

FLOWERING IN *PISUM*: EVIDENCE THAT GENE *Sn* CONTROLS A GRAFT-TRANSMISSIBLE INHIBITOR*

By I. C. MURFET† and J. B. REID†

Abstract

Under an 8-hr photoperiod scions of an early pea variety of genotype *lfe sn hr* grafted to stocks (with cotyledons) of a late variety of genotype *lfe Sn hr* flowered several nodes later than either intact, decotyledonized, or self-grafted plants of the early variety. The graft early/late was not later than the self-graft early/early when the scion and stock cotyledons were maintained under an 18-hr photoperiod. These results provide strong evidence that gene *Sn* is responsible for the formation of a graft-transmissible flower inhibitor under short days and an indication that *Sn* activity is suppressed under long days.

Introduction

Formation of a graft-transmissible flower inhibitor in the cotyledons of late varieties of peas was first proposed by Barber and Paton (1952) and Paton and Barber (1955) and several other workers have supported their proposal (e.g. Sprent and Barber 1957; Johnston and Crowden 1967; Paton 1969; Amos and Crowden 1969). However, the proposal has been challenged by Haupt (1969) and Köhler (1965) who claim that there is no need to postulate a flower inhibitor in peas as most of the data can either be explained solely in terms of a flower promoter or as an indirect effect on flowering resulting from interference with the normal growth rate. The latter interpretation certainly seems to apply to the reduced flowering node in late varieties grown as cuttings or with cotyledons excised and this effect becomes untenable as evidence of a cotyledonary inhibitor (Murfet 1973*b*). Again the flowering node of 11 reported for the graft Massey/Telephone by Paton and Barber (1955) could just as well be taken as evidence for a florigen in Massey cotyledons since the decotyledonized Massey plants flower at the same node in one of their two experiments. More recently Murfet (1971*c*) has provided additional evidence that the *Sn* gene ($Sn = S_2$ as redefined by Murfet 1971*b*) forms an inhibitor not only in the cotyledons but the shoot as well. Nevertheless the graft evidence of both Paton (1969) and Murfet (1971*c*) suffers from the disadvantage that no decotyledonized plants were grown for direct comparison. The present work was therefore designed to obviate this disadvantage and strengthen the evidence that gene *Sn* is responsible for the production of a graft-transmissible flower inhibitor.

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† Botany Department, University of Tasmania, Box 252C G.P.O. Hobart, Tas. 7001.

Materials and Methods

The two pure lines used in this work were developed from the crossing programme at Hobart and have been genotyped for four major gene loci controlling flowering, namely *Lf*, *E*, *Sn*, and *Hr* (Murfet 1971a, 1971b, 1973a). Line 53 (genotype *lf e Sn hr*) is a late variety developed from a cross between line 7 (Acacia) and line 22 (a selection from cultivar Massey). Line 58 (*lf e sn hr*) is an ED (early developing—see Murfet 1971a) form of early variety selected from a cross between line 53 and line 22. The plants were grown in the glasshouse in 3-litre cans using a growth medium of vermiculite and dolerite chips in a ratio of 1:1. Nutrient solution was supplied twice weekly. Flowering node refers to the node at which the first flower bud was initiated counting from the cotyledons as zero. Data are taken from main shoots only and any laterals were regularly excised.

The experiment involved two photoperiods—18 hr (14–15 hr of daylight with the remainder from a mixed incandescent–fluorescent source giving approximately 430 lux at plant height) and 8 hr (daylight) and four treatments—intact line 58 controls (58), decotyledonized line 58 plants (58⁻), self-grafts of line 58 (58/58), and the experimental graft of 58 scions on 53 stocks (58/53). The grafting technique is described by Murfet (1971c). The scion and stock were grafted together at the epicotyl. The seeds were germinated in the dark until the grafting and cotyledon excision were performed on day 5. (At this time the plumules were 8–16 mm long.) The plants, including the cotyledons of the stocks and intact controls, were then exposed to the appropriate photoperiod. Twenty-four plants were used per treatment per photoperiod. Slow and stunted grafts were rejected, the data in Figure 1 being for vigorous grafts only. The scoreable plants numbered 10 for 58/58 in long days, 9 for 58/58 in short days, and 15 for 58/53 in both long and short days. Night temperatures were in the order of 18–20°C and day temperatures 25–32°C.

