

INVESTIGATIONS ON THE INHERITANCE OF THE GLUCOSINOLATE CONTENT
IN SEEDS OF WINTER OILSEED RAPE (BRASSICA NAPUS L.)

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To breed quality winter oilseed rape with a low content of glucosinolates was a long way. Today the demand for so called industrial rape varieties confronts the breeder with a new task. It is necessary to combine new characters as specific fatty acid composition or very high oil content with low glucosinolate content. Only rapeseed meal with low glucosinolate content produced at an important amount with industrial rape can be used for animal feeding purposes.

Special knowledge about the inheritance of these features is necessary for the development of effective breeding methods. The following paper deals with investigations on the inheritance of the glucosinolate content. By crossing two doubled haploid lines and analysing the following generations the number of effective factors and their action were determined. Furthermore quantitative genetic parameters were estimated from a complete F1 diallel progeny of 8 parent lines.

MATERIAL AND METHODS

Crossing experiment: The doubled haploid lines A633 (8 $\mu\text{mol/g}$, P1) and A13 (116 $\mu\text{mol/g}$, P2) were crossed and the following generations were tested in 1989/90 in a field trial: P1, P2, F1, F1r, F2, F2r, BC1 and BC2. F1 is equivalent to (P1 x P2) and F1r is equivalent to (P2 x P1) and so for F2 and F2r. BC1 and BC2 are the backcrossing generations (F1 x P1) and (F1 x P2) respectively.

100 single plants of each non segregating population, three families with 200 plants of the F2 (F2A, F2B, F2C) and 300 plants per remaining generation were analysed.

From frequency distribution of the different generations the number of effective factors and their action were estimated after MEREZKO (1983). With generation mean analysis we proved the validity of MATHER and JINKS' additive-dominance model and computed additive and dominance effects (MATHER, 1949, PETERKA, 1973).

Diallel: 8 doubled haploid lines of different descent with glucosinolate content from 7 to 108 $\mu\text{mol/g}$ seed were crossed in a complete diallel crossing design. The 8 parent lines, 28 F1 and 28 reciprocal F1 were evaluated in a randomized block design with 3 replications in 1988/89 and 1989/90. The glucosinolate content was analysed on 5 plants per plot.

With GRIFFING's model 2, method 1 and 3 (GRIFFING, 1956a + b) expanded by NEUMANN and CLEMENS (1986) combining ability, genetic effects and heritability were estimated (inbreeding coefficient = 1).

RESULTS

Crossing experiment: The mean glucosinolate content of all generations is shown in table 1. Tested with χ^2 -test there were no differences in frequency distribution between F1 and F1r or F2A, F2B, F2C and F2r ($P = 0.21$ and $P = 0.63$). So the generations were summerized.

Table 1: Mean glucosinolate content and variance of different generations

generation	nb. of plants	glucosinolates ($\mu\text{mol/g}$ seed)	std.dev. s
P1	98	8	3.31
P2	110	116	11.51
F1	196	77	10.22
F2	853	79	20.32
BC1	298	43	17.59
BC2	297	102	14.16

In fig.1 you see the distribution of generation means in comparison to the mean parent value. In scaling-tests no significant deviations from the additive-dominance model were found, that means epistatic effects do not appear.

Following parameters were estimated:

$$\begin{array}{llll}
 \text{mean parent} & m & = (P1+P2)/2 & = 61.8 \\
 \text{additive effects} & [d] & = (P2-P1)/2 & = 53.8 \\
 \text{dominance effects} & [h] & = F1-m & = 15.4 & [d]/[h] = 0.29 \\
 & & = 2(F2-m) & = 34.9 & = 0.65
 \end{array}$$

The frequency distribution of parents and progenies is presented in fig.2. Supposed that in class 1 only segregants with the genotype of P1 appear the F2 and BC1 show a segregation for 4..5 independent factors. From the frequency of class 14 in F2 and BC2 can be concluded that 2..3 of the 4...5 factors have a dominant action (MEREZKO, 1983).

A similar result was found by estimating the number of effective factors n with the formular of CASTLE and WRIGHT (FRANKE; FUCHS, 1980).

$$n = (P1-P2)2/8(\sigma_{F2}^2 - \sigma_E^2) = 4,46$$

The environmental variance σ_E^2 was computed as the mean variance of parents and F1 generations.

