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PRELIMINARY NOTES ON CURRENT RESEARCH

C. BALL.

An "unstable" state associated with gene suppression.

During experiments involving reversion of the gene meth$^-$ in strains bi_1; $meth_1$ a number of very small colonies (not scored in the part of the system examined by Lilly) were recovered. By subculture and spreading of conidia, normal colonies were obtained on CM and on MM + biotin + methionine, but on MM + biotin there was a great range of colony sizes. Many vigorous colonies arose as faster growing sectors from what initially were poorly growing areas and the range of colony sizes appeared, mainly, to be the result of differing times of appearance of such sectors.

One series has so far been studied further by several means.

1. Vegetative propagation.

   (a) After conidial platings on CM the range of colony sizes could again be obtained by conidial subculture and plating on MM + biotin.

   (b) Further subculture to MM + biotin from colonies of different sizes, in all cases again yielded a range of colony sizes. In certain of these lines, however, viability was observed to decrease.

   (c) At 25^{o}C the proportion of colonies on MM + biotin, with extremely slow growth rate was reduced and germination occurred earlier than at 37^{o}C.

2. Genetic analysis

Crosses to strains $ribo_1$ y nic_8 and pro_1 $paba_6$ y w_3 enables recovery of segregants with the phen types meth$^-$, meth$^+$ and "unstable suppressor" meth$^-$ in frequencies not inconsistent with the segregation of an unlinked genic suppressor of meth$^-$.

The situation described here is in certain respects similar to that found in Salmonella (Dawson and Smith Keary, Heredity, 1963; Genetical Research, 1964). The possibility of closer analogy is being examined.

R. N. NEDER.

Temperature sensitive lethals in A. nidulans.

Initially the study was aimed at the isolation of variants with respect to growth ability on different types of media at different temperatures.

The temperatures used were 25^{o}C, 30^{o}C, 37^{o}C and 45^{o}C. The strain used was bi_1 $meth_1$ and media: CM, MM + biotin + methionine and MM + biotin + methionine + supplements.

Conidial suspensions were irradiated with U.V. to 1% survival and survivors incubated on CM at each of the temperatures used. Subsequently, replication was made from these (inoculated into a master plate matrix) into MM + bi + meth at the temperature in question, and also into CM and MM + bi + meth at the other temperatures. An equal number of non-irradiated control colonies were analysed in each case and in all 8,000 colonies from treatments and controls were studied.

The series so far studied in most detail is that in which survivors were incubated at 25°C. On the basis of subsequent behaviour (growth or no growth) after replication to the two types of media, two general categories could be distinguished: (a) Variants that were irreparable in that the same behaviour was found on both types of media. These constituted 3.7% of the survivors analysed. (b) Variants that were reparable in that they were auxotrophic for characters, other than bi and meth, either at one temperature only or at more than one temperature. The former constituted 0.5% of the survivors analysed and the latter 0.5%.

No lethals were recovered in controls.

The present study is directed at establishing the genic nature (or otherwise) of these variants. So far a number of diploids have been synthesised between normal strains and variants exhibiting lethality at 45°C only. Complete recessivity of the character in each case was shown.

J. L. de AZEVEDO.

The Centromere of Chromosome VII of Aspergillus nidulans.

Diploid fluffy sectors from a diploid strain having mal1 and nic8 markers in coupling with a morphological fluffy marker in chromosome VII (see Ball and de Azevedo A.N.L. No. 5), were analysed. These sectors turned out to be of three types:

a) mal$^+$ nic$^+$ fl.

b) mal$^+$ nic$^-$ fl.

c) mal$^-$ nic$^-$ fl.

These results indicate that probably the three markers are on the same arm in chromosome VII and fluffy (fl 1) is distal in relation to nic8 and and mal 1.

mal1   nic8   fl1

——o————————————————

A Technique to detect Recessive Lethals in Aspergillus nidulans.

Two diploid strains, carrying appropriate colour or morphological markers (yellow, white, chartreuse and fluffy) were treated with UV light (1 - 5% survival) and surviving colonies were plated on minimal medium plus the requirement carried by the haploid strains which form the diploids + para-fluorophenylalanine (pfa). The absence of one type of colour or morphological sector, would indicate possible recessive lethal on the chromosome which carries the marker responsible for this type of sector. If no sectors are produced on medium with pfa, this would indicate that a recessive lethal was induced on chromosome III, in repulsion to the phen$^-$ marker since phen$^-$ sectors are inhibited by pfa.