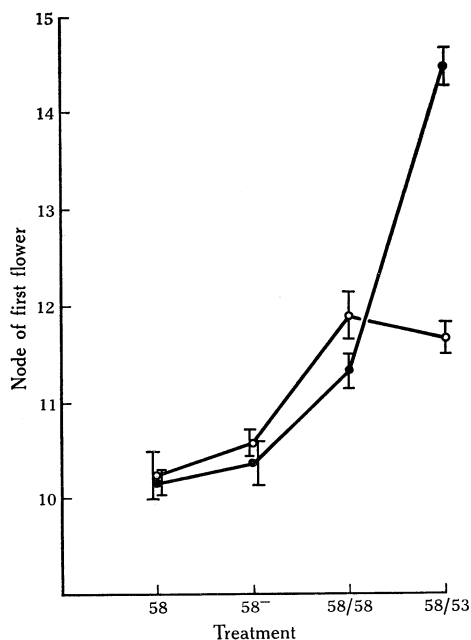


Fig. 1.—Mean node of first initiated flower ($\pm$ S.E.) for plants of the early line 58 grown as intact controls (58), decotyledonized (58⁻), self-grafted (58/58), or grafted as scions to stocks of the late variety line 53 (58/53). Grafting and cotyledon excision were performed on day 5.

○ Photoperiod 18 hr.
● Photoperiod 8 hr.

Results and Discussion

One point, the short-day graft 58/53, stands out from all other results in Figure 1. The large (4.3 nodes) delay in the flowering of the 58 scions cannot be attributed to either the absence of 58 cotyledons (58⁻) or the act of grafting itself (58/58), although self-grafting has caused a significant delay. These results point strongly towards a

positive delaying action by the 53 cotyledons (genotype *lf e Sn hr*) which may be attributed to the formation of a graft-transmissible flower inhibitor by the *Sn* gene under short days. Any floral stimulus from the 58 scions (or 53 cotyledons—see Murfet 1973*b*) is unable to achieve a hormonal balance in favour of flowering until the supply of inhibitor from the 53 cotyledons falls off. Alternatively the results could be explained without postulating an inhibitor if the 53 cotyledons were to act under short days as a sink for a floral stimulus produced in the 58 scions but this seems a rather unlikely role for cotyledons. Comparisons between plants with and without cotyledons have to be approached with caution because of differences in the growth pattern. However, our observations suggest that within the 58⁻ treatment reduced growth tends, if anything, to raise the flowering node. The indirect effect of altered growth pattern is therefore tending to reduce the flowering difference between the 58⁻ and 58/53 treatments and we may conclude that the remaining difference does truly reflect a hormonal action of the 53 cotyledons.

The delaying action of the 53 cotyledons is totally lacking in long days (compare 58/53 with 58/58), indicating a lack of *Sn* activity under long days. We have now repeated the graft 58/53 four times under short days and twice under long days. The results on each occasion have been closely consistent with the values given in Figure 1. However, the long-day self-graft 58/58 was 0.76 of a node earlier than graft 58/53 in the repeat experiment (difference significant at the 0.01 level). The implication of some *Sn* activity in long days is not necessarily correct as the slight delay may be caused by inhibitor formed during the first 5 days whilst the 53 cotyledons were in the dark. The relatively small delays reported by Paton and Barber (1955) for the graft Massey/Telephone could be due to the use of a photoperiod at least 4–5 hr longer than the 8 hr short day used here. The present experiment was designed primarily to check on the nature of *Sn* activity and we hope to report more fully on the conditions governing *Sn* activity in a later paper.

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