

Genetics of yellow berry in wheat (Triticum aestivum)

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Summary. The inheritance of yellow berry, a grain disorder in durum and bread wheats, was studied in six intervarietal crosses in bread wheat. The trait was found to be controlled by either two or three dominant genes. Monosomic analysis using 'Chinese Spring' monosomic series showed the presence of two major dominant genes on chromosomes 1A and 7A, and four modifiers on 4A, 4B, 6A and 6D, which influence the expression of yellow berry in bread wheat.

Key words: Triticum aestivum – Monosomic analysis – Inheritance – Gene location

Introduction

Yellow berry (Yb) is an undesirable grain disorder in wheat in which the normal vitreous and hard textured kernels assume a yellowish and soft appearance. This condition is also reported to be negatively correlated with seed protein content in durum, bread wheat and triticale (Alessandroni et al. 1976; Dhaliwal et al. 1981; Sharma et al. 1983). Waines et al. (1978) compared the isoelectric focussing patterns of albumins and gliadins of kernels with and without yellow berry in eight varieties of wheat and found that the protein differences between yellow berry and vitreous kernels were purely quantitative and not qualitative. The reduced protein content in yellow berry kernels adversely affects the nutritional and cooking quality of such wheat and durum products as macaroni, spagetti and semolina. Nothing has ever been reported on the genetics of yellow berry in wheat, therefore, in the present study an attempt was made to identify and localize the genes controlling yellow berry in wheat.

Materials and methods

Three varieties of bread wheat ('WL 711', 'HD 2281' and 'WG 357') with average protein content and vitreous kernels, and three strains with high protein content and vitreous kernels, obtained from the Centro Internacional de mejoramiento de maiz y Trigo (CIMMYT) Mexico ('PH 132', 'PH 133' and 'PH 138'), were crossed with 'HD 2009', a high yielding yellow berry susceptible wheat variety obtained from the Indian Agricultural Research Institute, New Delhi. The parentage of the varieties is given in Table 1. A variable number of individual F2 plants from each of the six crosses were harvested at random for raising F₃ families. About 50 plants from each of the F3 lines derived from single F2 plants were evaluated for the yellow berry disorder. Vitreous kernels were given the score '0' whereas kernels displaying varying degrees of the yellow berry disorder were graded with scores 1 to 4. The score I was assigned to kernels having small yellow specks and 4 to completely yellow berry kernels; 2 and 3 indicate intermediate situations.

In order to locate the yellow berry genes, 'WL 711' was crossed as male parent with each of the 21 monosomic lines of 'Chinese Spring'. 'Chinese Spring' is highly prone to yellow berry. The F₁ monosomic plants were identified by counting the chromosomes at metaphase I or anaphase I. Except from the cross involving monosomic 2D all grains harvested from confirmed monosomic F₁ plants were evaluated for the yellow berry disorder. The F₂ seeds from each of the 20 F₁ monosomic hybrids were evaluated for yellow berry. Sixty kg N/ha was applied to all plants as there is a maximum expression of the yellow berry disorder in the susceptible genotypes at this level of fertilizer application.

Table 1. Wheat varieties and their parentage

'HD 2281' HD 2160/249 'PH 132' Fnd-Cno 67 ² × Ron No. 2-Fnd 'PH 133' Fnd-Cno 67 ² × Ron No. 2-Fnd	., ,,	Fnd-Cno $67^2 \times$ Ron No. 2-Fnd Fnd-Cno $67^2 \times$ Ron No. 2-Fnd	
'PH 138' Cal-NH×Za 75/Cal-NH	,	Cal-NH×Za 75/Cal-NH	

Table 2. Incidence of yellow berry in F₃ families from six crosses each involving a wheat variety with vitreous kernels crossed with 'HD 2009', a yellow berry susceptible variety

Cross	F ₃ families	Segre-	χ² ,			
	Yellow berry plants only	Segre- gating for YB	Vitreous plants only	Total	gation	value
'HD 2009'×'WL 711'	20	112	8	140	15:1	0.06
'HD 2009'×'HD 2281'	26	72	2	100	63:1	0.12
'HD 2009'×'WG 357'	6	84	1	91	63:1	0.12
'HD 2009'×'PH 132'	28	73	1	102	63:1	0.22
'HD 2009'×'PH 133'	5	91	1	97	63:1	0.18
'HD 2009'×'PH 138'	1	90	9	100	15:1	1.29

Table 3. Monosomic analysis for yellow berry in crosses of 'Chinese Spring' monosomics with vitreous wheat – 'WL 711'

