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The influence of the group 1 chromosomes of wheat on ear-emergence times and their involvement with vernalization and day length

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By using aneuploid lines of wheat in the variety Chinese Spring, each of the homoeologous group 1 chromosomes was found to delay ear emergence. More than one gene per chromosome was probably involved, and, because of the delays in ear emergence, at least one of them may be responsible for producing an inhibitor of flowering. The genes interacted with each other and with vernalization and day length. The genes on chromosome 1A were the most potent and 1B the least. The genes on the group 1 chromosomes may be related to the genes for vernalization and day-length sensitivity found on the homoeologous chromosome 1H of barley. Reciprocal monosomic analyses of three varieties detected allelic variation between homologues of group 1 for ear-emergence time.

Keywords: day length, ear-emergence time, flowering inhibition, group 1 chromosomes, vernalization, wheat.

Introduction

The homoeoallelic series of major photoperiodic and vernalization genes (*Ppd1*, *Ppd2* and *Ppd3*, and *Vrn1*, *Vrn4* and *Vrn3*), located on the group 2 and 5 chromosomes, respectively, usually features in any description of the genetical regulation of ear initiation and development in wheat (Tsunewaki, 1966; Pugsley, 1971; Klaimi & Qualset, 1973; Law *et al*., 1976; Maystrenko, 1980). This is because these genes appear to be responsible for the major adaptive divisions of wheat into winter and spring types and those which are day-length insensitive and sensitive. Genetical studies of the differences between these divisions invariably pinpoint one or more genes from these two groups as having a decisive role. This has tended to reinforce their pre-eminent position as determinants of ear initiation and development to the exclusion of other genes on other chromosomes which also influence these characters. This is despite the fact that almost every chromosome of wheat has been cited at one time or another as having some effect (Kuspira & Unrau, 1957; Morrison, 1960; Halloran & Boydell, 1967a,b). The role that such genes might have in wheat breeding as well as in the overall control of the flowering process has therefore received little attention. Because of this there is a need to identify and characterize these additional genes to establish the full panoply of genes controlling the flowering of wheat.

As a first step, a systematic study of each homoeologous group of chromosomes, using aneuploids and 'alien' chromosome addition and substitution lines to look at chromosome dosage effects, is a rapid way of locating critical chromosomes. This could pave the way for the second phase of investigation, the identification of allelic variation through the study of varietal differences. This approach has been used successfully to investigate the control of ear-emergence times by the chromosomes of homoeologous group 6 (Islam-Faridi *et al*., 1996). The study not only identified critical chromosome arms controlling day-length response, but also the presence of separate genes whose increased dosage delayed ear emergence possibly through the production of an inhibitor. A similar study of homoeologous group 3 has also been undertaken revealing the presence of earliness *per se* genes, i.e., genes

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acting independently of day length and vernalization (Miura & Worland, 1994).

This paper reports a series of experiments designed to reveal the genes on the group 1 chromosomes influencing ear emergence and their reaction to vernalization and day length.

Materials and methods

Three of the four experiments described in this paper used the homoeologous group 1 aneuploid lines of Chinese Spring (CS), derived by Sears (1954), and the addition and substitution lines of chromosome 1U of *Aegilops umbellulata*, again in CS. The fourth experiment employed reciprocal F_2 monosomic populations derived by crossing the monosomics of homoeologous group 1 from the varieties Bersee, Cappelle-Desprez and Koga II.

Experiment 1

This consisted of all six CS nulli-tetrasomic lines of homoeologous group 1, the three CS tetrasomics, the addition and substitution lines of 1U into CS, and the CS euploid. It is possible to arrange these genotypes into a two dimensional matrix such that rows represent chromosomes 1A, 1B, 1D and 1U, and columns 1A1B1D, 1A1B, 1A1D and 1B1D. This design, given in Table 1, requires that the CS euploid should be represented three times, and because only one CS euploid was used in the experiment an appropriate correction has to be made to the number of degrees of freedom in the analysis of variation. The design is similar to the North Carolina design 2 so that deviations of the means of rows and columns from the overall mean provide estimates of the additive effects of each chromosome (rows), and the additive effects of combinations of chromosomes (columns), whereas departures from the predicted genotypic values based upon the row and column estimates give a direct estimate of interaction between chromosomes.

