GENES CONTROLLING CHIASMA FREQUENCY IN *HORDEUM*

M. D. GALE and H. REES
Department of Agricultural Botany, University College of Wales, Aberystwyth

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1. Introduction

The following is an account of variation in chiasma frequency at meiosis within the genus *Hordeum*. Small but significant differences in chiasma frequencies between five *Hordeum* subspecies are shown to be genotypically controlled. Comparisons between parents, their hybrids and derivatives provide information about the nature of the gene systems controlling the variation. By means of " morphological " markers, an assay of the chromosome complement was also carried out in order to locate genes that control the chiasma frequency variation.

2. Materials and methods

a. *The Subspecies*

The five subspecies used, all diploids with $2n = 14$, are listed in table 1. Crosses between all five are easily accomplished and produce fully fertile

Table 1

The five Hordeum *subspecies*

Hordeum agriocrithon	ex. R. Davies, Welsh Plant Breeding Station
H. spontaneum (*vulgare*, *ss. spontaneum*)	ex. Dr W. Bowden, Ottawa
H. hexastichum	from D. B. James, Agricultural Botany Department, University College of Wales, Aberystwyth
H. distichum	
H. intermedium	

offspring. The interfertility, amongst other things, has led to the view that the five forms merit subspecific rather than the specific rank previously accorded (see Bell, 1965; Åberg, 1940). Custom and tradition and, perhaps, convenience account for the retention of specific nomenclature.

b. *The Method*

Experimental design. In 1966 seeds from a diallel cross, comprising the five parents and F_1 reciprocals, were sown eight to a pot in two replicates. The randomised pots were kept in a greenhouse for six weeks and then transferred outdoors. Heads were fixed for cytological examination from two plants of each genotype in each replicate.

In 1967 the seed from the reciprocal F_2's were pooled because no reciprocal differences were revealed by analysis of the F_1 results. Seeds from this " half diallel ", comprising parents and F_2, were sown in two-inch pots, kept in a warm greenhouse for two weeks and then transplanted into the field. The lay-out comprised eight blocks (A-H) each containing two randomised

plots of each parent and F_2 families. Each plot contained a row of five plants at one-foot intervals, two feet between rows, the whole experiment surrounded by a guard row. Heads were fixed in each of two plants in each family in blocks A-D.

Cytological. The heads were fixed in Carnoy's fluid and staining was by aceto-carmine. The chiasma frequency per plant was expressed as the mean of 20 pollen mother cells, scored at first metaphase.

Morphological. The 600 plants comprising blocks A-D in 1967 were classified in respect of the eleven characters below:

(i) Shattering capacity.
(ii) DDT resistance.
(iii) Height.
(iv) Intermedium head type.
(v) Rachilla hair type.
(vi) Seed colour.
(vii) Tiller number.
(viii) Kernel row number.
(ix) Glum-awn length.
(x) Awn length.
(xi) Spike density (spike internode length).

Whereas the genes controlling some of the characters are well mapped there is controversy and disagreement about the number, the action and location of genes controlling others. For some, also, the classification of phenotypes is by no means straightforward. For these reasons it is necessary to explain, in some detail, the methods adopted for scoring and the nature of the segregations encountered in the present material.

(i) *Shattering capacity*

Brittle rachis or " shattering " genes are a feature of wild and primitive barleys. Most workers (*e.g.* Takahashi, 1955) attribute control to two genes, Bt_2 or Bt_3 and Bt, which are complementary and situated on chromosomes 3 and 5 respectively. The results of the present work agree with this interpretation, *i.e.* control by two independent complemenatary *loci*, giving a 9 brittle : 7 non-brittle F_2 ratio.

(ii) *DDT resistance*

Resistance to DDT is controlled by a single recessive gene (*ddt*) located on or near the satellite of chromosome 7 (Hayes and Rana, 1966). This character was scored by spraying green leaves with a 0·2 per cent. solution of Strykol DDT, marking them with a paper tab and inspecting them after ten days for unnatural yellowing. Although plants in blocks A-D did not give a clear 3 susceptible : 1 resistant F_2 ratio, blocks E-H gave such a ratio when sprayed as whole plants. An excess of resistant plants in blocks A-D was presumably due to misclassification of leaves not sufficiently wetted by the spray.

(iii) *Height*

Plant height in barley is generally treated as a very complex character much influenced by the environment and consequently displaying low

heritability (*e.g.* Jogi, 1956). At least part of the variation would appear to be governed by the "*H*" *locus* on chromosome 2 (Mann, 1953). In the present work height was treated as a continuous variable, no attempt being made to split the phenotypes into arbitrary, and dubious, tall and short classes.

(iv) *Intermedium head type*

This type of spike is shown by *H. intermedium*, a subspecies with awnless fertile lateral florets. The I^h allele on chromosome 4 governs the production of these fertile laterals in the presence of the *V* allele for two-rowed spike type (Murty and Jain, 1960).

(v) *Rachilla hair type*

Short rachilla hairs are determined by a recessive allele on chromosome 7 (Doney, 1961). The two rachilla types were easily distinguishable and the F_2 ratio fitted a 3 long : 1 short segregation.

(vi) *Seed colour*

Black, as opposed to non-black, lemma and pericarp is reported by most workers (*e.g.* Litzenburger and Green, 1951) to be due to a single gene difference with "black" dominant over "non-black" types. The gene "*b*" is carried on chromosome 5. Das (1957), however, reported a two-factor mode of inheritance. Results obtained in the present work show a monofactorial type of inheritance.

