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Dominance modification in Neurospora crassa.

The aberrant ascus locus peok (biscuit) has been described previously by Srb and Basl (1969 Genet. Rer. 13:303). Among the mony mutant representatives at this locus are 5 ascus dominant alleles (17-088, 19-722, 21-804, 22-335 and 22-384) which, in heterozygous crosses (+/pk), show different characteristic frequencies of linear asci. Thus, it seemed of interest to look for modifiers that act on dominance relations in such heterozygotes. A technique of mutagenesis was employed in order to isolate mutants of wild type (74A) resistant to the colonialising action of sorbose, a chemical known to phenocopy the ascus abnormality when wild type crosser ore subjected to its action. The idea was to determine whether or not on "indirect selection method" would provide strains capable of modifying the dominant peok phenotype at the ascus level, in heterozygous crosses.

By this method, 209 rot-boss-resistant mutants were isolated, and 56 of these were crossed to the most dominant peak allele (17-088), which giver 96.5% abnormal asci when crossed to 74A. Modifiers were then identified or occurring in those strains that gave a repeatable, statistically significant increase in the percentage of linear asci over that observed in control crosses. Four genic modifiers (pk-mod-1 — pk-mod-4) hove been identified in this woy. The results of crossing 3 of the modifiers, and 74A, to 17-088 and to the other 4 dominant peaks ore shown in Table 1.

Table 1.

Strain	Dominant Peaks									
	17-088		19-722		21-804		22-335		22-384	
	L/T	%L	LIT	%L	L/T	%L	L/T	%L	L/T	%L
+	9712650	3.66	254/1471	17.27	78411013	77.39	2521950	26.53	145/497	29.18
ok-mod-	1290/11,521	11.20*	124/588	21.1	593/642	92.5*	355/605	58.7*	2661442	60.2*
ok-mod-2	502/4106	12.23*	48/264	18.18	3391341	99.41*	216/451	47.89*	1631239	68.20*
pk-mod-3	506/5186	9.76*	135/381	35.43*	2251270	83.33	55/472	11.65	1391239	58.16*

* Indicates a significant increase over the control value L/T = number linear asci/total number asci scored
%L = percentage linear asci

It is clear from these results that modifiers first identified in reference to one of the dominant peak alleles do not affect the dominance relations of the other dominant peak alleles in the same way. A difference in specificity of the modifying effect is seen between pk-mod-I and pk-mod-4 and between pk-mod-2 and pk-mod-3, whereas pk-mod-1 and pk-mod-2 seem to hove the some specificity.

Table 2.

Strain		Dominant Pk (17-088) L/T	%L
pk-mod-l;	pk-mod-3	102/822	12.41
pk-mod-3;	pk-mod-3	30/822	12.88

In order to investigate whether the modifiers might act in an additive manner or not, double modifier strains were set up and these were tested against 17-088, as shown in Table 2. A comparison of the figures for the double modifiers with those of the modifiers crossed singly with 17-088 (see Table 1) indicates that there is no rignificont difference in the modification.

It was then considered that a modifier might have on effect if it were in the homozygous condition. Crosses of the type (pk-mod-l; pk/pk-mod-l; pk^+) were set up and scored for linear versus non-linear asci. With dominant pk (17-088) the result was 175 linears out of a total of 2731 asci; that is, 6.4% linears, indicating a decrease over the effect of the modifier in heterozygous condition.

Further experiments are under way to test whether or not ony of the modifiers ore allelic, and to amplify existing results. (This work was supported by grant GM-12953, National Institutes of Health, USPHS). - - - Sectiion of Genetics, Development and physiology, Cornell University, Ithaca, New York 14850.