The six CS nulli-tetrasomic combinations can also be grouped into reciprocal pairs such that each pair if crossed would produce the euploid, i.e. CS Nulli 1B Tetra 1D or 1A1D1D × CS Nulli 1D Tetra 1B or 1A1B1B = the CS euploid or 1A1B1D. This is identical to saying that the two nulli-tetrasomics are the parents and the euploid is the F_1 between them. The departure of the euploid from the midpoint between the two reciprocal nulli-tetrasomics therefore provides a further measure of between-chromosome interaction, in this case, between chromosomes 1B and 1D.

Table 1 Experiment 1. Mean days to ear emergence in the field of Chinese Spring (CS) euploid, the six CS nulli-tetrasomics and three CS tetrasomics of homoeologous group 1, and CS addition and substitution lines of chromosome 1U from *Aegilops umbellulata* arranged so that rows represent the single homologous pairs of chromosomes 1A, 1B, 1D and 1U, and columns, combinations of homologous pairs, 1A1B1D, 1A1B, 1A1D and 1B1D. Estimates, in bold type, of additive effects of rows and columns are also given

	1A1B1D	1A1B	1A1D	1B1D	Total	Additive effects
	Tetra 1A	N1DT1A	N1BT1A	Euploid		
1A	1A1A1B1D	1A1A1B	1A1A1D	1A1B1D	91.00	**+0.91**
	25.20	23.20	22.60	20.00		**±0.58**
	Tetra 1B	N1DT1B	Euploid	N1AT1B		
1B	1B1A1B1D	1B1A1B	1A1B1D	1B1B1D	86.00	**−0.34**
	25.20	21.00	20.00	19.80		
	Tetra 1D	Euploid	N1BT1D	N1AT1D		
1D	1D1A1B1D	1A1B1D	1D1A1D	1D1B1D	85.00	**−0.59**
	21.60	20.00	22.40	21.00		
	1U add	1D(1U)sbst	1B(1U)sbst	1A(1U)sbst		
1U	1U1A1B1D	1U1B1D	1U1A1D	1U1A1B	87.40	**+0.01**
	23.20	23.40	22.00	18.80		
Total	95.20	87.60	87.00	79.60	349.40	
Additive	**+1.96*****	**+0.06**	**−0.09**	**−1.94*****		Av 21.84
effects	**±0.58**					±0.26

***$P < 0.001$.

The experiment was grown from a spring sowing in the field at the former Plant Breeding Institute, Cambridge. Each genotype was represented by five plants, whose chromosome composition was checked cytologically by examination of root-tips obtained from germinating seeds. The plants were grown in small pots in a heated glasshouse until about the second leaf stage when they were transplanted to the field using a single plant randomization layout. The date of ear emergence was scored for each plant and the days to ear emergence from an arbitrary date calculated.

Experiment 2

This experiment followed the procedure of Experiment 1 closely, but consisted of the long-armed ditelocentric lines for each of the group 1 chromosomes as well as their tetrasomics. Duplicate lines of the CS euploid were also grown in the experiment. Six plants were used for each line or genotype and these were germinated and grown under glasshouse conditions for a short period before being transplanted into the field.

