Theor Appl Genet (1990) 80:281–287

THEORETICAL AND APPLIED GENETICS

Dosage effects of chromosomes of homoeologous groups 1 and 6 upon bread-making quality in hexaploid wheat

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Received January 12, 1990; Accepted February 23, 1990
Communicated by J. W. Snape

Summary. The endosperm storage proteins, glutenin and gliadin, are major determinants of bread-making quality in hexaploid wheat. Genes encoding them are located on chromosomes of homoeologous groups 1 and 6. Aneuploid lines of these groups in spring wheat cultivar 'Chinese Spring' have been used to investigate the effect of varying the dosage of chromosomes and chromosome arms upon bread-making quality, where quality has been assessed using the SDS-sedimentation test. Differences between the group 1 chromosomes for quality were greater than those between the group 6 chromosomes. The chromosomes were ranked within homoeologous groups for their effect on quality as follows (> = better quality): *1D* > *1B* > *1A* and *6A* > *6B* = *6D*. The relationship of chromosome dosage with quality was principally linear for four of the chromosomes, but not for *6B* and *6D*. Increases in the dosage of *1B*, *6A* and, especially, *1D*, were associated with significant improvements in quality, whereas increases in the dosage of *1A* were associated with reductions in quality. The effects of *1A* and *1D* were such that the best genotype for quality was nullisomic *1A*-tetrasomic *1D*. For group 1, effects of the long arm appeared in general to be more important than effects of the short arm. For group 6, effects were found associated with the long arms as well as with the short arms, a surprising result in view of the absence of genes encoding storage proteins on the long arms. Significant interactions were found between chromosomes and genetic backgrounds, and between individual chromosomes. Analysis of trials grown over two years demonstrated that, although additive environmental differences over years and genotype × years interaction were present, they were relatively small in magnitude compared with purely genetic differences.

Key words: *Triticum aestivum* – Glutenin – Gliadin – Aneuploid

Introduction

Most studies of the relationship between endosperm storage proteins and bread-making quality in hexaploid wheat *Triticum aestivum* L. have sought to characterise the effect of allelic variation in genetic lines or commercial cultivars (Sozinov and Poperelya 1980; Payne et al. 1984, 1987a). The result has been the development of valuable methods for obtaining improvements in quality, by the identification of alleles associated with high quality, and their consequent assembly into favourable combinations during a breeding programme (Payne et al. 1984). Less attention has been directed towards the influence on quality of varying the dosage of genes known to affect quality.

The principal series of homoeoloci encoding storage proteins that govern bread-making quality in hexaploid wheat are located on chromosomes comprising homoeologous groups 1 and 6 (Payne 1987, for review): *Glu-1*, located on the long arms of chromosomes of group 1, encoding high-molecular-weight (HMW) subunits of glutenin; *Gli-1*, located on the short arms of chromosomes of group 1, encoding ω-gliadins, most of the γ-gliadins and some of the β-gliadins; *Gli-2*, located on the short arms of chromosomes of group 6, encoding α-gliadins, most of the β-gliadins and some of the γ-gliadins; and *Glu-3*, tightly linked to *Gli-1* on group 1, encoding low-molecular-weight (LMW) subunits of glutenin. Several additional loci encoding endosperm proteins are located on the short arms of group 1, allelic variation at which has not yet been shown to influence quality. These loci include *Gli-3*, encoding ω-type gliadins and D subunits of LMW glutenin (Payne et al. 1988); loci encoding further minor classes of gliadins (Akhmedov and Metakovsky 1987); and *Tri-1*, encoding triticins that are classified as globulins (Singh et al. 1988) rather than as prolamins.

Studies of dosage have been primarily concerned with the effect of reduction in the dosage or, indeed, of complete removal, of particular loci, chromosome segments or whole chromosomes. From such studies it has emerged that the following phenomena detrimentally affect quality: (i) replacement of certain active alleles by null alleles, particularly at *Glu-D1* (Payne et al. 1987b; Lawrence et al. 1988); (ii) replacement of part or all of the short arm of *1D* and, to a lesser extent, of *1B*, carrying *Gli-1*, by part of the short arms of homoeologous chromosomes from rye (Koebner et al. 1984) and *Aegilops umbellulata* (Harris 1983; Rogers et al. 1987); (iii) complete removal of the short arm of *1D* in a ditelosomic line (Maystrenko et al. 1973); and (iv) reduction of the dosage of chromosome *1D* to the monosomic level (Welsh and Hehn 1964). Also, the replacement of *Gli-B3* by a segment of chromosome *1U* from *Ae. umbellulata* has been implicated in lowering quality (Harris 1983; Rogers et al. 1987).