(vii) *Tiller number*

Genetic control of tiller number has been established by Murty and Jain (1960). They found correlations between tiller number and segregation at the *N/n locus* (hulled/naked grain) on chromosome 1. In the present work, tiller number has been treated as a continuous variable.

(viii) *Kernel row number*

Kernel row number is determined mainly by the *V/v locus* on chromosome 2 but there are modifying *loci* such as the *I/i locus* on chromosome 4 (Woodward, 1949). Crosses with *H. hexastichum* as the six-rowed parent gave F_2 progenies with 3 : 1 segregations. Crosses with *H. agriocrithon* as the six-rowed parent gave, on the other hand, ratios of 15 : 1 non six-rowed to six-rowed, the non six-rowed class including intermediate as well as two-rowed phenotypes. Although two genes are clearly involved, the precise basis of the control is not clear from the data available.

(ix) *Glume-awn length*

In the present work segregating F_2's gave a ratio of three long to one short awn. This character is controlled by the E_2/e_2 *locus* on chromosome 1 (Doney, 1961).

(x) *Awn length*

Segregating F_2's gave good 3 : 1 short and intermediate to long awns (*cf.* Takahashi *et al.*, 1953). A gene controlling awn length, *lk5*, has been

mapped in chromosome 2 (see Litzenburger and Green, 1951; and Woodward, 1957). Whereas the long awned phenotypes were fairly easily identified the short and intermediate forms were not. Consequently, for correlation purposes, the awn length character was treated as a continuous variable.

(xi) *Spike density* (*spike internode length*)

Reports differ concerning the inheritance of this character, much confusion arising from the pleiotropic effects of the genes for spike density on the length of the spike, internode number and length of the coleoptile (Takahashi, 1951). Litzenburger and Green (1951) found the character to be inherited monofactorially with lax dominant over dense spike. The gene, *Rin*/*rin*, affecting spike density, was located on chromosome 2 by Woodward (1957). Aziz and Mir (1960) say that more than one gene may be involved.

In the present work, although lax tends to be dominant over dense spike, no clear cut segregation was observed and the character has been treated as a continuous variable, classified on the basis of spike internode length.

The analyses. These fall into two groups. The first is concerned with partitioning the heritable variation in chiasma frequency into its component parts *viz.* into additive and non-additive variation. The analyses consist of classical biometrical methods developed by Jinks (1954) and Morley-Jones (1965).

The purpose of the second type of analysis was to assay the chromosome complement for genes controlling the chiasma frequency variation. Essentially, the assay depends on establishing correlations between differences in chiasma frequency and the segregation of the marker genes located on different chromosomes. Details follow in the next section.

3. Results

Table 2 gives the mean chiasma frequencies for the parent, F_1 and F_2 families. As mentioned earlier there was no significant difference between reciprocals of the F_1 families sown in 1966. For this reason the values from reciprocals were pooled in table 2 and only a " half diallel " carried forward to the F_2.

(i) *Parents*

An analysis of variance of the means of subspecies over two seasons appears in table 3. This table shows:

(*a*) There is a highly significant difference between subspecies ($P = < 0{\cdot}001$).
(*b*) There is a significant variation due to seasons ($P = 0{\cdot}05$-$0{\cdot}01$).
(*c*) The difference between seasons is independent of the genotype, as indicated by the insignificant mean square for interaction.

The cause of the seasonal difference is not known. It could be due to a difference in temperature (*e.g.* Dowrick, 1957; Henderson, 1962) or even in soil fertility (see Law, 1963).

The variation between subspecies is attributed to genotypic control as distinct from structural differences between chromosomes. The grounds for this conclusion are that F_1 hybrids and their F_2 derivatives show no sign whatever of structural heterozygosity. The conclusion is amply confirmed by the results of the assay described in a later section.

TABLE 2

The mean chiasma frequencies of parent and F_1 families (1966) and of parent and F_2 families (1967). 1966 results from four plants of each genotype, 1967 results from eight plants of each genotype except for 1 × 2 (five plants) and 2 × 5 (four plants)

	Subspecies	1	2	3	4	5	Array totals
1966	1. *H. agriocrithon*	14·50	14·13	14·30	14·42	14·30	71·65
	2. *H. spontaneum*		13·88	13·82	14·18	13·87	69·88
	3. *H. hexastichum*			13·78	14·07	14·00	69·97
	4. *H. distichum*				14·00	13·95	70·62
	5. *H. intermedium*					14·13	70·25
1967	1. *H. agriocrithon*	15·64	14·61	14·85	15·28	14·26	74·64
	2. *H. spontaneum*		13·62	14·08	14·39	14·74	71·44
	3. *H. hexastichum*			14·19	14·68	14·24	72·04
	4. *H. distichum*				14·33	14·44	73·12
	5. *H. intermedium*					14·14	71·82

TABLE 3

An analysis of variance of the chiasma frequencies of the parental subspecies grown in 1966 and 1967

Item	d.f.	S.S.	M.S.	V.R.	P.
Subspecies	4	16·40	4·10	13·66	< 0·001
Years	1	1·43	1·43	4·77	0·05-0·01
$S \times Y$	4	2·94	0·74	2·45	
Error	50	15·00	0·30		

TABLE 4

The " half-diallel " analysis of variance; parents and F_1

Item	d.f.	S.S.	M.S.	V.R.	P.
a	4	0·5509	0·1877	21·85	< 0·001
b	10	0·1233	0·0123	1·95	0·05
b_1	1	0·0071	0·0071	1·31	
b_2	4	0·0594	0·0149	2·37	0·10-0·05
b_3	5	0·0568	0·0114	1·81	
error	45	0·2835	0·0064		

