Supplemental data on *fasciata* genes in *Pisum* resources

Święcicki W.K. Inst. of Plant Genetics, Polish Academy of Sciences, Poznań, Poland

Fasciation is one of the most exciting characters in *Pisum*. It was described for the first time in 1597 (3), with different names having been used for the phenotype by various individuals *(Pisum umbellatum*, Mummy pea, Crown pea, Pois turc, Pois couronne, var. *coronatum*). Breeders have been interested in possibility of modifying stem architecture, flowering physiology and maturation using this source of genetic variation. Unfortunately, the theoretical advantages (most of the pods being produced on the upper portion of the plant) had associated disadvantages (lodging and drought stress). Thus, despite the availability of high yielding *fasciata* genotypes (5) few cultivars have been released (e.g. Buława/POL, Ornamenta, Rosacrone and Golf/GER, Novella/USA).

The *fasciata* phenotype appears to be much more important for geneticists. It was one of the seven Mendelian characters, and other cases of spontaneous mutation have been identified. The use of mutagens have led to the production of a considerable number of additional mutants (1, 5). Marx and Hagedorn (9) reviewed the literature dealing with the anatomy, morphology, expression and inheritance of fasciation in pea. Gottschalk induced and discribed a similar mutation type: *dichotomous branching*. The fasciation in this mutant type involved only a few nodes, resulting in a forked stem (1, 5). Gottshalk indicated that the gene *bif1* was present in mutants 1201A and 239CH and that *bif2* (polymeric to *bif1*) was present in the mutant 157A. Both genes showed incomplete penetrance. Gottshalk (4) also described a mutant, 37B, which proved to be an allele of the gene *bif1* with full penetrance.

Lamprecht (7) proposed that the *fasciata* mutation was controlled by two recessive genes (polymeric and duplicate): *fa* (12) and *fas* (6). The former has been mapped on linkage group IV (6) and the latter on linkage group III (2). WL 6 (Wt 10006 in the Wiatrowo Genebank) is the type line for both genes. In contrast, Marx and Hagedorn (9) concluded that the *fasciata* phenotype is controlled by one recessive gene but exhibits variable expressivity and incomplete penetrance. In the original test cross at Wiatrowo (WL 6 x Wt 3527 and reciprocal) the following F_2 segregations were found – 79 *Fa* : 20 *fa*, $\chi^2 = 1.21$, 77 *Fa* : 18 *fa*, $\chi^2 = 1.56$). Sidorova (11) tested for allelism among independent *fasciata* mutations (induced by different mutagens) and stated that at least 2-3 loci, supplemented by several modifiers, control this character. Rod and Vagnerova (10) suggested that the *fasciata* phenotype is controlled by three multiple alleles. Allelism tests were also made by Loennig (8) among 12 *fasciata* lines including WL 6. His results indicated that two of Vasileva's mutants, Mut I/74 and Mut II/87, were allelic but differed from the type line at one locus. The symbol used for the postulated new locus was *Fa2*.

After studying many *fasciata* mutants, Gottschalk concluded that three to four independent genes with multiple alleles are responsible for the character (5). Gottschalk stated that "These interpretations are not necessarily in contradiction to each other... certain observations indicate that the fasciated pea mutants are not a uniform group. They are heterogeneous in their morphology as well as in their genetic constitution." These analyses also included the line WL 5544 (weakly fasciated and bifurcated with unstable penetrance) but did not investigate relationships between *fasciata* and *dichotomous branching*.

The above, contrasting opinions regarding the genetic basis of the *fasciata* phenotype are rather frustrating. The character could be a suitable gene marker (particularly because linkage group IV is very poor in markers), but the genotype of many of the *fasciata* lines in genebanks is unknown. Moreover, the only tester line for linkage group IV, WL 1143, has a very weak *fasciata* expression. In order to clarify the genetic basis of faciation in pea lines held at the Wiatrowo Genebank, I performed complementation tests among them in all possible combinations.

Seventeen lines were identified as possessing a fasciated or similar phenotype (Table 1). These lines included material from Blixt, Gottshalk and Marx. The extensive nature of the full diallele test (272 combinations) required that the crossings and examination of the F_1 plants be conducted over a period of six years (1996 through 2001) in field at Wiatrowo. The F_1 plants, excluding those involving Wt 12 185 and Wt 10 785, were all fasciated. The type line for *fa*, WL 6, was included among the 17 lines, indicating that in

Table 1. Fasciata lines in locus identity test crosses.

Wiatrowo's catalogue number	Name and/or donor number	Donor and /or origin	Donor country	Type *)
Wt 10006	Mummy pea, WL 6		SWE	SE
Wt 10007	fasciata	Paloma,500r Nf+ 0.014% NEU	POL	MU
Wt 10285	Golf	K.Behm, Hamburg	GER	CV
Wt 10300	fasciata	Paloma, 0.014% NEU	POL	MU
Wt 10467	W_6 15 200	USDA, Pullman	USA	XD
Wt 10468	W_6 15 202	USDA, Pullman	USA	XD
Wt 10469	W_6 15 203	USDA, Pullman	USA	XD
Wt 10471	W_6 15 199	USDA, Pullman	USA	XD
Wt 10472	W_6 15 289	USDA, Pullman	USA	XD
Wt 10473	W_6 15 290	USDA, Pullman	USA	XD
Wt 10 783	251 A	Gottschalk	GER	MU
Wt 10784	489 C	Gottschalk	GER	MU
Wt 10785	37B, dichotomous branching	Gottschalk	GER	MU
Wt 11139	K.872/Wt	(LUFK x Wt 11 145)x WL30	POL	XD
Wt 12163	Wyola 50079			MU
Wt 12185	fasciata	Kaliski 500r Nf+ 0.014% NEU	POL	MU
Wt 12 187	K.657, WL 1120	Blixt,L.578 x L.668	SWE	XD

***) SE – selection, MU – induced mutant, XD – cross-derivative, CV - cultivar.**

all lines except Wt 12 185 and Wt 10 785 the fasciated phenotype is controlled by the *fa* gene. In cross combinations with Wt 12 185 the F_1 plants were normal suggesting that the second *fasciata* gene from a different locus is involved. When Wt 10 785 was used as one parent and *fa* lines as the second the F_1 plants displayed the *dichotomous branching* phenotype. This result suggests that the *dichotomous branching* mutation is caused by an allele at *Fa* with the following dominance ($Fa - fa^{bif} - fa$).

There remain several aspects of the genetics of the faciated phenotype still to be investigated. The second *fasciata* gene (in Wt 12 185) needs to be localized on the pea linkage map. Further allelism tests need to be conducted including mutant lines from Sidorova, Loennig, and Rod and Vagnerova. Finally, the relationship of the *bif2* mutations to fa^{bif} and other fasciated mutations needs to be examined. However, it appears that a tester line for linkage group IV can now be constructed using an *fa* gene with clear expression together with other markers on that linkage group.

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