The current paper assesses the effect of varying the dosage of whole chromosomes and chromosome arms, both in the decreasing direction, i.e. below the disomic level, as in the previous studies cited above, and in the increasing direction, above the disomic level, in an attempt to identify beneficial effects on quality that might be open to exploitation via cytogenetic manipulation or molecular biology.

Materials and methods

The study was principally based upon a trial of aneuploid lines of spring wheat cultivar 'Chinese Spring' (CS) grown during 1988 (trial T88). The CS aneuploids were originally produced by Dr. E. R. Sears, University of Missouri, and seed was kindly provided from stocks maintained by T. E. Miller and S. M. Reader at the Cambridge Laboratory.

T88 consisted of 32 aneuploid lines involving chromosomes from homoeologous groups 1 and 6, as shown in Table 1, plus CS euploid controls. The abbreviations for the aneuploids given in Table 1 (NT, T, etc) will be used throughout the text. Six samples of CS were grown, where three were arbitrarily assigned to each group of aneuploids (1 and 6), enabling a number of *a priori* comparisons to be carried out during analysis (see 'Results'). A randomised complete block design was used, where each line was represented by a single plot of six plants in each of five independently randomised replicate blocks. Individual seeds were initially germinated on moist filter paper. Roots were taken from seedlings of monosomic families for cytological checking of Feulgen-stained mitotic preparations, and individuals with other than 41 chromosomes were discarded. Seedlings were planted into 4-cm pots and subsequently transplanted into soil in five unheated irrigated glasshouse compartments. Spacing was 15 cm between plants within a row (plot) and 20 cm between rows. Guard rows were sown at the ends of replicate blocks to prevent edge effects. During growth, only one fertiliser application was made, shortly after ear emergence, to guard against artifically boosting the protein content, which might obscure differences in quality due to differences in protein composition. Individual ears were bagged prior to anthesis to guarantee self-pollination. Harvesting was carried out at maturity, and grain yield (GY) data was obtained for individual plants.

Table 1. Genotypes grown in trial T88[a]

	Group 1		
Nullisomic-tetrasomic (NT)	N1A-T1B N1A-T1D	N1B-T1A N1B-T1D	N1D-T1A N1D-T1B
Tetrasomic (T)	T1A	T1B	T1D
Monosomic (M)	M1A	M1B	M1D
Ditelosomic (DT)	DT1AS DT1AL	DT1BS DT1BL	– DT1DL
'Double ditelosomic' (DDT)	–	–	DDT1D[b]
	Group 6		
Nullisomic-tetrasomic (NT)	N6A-T6B N6A-T6D	N6B-T6A –	N6D-T6A N6D-T6B
Tetrasomic (T)	T6A	T6B	T6D
Monosomic (M)	M6A	M6B	M6D
Ditelosomic (DT)	– –	DT6BS –	DT6DS DT6DL

[a] Plus six independent euploid controls

[b] This line is not a true double ditelosomic; one telosomic pair represents chromosome arm *1DL*. The other is not a true telosome; instead, it appears to be deleted long arm separated by the centromere from, presumably, a small segment of the short arm

Quality tests

Shortly after harvesting, seed was milled using a Tecator Cyclotec sample mill, and the flour was conditioned to 14%–15% moisture content. Grain protein contents (PC) based on 14% moisture content were determined using near infrared reflectance spectroscopy.

Due to the very low GY from some of the aneuploids, it was necessary to apply a reliable quality test that could be performed on small amounts of material. For this purpose, a procedure was adapted from a test used to screen early generation durum wheat breeding lines, for gluten strength, using 1-g flour samples (Dick and Quick 1983), and termed the SDS-microsedimentation test (MST). Preliminary tests, in which the volume of a stock solution of SDS/lactic acid and weight of flour samples were varied, suggested that 0.7 g samples, 4 ml of H_2O and 12 ml of stock solution would give the greatest differentiation between MST values for this material. The results were expressed as height in cm of sediment formed in a glass test tube (150 mm long × 16 mm OD × 14 mm ID) after 30 min. Two replicate MST determinations were made on each flour sample.

