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Short Communication

Trisomic analysis for qualitative characters in tetraploid wheat

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Cytogenetic analysis in wheat through the use of aneuploids has led to chromosomal location of several genes controlling morphological and biochemical characters. Aneuploids developed in wheat variety Chinese Spring have facilitated monosomic analysis in hexaploid wheat. Such studies in durum wheat have not progressed because loss of one chromosome (2n-1) resulted in reduced fertility and vigour [1]. On the contrary, Blanco *et al.* [2] found that durum trisomics (2n+1) with high transmission rate and fertility can be utilized effectively in the same way as monosomics and conversion technique gives the possibility of obtaining desired identified trisomic lines. In present study 14 trisomic lines of *Triticum durum* var. MACS-9 developed by Agharkar Research Institute were used to assign genes governing three qualitative characters *viz*., glume pubescence, grain colour and spike type on their respective chromosomes. These lines were developed by conversion technique using tetrasomic of *T. aestivum* var. Chinese Spring. These trisomic lines are morphologically indistinguishable and can be identified cytologically.

The material in this study comprised the trisomics ($13^{II}+1^{III}$) of durum wheat variety MACS-9 and a contrasting genotype H-5092. MACS-9 is a tall glabrous glume, yellow grain variety developed by Agharkar Research Institute for rainfed conditions of Peninsular Zones. H-5092 is a semidwarf, pubescent glume, red grain genotype with sphaerococcoid type of spike. Using trisomics as female parent, each trisomic line was crossed with H-5092. Cytologically identified trisomic F_1 plants were selfed to obtain F_2 seeds. At the same time, disomic MACS-9 and H-5092 were also crossed and advanced to F_2 generation. F_2 populations were raised on ARI farm at Hol.

F_2 data on glume pubescence, grain colour and spike type were collected. Chi-square test was used to compare the segregating pattern of trisomic derived F_2 with that of disomic F_2 populations for identifying the critical chromosome. It was presumed that F_2 involving critical cross combination will deviate significantly from disomic F_2 population due to presence of extra chromosome. Observations on chromosomal location of genes responsible for glume pubescence, grain colour and spike type are presented in Table 1.

Glume pubescenece

Glume pubescenece is well known, wide spread and easily identifiable dominant character. F_2 population of MACS-9 (glabrous glumes) and H-5092 (pubescent glumes) segregated in 3 pubescent : 1 glabrous ratio indicating monogenic dominance of pubescence over glabrousness. In all trisomic crosses except 1A, the segregation was in accordance with 3:1 ratio. Earlier studies had also revealed similar mode of inheritance and mapping of the gene *Hg* for glume pubescence on chromosome 1A [3, 4].

Grain colour

F_2 segregation of H-5092 X MACS-9 and all trisomic lines except 3 A resulted in 3 red : 1 amber coloured grain ratio, indicating monogenic inheritance. In F_2 progenies of trisomic 3A x H-5092 chi-square values ($c^2 = 5.949$, $P<0.02$) deviated significantly suggesting the presence of genes controlling grain colour, on chromosome 3A. These results are in accordance with the earlier reports [5, 6].

Sphaerococcoid type spike

Sphaerococcoid spike types were earlier reported in durum wheats due to induced mutations [7,8]. The

Table 1. Inheritance of glume pubescence, grain colour and spike type in tetraploid wheat

Trisomic line	Glume pubescence				Grain colour					Spike type				
	Plants with hairy glumes	glabrous glumes	Total No.of plants	c^2 (3:1)	P-value	Plants with red grains	Yellow grains	Total No.of plants	c^2 (3:1)	P-value	Plants with Durum type spikes	Sphaero-coccum spikes	Total No.of plants	c^2 (15:1)	P-value
1A	32	20	52	5.010	0.02-0.05	37	8	45	1.250	0.20-0.30	97	5	102	0.316	0.50-0.70
2A	43	13	56	0.095	0.70-0.80	41	14	55	0.006	0.95-0.98	48	8	56	6.170	0.01-0.02
3A	26	9	35	0.009	0.90-0.95	40	25	65	5.949	0.01-0.02	63	5	68	0.141	0.70-0.80
4A	37	12	49	0.006	0.95-0.98	68	25	93	0.176	0.50-0.70	90	7	97	0.156	0.50-0.70
5A	32	10	42	0.031	0.70-0.80	69	14	83	2.925	0.10-0.05	81	2	83	2.091	0.10-0.05
6A	38	13	51	0.059	0.70-0.80	80	23	103	0.392	0.50-0.70	98	4	102	0.940	0.70-0.80
7A	40	15	55	0.152	0.70-0.80	77	26	103	0.003	0.95-0.98	96	8	104	0.369	0.50-0.70
1B	19	6	25	0.120	0.70-0.80	50	24	74	2.180	0.10-0.20	72	3	75	0.401	0.50-0.70
2B	38	19	57	2.110	0.10-0.20	67	18	85	0.655	0.30-0.50	82	4	86	0.496	0.50-0.70
3B	41	17	58	0.575	0.70-0.80	81	30	111	0.243	0.50-0.70	105	8	113	0.133	0.70-0.80
4B	37	17	54	1.209	0.20-0.30	78	30	108	0.777	0.30-0.50	98	9	107	0.853	0.30-0.50
5B	43	13	56	0.094	0.70-0.80	86	24	110	0.594	0.30-0.50	105	6	111	0.044	0.80-0.90
6B	41	14	55	0.005	0.95-0.98	80	27	107	0.003	0.95-0.98	104	5	109	0.514	0.30-0.50
7B	38	12	50	0.026	0.70-0.80	88	19	107	2.987	0.10-0.05	102	4	106	1.109	0.20-0.30
Disomic MACS 9 x H-5092	84	21	105	1.402	0.20-0.30	37	11	48	0.111	0.70-0.80	99	6	105	0.051	0.80-0.90

P value at 0.05 level = 3.84.

sphaerococcoid genotype H-5092 used in the present study is a derivative of *T. durum* x *T. turgidum* cross. F_2 population of the cross MACS-9 x H-5092 segregated in 15 durum:1 sphaerococcoid type spikes, indicating control by two recessive genes. Joppa [9], found that two recessive genes were necessary to produce this phenotype. With exception of trisomic 2A, crosses of all trisomic lines with H-5092 also fitted in 15 durum:1 sphaerococcoid ratio. Out of 56 plants from the cross-trisomic 2A X H-5092, 8 plants showed sphaerococcoid type spikes and this F_2 data deviated significantly from the expected 15:1 ratio (c^2–6.17, P<0.02) indicating presence of genes responsible for sphaerococcoid type of spike on different arms of chromosome 2A. In hexaploid wheat, presence of this character was reported on chromosome 3D [10, 11]. On the contrary H-5092 being tetraploid in nature, the genes governing this character are not expected to be present on D genome chromosomes. Schmidt and Johnson [12] also reported that presence of sphaerococcum effect is not restricted to D genome. Similarly, the different sources of the genes may also affect the location of the character. Hence, it can be concluded that the genes responsible for this character may be present on chromosome 2A.

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