Experiment 3

The design used in the first experiment was repeated in this experiment. However, because of restrictions in available space, the CS tetrasomics and the CS 1U addition line were not included. One of the columns estimating the combined effect of the 1A1B1D chromosomes was therefore missing from the analysis. Also unlike experiment 1, three sets of the CS euploid were introduced so that the design was completely orthogonal. The experiment was undertaken in two controlled environment cabinets, one having long-day, the other short-day conditions. In each of the cabinets, the plants received 8 h of high intensity fluorescent light combined with low level incandescent lighting, consisting of 8 h, synchronized with the main lighting, for the short-day and continuous for the long-day cabinets. Temperatures in each of the cabinets were kept at a constant 18°C, day and night. Half of the plants were vernalized prior to their transfer to their respective cabinets by treating germinated seeds for five weeks at 4°C under short-day conditions. The remaining unvernalized seedlings, having been germinated from seed four days before at 25°C, were also transferred to the cabinets at the same time. Four environmental treatments were therefore given to the genotypes: unvernalized short days, unvernalized long days, vernalized short days and vernalized long days. In each of these treatments, each of the 12 genotypes was replicated four times.

The analysis of each treatment was carried out separately. Estimates of additive chromosomal effects and between-chromosome interactions were derived for each treatment, in the manner described for experiment 1. These were then combined to give estimates of the overall chromosomal and interaction effects, and their interactions with vernalization, day length and vernalization/day length.

Experiment 4

An experiment was undertaken in the field to detect chromosomal differences in the control of the days to ear emergence between the winter wheat variety, Cappelle-Desprez, the facultative wheat variety, Bersee, and the spring variety, Koga II. Because this survey included the homoeologous group 1 chromosomes, the results of the investigation of these chromosomes are reported here. The method of reciprocal monosomic analysis was used to investigate the differences between the three varieties. Monosomic series exist in all three varieties, having been developed at the former Plant Breeding Institute in Cambridge, by recurrent backcrossing to the original monosomic series in Chinese Spring for at least eight generations. All monosomic lines had been checked to confirm that they were authentic. By intercrossing reciprocally the same monosomic from two varieties, two F_1 monosomic hybrids can be selected cytologically, which have identical backgrounds but whose hemizygous chromosomes originate from different varieties. Any phenotypic differences between the two monosomic hybrids must reflect allelic differences between the two hemizygous chromosomes. This is the basis of the reciprocal monosomic method (McKewan & Kaltsikes, 1970; Law *et al*., 1987). By selfing each of the F_1 monosomic hybrids, F_2 monosomic hybrid families are produced, the means of which can be compared with similar expectations of detecting allelic differences as the F_1 monosomic hybrid plants.

In this experiment, F_2 monosomic seed was sown directly into the field in the early spring. The field design consisted of randomized plots, each being a row of seven plants spaced 15 cm apart. Between-row distances were 30 cm and each F_2 family was represented by 10 plots. The mean days to ear emergence was recorded for each plot. Included in the experiment were control F_2s for each of the crosses to provide a measure of the effect of chromosome dosage.

Results

Chromosome dosage of homoeologous group 1 chromosomes

The mean days to ear emergence for each of the genotypes in the first experiment is given in Table 1. One of the striking features of the results is that all three tetrasomics and the addition line with 1U give ear-emergence times greater than the CS euploid. Increased chromosome dosage therefore delays ear emergence.

As already mentioned, for the design into columns and rows to be complete and orthogonal, the CS euploid has to be represented three times. Because only one CS euploid observation was available, the maximum number of degrees of freedom available for comparing genotypic variation is only 11 and not 13. Two degrees of freedom have therefore been removed from the interaction item in the analysis of variance (Table 2). The column item in the analysis is highly significant, the row and interaction items being insignificant. The estimates of combined chromosome additive effects (column estimates) show that for the columns, the 1A1B1D and 1B1D estimates are significant, and opposite in sign, the 1A1B1D combination delaying ear emergence, 1B1D accelerating it. None of the single chromosome estimates (row estimates) is significant, although the rankings place 1A as having the greatest delaying effect, 1B and 1D being very similar in accelerating ear emergence, and 1U being intermediate.