In a total of 200 control strains, no recessive lethals were located in the chromosomes tested. In a total of 200 treated strains, a frequency of 28% of recessive lethals was found for all the genome.

Diploid strains with recessive lethals, tested on several different media have shown that 95.5% of the recessive lethals were irreparables by the media used, and 4.5% were found to be reparables (auxotrophic mutants).

B. W. BAINBRIDGE.

The Arginine Crossing Technique.

It has been observed that large perithecia develop at the junction between colonies of y; w_2; arg_1 and colonies of certain other strain (Clutterbuck, 1963, unpublished). This effect usually occurred on complete medium to which no extra arginine has been added.* Perithecia from the junction were found to be predominantly hybrid. These observations have been used to develop a technique for obtaining hybrid perithecia.

The basic technique was to streak strain y; w_2; arg_1 across a thick CM plate (20 ml. of medium). The second strain was streaked at a very acute angle to the first strain so that mixed spores occured at one end and the two strains were a few mm. apart at the other. Alternatively, spores were mixed at random on the plate. After seven days incubation at 37°C large mature perithecia formed at the junction between the strains or in regions of mixed spore heads. Fifty perithecia have been tested from ten crosses and 40 perithecia were found to be hybrid. Six of the crosses had 100% hybrid perithecia and the lowest frequency of hybrids was 25%.

The original strain y; w_2; arg_1 carried ve^+. This morphological character prevented accurate multi-point replication. A strain was therefore isolated which had the mutant character, ve (Käfer A.N.L. No. 3, 1962). The new strain y; arg_1; ve was a segregant from the cross y; w_2; arg_1 x bi_1; cys_2; Act_1; ve. (This latter strain was derived from a cross between $ribo_1$; y; Act_1; nic_8 x bi_1; w_3; cys_2 (Warr, 1964, Ph.D. Thesis)). The new strain produced the same perithecial effect as the original strain.

Preliminary results involving y; arg_1; ve crosses have suggested that the perithecial effect is due to the presence of the arg_1 allele or to effects closely associated with it_1. The absence of the effect on CM, to which extra arginine had been added, suggested that the effect may be due to partial inhibition of the arg_1 strain on medium containing a limiting balance of arginine and lysine. Under these conditions the arginine strain produces immature perithecia.

The advantages of this technique are as follows:

1. Large, predominantly hybrid, perithecia can be obtained in 7 days on CM.

2. Petri dishes need not be sealed.

3. Almost any strains may be crossed provided that one strain has the arg_1 allele.

4. The technique is particularly useful for crossing strains which have a high reversion rate. Selection for the revertant type can be reduced to a minimum by growing the two

strains closely together and selecting perithecia only from the junction. The technique has been used extensively for crossing crinkled types to wild type, (Bainbridge, Ph.D. Thesis, 1964). In this case, reversion of crinkled occurs frquently and the techniques is essential to avoid crossing the revertant instead of the crinkled type.

* <u>CM</u> : Yeast extract (Difco), 1g.;
Peptone (Difco, 1 g.;
Casein hydrolysate (Difco), 1 g.;
Adenine HCl, 0.15 g.;
Glucose, 10 g.;
Vitamin solution, 1 ml.
Agar, 15 g.;
Water, 1-litre.;
pH to 6.5.

Addition of arginine : 0.04% arginine (final concentration).

G.J.O. Jansen.

It was shown previously that UV irradiation of diploid conidia induces mitotic recombination in the $paba_1$ cistron (Jansen (1964), ANL 5, 6 and Genetica 35, 127-131). A genetic analysis was made of PABA independent recombinants obtained from UV-irradiated diploid conidia that were heteroallelic at the $paba_1$ locus. Approximately 75% of the colonies may have been of UV-induced origin. Out of a random sample of 36 recombinants 31 colonies were completely analysable. Of these, 30 recombinants were shown to contain a paba strand in addition to a $paba^+$ one. In one recombinant two $paba^+$ strands were present. In 29 cases the paba strand carried either one (15x) or the other (14x) parental paba allele. In only one case the paba strand contained the double mutant. These data may be explained in two ways: either recombination at the $paba_1$ locus was usually reciprocal, but non-random segregation of chromatids occurred; or random segregation of chromatids occurred, but recombination was usually non reciprocal. As there is no reason to suppose that preferential segregation occurred and since the phenomenon of non-reciprocal recombination (gene conversion) appears to be of widespread occurrence in fungi, our data suggest that UV irradiation predominantly induced gene conversion at the $paba_1$ locus. The analysis of an additional number of recombinants is in progress.