Diallel: Table 2 gives the results of the analysis of variance. The main part of the whole variability is caused by general combining ability effects (gca). But also specific combining ability effects (sca) appear. No significant reciprocal effects were found. The environmental influence caused by the specific conditions of the two years is small but significant for sca and reciprocal effects.

Table 2: Analysis of variance for the diallel (mod.2, meth.3)

source	mean square	d.f.	F	lowest sig. F
gca	29757.16	7	29.14	2.49
sca	964.30	20	10.23	2.33
recipr.eff.	57.90	28	1.72	1.88
gca x years	71.70	7	0.81	2.76
sca x years	97.48	20	2.67	2.10
rec.eff. x years	33.63	28	2.67	1.53
error	32.36	220		

With these results it is possible to estimate the genetic parameters shown in table 3. The high heritability in broad sense (h^2_b) reflects the low environmental influence and the high heritability in narrow sense (h^2_n) the dominating additive effects.

Table 3: Quantitative genetic parameters estimated from the 8x8 diallel

parameter	meth.1, mod.2	meth.3, mod.2
additive effects	769.41	800.52
nonadditive effects	72.11	72.23
genotyp. variance	841.52	872.75
phenotyp. variance	882.26	914.33
envir.eff.on genotype	9.53	9.21
h^2_b	0.95	0.95
h^2_n	0.87	0.88
degree of dominance	0.43	0.42

DISCUSSION

All quantitative genetic methods used make a number of assumptions which are not met unrestricted by the experimental material. MEREZKO's method demands equal environmental variability of all genotypes. In our experiment this is only true for P2 and F1. P1 has quite a lower variability. This method and also the generation mean analysis suppose equal effects of all genes. With 4 active factors the mean effect of one homozygous dominant factor is about 27 $\mu\text{mol/g}$ seed. But the dominant action found for 2.3 factors is a sign for unequal gene action. The computed effects [d] and [h] are only netto effects and a different contribution of each gene is possible. Nearly the same F1 and F2 mean causes different [h] and [h]/[d] values. The reason can be environmental influence or a not fully lineal scale. In other F1 investigations different degrees of dominance were also found from year to year.

The consideration of parents in diallel progenies leads to an unproportional high amount of homozygous individuals in comparison to random mating populations. Therefore GRIFFING proposes method 3 in order to get unbiased estimations. In our experiment we found nearly no differences between method 1 and 3.

The selected 8 parent lines for the diallel represent the whole variability of the doubled haploid population. Because of the breeding aims the frequency of low glucosinolate containing lines in the population is much higher. But the breeder is interested in possibilities of transmitting the low glucosinolate genes and not so much in the genetical situation of the whole doubled haploid population. So the estimated parameters can be used for making the breeding methods more effective.

Because assumptions made for the different models are not met in all cases our estimated parameters can be incorrect. But in all experiments we got similar results and we can conclude that they reflect the real genetical situation .

Other authors found dominant or partial dominant gene action for glucosinolate content, too. They generally detected only 2...3 independent factors in the investigations in contrast to our results (LEIN, 1972, RÖBBELEN, 1976, MOU TONGMIN; LIU HOULI, 1988).

KONDRA and STEFANSSON (1970) found 4...5 factors in investigating the inheritance of glucobrassicinapin and progoitrin.

CONCLUSIONS

1. In segregating F2 populations the glucosinolate content is continuously distributed between both parents. Segregation classes are not discernible. F1 and F2 means are higher than mean parent.
2. Low glucosinolate content is inherited by 4...5 independent recessive factors. An influence of the cytoplasm could not be found.
3. The factors act in additive manner. Epistasis is not important. In addition to dominating additive effects dominance effects are important.
4. The heritability is high ($h^2_b = 0,95$, $h^2_n = 0,88$). The environmental influence is low. The variability of high content is higher than of low content.