TABLE 5

The " half-diallel " analysis of variance; parents and F_2

Item	d.f.	S.S.	M.S.	V.R.	P.
a	4	2·3427	0·5857	8·72	< 0·001
b	10	1·1362	0·1136	1·69	0·10-0·05
b_1	1	0·0998	0·0998	1·49	
b_2	4	0·4258	0·1064	1·58	
b_3	5	0·6106	0·1221	1·82	
error	98	6·5856	0·0672		

(ii) *Diallels*

Analyses of Variance. The half diallel analyses presented in tables 4 and 5 are derived from Morley-Jones (1965) based on a genetical model by Hayman (1954). The " *a* " item relates to additive effects and " *b* " to

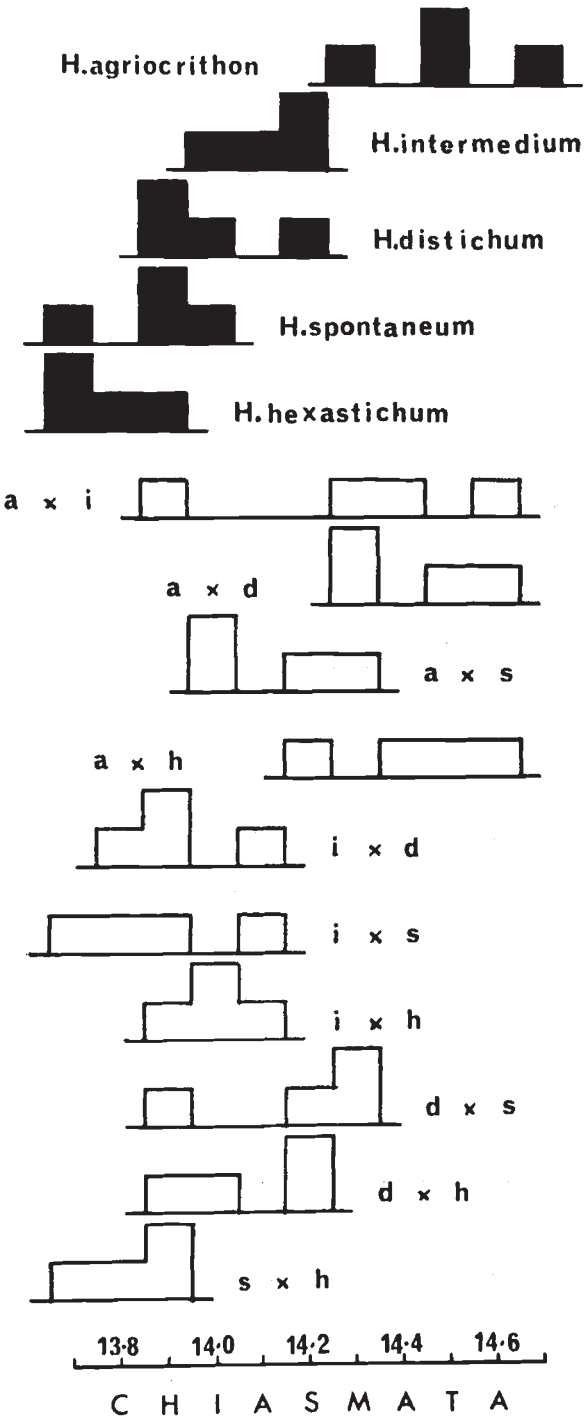

FIG. 1.—The chiasma frequency distribution in the *Hordeum* subspecies and their F_1 hybrids.

total non-additive effects. If we assume no non-allelic interaction, a reasonable assumption in the light of the *Wr/Vr* analysis (fig. 2), "b" is broken down to,

"b_1", mean dominance,
"b_2", additional effects due to dominant genes restricted to certain of the parental lines,
"b_3", residual dominance effects.

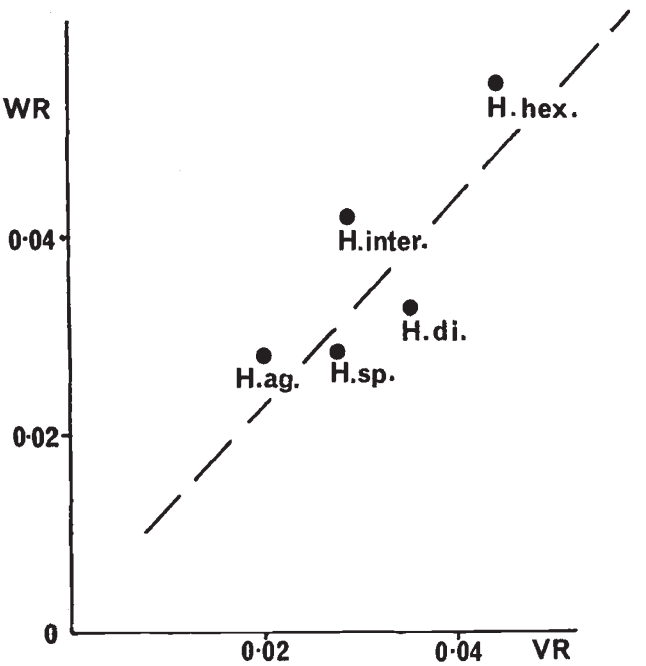

FIG. 2.—The array covariance (*Wr*) plotted against the array variance (*Vr*) for parent and F_1 chiasma frequencies.