JOHANNA C. SOBELS.

Mutagenic effects of DNA in Aspergillus nidulans.

Spores of *A.nidulans*, y;Acr$_1$,ad$_1$;pyro$_4$ were treated with DNA extract prepared from mycelium of a green prototroph strain.*
Three series were run: (1) 3.7×10^6 spores were treated with an extract containing 0.068% DNA. (2) 7.4×10^6 spores were treated with DNA that was slowly cooled after having been kept for 5 min. at 100°C. (3) 3.7×10^6 spores treated with DNA inactivated by DNA-ase served as control.

In series (1) only one haploid, wildtype mutant colony was recovered. Further genetic analysis showed complete reversion to Y, ad$_1^+$, pyro$_4^+$ and partial reversion to Acr$_1^+$.- In series (2) 24 diploid, pheno-typically wildtype colonies were observed. All of these showed somatic segregation for y.- No mutations were obtained from the control series (3).

Out of the 24 diploids obtained in series (2), three were tested in detail by mitotic, and seven by meiotic segregation. They all showed recombinations of either the 4 original markers of the treated strain, or reversions of these in any combination, with, in addition, new auxotrophic mutations, as: bi,nic/tryp/indol, paba, phen, pro, lys, meth, or some spore pigment mutations as grayish green or white. It may be noted that from the 66350 ascospores plated in the tests for meiotic segregation, only 762 (1.15%) germinated; they were all mutant, about one half being diploid and the other half haploid. Retested, the majority was of unstable genetic character.

The experiments were repeated with a commercial DNA preparation (herring-sperm, N.B.C., Cleveland, OhiO.)- In one series of experiments spores of the same stock were treated. Very similar results were obtained, i.e. a high frequency of diploids and multiple reverse and forward mutations, as in the above experiment with Aspergillus DNA.

In other experiments spores of the genetic constitution $y, ad_{20}; Acr_1; ribo_2$ were plated on media supplied with the heated and slowly cooled sperm DNA. Whereas no mutations were found in the controls, of the approximately 490 spores plated on the DNA medium, 161 germinated, 28 gave mutant colonies and 2 diploid colonies.- 148 spores which germinated directly produced 20 mutant colonies. The remaining 13 spores, characterized by delayed germination (5 days), produced as many as 8 mutant colonies and in addition the 2 diploids. The great majority of all mutant colonies, as well as a proportion (29) of the colonies which first appeared to be normal, continued to produce mutated sectors.

DNA treatment thus produces mosaicism and prolonged instability of the genetic material.

* Prepared by W.F.F. Oppenoorth.

A.J. CLUTTERBUCK.

A fawn conidia mutant in Aspergillus nidulans.

The mutant arose spontaneously in the bi1 strain of the Glasgow stocks. The light brown pigmentation of the conidia is autonomous in heterokaryons and recessive in diploids. It is unaffected by any common variables in growth conditions, and is hypostatic to white, but epistatic to yellow, and wholly or partially epistatic to chartreuse.

The mutant, designated "fw", has been located on chromosome VIII by haploidisation, and the original mutant strain shows no evidence of translocations. Meiotically, it is situated approximately 25 units from orn7, and is unlinked to arg3. In the course of further mapping, fw has been used to establish the position of co relative to orn7 and has also been found to be linked, at a distance of 13 units, to a morphological marker found in bi1 y, and believed to be ve^+ (Käfer ANL 3). The resulting map of the left arm of chromosome VIII is as follows.

