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PUBLICATIONS

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G. MORPURGO. Induction of mitotic crossing-over in Aspergillus nidulans by bifunctional alkylating agents. Genetics 48, 1259, 1963.

CURRENT RESEARCH PROJECTS

A.T. BULL.

Action pathways of mutagenic base analogues.

B.M. FAULKNER

1) Induction and behaviour of cytoplasmic mutations.
2) Resolution of the cytoplasmic contributions to differentiation patterns in lines carrying genetically marked cytoplasms.

PRELIMINARY NOTES ON CURRENT RESEARCH

G.J.O. JANSEN

UV-induced intragenic recombination in Aspergillus nidulans

A study was made of UV-induced mitotic recombination within the paba[1] region of Aspergillus nidulans. For that purpose hetero- and homoallelic diploid strains were synthesized, using two allelic paba mutants isolated in our laboratory some years ago. PABA-independent reversions from these strains were selected by plating conidia in the absence of PABA. Plates were incubated at 37°C.

Irradiation of heteroallelic conidia with low UV doses (permitting high survival) usually causes a slight increase in the reversion frequency over the spontaneous level. Incubation of heteroallelic conidia in PABA-supplemented liquid minimal medium (MP medium) for 1 hour at 37°C also increases the reversion frequency over the control level. Combination of UV irradiation with either pre- or postincubation in MP medium for 1 hour at 37°C reveals a synergistic action of the two successive treatments. Results of a typical experiment with a heteroallelic strain are given in the table.

treatment	$paba^+$ per 10^6 survivors (survival approx. 100%)
Control	1
UV	4
MP	17
MP - UV	69
UV - MP	54

Similar experiments with the homoallelic diploids show that UV irradiation induces reversions in homoallelic conidia as well. However, the UV-induced reversion frequency in these conidia is much lower than in heteroallelic conidia and is not significantly influenced by pre- or postincubation in MP medium.

Assuming that the probability of backmutation in the heteroallelic condition is not higher than in the homoallelic condition, the reversions obtained from a heteroallelic strain can only partly be attributed to backmutation (or to the induction of dominant suppressors) of either paba allele.

It is concluded that in heteroallelic conidia UV irradiation induces recombination between both paba alleles and that the frequency of this UV-induced mitotic recombination is enhanced by pre-or postincubation of the conidia in PABA- supplemented medium. The presence of PABA seems to stimulate metabolic processes involved in the UV-induced recombination event (Jansen, manuscript in preparation).

CURRENT RESEARCH PROJECTS

G. MORPURGO.

Effects of monofunctional and bifunctional mustards in inducing mitotic crossing-over. The purpose is to test the hypothesis that the very high frequency of mitotic crossing-over induced by treatment with the bifunctional mustard methyl bis (β chloroethyl)-amine is due to the cross-linking of non-homologous chromatids and hence to effective exchange of parts. First results strongly support this hypothesis.

M.G. PETRELLI, R. RICCI and G. SERMONTI.

Study of suppressor mutations of pfp-r (p-fluoro-phenylalanine resistance).

G. MORPURGO.

Induction of mitotic crossing-over by means of fluorouracil (FU) and fluorodeoxyuridine (FUDR)

Conidia of a diploid strain with the following genotype in the first chromosome:

$$\frac{su1\ ad20\ ribo1\ pfp\text{-}r\ +\ \circ\ pro1\ +\ ad20\ +\ bi1}{+\ +\ +\ an1\ +\ paba1\ +\ y\ +}$$

were plated on a medium supplemented with various quantities of FU and FUDR. The conidia formed on such a medium were seeded on Petri dishes on MM medium supplemented with riboflavin and para-fluorophenylalanine, on which only cross-over segregants can produce colonies. Both FU and FUDR produce a 20-30 fold increase over the spontaneous level in the mitotic crossing-over frequencies at a concentration of 0,00568 and 0,00285 mM respectively. Action of FU is completely abolished by excess of thymine or uracil, and action of FUDR by an excess of uridine. Reconstruction experiments have completely ruled out the possibility of selection.

A.T. BULL and B.M. FAULKNER

Mutants of A.nidulans obtained following treatment with 8-aza-guanine (8AG).

Conidia from wild type strain 13 (Birmingham) and mutant strain 13y (yellow conidia, Birmingham) were inoculated into liquid Czapek MM containing 8AG at 10^{-8} to 10^{-2}M. Growth of both strains was inhibited by 10^{-2} and 10^{-4} M 8AG; lower molarities had a stimulatory effect. Growth inhibition was reversed by guanine, adenine andxanthine (c.f. MORPURGO, Aspergillus News Letter, No.2) but not by uric acid.

