GENETICS OF PLANT HEIGHT IN SPRING WHEAT

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Genetics of plant height was studied in crosses of three isogenic lines of wheat (Tritleum aestivum L.) having different levels of height with a tripple dwarf variety Olsen dwarf. Analysis of data revealed two to three genes responsible for plant height. Genetic components estimated from generation means (parental F₁ and F₂ indicated both additive and non-additive gene action for the inheritance of plant height.

INTRODUCTION

Short statured wheat varieties are resistant to lodging and thus ensure hetter development of yield components consequently resulting in enhanced grain yield. A better understanding of the genetics of plant height is, therefore, essential for the breeders to synthesize new cultivars incorporating the height reducing genes. Isogenic analysis is an efficient and reliable technique for the inheritance study of height. Although studies have already been conducted in the past (Markle and Atkins, 1964; Jerrel et al., 1967; Amaya et al., 1972; Fick and Qualset, 1973; Saakyan, 1981; Pochaba, 1985), these are usually based on the generations of the crosses between varieties and not on the generations of the crosses of isogenic lines having different height levels. For this reason the present study was conducted and the results are reported in the present paper. Genetic information thus derived would be a valuable tool for tailoring new dwarf varieties of wheat.

MATERIALS AND METHODS

The experimental population was derived from three crosses of isogenic lines (Isogenic line I, Isogenic line II and Isogenic line III) of wheat (Triticum asstivum L.). The F₁ and F₂ generations of these crosses alongwith their parents were space planted in the experimental area of the Department of Plant Breeding and Genetics, University of Agriculture, Faisalabad during the year 1985-86 using randomized complete block design of layout with three replications. Two

hundred and forty plants from each F_2 population and 24 plants from each of F_1 and the parents were selected at random. The data were recorded for :

1, Plant height (cm):

Length of the central tiller from the soil surface upto the tip of the spike excluding awns.

2. Number of nodes per culm;

The number of nodes of the central tiller of selected plants.

3. Internode length (cm):

The length of individual first three internodes (below peduncle) of the central tiller.

Analysis of variance was run as per method given by Steel and Torrie (1980). The data of plant height were analysed to get an estimate of the minimum number of genes involved in controlling this character using the formula given by Sewall Wright (1921).

Simple correlation coefficient between internode length and plant height was calculated by the following formula:

$$r = \frac{\leqslant XY - \frac{\leqslant X. \leqslant Y}{n}}{(\leqslant X^2 - (\leqslant X^2) + (\leqslant X^2 - (\leqslant X)^2)}$$

RESULTS AND DISCUSSION

The analysis of variance (Table 1) indicated significant differences among generations for plant height and internode length while for number of nodes the results were non-significant meaning thereby the differences of height in the lines were not due to the number of nodes.

The mean values of all the F₁ hybrids were greater than the respective mid parents (Table 2) showing positive heterosis and partial dominance of positive genes for plant height. Similar findings were reported by Markle and Atkins (1964). Pochaba (1985), however, observed incomplete dominance of short

Table 1 Mean performance of parents and analysis of variance for verious characters.

Character	Isogenic line I	lsagenic line II	Isogenic line III	Olsen	Replication mean squares	Generation mean aquares
Plant height (cm)	117.55	107.39	79.6	49.81	28.10**	11.23**
	2	7 00	¥ 04	y N	0 375**	0 03 NS
	1000				Z'	
Ist internode length (cm)	22.92	22.00	18.4	10.6	0 202	37 59**
2nd internode length (cm)	17,18	15,00	10.15	9.08	9.47**	13 52**
3rd internode length (cm)	12,4	10.58	7.44	6.06	0.645 0.645	10.69**

NS = Non-significant

Significant at 5% level of probability
 Significant at 1% level of probability

Table 2. Means (X), standard deviations (S, D_i) for parental, F_1 and F_2 generations and number of genes controlling plant height in three wheat croses.

Crosses		 Isogenic line I x Olsen dwarf 	 Isogonic line II x Olson dwarf 	
	×·	117.55	107.39	79.60
P,	S.D.	3,14	3.34	3,00
Р2	×.,	- 1	49.81	49.81 2.78 64.6 5.24 63.24
	S.D.	2.78	2.78	2.78
F,	×	49.81 2.78 87.57 3.96 84.72 15.49	49.81 2.78 76.75 3.26 77.24 13.98	64.6
	SD.	3.96	3.26	5.24
\mathbf{F}_{2}	×·	84.72	77.24	63.24
	S.D.	15.49	13.98	7.87
Number of genes for	height	3	2	ψ

plant stature in wheat,

The mean heights of all the F₂ populations approximated to mid parents (Table 2) which indicated the presence of additive gene effects for the inheritance of this character, an observation consistent with the findings of Amaya et al. (1972). Additive gene action points to the possibility of obtaining desirable plant statute with relative ease from the population under study.

Minimum number of genes involved for the inheritance of plant height in the crosses Isogenic line I x Olsen dwarf and Isogenic line III x Olsen dwarf were three while in the cross Isogenic line II x Olsen dwarf were two (Table 2). These results find support from the studies conducted by Saakyan (1981) and Pochaba (1985). However, Fick and Rualset (1973) observed four genes responsible for plant height in wheat.

As is evident from the data in Table 1, the length of the first, second and third internode below pedancle for the tail line (Isogenic line I) was greater as compared to the dwarf line (Isogenic line III). Moreover, the tall genotype also had greater mean internode length than the dwarf one. The values of simple correlation coefficients of plant height with the first, second, and third internode length below penducle were 0.963, 0.968 and 0.964, respectively and were positive and significant. These observations suggest that height in wheat primarily increased through increasing internode length rather than the number of nodes. These conclusions are in agreement with the findings of Markle and Atkins (1964) and Jerrel et al. (1967).

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