. Bull. **572**: 59 pp.

1956 The systematics, cytology and genetics of wheat. Handb. Pflanzenzücht., Second edition, **2**: 164–187.

1958 The aneuploids of common wheat. Proc. 1st Intern. Wheat Genet. Symp. Winnipeg, Canada. Pp. 221–229.

1959 Neatby's virescent, a chlorophyll aberration in common wheat. (Abstr.) Genetics **44**: 534.

SMITH, S. G., C. L. HUSKINS, and G. F. SANDER, 1949 Mutations in polyploid cereals. II. The cytogenetics of speltoid wheats. Can. J. Res. C. **27**: 348–393.

1950 Mutations in polyploid cereals. III. The cytogenetics of compactoid wheats. Can. J. Res. C. **28**: 153–182.

THRELKELD, S. F. H., 1962 Study of a gene controlling height in *Triticum vulgare* var. Thatcher. Can. J. Genet. Cytol. **4**: 147–150.

UNRAU, J., W. E. SMITH, and R. C. MCGINNIS, 1950 Spike density, speltoidy and compactoidy in hexaploid wheat. Can. J. Res. C. **28**: 273–276.