Results

Correlation between characters

No correlation was found between PC and MST, suggesting that overall differences between genotypes for MST were due to qualitative differences in protein composition rather than to quantitative differences in protein

amount. To ensure against misinterpretation, MST was analysed both with and without PC as a covariate, and the conclusions differed appreciably in only one case (considered below). In general, only analyses unadjusted for PC are reported.

Individual chromosome effects

The mean performance of each genotype is given in Table 2. For analysis, the NTs and Ts of each homoeologous group, plus three of the CS euploids, provide a balanced and orthogonal set of genotypes for identifying the effects of individual chromosomes and combinations of chromosomes, in an extension of the design of Pink and Law (1985). In theory, with the families arranged as shown in Table 2, each chromosome appears in four 'backgrounds', i.e. the three pairwise combinations of the remaining two chromosomes in the relevant homoeologous group, plus the combination of all three chromosomes. For each chromosome, the mean over the four backgrounds can be compared to determine which chromosome is best for quality; similarly, the means of the backgrounds can be compared. Interactions between chromosomes can also be tested. In practice, background *6A-6D* has had to be excluded due to the enforced absence of N6B-T6D from the trial (Table 1).

An analysis of variance of the data is given in Table 3, including orthogonal comparisons between chromosomes. Previous evidence suggested that valid comparisons would be *1D* against the mean of *1A* and *1B*, and, given that *1A* carries a null allele at *Glu-A1*, *1B-1D* against the mean of *1A-1B* and *1A-1D*. Also the euploid background can be compared with the mean of the three pairwise comparisons *1A-1B*, *1A-1D* and *1B-1D*. No a priori comparisons have been calculated for group 6, but differences can be compared using the appropriate standard error of the difference between means. The analyses clearly demonstrate the superiority of *1D* over the mean of *1A* and *1B*, and of *1B* over *1A*. The best background is *1B-1D*, and *1A-1D* is superior to *1A-1B*. For group 6, *6A* is clearly superior to *6B* and *6D*, whereas *6B* and *6D* do not differ from one another. For the backgrounds, *6A-6B-6D* is the best, and *6A-6B* is marginally better than *6B-6D*.

For both groups of chromosomes there are significant interactions between chromosomes and backgrounds (Table 3, chromosomes × backgrounds items). The best genotype in the trial, and, therefore, the one providing the most encouragement for exploiting the observed results, is N1A-T1D.

Relationship with dosage

The above analyses demonstrate that the rank order of chromosomes is *1D*>*1B*>*1A*, *6A*>*6B*=*6D*, but do not directly address the effect of varying the dosage of indi-

Table 2. Mean MST values for NTs, Ts and CSs in T88

Genetic background	Group 1			
	Chromosome			Background means
	1A	*1B*	*1D*	
1B-1D	CS 5.79	N1A-T1B 8.81	N1A-T1D 9.45	8.07
1A-1D	N1B-T1A 1.93	CS 6.26	N1B-T1D 6.58	4.92
1A-1B	N1D-T1A 2.33	N1D-T1B 3.49	CS 6.13	3.98
1A-1B-1D	T1A 5.43	T1B 6.93	T1D 8.27	6.88
Chromosome means	3.87	6.37	7.61	
Genetic background	Group 6[a]			
	Chromosome			Background means
	6A	*6B*	*6D*	
6B-6D	CS 6.06	N6A-T6B 5.13	N6A-T6D 4.15	5.11
6A-6B	N6D-T6A 6.08	N6D-T6B 4.90	CS 6.06	5.68
6A-6B-6D	T6A 7.08	T6B 6.06	T6D 6.15	6.43
Chromosome means	6.41	5.36	5.45	

[a] Background *6A-6D* omitted due to absence of N6B-T6D

Table 3. Analysis of variance testing differences between chromosomes and between backgrounds in T88[a]

Source of variation	*df*	MS[b]
Replicate blocks	4	0.12[NS]
Genotypes	37	13.09***
Group 1 chromosomes	2	72.52***
1D vs (*1A*+*1B*)	1	82.42***
1A vs *1B*	1	62.62***
Group 1 backgrounds	3	50.26***
1A-1B-1D vs (*1B-1D*+*1A-1D*+*1A-1B*)	1	17.17***
1B-1D vs (*1A-1D*+*1A-1B*)	1	126.97***
1A-1D vs *1A-1B*	1	6.63***
Group 1 chromosomes × backgrounds	6	3.12***
Group 6 chromosomes	2	5.01***
Group 6 backgrounds	2	6.54***
Group 6 chromosomes × backgrounds	4	1.71***
Deviations	18	7.77***
Replicate blocks × genotypes interaction	146 (2)	0.27

