

A non-specific mutagenic gene in cyclamen¹

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Summary

In former work a dominant gene was described which prevents the vegetative mutation from white to violet flower colour, while in recessive condition this gene enables the mutation to take place. The present work deals with the vegetative mutation from very light pink to scarlet red. Evidence is presented that this mutation is determined by the same mutagenic gene as the mutation from white to violet.

Introduction

In cyclamen a frequently observed vegetative mutation involves the change from white to violet flower colour. This mutation may express itself in a longitudinal part of a petal, in one of more whole petals, in a whole flower, or in all flowers of a plant. A change from yellowish to dark purple colour of those stamens, which are situated opposite the mutated petals, always accompanies the mutation in the petals. This is comprehensible, since in cyclamen petal and stamen arise from the same primordium (Jeane de Vries, unpublished). The mutation from white to violet, followed by selection, has given rise to the violet cv. 'Sylphide', which is perfectly constant and never mutates back.

In earlier work (Wellensiek, 1960) it was reported that in certain families of 'White' the mutation in question occurs in each successive generation, while it never occurs in others. True breeding 'ever mutating' and 'never mutating' lines were selected. A genetical analysis followed, involving F_1 , F_2 , both backcrosses and F_3 . This led to the conclusion that the possibility of mutating depends on a recessive mendelian gene, or the other way round: that a dominant gene prevents the mutation by acting as a stabilizer. The case as such can be considered a mendelian mutation.

The present paper deals with another mutation, namely from very light pink ('light') to scarlet red, for the rest behaving identically to the mutation from white to violet as to the degree of its occurrence and the accompanying change of colour of the stamens. An observation, not previously made, is that the mutation may involve both sides of a petal, but sometimes occurs in the upper side, sometimes in the lower side only. The special problem was whether both mutations, from white to violet and from 'light' to scarlet red, depend on the same mutagenic gene.

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Materials en methods

In principle the method was to introduce a mutagenic gene from 'light' into a never mutating 'white' and to observe whether in F_2 the mutation from white to violet would occur. For this purpose a cross was made between the diploids 'never mutating white' and 'ever mutating light'. The 'never mutating white' was a selected line, in which not a single mutation had occurred during 4 successive generations. The 'ever mutating light' was a selection from a cross between 'Firefly' and the well known 'White with purple base'. 'Firefly' has been grown by Messrs Sutton & Sons in England since 1923. It was introduced in the Netherlands by J. Doorenbos in 1952 and found to be diploid by Legro (1959). The last feature made it very attractive for a diploid breeding-programme (Wellensiek, 1961). Van Bragt (1962) very carefully analysed some 'Firefly' crosses as to their flower colour pigments and in 'Firefly' \times 'White with purple base' found 5 F_2 plants with 'very pale pink' flowers. These may be identical to my 'light', but there is no absolute proof, since the cross 'Firefly' \times 'White with purple base' was continued for practical breeding purposes only and no further chemical analyses were carried out. Facts are that the parent of the present cross was selected for 'light' flower colour, that it bred true for this characteristic, apart from the fact that in 4 successive generations the mutation to scarlet red was observed. This certainly makes it an ever mutating line.

'White' and 'light', although no doubt genetically different, are phaenotypically so similar and so modifiable that a sharp distinction is impossible. Their colours overlap. The mutations violet and scarlet red, fortunately, are very distinct and classification is quite simple.

The cross 'never mutating white' \times 'ever mutating light' was made in 1967. The ordinary way of growing cyclamen for seed was followed, i.e. one generation in two years. The F_1 grew in 1968/1970, the F_2 in 1970/1972.

Experimental results and discussion

The 24 F_1 plants and the 334 F_2 plants varied in flower colour between white and 'light', but a clear distinction could not be made. During the period of flowering of 7 months, every flower was recorded. Mutants did not occur in F_1 , but were found in F_2 , surprisingly both to violet and to scarlet red. The F_2 results are:

not mutating	322 plants = 96.4 %
mutating to violet	8 plants = 2.4 %
mutating to scarlet red	4 plants = 1.2 %

In several cases the original flower colour of the violet mutants seemed to be white and that of the scarlet red mutants 'light', but there is no absolute certainty due to classification difficulties.

The results indicate that a mutagenic gene from 'light', introduced into a non-mutating white parent, in F_2 gives some plants which mutate to scarlet red, but also some plants mutating to violet.

In an attempt to explain these results, we start from the presence of the mutagenic gene pair, coded *mut mut*, in the ever mutating 'light' parent. Its dominant allele *Mut Mut* in the never mutating white parent prevented mutation. In $F_1 = Mut mut$ no mutation could occur, but in the F_2 *mut mut* segregated for 25 % and enabled mutation. When *mut mut* was combined with the genotype of white, the mutation was

from white to violet. When it was combined with the genotype of light, the mutation was from light to scarlet red.

The difference between white and violet is monogenic, white being *ww*, violet *W*. (Wellensiek et al., 1950) so that in F_2 25 % *ww* could be expected. The chance of combining *mut mut* and *ww* then would be 6.25 %, or 21 plants. This makes the percentage of mutation $8/21 = 38$.

A similar reasoning for the mutation from 'light' to scarlet red is not justified, since the genetical difference of the two types is unknown. Presuming a monogenic difference, the percentage mutation would be $4/21 = 19$.

The observed mutations of 38 % and 19 % for violet and red, respectively, are rather different from the mutations in the ever mutating parental lines white and 'light', which in 1970/72 were $15/86 = 17$ % and $22/68 = 32$ %, respectively. However, considering the great variation from year to year, the small numbers of mutants, and the unpredictability of mutations, a close agreement could hardly be expected.

The combination of *mut mut* with the double recessive genotype white and 'light' could be expected in 1.56 % or 5 F_2 plants. Such plants could probably mutate both to violet and to scarlet red. It is not surprising that they were not found.

The final conclusion is that the mutagenic gene from the 'light' parent, where it may induce the mutation to scarlet red, in F_2 plants with a suitable genotype induces the mutation to violet. Both mutations depend on the same mutagenic gene, so that its action is not specific to one gene. This conclusion would not be justified, if white and 'light' would both carry the mutable gene pair *ww*, while the phenotype of the mutants would depend on the rest genotype. Such a situation seems rather improbable. Whether the mutagenic gene influences other mutations as well, in other words, whether the dominant allele acts as a general stabilizer, remains an open question.

At least six other cases of mutagenic genes, although not indicated by this term, have been mentioned in the literature (Wellensiek, 1960, 1965). No other case has been described of one mutagenic gene unspecifically determining more than one mutation.

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