ve ——— fw ——— orn7 — co ——————— arg3

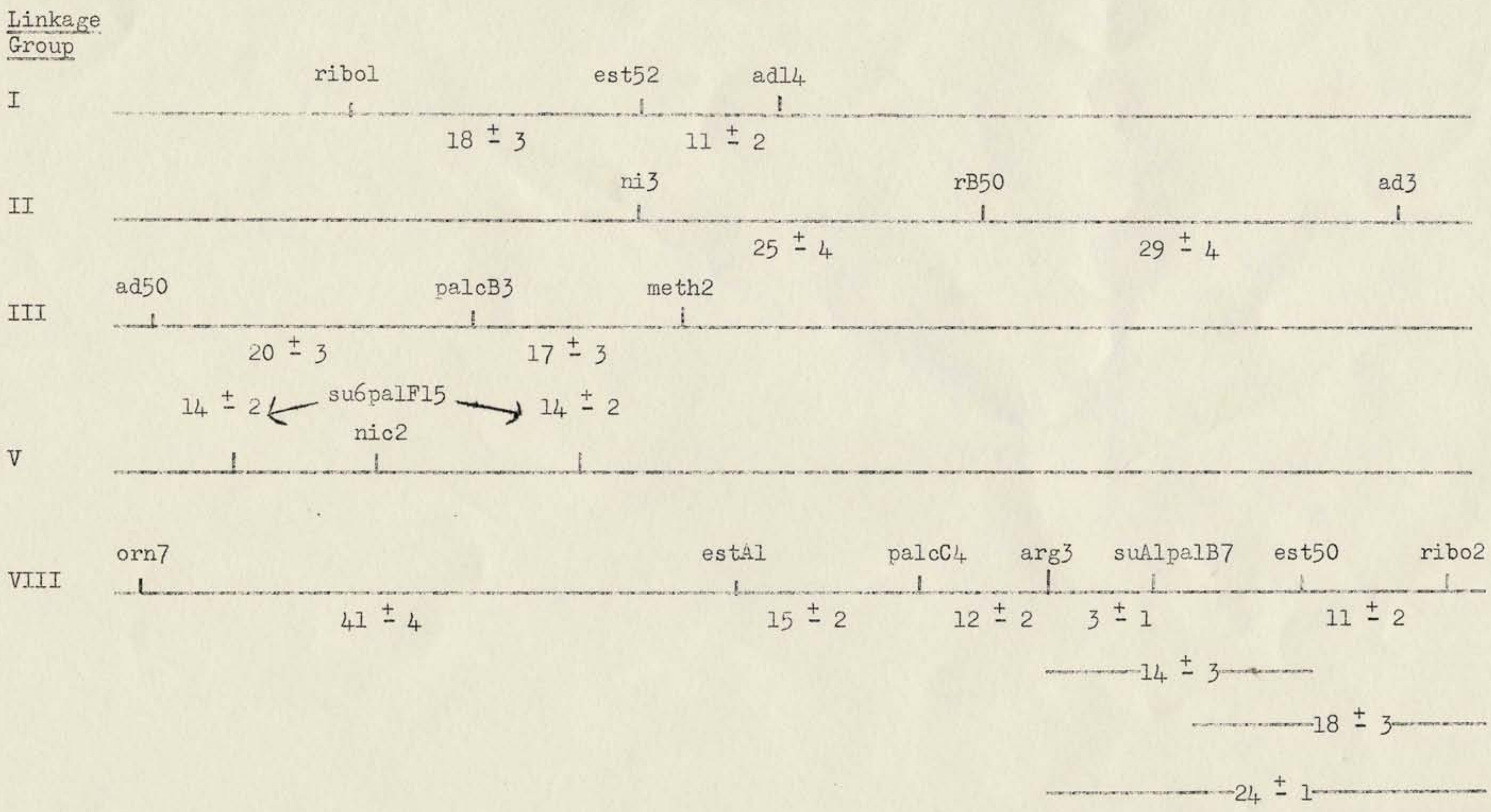
13 ——— 25 ——— 0.5

Supplementary list of located or partially located mutants in *A. nidulans*.

Locus symbol	Phenotype	Linkage group	Strain	Mode	Year	Reference
ad50	adenine	IIIL	bil	UV	1964	Rivera
r 50	enhanced ability to split α -napthyl-phosphate at pH 4.8.	IIR	bil	UV	1965	Dorn
r 51	enhanced ability to split α -napthyl-phosphate at pH 4.8.	VIII	bil	UV	1965	Dorn
suA1palB7	partially restores alkaline phosphatase activity (pH 8.2) in the palB7 mutant.	VIII	bil; palB7	spont.	1962	Dorn
su6palF15	partially restores alkaline phosphatase activity of the palF15 mutant.	V	bil; palF15	spont.	1963	Dorn
palcB3	reduced ability to split α -napthyl phosphate at both pH 8.2 and 4.8.	IIIL	bil	UV	1962	Dorn
palcC4	reduced ability to split α -napthyl-phosphate at both pH 8.2 and 4.8.	VIII	bil	UV	1962	Dorn
estA1	reduced ability to split α -napthyl-acetate at pH 6.4.	VIII	bil	UV	1964	Rivera