Following growth in the 8AG supplemented liquid MM both strains were screened for morphological variants on solid MM. Five classes of variants were isolated initially:-

class 1. pink, fluffy mycelium; perithecia bearing Hülle cells which autolyse; pink pigment secreted into medium.

class 2. perithecia absent; little extracellular pigment compared with wild type.

class 3. mycelium covered with web of aerial hyphae; reduced production of perithecia; red-pink pigment produced extracellularly.

class 4. increased production of perithecia; few conidial heads; little extracellular pigmentation compared with wild type.

class 5. as class 1 but Hülle cells normal.

Class 1 and 2 variants were obtained from strain 13 in 7 and 1 separate treatments respectively. Clas 1, 3, 4 and 5 variants were obtained from strain 13y in 1, 4, 2 and 1 separate treatments respectively.

Heterokaryon tests of class 1, 2 and 5 variants indicate that they are nuclear in origin.

Subsequent to their isolation class 3 and 4 variants have given

rise among their asexual spore progenies to a number of new phenotypic classes. Several of these new phenotypic variants persistently segregate among their asexual offspring and are prospectively cytoplasmic in origin. Heterokaryon tests of the persistently segregating variants have not yet been attempted.

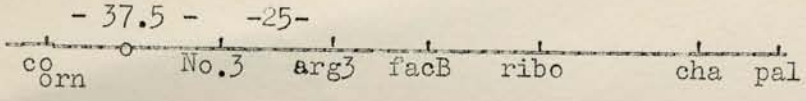
J.L. DE AZEVEDO

The chromosome VIII map of Aspergillus nidulans

A mutant unable to utilise nitrate, but which grows when the nitrate is replaced by nitrite or NH_4 sources, has been induced with UV treatment in a bi1 strain of A. nidulans. The mutant presents a residual growth on minimal medium and grows quite well when large amounts of certain amino acids (arginine, proline, glutamic acid aspartic acid, threonine, alanine, glycine) or guanine are added to the minimal medium. By mitotic analysis the gene was located on chromosome VIII on the same arm as cha and proximal to cha and ribo2. Meiotic analysis gave the following relevant results.

compact (co) - NO3 : 37.5 (622 ascospores analysed)
Arg 3 - NO3 : 25 (160 ascospores analysed)

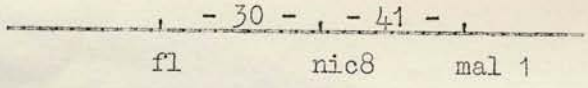
The new marker permits, therefore, the joining of the three groups of meiotically linked loci (Apirion, Dorn and Forbes - Aspergillus News Letter no. 4) on chromosome VIII.



C. BALL and J.L. DE AZEVEDO

A "fluffy" mutant in Aspergillus nidulans

A morphological mutant characterised by a mass of aerial mycelium has been recovered (probably induced by acridines) in a bi 1 meth 1 strain of Aspergillus nidulans. Mitotic analysis showed that the marker responsible for the fluffy character is located on chromosome VII. Meiotic analysis showed that the new marker is linked with the nic 8 gene (30% recombination) but recombines freely with the mal 1 gene. If the linkage value between nic 8 and mal 1 is 41 (Roberts - Aspergillus News Letter No.2.) the actual situation on chromosome VII is:



The fluffy character is recessive; diploid heterozygous colonies produce fluffy sectors (haploid and diploid). By analysis of these sectors it will be possible to locate the centromere of chromosome VII of A. nidulans.

B.W. BAINBRIDGE

The Mechanism of Crinkled Reversion

The segregation of an abnormal type, crinkled, from crosses

heterozygous for T (III - VIII) has already been reported. (Bainbridge ANL 4, 20). It was suggested that crinkled strains possessed duplicated chromosome material.

Chromosomes III and VIII were implicated by stock strain pedigrees. Recent results have shown that certain crinkleds appear to be heterozygous for markers located in the right arm of chromosome III. A cross between a phenotypic cys_2^+ crinkled and a cys_2^+ normal strain segregated equal numbers of cys_2^+ and cys_2 normals. This suggested that the parental crinkled was cys_2^+/cys_2. It seems safe to conclude that the main segment involved in the translocation is part of the right arm of chromosome III which has been translocated on to chromosome VIII.

Reversion of heterozygous crinkleds yielded revertants which were either cys_2^+ or cys_2. Out of 16 revertants from one crinkled, 11 were cys_2^+ and 5 cys_2. Five of these revertants, three cys_2^+ and two cys_2, were backcrossed to translocated and untranslocated strains. In all five cases either 2:1 (translocated cross) or c 20:1 (untranslocated cross) ratios were obtained or the ratios were reversed relative to the translocation state. In four cases out of five, there was a direct correlation between the behaviour of a revertant in the crosses and its response to cysteine, i.e. cys_2 x unT always gave 2:1 and cys_2^+ x unT normally gave c 20:1. The result suggested that there was suppression or loss of either member of the translocated segment.