Perhaps the most informative comparisons that can be made from this analysis of the experiment are between the column estimates. This really follows on from the delayed ear emergence of the tetrasomics. Thus the comparison between 1A1B1D and 1B1D is in effect a measure of chromosome 1A against its absence, i.e. 1A1B1D vs. 1B1D or 1A vs. zero $= +3.90 \pm 0.82$, $P < 0.001$. In other words, the effect of the 1A chromosome is to delay ear emergence. Similar comparisons can also be made for chromosome 1B, i.e. 1A1B1D vs. 1A1D or 1B vs. zero $= +2.05 \pm 0.82$, $P < 0.05$; 1A1B1D vs. 1A1B or 1D vs. zero $= +1.90 \pm 0.82$, $P < 0.05$. Although these comparisons are not independent of each other, they nevertheless show that all three chromosomes delay rather than accelerate ear emergence, 1A having twice the delaying effect of 1B and 1D. The negative column and row estimates, arising because they are expressed as deviations from the mean, are therefore a reflection of the reduced effects of 1B and 1D relative to 1A on delaying ear emergence.

The analysis of the three reciprocal pairs of nullitetrasomics and the position of the CS euploid in relation to their midpoints is also of interest because it provides a separate test of between-chromosome interaction. Although this is not such a robust test in this case, because of the availability of only one value for the CS euploid, all three comparisons give departures from the midpoint which are negative, i.e. 1A vs. 1B $= -1.20 \pm 1.03$; 1A vs. 1D $= -2.10 \pm 1.03$, $P < 0.05$; 1B vs. 1D $= -1.70 \pm 1.03$. Even though only one of these comparisons is significant, the consistent negativity suggests that there may be a level of between-chromosome interaction which, rather surprisingly, opposes the activity of each chromosome. As the dose series indicate, this is positive and delays rather than accelerates ear emergence.

Arm locations

The second experiment was designed specifically to test whether the delaying effects of each of the group 1 chromosomes could be assigned to particular arms. The results of the experiment are shown in Table 3 and differ from the previous experiment in

Table 2 Analysis of variance of experiment 1

Item	d.f.	MS	VR
Columns	3	50.78	9.49***
Rows	3	8.61	1.61
Interaction	7	10.57	1.97
Error	56	5.35	

***$P < 0.001$.

Table 3 Experiment 2. Mean ear-emergence times of Chinese Spring (CS) euploid, tetrasomics and long-arm ditelosomics of homoeologous group 1 grown from a spring sowing in the field

Lines	Ear emergence	Deviation from CS euploid
CS tetrasomic 1A	84.33	4.33***
CS tetrasomic 1B	80.33	0.33
CS tetrasomic 1D	80.17	0.17
CS ditelosomic 1AL	75.80	−4.20***
CS ditelosomic 1BL	79.67	−0.33
CS ditelosomic 1DL	81.33	1.33
CS euploid	80.00	
CS euploid	80.00	

***$P < 0.001$.

showing significant effects for chromosome 1A only. This may indicate that the conditions of this experiment were more favourable to vernalization than the earlier experiment. The data show, however, that the CS tetrasomic 1A is delayed compared to the CS euploid, confirming previous results, but that CS ditelosomic $1A^L$ is significantly earlier. Indeed, the difference between the tetrasomic and the ditelosomic is more than 8 days. The short arm therefore appears to have a major effect on delaying ear emergence. Because neither CS ditelosomic $1A^S$ nor CS nullisomic 1A is available at the moment, it is impossible to establish whether the short arm is solely responsible for the delay in ear emergence.

Group 1 chromosomes and their response to vernalization and day length

The analysis of ear-emergence times for the 12 genotypes, the six CS nulli-tetrasomics of group 1, three substitutions of 1U into CS and three separate CS euploids, in each of the four environments (unvernalized short days, unvernalized long days, vernalized short days and vernalized long days) is presented in Table 4. The rows item in this analysis refers to differences between the additive effects of the chromosomes, 1A, 1B, 1D and 1U, the columns to differences between the additive effects of the chromosome combinations, 1A1B, 1A1D and 1B1D, and the interaction to departures from additivity for the rows and columns. The analysis indicates that all three items are highly significant in each of the four environments. Unlike the first experiment, the differences between the chromosomes, 1A, 1B, 1D and 1U, are significant, but more strikingly, the interaction between chromosomes is a significant feature which was not so apparent in the earlier experiment. In general the MSs for rows, columns and interactions are smaller in the vernalized treatment than the unvernalized; a similar relationship applies to long days vs. short days. As might have been anticipated, both of the major environmental variables therefore interact with genotype, long days and vernalization reducing the variation between genotypes compared to short days and no vernalization.