Locus symbol	Phenotype	Linkage group	Strain	Mode	Year	Reference
est50	enhanced ability to split α -napthyl-acetate at pH 6.4.	VIII	bil	UV	1964	Rivera
est52	enhanced ability to split α -napthyl-acetate at pH 6.4.	IL	bil	UV	1964	Rivera

RECENT MEIOTIC LINKAGES*



* Irrelevant chromosomes have been omitted.

JEAN M. FOLEY, N.H. GILES, AND C.F. ROBERTS.

Suggested modifications of techniques.

(1) The standard procedure for the synthesis of heterokaryons becomes laborious when large numbers of combinations have to be done. We find the following modified procedure rapid and efficient. Loops of conidia, taken from slopes or dense suspensions, are mixed in small drops (<.05 ml) of Pritchard's CA medium or the surface of thick plates of fresh MM supplemented for the mutants under test. The plates are incubated for 5-7 days when heterokaryons are obtained as vigorous outgrowths in about 75% of the cases. If there is no heterokaryon, blocks of agar are taken from the background growth in the original spots and the mycelium and conidia dug into fresh plates. The second transfer also succeeds in about 75% of the cases.

We have found this technique very effective for inter-genic complementation tests with adenine, histidine, and tryptophane mutants. We agree with Pateman's group that heterokaryon tests are sufficient for this purpose and that the isolation of diploids is unnecessary.

In the case of inter-allelic complementation among adenylosuccinase mutants we have found that the technique works well if the adenine is increased from the usual 100 to 500 μg/ml.

(2) The following procedure was found very effective in increasing the recovery of haploid segregants after p-Fφ al-induced mitotic haploidisation. Suspensions of conidia of the heterozygous diploid (master strain/mutant) are stabbed into thick plates of CA + pFφ al and incubated 2-3 weeks. The poorly conidiated growth on these plates is replicated with velveteen to CA, and after incubation many discrete white, yellow or green sectors are readily visible.

C.F. ROBERTS.

Failure of the membrane technique for growth of mycelium on solid media.

This technique (Roberts, Genetical Research 5, 211, 1964) was suggested as a way of growing heterokaryons for the extraction of enzymes. Subsequent work with adenylosuccinase mutants has shown that the technique may not be suitable in the case of inter-allelic complementation (see Table). The enzyme could be obtained from a complementing diploid grown overnight in liquid culture, but not from the same diploid grown between cellulose membranes on solid medium for 5 days. It is evident that the difference in physiological conditions places severe restrictions on the amount of complementation enzyme formed. However, the good recovery of enzyme from the other heterokaryons and diploids indicates that the technique may be useful for inter-genic studies.

ADENYLOSUCCINASE ACTIVITY IN HETEROKARYONS AND

HETEROZYGOUS DIPLOIDS

Genotype	Grown solid medium		Grown liquid culture
	HK	Diploid	Diploid
+/+	2.10	1.52	2.4-3.2
+/74	1.47	0.56	--
56/+	1.44	0.57	0.28
56/74 (complementary)	<.10	<.10	0.84
+/74	0.88	0.59	--
3/+	1.67	0.64	1.00
3/74 (non-complementary)	<.10	<.10	<.10

Spp activity = OD 280 mμ/10 min/mg P.

A.T. BULL.

The Melanin of *A. nidulans*.

A notable effect of 8-azaguanine treatment was a disturbance of mycelial pigment synthesis. Wild-type strains had dark brown-black pigmentation and among the mutants were pink, purple-brown and hyaline variants. Analysis of these pigments indicates that the wild-type produces a melanin (sensu "a dark pigment produced by a tyrosinase acting upon a phenolic substrate with the utilisation of oxygen"). The nitrogen content of this melanin is $7.3 \pm 0.5\%$ and degradation by alkali fusion and permanganate oxidation yields indole and pyrrole products. The pink pigment autoxidises under alkaline conditions to give an increasingly purple solution; eventually melanin precipitates. UV Spectra of these pink and purple pigments were indicative of the melanin intermediates dopachrome and melanochrome. This is believed to be the first established case of an indole-melanin in fungi. Enzyme studies have supported this conclusion.