[a] Standard error of difference between genotype means = 0.3258; between group 1 chromosomes = 0.1629; between group 6 chromosomes and between backgrounds for both groups = 0.1881

[b] NS = $P>0.05$; *** = $P<0.001$

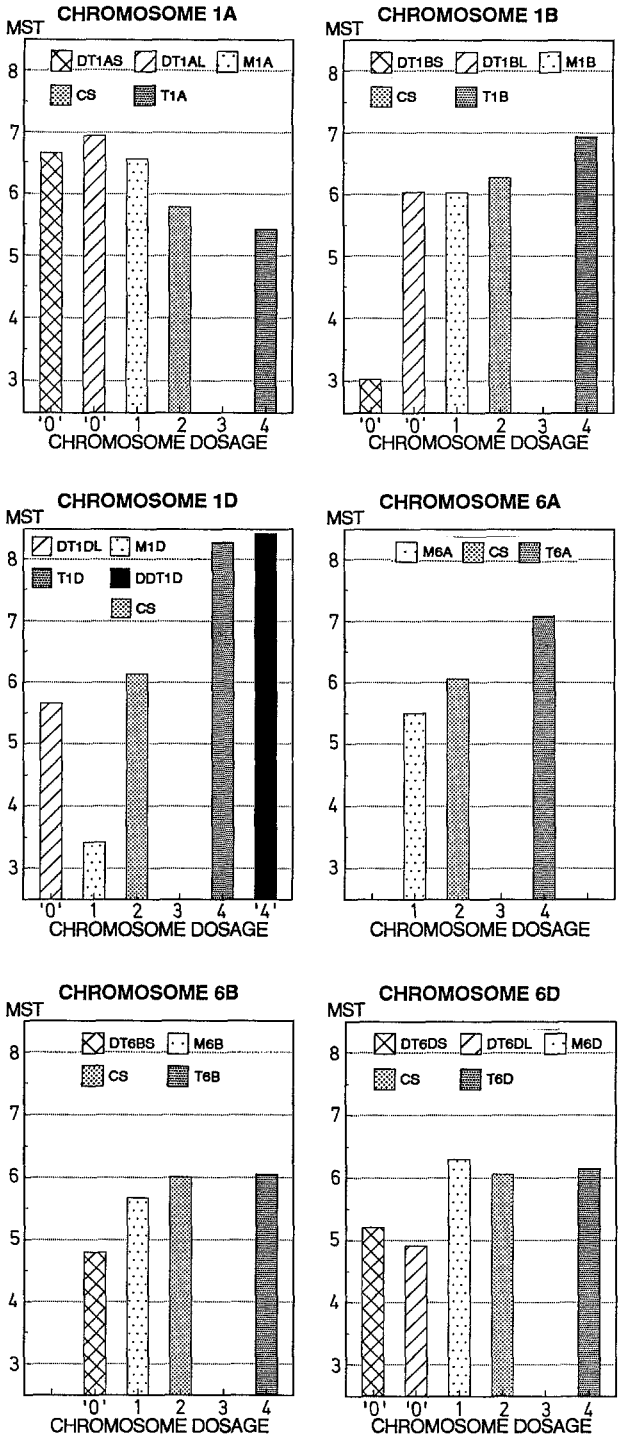

Fig. 1. Dosage effects of individual chromosomes and chromosome arms upon MST in T88. '0' and '4' on the x-axes represent putative dosage values only (see text)

vidual chromosomes. A consideration of monosomics, euploids, tetrasomics and ditelosomics, shown in Fig. 1, enables direct comparisons of dosage differences. In all cases except for *6B* and *6D*, the relationship of MST with dosage of whole chromosomes is principally linear. The steepest gradient is that for *1D*, demonstrating that increases in the dosage of *1D* provide the greatest improvement in quality; *1B* also shows an improvement in quality upon increases in dosage. The results for *1A* contrast sharply with those for *1B* and *1D* in showing a surprising reduction in quality upon increase in dosage. For group 6, only *6A* shows an appreciable improvement in quality upon increase in dosage, although *6B* shows a slight improvement upon increase from the monosomic level. However, *6D* shows a small reduction upon increase from this level.