Previous results had suggested that reversion could be due to a suppressor mechanism. The original theory was based on experiments in which it was thought that cys_2 could be distinguished nutritionally from S_1. This was later found to be incorrect. More detailed experiments have led to the rejection of the suppressor theory. The rejection is based on the following crosses.

CROSS			NORMAL PROGENY	
Revertant		Normal	"unsuppressed" allele	"suppressed" allele
R6 cys_2^+	x	cys_2^+	84	0
R12 cys_2^+	x	cys_2^+	112	0
R14 cys_2^+	x	cys_2	50	0
			500	0
			9500	0

It will be seen that in three crosses with different revertants the theoretically "suppressed" allele has not been recovered. This suggests that the mechanism of reversion involves "drop off" of the relevant chromosome piece.

C.F. ROBERTS

Initial stages in galactose metabolism

The accumulation of C^{14} galactose by intact mycelium was a reversible, energy requiring process which was a constitutive property of the organism. All of the gal mutants tested (gal 1 to gal 5 loci) accumulated the sugar.

Isotopic techniques have enabled identification and assay of the

enzymes galactokinase and Gal-1-P uridyl transferase; these were present at readily detectable levels in the non-induced WT and increased 5 and 20 fold respectively upon induction. (Mycelium was induced by growth in liquid medium at pH 6.5 with 2% glycerol and 1% galactose.)

Mutants at the gal 5 locus had less than 2% of the transferase activity of the WT. Mutants at the gal 1 locus had normal constitutive levels of Kinase and transferase but the amount of neither enzyme increased upon induction. The gal 1 mutants were permeable to galactose and recessive; they may lack epimerase (c.f. ga 10 mutants in yeast, Douglas & Hawthorne, Genetics, 1964) though the possibility that they are regulatory mutants has not been excluded.

A pH effect on the phenotype of galactose mutants

Strain bi 1;w 3 utilises galactose in the range of pH 4.0-7.5 when tested on solid media. Total galactose mutants grew poorly or not at all in the range pH 4.0-6.0 (0.05 M citrate) irrespective of the sample of galactose or agar used in the test media. The most severe mutant phenotypes were observed at pH 5.0.

At pH 6.5-7.0 (0.01 M phosphate) the phenotypes were modified by the sample of galactose or agar employed, though the mutants were distinguishable from the WT after 3-4 days incubation. Generally the most severe mutant phenotypes were yielded with BDH agar and Kerfoote's galactose (c 1% glucose) and the least with Difco agar and Sigma galactose (glucose free). At pH 7.5 (0.01 M phosphate) the mutants generally grew like the WT.

Experiments with mixtures of Sigma galactose and small amounts of glucose (Difco agar) have failed to support the hypothesis that at pH 6.5-7.0 the total phenotype results from the presence of small amount of carbon source in addition to galactose. Trace salts had no effect on the phenotypes and the mineral salts medium was the usual one.

Non-allelic mutants respond differently to pH, thus the pH effect could be in the accumulation of inhibitory intermediates. It is also possible that the organism has more than one system for galactose metabolism and these operate at different pH's.

From the practical point of view variation in pH may help to accentuate the phenotypes of other sugar mutants.

E. KÄFER-BOOTHROYD

Evidence for a paracentric inversion from mitotic crossing over in A. nidulans.

Mitotic cross-overs were selected on both arms of linkage group I in a diploid heterozygous for the following markers in coupling: sulad20 (= su), ribo1, pro1, paba1 and y. "Suppressed" su/su diploids turned out to be of three types; prototroph, ribo1/ribo1 and ribo1 pro1/ribo1 pro1, all still y/+. On the other hand only two types of yellow (y/y) cross-overs could be found, y/y only and y paba1/y paba1 (both still su/+). It is concluded, that a paracentric inversion is present, which transfers pro1 to the left arm of linkage group I and has breakage points proximal to ribo1 and paba1. Whether the test diploid is heterozygous or homozygous for the inversion is not yet clear, nor is anything known about the origin and pedigree of the inversion, since pro1 has not often been used in routine tests for translocations.

A. PUTRAMENT

Prolonged appearance of adenine-independent revertants in heteroallelic diploids ad9/ad15 of Aspergillus nidulans.

When diploid conidia of genotype

pro3	ad9	+	+	y	+	+
+	+	ad15	pabal8	+	bi1	phen2

are plated on adenine-less medium, adenine-independent colonies begin to appear after 48 hours and continue to appear for several days.