Monosomic for chromosome	F ₁ monosomic		F ₂		χ² value	
	Vitreous/ yellow berry	Yellow berry grade	YB:Vitreous	Total	(15:1)	
Mono-1A	Vitreous	(0)	94:12	106	4.65*	
-1B	YB	(4)	68:8	76	2.37	
-1D	YB	(4)	64:5	69	0.12	
-2A	YB	(4)	89:3	92	1.39	
-2B	YB	(4)	56:4	60	0.02	
-2D	_	_	_	_	_	
-3A	YB	(4)	57:1	58	2.03	
-3B	YB	(4)	43:2	45	0.24	
-3D	YB	(4)	58:7	65	2.29	
-4A	YB	(1)	56:8	64	4.27*	
-4B	YB	(1)	46:7	53	4.39*	
-4D	YB	(4)	75:3	78	0.76	
-5 A	YB	(4)	63:6	69	0.70	
-5B	YB	(4)	67:2	69	1.33	
-5D	YB	(4)	115:4	119	1.69	
-6A	YB	(1)	74:10	84	4.58*	
-6B	YB	(4)	49:6	55	2.03	
-6D	YB	(1)	31:10	41	23.06**	
-7 A	Vitreous	(0)	70:14	84	15.55 **	
-7B	YB	(4)	52:5	57	0.61	
-7D	YB	(4)	48:4	52	0.18	
Total (excluding 1A and 7A)			1,111:95	1,206	5.423*	
Total (excluding 14 4B, 6A and 6D	A, 7A, 4A)		904:60	964	0.001	

^{*} P≤0.05; ** P≤0.01

Results and discussion

Screening of F_1 kernels for yellow berry or vitreousness in all six crosses revealed that the yellow berry disorder is controlled by dominant gene(s). Table 2 shows the incidence of yellow berry in F_3 families of the six crosses. In all these crosses when yellow berry susceptible true breeding and segregating families were pooled and the data tested for fitness to genetic

segregations it was observed that yellow berry was under the control of two or three dominant genes. Evaluation of kernels in the monosomic F_1 plants from 'Chinese Spring' monosomics \times 'WL 711' crosses is given in Table 3. The seeds of the F_1 family for 2D monosomic did not germinate and therefore observation for this family in the F_1 and F_2 could not be recorded. Grains harvested from monosomic F_1 hybrids in all crosses except 1A and 7A had the yellow berry

disorder. Vitreous kernels in F1 monosomics for 1A and 7A chromosomes and grade 1 for yellow berry (traces) in 4A, 4B, 6A and 6D monosomic F₁s shows that this trait is controlled by two major dominant genes (duplicate genes) along with a few inhibitors of yellow berry or modifiers for vitreousness. Vitreous kernels in 1A and 7A monosomic F₁ plants were due to the absence of one of the two dominant duplicate genes for yellow berry and the presence of modifier/inhibitory genes. The differential inheritance of yellow berry susceptible variety 'HD 2009' when crossed with six resistant cultivars also stresses the presence of some modifiers which influenced the expression of yellow berry. For confirmation of this theory, the F₂ populations from the identified monosomic F₁ plants of all twenty crosses were grown. Assuming a negligible proportion of nullisomics in the selfed progeny of monosomic F₁ plants and the absence of a dosage effect in the genes controlling yellow berry, F2 families were tested for duplicate gene (15:1) segregation. F2 families of monosomic 1A and 7A deviated from the predicted disomic segregation ratio indicating these as the critical monosomics. Thus, it is evident that the two major dominant genes controlling yellow berry disorder are present on chromosome 1A and 7A. F₂ segregation for the other four of the remaining 18 crosses also deviated significantly from the predicted 15 susceptible: 1 vitreous ratio, but only the results of 6D were significant at P=0.01. Deviations from the 15:1 segregation in the F₂ generation involving 4A, 4B, 6A and 6D monosomics which had traces of yellow berry in F1 monosomic plants indicated that all four chromosomes have modifiers for vitreousness. The pooled ratio of yellow berry and vitreous kernels from all crosses, excluding 1A and 7A, deviated $(X^2 = 5.423)$ from the expected 15:1 ratio but on the other hand pooled analysis, excluding 1A, 7A, 4A, 4B, 6A and 6D, fits very well indicating that the classification of plant progenies for yellow berry and vitreous kernels was satisfactory.

Yellow berry is highly negatively correlated with grain protein content and for a 10% increase in yellow berry there is about 1% decrease in protein content.

The genes for gliadins, the major storage proteins in wheat, are also reported to be located on all chromosomes of homoeologous group I and 6 (Wrigley and Shepherd 1973; Kasarda et al. 1976; Sozinov et al. 1979; Brown and Flavell 1981). The fact that a major gene and a number of modifiers for yellow berry or vitreousness are also located on chromosomes IA, and 6A and 6D, respectively, indicates that some of the genes controlling yellow berry could probably be the genes coding for or controlling the synthesis of gliadin polypeptides. The durum wheats, in which yellow berry adversely affects its products, can be improved by introgression of major dominant genes for yellow berry present amongst the bread wheats.

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