Each ditelosomic genotype can be regarded, putatively, as representing a nullisomic dosage, but in few cases does it appear that only one arm of a particular chromosome is involved in the control of quality (Fig. 1). For *1A*, although DT1AL fits the linear relationship better than does DT1AS (and, indeed, indicates that loss of *1AS* gives the result expected in a line nullisomic for *1A*), the line DT1AS also shows an increase in quality over the euploid. This demonstrates that loss of *1AL* is also having a significant effect, and that the effects of individual arms are not additive. For *1B*, loss of *1BS* in DT1BL shows only a small reduction in quality compared with the euploid, but loss of *1BL* in DT1BS shows a drastic reduction in quality, suggesting that virtually all the effects of dosage of *1B* are due to variation in the dosage of *1BL*. A similar case might apply to *1D*, where DT1DL also shows only a small reduction compared with the euploid. Unfortunately, seed of DT1DS was not available at the time of the trial. For *6A*, neither DT could be scored for quality (Table 1). For *6B*, only DT6BS was available and showed a reduction in quality associated with the loss of *6BL*, surprising given the absence of loci encoding storage proteins on this arm. For *6D*, DT6DS also suggests an effect of the long arm, although here, DT6DL was available and indicated, if anything, a larger effect of the short arm.

As noted earlier (footnote b, Table 1), line DDT1D is not a true double ditelosomic, but contains a pair of long arms plus a pair of shortened long arms, and is, therefore, essentially tetrasomic for most of the long arm. It shows an increase in staining intensity on SDS-PAGE gels of the proteins encoded by *Glu-D1*. *Gli-D1* is thus absent in this line. As shown in Fig. 1, this genotype has a very high MST volume, as high as T1D. Increases in the dosage of part of the long arm may thus be solely responsible for these improvements. However, DDT1D showed quite a high PC, and correction for this lowers its MST value to slightly below that of T1D (results not shown).

Interactions between chromosomes

The experiment was designed to allow an alternative partition of the variation that enables interactions between chromosomes to be quantified. Each independently in-

Table 4. Weighted least-squares model fitting to genotype means for MST in T88[a]

Chromosomes	Parameters	Perfect fit model	2 parameter model[b]	χ_1^2 for 2 parameter model
1A, 1B	m	5.37***	5.51***	2.216[NS]
	$[d_1]$	3.44***	3.44***	
	$[c_1]$	0.42[NS]		
1A, 1D	m	5.89***	6.01***	1.719[NS]
	$[d_2]$	3.56***	3.56***	
	$[c_2]$	0.37[NS]		
1B, 1D	m	5.04***		
	$[d_3]$	1.55***		
	$[c_3]$	1.10***		
6A, 6B	m	4.70***		
	$[d_4]$	0.43**		
	$[c_4]$	1.36***		
6A, 6D	m	5.12***		
	$[d_5]$	0.97***		
	$[c_5]$	0.91**		

[a] NS = $P > 0.05$; ** = $P < 0.01$; *** = $P < 0.001$
[b] Two parameter models (m, $[d]$) fitted where $[c]$ is NS

cluded CS euploid family can be regarded as the F_1 between a pair of NTs, as follows: N1A-T1B × N1B-T1A, N1A-T1D × N1D-T1A, N1B-T1D × N1D-T1B, N6A-T6B × N6B-T6A and N6A-T6D × N6D-T6A. The sixth combination, N6B-T6D × N6D-T6B, was not available for analysis due to the absence of N6B-T6D from the trial. The difference between the means of the parental NTs in each case is an additive genetic difference, and the deviation from the parental mean of the mean of the CS euploid is a genetic interaction between the two chromosomes for which the parents differ. The differences can be explored by fitting biometrical models to genotype means as described by Mather and Jinks (1982). In the crosses considered here, three parameters are sufficient to describe the variation in each cross: m, the common genetic effects, equal to the mid-parental value; $[d]$, the additive genetic difference; and $[c]$, representing the interaction between chromosomes. m and $[d]$ are as defined by Mather and Jinks (1982), whereas $[c]$ is a new parameter for describing chromosome interactions, although analogous in this case to potence (net dominance), $[h]$, in a conventional cross between inbred lines. With only three statistics available from each cross, the full parameter model is a perfect fit one.