The tables show,

(*a*) A highly significant "a" item for both F_1 and F_2 generations ($P = < 0{\cdot}001$), testifying to a control exercised by genes with additive effects.

(*b*) "b" to be just significant at the 5 per cent. level in the F_1 analysis, approaching significance in the F_2 analysis ($P = 0{\cdot}10\text{-}0{\cdot}05$). The conclusion is that genes with non-additive effects play a smaller part in controlling the chiasma frequency.

The F_1 analysis also shows that "b_2" contributes most to the "b" sum of squares. Table 4 indicates that this is due to genes carried by *H. agriocrithon*, which has the highest chiasma frequency, showing dominance throughout the array. Although such dominance effects are not widespread or pronounced in the F_1 there is enough to justify further analysis by the *Wr/Vr* method of Jinks (1954).

Wr/Vr analysis. The array covariances are plotted against the array variances in fig. 2. Bearing in mind the small number of lines involved,

the regression, although not quite significant at the 5 per cent. level, tends to confirm the dominance effects inferred from the analysis in table 4. The positions of the points along the regression line confirm, also, the direction of dominance to be towards a high chiasma frequency with *H. agriocrithon*, the species with the highest frequency, nearest to the origin and, furthest away, *H. hexastichum*, the species with the lowest chiasma frequency.

To summarise, the analyses show that the variation is controlled mainly by genes with additive effects and, to a lesser extent, by genes with non-additive effects. A significant component of the non-additive variation is attributable to genes displaying normal dominance.

These findings, coupled with the continuous nature of the variation displayed (fig. 1), are readily interpreted as a reflection of control by many genes, comprising possibly a classical polygenic system made up of factors with small, similar and supplementary effects (Mather, 1949). In the following section are set out the results of a scheme devised to locate by assay the genes involved.

(iii) *The Chromosome Assay*

As with previous assays (Mather and Harrison, 1949; Breese and Mather, 1957, 1960; Thoday, 1961, 1966) the procedure was, first, to pick out, with the aid of marker genes, the chromosomes whose genic activity contribute substantially towards the chiasma frequency variation and, subsequently, to explore in more detail the sites of activity within these chromosomes. As compared with *Drosophila* the barley complement is crudely marked and the assay, in consequence, that much cruder.

(a) *The Complement*

Gene markers controlling the 11 characters listed under Materials and Methods cover six out of the haploid set of seven chromosomes. An initial survey took into account the first nine characters in this list. Correlations were sought between their segretation and that of chiasma frequencies in F_2 progenies. Of the nine characters used only one, kernel row number, was correlated with the chiasma frequency. Control of this character depends on at least one gene located in chromosome 2. On the assumption that the correlations may reflect a " concentration " of chiasma frequency genes in chromosome 2 two more characters marking that chromosome, awn length and internode length, were surveyed. These two, in addition to kernel row number and height, which was also included in the initial survey, provide a means not only for confirming that genes influencing chiasma frequency are found in chromosome 2. They may be used to indicate where, within that chromosome, such genes are located. Details of the correlations between markers and chiasma frequency are given below.

(b) *Chromosome 2*

(i) *Kernel Row Number*

It will be recalled that two types of segregation were found in the F_2 families from crosses between two- and six-rowed parents. Crosses with *H. agriocrithon* as the six-row parent gave segregations indicating control by two genes. One of the genes is on chromosome 2. The location of the second gene is not known.

In table 6 are the mean chiasma frequencies for the different segregating phenotypes in the only two F_2 families providing at the same time sufficient data and, as well, being derived from parents differing both in chiasma frequency and kernel row number. Both " *a* " and " *b* " are F_2's from crosses between high chiasma frequency/six-rowed and low chiasma frequency/two-rowed parents. Because the genes for high chiasma frequency carried by

TABLE 6

The mean chiasma frequencies of two-row and non-two-row segregates in two F_2 families

	F_2 cross	Two-rowed	Non-two-rowed
a	*H. agriocrithon* × *H. spontaneum*	14·43	14·90
b	*H. agriocrithon* × *H. distichum*	14·95	15·45
Mean		14·69	15·18

H. agriocrithon are dominant we should expect, if there is linkage between kernel row number and chiasma frequency, the two-rowed segregates to have a lower mean chiasma frequency. An analysis of variance confirms this (P = 0·05-0·01). This evidence provided the first, albeit tenuous, indication of a possible linkage between genes influencing chiasma frequency with a marker gene in chromosome 2.

Awn length : Chiasmata

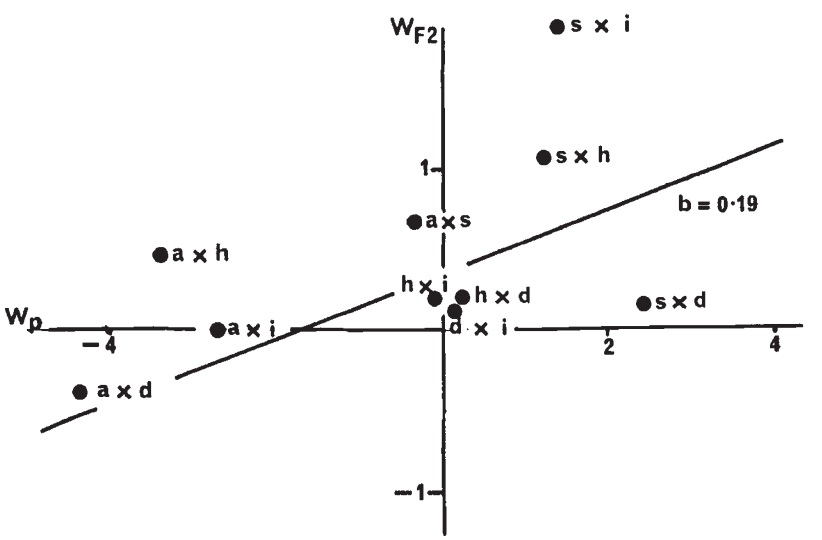

FIG. 3.—Mean awn length plotted against the mean chiasma frequency for parents, indicated by the appropriate initial letters, and for individual F_2 plants derived from these parents.