A more detailed analysis, however, emerges by considering, for each of the environments, the estimates of additive chromosome effects (rows), additive chromosome combination effects (columns) and the between-chromosome interaction for each of the 12 genotypes. Each of these estimates will have an appropriate error, so that it is possible to consider each estimate and determine its overall mean and interactions with the different environments. Because there are four environments, three different genotypic/environment interactions can be derived for each genotypic estimate, i.e. unvernalized vs. vernalized, short days vs. long days and the interaction unvernalized vs. vernalized/short days vs. long days. The results of these calculations are given in Table 5.

The overall means for the additive effect of each of the chromosomes indicate that 1A has the greatest delaying effect on ear-emergence time followed by 1D, then 1U and finally 1B. This ranking is reflected to some extent in the estimates of the effects of combined chromosomes with 1A1B and 1A1D giving positive and 1B1D negative effects. These rankings of mean chromosome effects are very similar to those obtained in experiment 1.

For both sets of chromosome estimates the interactions with vernalization and day length are significant and when the sign of the mean is taken into account, all the interaction estimates are positive. This means that vernalization and long days reduce

Table 4 Experiment 3. Analyses of variance of the days to ear emergence of Chinese Spring (CS) euploid, the six CS nulli-tetrasomics of homoeologous group 1, and the substitution lines of 1U from *Aegilops umbellulata* into CS grown under long-days unvernalized (LUV), short-days unvernalized (SUV), long-days vernalized (LV) and short-days vernalized (SV) conditions

Item	d.f.	LUV MS	SUV MS	LV MS	SV MS
Reps.	3	3.7	31.3	0.4	27.8
Rows	3	209.1***	321.5***	17.9***	227.6***
Columns	2	290.9***	585.7***	22.9***	141.9***
Interaction	6	33.3***	97.0***	6.9***	146.2***
Error	33	3.7	14.0	1.5	11.3

***$P < 0.001$.

the magnitude of the chromosome effects, the result also obtained by comparing the analyses of variance (Table 4).

For the interaction estimates, some of the mean effects are large, highly significant, and in many cases, because of the absence of interactions with vernalization and day length, consistent across environments. There is also a strong suggestion that, where interactions do occur, they mainly concern day length. If there is a pattern amongst the interaction estimates it would appear to relate to their direction. Thus, all three CS euploids (1A1B1D) have negative interaction values whereas the majority of the other significant interactions are positive. Although the results are not shown in the paper, the negative interactions are undoubtedly caused by the fact that the emergence times of each of the three CS euploids, irrespective of environment, are equal to or earlier than the earliest emerging nulli-tetrasomic of a reciprocal pair. This is exactly the pattern found in experiment 1 and is the opposite of that expected if the genes implicated in this study act only to delay ear emergence.

One apparent anomaly emerges from the interaction estimates. Again this relates to the three CS euploids. The interaction estimates for each of these identical genotypes should be the same, but they are not and differ depending upon their position in the table. Each interaction estimate is in fact a deviation of the predicted value from the observed based upon the column and row estimates. In the absence of any interactions, these provide accurate estimates of their additive effects. However, when there are interactions then the column and row estimates will involve interaction components. These will in their turn influence the predictions. This accounts therefore for the differences between the CS euploid interaction estimates. Unfortunately, it also means that the magnitude of many of the estimates will be influenced in the same way. This obviously complicates the interpretation of the data, particularly where the magnitudes of the effects are concerned. On the other hand, such correlated effects are unlikely to obscure the large effects of chromosome 1A, or the earliness of the CS euploid in relation to the reciprocal pairs of nulli-tetrasomics, or the incidence of interactions with the environment.