The results of the model fitting are shown in Table 4. In each of the five crosses there is a significant additive genetic component ($[d]$), but this varies considerably over crosses, being larger for group 1 than for group 6. For two of the group 1 crosses, there is no significant interaction between chromosomes [*1A* with *1B* ($[c_1]$), and *1A* with *1D* ($[c_2]$)], such that two parameter models apply, but there is an interaction for the third ($[c_3]$), where *1B* interacts with *1D* in the CS euploid to give a higher volume than expected on a purely additive model. The latter case also applies to the two group 6 crosses [*6A* with *6B* ($[c_4]$), and *6A* with *6D* ($[c_5]$)]. There is a general tendency over the five crosses for the magnitude of $[c]$ to be large where that of $[d]$ is small, which may be due to chromosomes from different genomes interacting with one another in an analogous situation to that of dispersed genes accompanied by heterosis observed in normal crosses. Selection for such dispersed genes might have been important during the evolution of wheat.

Genotype × years interaction

The group 1 NTs plus CS euploids have been previously grown in a pilot trial in 1987 (T87), and the results were reported in Rogers et al. (1988). Comparison of these with the results from T88 showed that the magnitude of estimates quantifying additive environmental differences over years and genotype × years interaction was small, even where they were statistically significant (results not shown). In T87, more grain was available than in T88, and a larger scale SDS-sedimentation test based upon 4.5 g of flour was carried out. For the group 1 NTs and CS euploids in T88, there was also enough grain for this test, in contrast to many of the other lines. This larger scale test and the smaller scale MST showed close general agreement, although they showed differences in detail. In particular, in T88, $[c_1]$ was significant for SDS, but not for MST, and $[c_2]$ was not only significant, but reversed in sign. Therefore, for the relevant crosses (N1A-T1B × N1B-T1A and N1A-T1D × N1D-T1A), caution should be exercised in interpreting gene action until the reasons for the differences between the two tests are known.

Discussion

The above results suggest that, for group 1, increases in the dosage of *1B* and, especially, *1D* produce improvements in quality, whereas increases in the dosage of *1A* reduce quality. Where *Glu-1* produces an active gene product, effects of the long arm are more important than effects of the short. The detrimental effect observed upon increasing the dosage of *1A* may be due to the presence on *1AS* of relatively inferior gliadins or LMW subunits of glutenin compared with those produced by the other group 1 short arms, although, of course, other loci may be responsible. The influence of group 6 is less marked than that of group 1, but increases in the dosage of *6A* clearly improve quality. Sozinov and Poperelya (1980) have previously demonstrated effects on quality of allelic variation at *Gli-2* located on the short arms of group 6, and

changes in the dosage of these loci may be responsible for the observed changes in sedimentation volume in the current study. However, some of the effects of group 6 are less readily explained than those of group 1, such as the absence of a clear relationship of dosage with quality for *6B* and *6D*, and the apparent effect on quality of the long arms of *6B* and *6D*, which are not known to carry loci encoding endosperm storage proteins. Interactions between chromosomes are important, but interactions of genotypes with years are of minor importance, as are additive environmental differences over years.

Varietal improvement

To exploit the described effects for varietal improvement requires the production of meiotically stable, acceptably yielding lines that carry favourable combinations of duplication(s) and/or deletion(s) of gene(s) affecting quality. The aneuploids themselves often suffer from agronomic defects, such as yield deficit and cytological and morphological instability, and are probably best regarded not as an end in themselves, but as the means of identifying promising target combinations and as the source of material with which to begin a crossing programme. In this context, line N1A-T1D is of prime interest, due to its outstanding performance in the small-scale quality tests. Its advantage might be utilised by replacing a limited number of loci on *1A* by homoeologous loci from *1D*, rather than by manipulating whole chromosomes. The problem is to determine which of the many loci carried by each chromosome should be manipulated. The results described in this paper suggest that the duplication of genes carried by both arms of *1D* might be beneficial for quality, although it would appear that duplication of genes on the long arm should provide greater gain than duplication of those on the short. Conversely, deletion of genes on both arms of *1A* would be expected to provide some benefit. The genes most likely to be responsible for the various effects on quality are those encoding glutenins and gliadins. Thus, replacement of *Glu-A1* by *Glu-D1*, whilst retaining the copy on *1D*, might lead to improvement, as might replacement of *Gli-A1* by *Gli-D1*.