(ii) *Awn length and chiasma frequency*

Although the segregation results in a mainly discontinuous variation in F_2 it will be recalled that, due to the difficulty of distinguishing between short awn (*lk*/*lk*) and intermediate (*Lk*/*lk*) phenotypes, awn length was treated as a continuous variable, no attempt being made to " split " the F_2 progenies into the three discrete classes.

The awn length is plotted against the chiasma frequency for individual plants in all segregating F_2 families in fig. 3. Also plotted are the means for awn length and chiasma frequency in the parent phenotypes. The graphs show that, in general, the awn length/chiasma frequency relationship in each F_2 is similar to that displayed by the two parents. For example, where

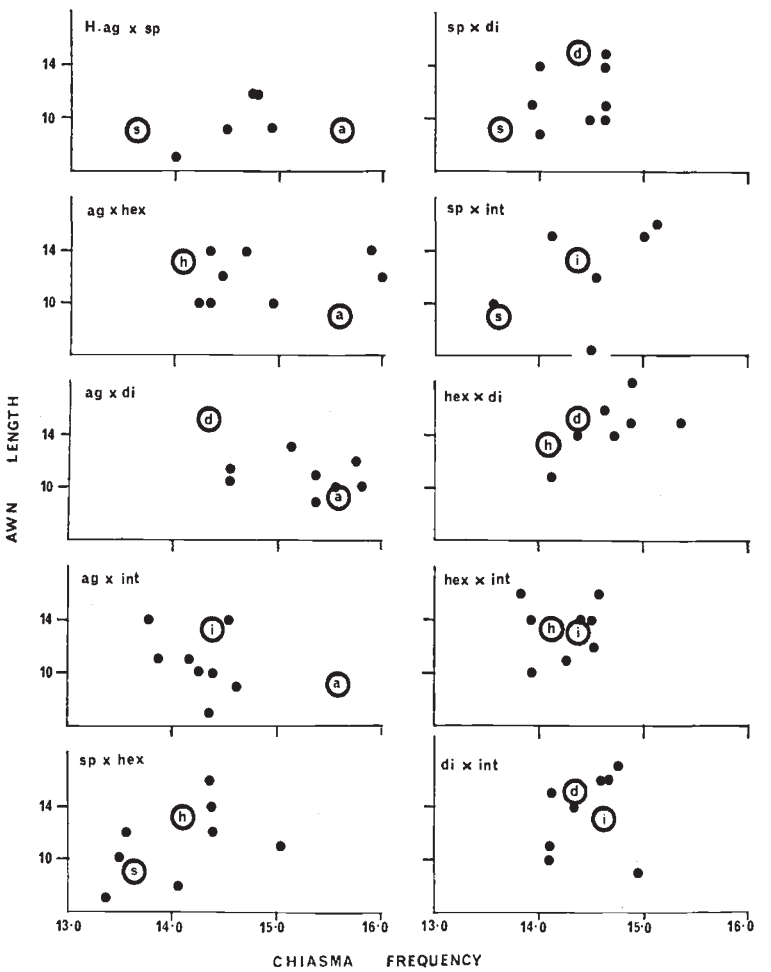

FIG. 4.—The covariance of awn length on to chiasma frequency for the F_2 (W_{F_2}) plotted against the parental covariance (W_p).

the combination of characters in the parents is high chiasma frequency/long awns and low chiasma frequency/short awns then awn lengths among the F_2 tend to increase with increasing chiasma frequency. In order to test the significance of the parental and F_2 correlations the method adopted was to compare the covariance of awn length on to chiasma frequency in each F_2 with that of the parental covariance. The parental covariance is $W_p = \Sigma Pxy$, where x and y are the mean parental values for the two characters, and the F_2 covariance is $W_{F_2} = \dfrac{\Sigma Px'y'}{n-1}$ where x' and y' are the values for the

two characters in individuals of the F_2 family, n being the number of individuals in that family. The covariances are presented in fig. 4. An analysis of variance shows that W_p/W_{F_2} gives a linear regression significant at the 5 per cent. level. In this analysis the probability value was halved because the W_p/W_{F_2} relationship is on *a priori* grounds expected to be positive.