Table 5 Experiment 3. Estimates of single and paired chromosome additive effects (rows and columns) and between-chromosome interaction effects for each of the 12 genotypes, and their interaction effects with vernalization (UV vs. V), day length (S vs. L) and vernalization/day length (UV vs. V/S vs. L)

	Mean	UV vs. V	S vs. L	UV vs. V/ S vs. L
Single chromosome				
1A	4.02***	1.09**	0.92*	−0.52
1B	−1.85***	−2.35***	−1.19**	−1.20**
1D	−0.58	−0.56	2.28***	1.12**
1U	−1.58***	1.82***	−2.01***	0.61
Paired chromosomes				
1A1B	2.04***	0.48	−0.74	−0.98*
1A1D	1.82***	1.16***	1.80***	0.82*
1B1D	−3.87***	−1.64***	−1.06**	0.16
Between-chromosome interactions				
1A1A1B	3.07***	−0.84	0.76	−1.63**
1A1A1D	−1.19*	0.45	0.62	0.91
1A1B1D	−1.88***	0.38	−0.14	0.72
1B1A1B	0.73	0.06	−0.08	−0.24
1B1A1D	−3.07***	−0.11	−1.25*	0.60
1B1B1D	2.35***	0.05	1.33*	−0.37
1D1A1B	−4.53***	−0.17	−2.36***	0.68
1D1A1D	3.07***	−1.11	2.48***	−1.39*
1D1B1D	1.46**	1.28*	−0.12	0.71
1U1A1B	0.73	0.95	1.67**	1.18*
1U1A1D	1.20*	0.76	−0.61	−0.13
1U1B1D	−1.93***	−1.71**	−1.07	−1.06

*$P<0.05$, **$P<0.01$, ***$P<0.001$.

Allelic variation

The differences between the CS group 1 chromosomes and 1U must reflect the different alleles at related loci on these chromosomes. There should therefore be every likelihood that similar variation can be found between homologues derived from different varieties of wheat. Indeed, if such variation is not available then it will be difficult to identify the number of genes involved and their location by studying only homoeologues. For this reason the results of the fourth experiment could be important to the future study of these genetic effects.

The mean ear-emergence times of the 1B and 1D reciprocal F_2 monosomic families derived from the group 1 monosomics of Cappelle-Desprez, Bersee and Koga II are shown in Table 6. The results obtained from the 1A F_2 families are not included in the Table because none of the differences between them was significant. Also included in the Table are the means of the control F_2s for the three intervarietal crosses. Any differences between the reciprocally derived F_2 monosomic families indicate that the hemizygous chromosomes carry different alleles. Likewise, because the 1B and 1D chromosomes are involved with three reciprocal pairs of crosses,

Table 6 Experiment 4. Mean days to ear emergence in the field for the 1B and 1D F_2 reciprocal monosomic crosses between the group 1 monosomics of Koga II (Koga), Cappelle-Desprez (Cap) and Bersee

Chromosome	Cross	Chromosome origin	Reciprocals	Difference between reciprocals	Mid-point (a)	Disomic mean (b)	Difference (a)–(b)
1B	Koga × Cap	Cap	13.48 ± 0.85	0.98 ± 1.26	12.99 ± 0.80	17.05 ± 0.47	−4.06 ± 0.93***
	Cap × Koga	Koga	12.50 ± 0.93				
	Koga × Bersee	Bersee	12.32 ± 1.07	0.45 ± 1.59	12.10 ± 0.79	11.83 ± 0.25	0.27 ± 0.83
	Bersee × Koga	Koga	11.87 ± 1.17				
	Cap × Bersee	Bersee	15.97 ± 0.94	−2.97 ± 1.44*	17.46 ± 0.72	23.58 ± 0.41	−6.12 ± 0.83***
	Bersee × Cap	Cap	18.94 ± 1.09				
1D	Koga × Cap	Cap	19.07 ± 1.53	3.05 ± 2.75	17.55 ± 1.37	17.05 ± 0.47	0.50 ± 1.45
	Cap × Koga	Koga	16.02 ± 2.28				
	Koga × Bersee	Bersee	12.77 ± 1.09	−0.46 ± 1.38	13.00 ± 0.69	11.83 ± 0.25	1.17 ± 0.73
	Bersee × Koga	Koga	13.23 ± 0.84				
	Cap × Bersee	Bersee	19.45 ± 0.74	−5.76 ± 1.20 ***	22.33 ± 0.60	23.58 ± 0.41	−1.25 ± 0.73
	Bersee × Cap	Cap	25.21 ± 0.94				