There is some evidence that *Gli-3* is important for quality, since replacement of a segment of *1BS*, carrying the locus, by an alien segment results in a reduction in quality (Harris 1983; Rogers et al. 1987); but there is currently no direct information on the effect of *Gli-A3* on quality. It may be fortunate in this regard that there is no *Gli-3* locus appreciably separated from *Gli-1* on *1D*. Instead, the class of proteins encoded by *Gli-A3* and *Gli-B3* appear to be encoded on *1D* by genes at or close to *Gli-D1*, in addition to the other gliadins and LMW glutenin subunits produced in that region (Payne et al. 1988). This may simplify the transfer of chromatin from *1D* to *1A* by restricting consideration to two loci, and at the same time means it might be possible to retain *Gli-A3*, although this may be difficult if, as is likely, *1AS* and *1DS* differ from one another by a chromosome rearrangement (Payne 1987). Other loci, for example *Tri-1*, might also have to be manipulated. It is not currently clear whether allelic variation at *Tri-1* affects quality, and Payne et al. (1987c) found that the allelic difference at *Tri-A1* between varieties 'Chinese Spring' and 'Hope' did not affect quality.

The particular alleles carried by the parental material might be important. For example, the *Gli-A1* allele of CS (designated *Gld 1A5* by Sozinov and Poperelya 1980) is associated with only average quality (Sozinov and Poperelya 1980). If an allele giving better quality were originally present in the parental lines and were replaced by an allele of *Gli-D1*, a loss in quality might follow, albeit dependent upon the particular *Gli-D1* allele transferred. Further, the *Glu-D1* allele carried by CS, *Glu-D1a*, encoding subunits 2 and 12 (Payne and Lawrence 1983), is associated with mediocre quality, and a larger increase in quality might be expected if a superior allele, such as *Glu-D1d*, encoding subunits 5 and 10 (Payne et al. 1984), were duplicated on *1A*. Perhaps most importantly, the *Glu-A1* allele of CS (*Glu-A1c*) is null, i.e. does not produce a HMW subunit of glutenin, and gives relatively poor quality. If the parental material carried an active *Glu-A1* allele, such as *Glu-A1a*, encoding subunit 1, or *Glu-A1b*, encoding subunit 2*, both known to give superior quality than *Glu-A1c*, then replacement of the *Glu-A1* locus by *Glu-D1* might not produce the desired improvement. In such a case it might be worth considering producing a line that carried active alleles at all three *Glu-1* loci, as well as extra doses of one of them, probably *Glu-D1*, in addition to, rather than at the expense of, its other *Glu-1* loci.

Interactions

Previous studies of the effects of interactions on quality have been concerned with individual loci rather than with whole chromosomes. Lorenzo et al. (1987) found interactions between *Glu-1* loci on SDS-sedimentation volume, concluding that the contribution of individual subunits to quality depends on the glutenin subunits contributed by the other two genomes. In particular, they suggested that the observed superiority in quality of subunit 1, encoded by *Glu-A1a*, compared with that of subunit 2*, encoded by *Glu-A1b*, was related to the presence of subunits 7 and 8, encoded by *Glu-B1b*. Odenbach and Mahgoub (1988) also found interactions between HMW glutenin subunits, suggesting in this case that subunits 7 and 8 (*Glu-B1b*) could only be effective in conferring good quality when subunits 5 and 10 (*Glu-D1d*) were present; and further, that the effects of HMW glutenin

subunits on quality were modified by the remaining genetic background. These studies contrast with those of Payne et al. (1984, 1987c), who found that the influence of both the *Glu-1* and *Gli-1* loci on quality was principally additive. The current study, being mainly concerned with whole chromosomes or chromosome arms, is not directly comparable to such studies. Nevertheless, it suggests that interactions between loci might influence quality, but does not unambiguously characterise them.

Exploitation of the above effects might be achieved by cytogenetic manipulation. However, in future, particular genes might be more readily duplicated or deleted within the genome using the techniques of molecular biology.

Acknowledgements. The authors are most grateful to Dr. J. W. Snape for the model fitting programme, and to the Agricultural Genetics Company, Cambridge, for financial support.

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