(iii) *Spike internode length* (Spike density)

Tests for correlations betwen chiasma frequency and internode length in F_2 are of the *parent covariance*/F_2 *covariance* type described above. Internode

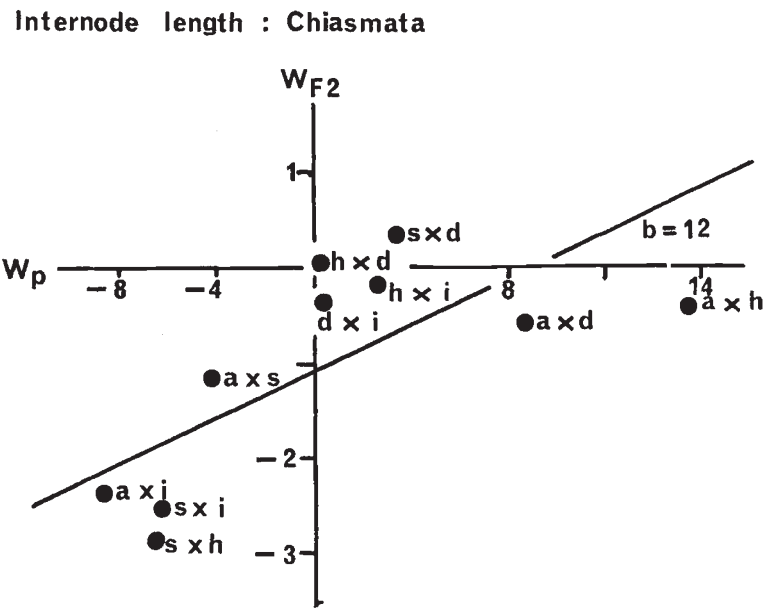

FIG. 5.—Mean internode length plotted against the mean chiasma frequency for parents and for individual F_2 plants.

length, like awn length, has been treated as a continuous variable with no attempt at sub-division into arbitrary classes. The internode lengths for parents and F_2's are plotted in fig. 5. In figure 6 are the F_2 covariances plotted against the parental covariances. On the expectation of a positive slope the regression is significant at the 2 per cent. level.

The correlations between the expression of kernel row number, awn length and of spike internode length with chiasma frequency among parental and F_2 phenotypes together provide very strong evidence for linkage between genes controlling chiasma frequency and genes on chromosome 2. In contrast, there was no indication of a linkage between chiasma genes and the other marker on chromosome 2, namely plant height.

(c) *Linkage between the markers*

Before concluding that genes controlling the chiasma frequency at meiosis are located in chromosome 2 it is first necessary to confirm that the three characters correlated with chiasma frequency are linked with one

another as would be expected from the previous reports showing all three to be controlled by genes in chromosome 2. The following " linkage tests " were carried out.

1. Between kernel row number and awn length.
2. Between kernel row number and internode length.

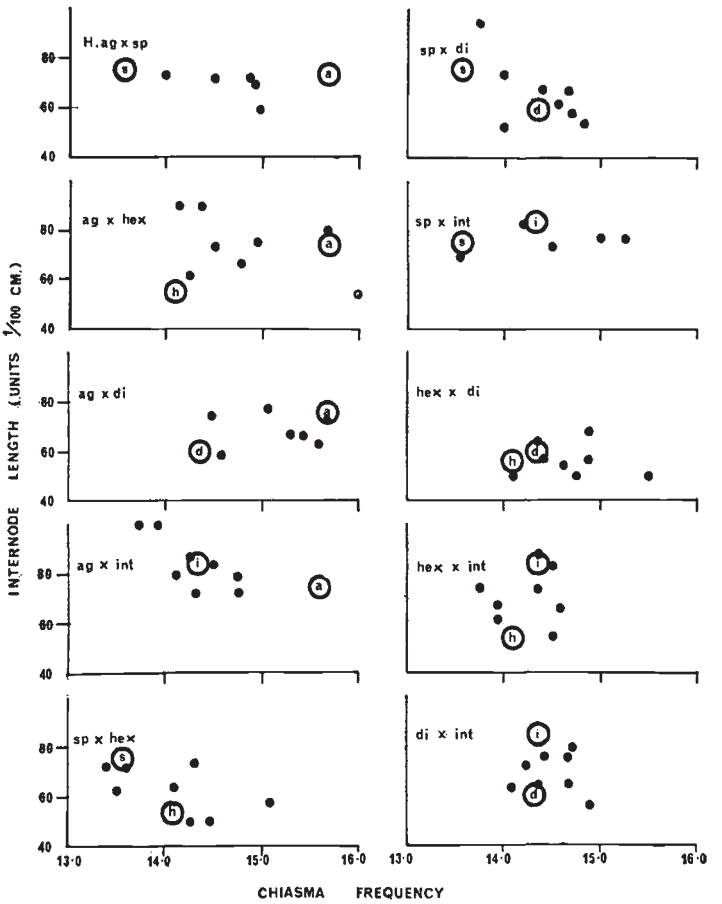

FIG. 6.—The covariance of internode length on to chiasma frequency for the F_2 (W_{F_2} plotted against the parental covariance (W_p).

(i) *Kernel row number/awn length*

Only three families segregate in respect of both characters. Two of these (table 7) are from crosses between six-rowed parents with " short " awns, and two-rowed parents with " long " awns. In the F_2's the two characters are associated in the same combination as in the parents *i.e.* increasing awn length with decreasing kernel row number. The association or correlation of characters is significant ($P = < 0{\cdot}001$ and $P = < 0{\cdot}03$, table 7). The probabilities are halved because the expectation in F_2 is for the same combination of characters as was shown by the parent.

In the third cross, between *H. hexastichum* and *H. spontaneum*, the correlations were not significant.

The significant correlations between kernel row number and awn length in two of the three families, however, is good confirmation of the expected linkage between their determinants.

TABLE 7

Awn length and kernel row number in parents and F_2 progenies

(a) *H. agriocrithon* (six-rowed with short awns—8·3 cm.) × *H. distichum* (two-rowed with long awns—15·0 cm.)

Awn length (cm.)	Two-rowed	Intermediate	Six-rowed
6	–	1	–
8	–	2	1
10	5	7	1
12	7	5	–
14	3	2	–
16	4	–	–
18	1	–	–
20	–	–	–
22	1	–	–
Total	21	17	2

Correlation coefficient (r_{39}) = 0·50 (P = <0·001).