The phenomenon seems to be similar to that discovered by M. Luzzati, L. Clavilier and P. Slonimski (C.R. Acad. Sci., v. 249, pp. 1412-1414, 1959, and other publications) in diploids heteroallelic for ad mutants in yeast.

Other ad mutants of A. nidulans will be tested for the same phenomenon.

Diepoxybutane induced mitotic recombination in Aspergillus nidulans

The following diploid was synthesised:

pro2	paba2	y	+	+	+	+	phen2	+
+	+	+	bi1	ad23	Acr1	w2	+	nic8

After treatment of the conidia of the diploid with 0.1M DEB survival is about 14%. Thirty per cent. of the colonies obtained from treated conidia (plated on complete medium) show recombinant sectors in respect of one or more markers. In some cases whole colonies are recombinant, no phenotypically wild-type sectors are recovered.

P. WEGLEŃSKI, Department of General Genetics, The University, Warsaw.

Genetic analysis of proline - 1 and proline - 3 cistrons in A. nidulans.

Four mutants from pro-3 cistron and ten from pro-1 cistron (all requiring proline or arginine or citrulline or ornithine) were mapped. The distance between the two cistrons is about .3 map unit. Complementation was found only among mutants from the two different cistrons. Spontaneous reversion frequencies for all mutants were established. Only suppressor type reversions both recessive and dominant were found. They are preliminarily mapped in chromosome III. Location of further suppressors is in progress.

JANINA KLIMCZUK, Department of General Genetics, The University, Warsaw.

Spontaneous and induced reversions of meth1 mutant of Aspergillus nidulans.

Reversions of meth1 mutant induced by HNO_2, U.V. and diepoxybutane (DEB)

were studied. The induced reversions seem to be of suppressor types A, B, C as were described for spontaneous reversions by Siddiqi (1962) and Lilly (1963). Reversions rates at the optimum of action of the mutagens used are as follows:

Mutagen	Survival %	Frequency of recombinants x 10^5		
		A	B	C
HNO_2	3.91	36.08	18.31	12.07
U.V.	0.97	76.52	150.31	38.98
DEB	1.82	191.49	230.76	82.34

DEB seems to be the most effective mutagen. There seems to be a selective killing effect of some mutagens on different meth1 suppressor strains which is studied now.

C. BALL

Some actions of the acridines on A. nidulans

Several acridines have been investigated with reference to:-

a) resistance, cross-resistance and reversal patterns within the series.

b) induction of abnormal morphological variants.

c) mutation as assayed by reversion of $meth_1$ in the strain bi_1 $meth_1$.

a) Resistance

The strains bi, bi acr_2 bi mg_1 bi Acr_1 were used. Resistance patterns to various acridines were as follows:

bi Acr_1 >> bi acr_2 ≃ bi mg_1 > bi.
acridine yellow bi Acr_1 ≃ bi acr_2 ≃ bi mg_1 > bi.
proflavine bi Acr_1 > bi acr_2 ≃ bi mg_1 ≃ bi.
acridine hydrochloride, 5 amino acridine, acridine orange, atebrin) bi Acr_1 ≃ bi acr_2 = bi mg_1 = bi.

Many of the less inhibitory acridines could reverse the inhibition of the more inhibitary ones when incorporated into the growth medium (MM + bi). Also an indication was obtained of partial dominance of mg_1 on acridine yellow medium and recessivity on acriflavine medium.

b) Abnormal morphologies.

After treatment of conidia of a bi_1 strain in MM + bi + 40 mg/l. acriflavine at 37°C for 24 hours, and then plating on complete medium, a 10% survival was obtained. Of these survivors, 20% were abnormal. Conidial sub-culture, from the centre of these colonies gave non-sectoring abnormal variants and abnormal variants sectoring normal growth. All of the non-sectoring types were single gene determined. One of the sectoring variants studied was shown to go through meiosis in low frequencies (1 in 20 hybrid perithecia) and was shown to have a duplication

of chromosome IV, loss of either homologue of chromosome IV, giving rise to normal sectors with faster growth rate.

c) Reversion

A significant increase in suppressor mutation frequency in the meth[1] system has been shown by nitrous acid treatment (Siddiqi) and also with DEB, UV, β propio-lactone (Ball and Kilbey, unpublished). Acridines have also been shown to induce suppressor mutations. Acridine concentration, high light intensity, oxygen and nitrogen, influence mutagenesis. Further interactions of these variables are being studied. Increase in rates of X 4 per plated conidium and X 15 per survivor for each of the revertant phenotype A and B have been obtained.