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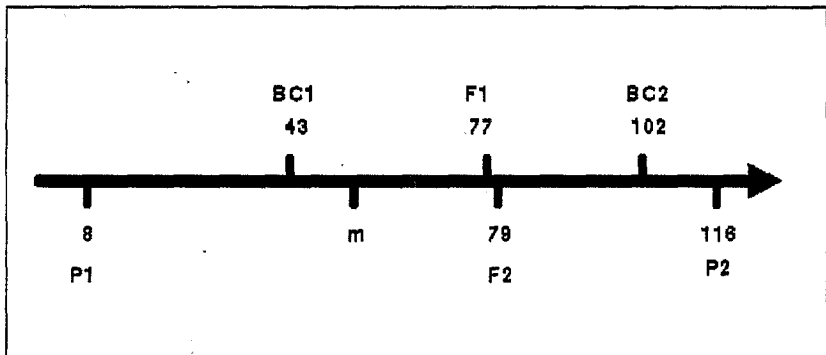


Fig. 1: Mean glucosinolate content of parents and progenies
(In umol/g seed)

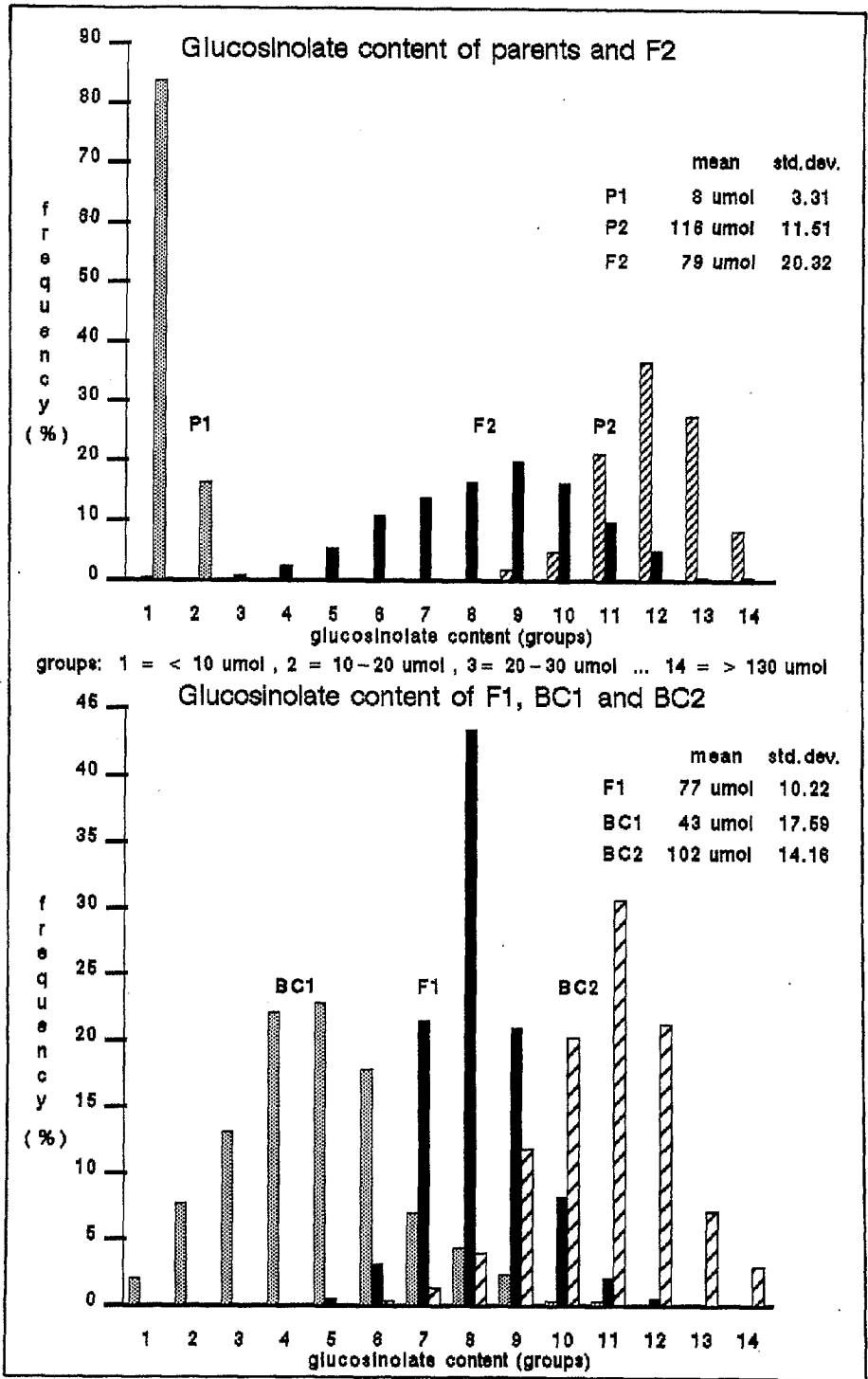


Fig. 2: Frequency distribution of parents and progenies