(b) *H. hexastichum* (six-rowed with relatively short awns—13 cm.) × *H. distichum* (two-rowed with long awns—15 cm.)

Awn length (cm.)	Two-rowed	Intermediate	Six-rowed
6	–	–	2
8	–	1	1
10	–	2	1
12	4	2	1
14	5	4	5
16	2	3	–
18	2	2	1
20	1	–	–
22	–	1	–
Total	14	15	11

Correlation coefficient (r_{39}) = 0·30 (P = <0·03).

(ii) *Kernel row number and internode length*

Linkage is nicely demonstrated by using the parent/F_2 covariance analysis described in the previous section. The covariances of internode length on kernel row number in F_2 (W_{F_2}) are plotted against the parent covariances (W_p) in fig. 7 for the four families segregating in respect of both characters. A positive regression is evident, and significant (P = <0·03).

The two tests above serve to confirm that all three characters are represented by markers on the same chromosome (chromosome 2) as had been previously reported.

(d) *The possibility of pleiotropy*

While the correlations demonstrated between chiasma frequency and the three morphological characters are compatible with linkage between their determinants they could be attributed to another cause, namely pleiotropy. That genes controlling each of the three morphological characters would, as well, have pleiotropic effects on chiasma frequency is extremely unlikely.

It is ruled out completely on the grounds that the chiasma frequency of the parental subspecies is quite independent of morphology. For example, both *H. agriocrithon* and *H. spontaneium* display short awns. The former, however, has a high, the latter a low, chiasma frequency.

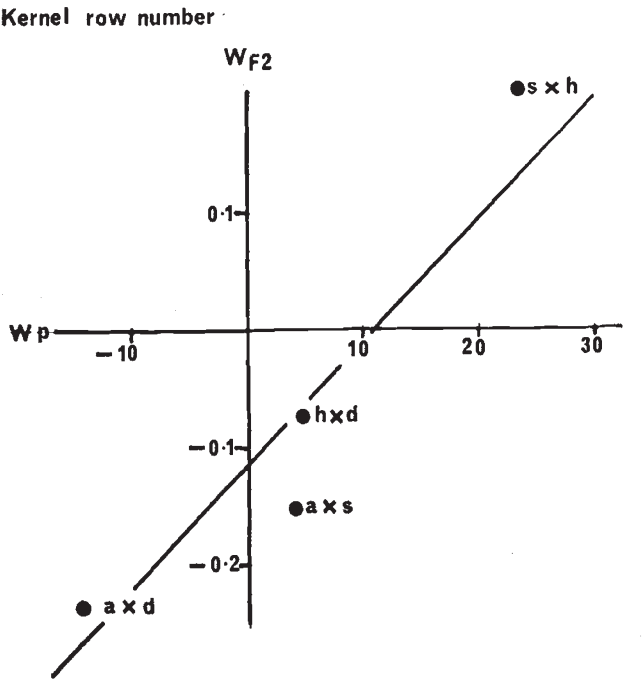

FIG. 7.—The covariance of internode length on kernel row number in F_2 (W_{F2}) plotted against the parental covariance (W_p) for the four F_2 families segregating for both characters.

(e) *The distribution of markers in chromosome 2*

Having disposed of pleiotropy and established therefore that genes controlling chiasma frequency are concentrated in chromosome 2 it is worthwhile trying to find out where on this chromosome they are located. Genes controlling awn lengths (*Lk/lk*), spike internode length (*Rin/rin*) and kernel row number (*V/v*) have been mapped in the long arm, covering a distance of not more than 40 units (Litzenburger and Green, 1951; Woodward, 1957). The gene affecting height (*H/h*) has been mapped outside the region spanned by *rin* and *v*. It will be recalled that there was no suggestion of linkage between chiasma frequency and plant height. It is possible that the concentration of genes influencing chiasma frequency in chromosome 2 are located, at least mainly, within the region *v-rin* in the long arm.

(f) *A measure of the activity of genes on chromosome 2 affecting chiasma frequency*

If genes controlling the variation in chiasma frequency were distributed uniformly throughout the *Hordeum* complement, each of the seven chromosomes would contribute a one-seventh share to the control of the variation. To detect a correlation between chiasma frequency and markers on a particular chromosome does not in itself, therefore, argue for a " concentra-

tion " of chiasma genes in that chromosome. The establishment of the correlation, *i.e.* of linkage, in one chromosome and not in others could reflect more upon the effective marking of that chromosome rather than upon a " concentration " of chiasma frequency genes. The assumption implicit throughout this paper that the linkage, in chromosome 2, is evidence for a " concentration " of chiasma genes in that chromosome is only justified therefore if it can be shown that genes on chromosome 2 contribute disproportionately more than do genes on other chromosomes to the chiasma variation.

In the present work one of the best of the markers in chromosome 2, indeed in the whole complement, is awn length. Although it is difficult to distinguish intermediate (*Lk*/*lk*) from short awned phenotypes (*lk*/*lk*) it is possible with reasonable confidence to classify the F_2 families into " shorts " (*Lk*/*lk* and *lk*/*lk*) and " longs ". This facility permits of a rough assessment of the extent to which chromosome 2 contributes to the chiasma frequency variation.