*$P < 0.05$, ***$P < 0.001$.

Cappelle-Desprez × Bersee, Cappelle-Desprez × Koga II and Bersee × Koga II, it is possible to do a three-way check to see if the differences between each of the reciprocals fit and behave in an additive way. For this to be the case, any one of the reciprocal differences should equal either the sum of, or the difference between, the other two reciprocal differences.

In the event only two of the reciprocal differences, Cappelle-Desprez 1D vs. Bersee 1D, and Cappelle-Desprez 1B vs. Bersee 1B, are significant, the Cappelle-Desprez chromosome having the greater delaying effect on ear-emergence time in both cases. Using the three-way check, both differences fit an additive model, i.e. for the 1D chromosome, Bersee 1D vs. Cappelle-Desprez 1D = Bersee 1D vs. Koga II 1D − Cappelle-Desprez 1D vs. Koga II 1D, or $-5.76 \pm 1.20 = 0.46 \pm 1.38 - 3.05 \pm 2.75$, which reduces to a nonsignificant -3.17 ± 3.30; similarly for the 1B chromosome, where the comparison again reduces to a nonsignificant -2.44 ± 2.49.

In the present experiment, the differences between the F_2 disomic means and the means of the reciprocal F_2 monosomics are shown in Table 6. This provides a test of chromosome dosage. Of the six differences depicted in the Table only two are significant, Cappelle-Desprez/Koga II 1B and Cappelle-Desprez/Bersee 1B. Both are negative, indicating that reduced chromosome dosage accelerates the time to ear emergence. This agrees with the results of experiments 1 and 2 where the CS group 1 tetrasomics were consistently later and CS ditelocentric $1A^L$ earlier than the CS euploid. For Cappelle-Desprez/Bersee 1B, the difference attributable to dosage is associated with a significant difference between the reciprocal F_2 monosomics. The allelic differences detected between Cappelle-Desprez 1B and Bersee 1B could therefore be caused by the same genes giving the dosage effect. For Cappelle-Desprez/Koga II 1B, the dosage effect is not associated with any evident allelic difference. In this case, the alleles are probably the same but show dosage effects.

Discussion

The group 1 chromosomes of wheat

The simplest explanation of the results of the four experiments is that there are genes on each of the homoeologous group 1 chromosomes, probably located on their short arms, whose action delays ear-emergence time. The effects of the genes vary between homoeologues, the gene or genes on 1A being the most potent, those on 1B, the least. Allelic differences can be detected between the homologues of different varieties. The genes interact between themselves and are influenced by both day length and vernalization. The delay in ear-emergence time

could be a consequence of the genes producing an inhibitor.

A similar 'inhibitor' explanation was advanced by Islam-Faridi *et al.* (1996) to account for the delayed ear-emergence times by the tetrasomics of the group 6 chromosomes. The effects in the group 6 case were much larger than the effects reported here. Moreover, there was a clear involvement of vernalization because the inhibition effects almost disappeared after vernalization. This is not apparent in the present study, where the interaction with both vernalization and day length is much more complex. Islam-Faridi *et al.* (1996) suggested that the group 6 genes acted as suppressors of the activities of the major *Vrn* genes which promoted ear emergence. Removal of the suppressor by vernalization would allow expression of the *Vrn* genes, thereby accelerating ear emergence. A similar role could be proposed for the group 1 genes, with vernalization and day length being involved in the removal of any suppressor effect. It should be emphasized that in neither of these instances is it yet possible to establish unequivocally a connection with the *Vrn* genes, or indeed between the group 1 genes and those found on the group 6 chromosomes. For this to happen, much more needs to be known about the genes and their products.