H. spontaneum crosses. The chiasma frequencies of " short " and " long " awned F_2 progenies from crosses involving *H. spontaneum* are given in table 8. *H. spontaneum* has short awns and a low chiasma frequency. As expected the short awned F_2 plants have a lower chiasma frequency than the long awned. The difference amounts to 0·43 chiasmata. 0·43 chiasmata represents 64 per cent. of the difference between the chiasma frequency of the parents, *i.e.* between *H. spontaneum* (13·62) and the other three species (14·29). Because the *Lk*/*lk* and *lk*/*lk* genotypes have been combined in this analysis the value is likely to be an underestimate of the contribution to the chiasma variation of genes on chromosome 2 linked to *lk*. Whether or not this is so there would appear to be no doubt that genes linked to *lk* on chromosome 2 contribute disproportionately to the chiasma frequency variation.

Crosses involving H. agriocrithon. H. agriocrithon has short awns and a relatively high chiasma frequency (15·64). As expected the long awned F_2 plants have the lower chiasma frequencies, 14·41 as compared with 14·91 for the " short " (*Lk*/*lk* and *lk*/*lk*). The difference between the classes, 0·50 chiasmata, represents 37 per cent. of the difference in the chiasma frequency of *H. agriocrithon* (15·64) as compared with the other parents (14·29).

In neither of the above comparisons are the data extensive enough nor precise enough to lend anything approaching accuracy to the estimates of variation attributable to chromosome 2. Together they do provide, however, firm grounds for concluding a large contribution by chromosome 2, a " concentration " of activity affecting chiasma frequency in that chromosome.

4. Discussion

That differences in chiasma frequencies between subspecies are genotypically controlled is not surprising. It is not clear what adaptive role, if any, they play in this instance because the subspecies are all strict inbreeders. Consequently differences in chiasma frequencies, other than very low frequencies causing chromosome loss and infertility, can have no relevance to adjustments in the amount of genetic recombination. It may be that the differences are relicts of an adaptive variation in genetic recombination between outbreeding ancestors.

Of greater interest is the information gained about the control of the

TABLE 8

The chiasma frequencies of "short" (Lk/lk *and* lk/lk) *and "long" awned* (Lk/Lk) *plants in F_2's derived from* (*a*) H. spontaneum *and,* (*b*) H. agriocrithon *crosses*

(*a*)	"Shorts"	"Longs"
	13·40	14·30
	14·30	14·60
	14·10	14·70
	15·05	14·05
	13·50	15·20
	14·35	15·00
	13·65	14·20
	14·70	
	13·95	
	14·65	
	14·05	
	14·40	
	14·55	
	13·55	
Means	14·15	14·58

Data pooled from *H. spontaneum* × *H. hexastichum* × *H. distichum* × *H. intermedium.*

(*b*)	"Shorts"	"Longs"
	16·00	14·45
	14·40	15·15
	14·95	13·85
	14·30	14·20
	14·70	
	15·80	
	15·30	
	15·35	
	15·55	
	15·85	
	14·60	
	15·80	
	14·60	
	14·60	
	14·25	
	14·30	
	14·65	
	13·90	
	14·30	
Means	14·91	14·41

Data from *H. agriocrithon* × *H. hexastichum* × *H. distichum* × *H. intermedium.*

chiasma frequency. It exhibits a continuous variation in heredity such as would arise by polygenic control, exercised mainly through genes with additive effects but partly, also, through genes with non-additive action including dominance. The dominance is in the direction of higher chiasma frequency, as in rye (Rees and Thompson, 1956; *cf.* Sun and Rees, 1964)

and *Drosophila* (Lawrence, 1958, 1963; Law, 1961). The assay described provides further information on this controlling system. We appreciate, of course, that the information from the assay is nowhere near as precise as that familiar to us from assays in other organisms, namely *Drosophila* (*e.g.* Mather and Harrison, 1949; Breese and Mather, 1957; Thoday, 1966) and wheat (Law, 1967; Kempanna, 1963). Partly this is because the marking of the *Hordeum* complement was lacking in respect both of the number and quality of marker genes. Equally important, we had no means by which to " block " recombination and thereby maintain and manipulate intact the marked chromosomes in the way achieved, for example, by inversions in *Drosophila*. Our assay, in fact, was crude and approximate. Even so it does provide evidence for a " concentration " of genes controlling chiasma frequency in one particular chromosome, *viz.* chromosome 2. The " concentration " may be interpreted in one of two ways. Either there is a large cluster of polygenes in chromosome 2 which determines, in large part, the chiasma frequency or, else, one or few genes with major effect on chiasma frequency are located in this particular chromosome. As Mather (1949) points out a continuous variation, such as displayed by the chiasma frequencies in *Hordeum*, does not always reflect control of the classical polygenic type, namely by genes of small, equal and supplementary effect. Whether, in this present instance, it be a major gene or a cluster of polygenes that is involved it is interesting to note that Kempanna's assay of wheat chromosomes (1963) revealed a disproportionate influence upon the chiasma frequency variation of homoeologous chromosomes 3A, 3B and 3D. It appears that in the two cereals one particular chromosome type is inordinately involved with control over the chiasma frequency.

Finally, the assay confirms the feasibility of dissecting and partitioning into manipulative components the genetic systems controlling *some* characters which display a continuous variation in heredity (Thoday, 1966).

5. Summary

1. Variation in chiasma frequency between *Hordeum* subspecies is controlled by genes with mainly additive effects. To a lesser extent control is exercised by genes with non-additive effects including dominance. High chiasma frequency is dominant to low.

2. The variation displayed is of a continuous nature. A chromosome assay revealed that a disproportionate contribution to the chiasma frequency variation is attributable to a gene, or to a cluster of genes, located in chromosome 2.

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