An interesting, if puzzling, feature of the results is the behaviour of the CS euploid in relation to the reciprocal pairs of CS nulli-tetrasomics. In every case, the value of the CS euploid is either equal to or earlier than the earliest of the CS nulli-tetrasomics. In other words, the interaction is negative. This was also found for the interaction estimates for each of the three CS euploids in experiment 3. This is surprising if the genes are actively involved in delaying ear emergence through the production of an inhibitor. Under such circumstances, the interaction would be expected to be positive. An identical result was obtained by Islam-Faridi *et al.* (1996) for the group 6 chromosomes, and although it was noted, no explanation could be offered to account for the result.

The extreme earliness of the CS euploid, transgressing the earliest CS nulli-tetrasomic of a reciprocal pair, is also unexpected. Two of the three reciprocal pairs in the first experiment showed this, as well as several pairs, including all three in the vernalization/short-day treatment, in the third experiment. If only single genes were involved then this would be akin to overdominance, where, for example, the heterozygote represented by the CS euploid is AABB(DD) and is earlier than the two homozygotes represented by the nulli-tetrasomics, AAAA(DD) and BBBB(DD). However, the more likely explanation would involve at least two genes in repulsion on each of the A and B chromosomes, say $+-$ for chromosome A and $-+$ for chromosome B. When combined as in the CS euploid and given directional dominance for the — alleles, then AABB(DD) would be earlier than either AAAA(DD) or BBBB(DD).

These considerations suggest that the model presented in the first paragraph and based solely on one type of gene is incorrect and that there are other genes involved. These could be separate genes affected by day length for instance, as well as separate genes which respond to vernalization. It may even be that such genes have an opposite effect to the genes delaying ear emergence, and actually accelerate ear-emergence times. If this were so then their activities could be responsible for the puzzling negative interactions of the CS euploid. However, none of these hypotheses can be tested until more detailed analyses can be undertaken to determine the numbers of genes located on the group 1 chromosomes. In this endeavour the allelic variation detected in experiment 4 could be valuable.

Comparisons with barley

Chromosome 1H of barley is homoeologous with the group 1 chromosomes of wheat. Two genes, one sensitive to day length, *Ppd-H2*, and the other to vernalization, Sh_3, have been located on this chromosome (Takahashi & Yasuda, 1971; Laurie *et al*., 1995). This therefore lends support to the many-gene hypothesis for the group 1 chromosomes of wheat.

More general comparisons with barley agree with the *Ppd* locations on the group 2 chromosomes (*Ppd-H1* on barley chromosome 2H) and the *Vrn* locations on group 5 (Sh_2 on barley chromosome 5H), but not for the vernalization gene, *Sh*, on barley chromosome 4H. A dominant gene for winter habit, designated *Vrn7*, has recently been located in an accession of *Triticum monococcum* on the translocated segment of $5A^mL$ derived from $4A^mL$ (Dubcovsky *et al*., 1996). This primitive translocation is homoeologous with the region carrying *Sh* on barley chromosome 4H indicating that *Vrn7* and *Sh* are probably identical loci. At present there is no evidence for *Vrn7* on chromosome 5A of hexaploid wheat. Nor is there any indication that related genes occur on chromosomes 4B and 4D. This lack of corroborative evidence could have a number of explanations, but perhaps the most likely is the lack of an in-depth search for such a gene within wheat.

The type of aneuploid survey undertaken for the group 6 chromosomes (Islam-Faridi *et al.*, 1996), for the group 3 chromosomes (Miura & Worland, 1994) and here for the group 1 chromosomes could reveal the presence of such a gene if carried out on the group 4 chromosomes.

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