A.T. BULL.

Wild-type and Mutant Tyrosinases.

Wild-type tyrosinase exhibits dual activity; *o*-hydroxylation of monophenols (tyrosine) and oxidation of *o*-dihydric phenols (DOPA) to corresponding quinones, with the subsequent formation of melanin. Activation (high and low temps.; hydrogen and hydroxyl ions; surface activating agents) and inhibition (cyanide) studies strongly suggest two distinct active sites for this enzyme. Of special interest is the tyrosinase of a colourless mutant which, while retaining the capacity to carry out the oxidation of diphenols, has lost the ability to *o*-hydroxylate monophenols. This latter feature is not due to the presence of an endogenous inhibitor, or, to the absence of a co-factor. Kinetic and electrophoretic studies are planned to test whether the two activities are properties of a single protein, or of two proteins, and to examine the nature of the mutant enzyme.

A.T. BULL & A. WESTBROOK.

A study of the significance of tyrosinase in terminal respiration of wild-type *A. nidulans* has been initiated. Endogenous respiration is influenced by the presence of tyrosinase substrates and inhibitors (e.g., Na diethyl dithiocarbamate). Indications are that tyrosinase becomes important in respiration at a certain stage in development and it is noteworthy that certain TCA cycle inhibitors and uncoupling agents are inducers of this enzyme. Furthermore, the end product of tyrosinase activity, melanin, constitutes up to 17% of the dry weight of old mycelia.

These studies form part of the wider investigation concerned with the metabolic bases of asexual and sexual spore development.

A. PUTRAMENT

Prolonged appearance of pABA-independent recombinants in heteroallelic diploids paba2/paba18.

The phenomenon of prolonged (or delayed) appearance of intragenic mitotic recombinants observed previously by Luzatti et al. in yeast and by the present author in ad9/ad15 diploids of A. nidulans (Aspergillus Newsletter, No. 5, 1964) occurs also in some paba2/paba 18 diploids. There is some indication that the ability to recombine could be in some way connected with the structure of chromosome and/or the character of outside markers. During 6 days after plating conidia of diploids

    + pro1 ad9 + paba18 y + +
- -
pro2 + + paba2 + + bi1 phen2

and

pro1 ad9 + + paba18 y + +
- -
  + + ad15 paba2 + + bi1 phen2

give rise to about 40 recombinant colonies per 10^6 conidia plated. Whereas diploids

pro2 ad17 paba2 + y + + +
- -
pro2 + + paba18 + bi1 phen2 lys5

and

  + ad9 paba2 + y + + +
- -
pro2 + + paba18 + bi1 phen2 lys5

give rise to about 2 recombinant colonies per 10^6 conidia plated.

Conidia of all diploids were washed in saline, and plated on minimal medium supplemented with proline, adenine and biotine. The density of plating was 1×10^5 to 4×10^5 conidia per plate.

CLARA CALVORI AND GIORGIO MORPURGO.

On the nature of U.V. and HNO_2 induced mutations in Aspergillus nidulans.

This research was planned to investigate the nature of spontaneous and induced mutations in a chromosomal organism, namely *Aspergillus nidulans*.

A system to efficiently select forward and back mutations, developed in our laboratory, was used. The mutants were selected on the basis of their resistance to parafluorophenylalanine (PFP) and were easely selected on a medium supplemented with PFP. All PFP resistant mutants map in the same cistron. Back mutations are selected on a medium supplemented with aminotyrosine and phenylanthranilic acid. On such a medium only the wild-type and the back mutants can develop,the PFP resistant mutants being totally incapable of growth.

A rapid method permits to distinguish among true back mutations and external suppressors.

15 U.V. and 14 HNO_2 induced mutations have been tested in order to examine the rate of spontaneous, U.V. and HNO_2 induced back mutations.

The pattern of spontaneous and induced reverse mutations is quite different in the case of U.V. and HNO_2 induced mutations, and the general conclusion can be reached that at the molecular level U.V. and HNO_2 induced mutations are different.

Most of the U.V. induced mutations are back mutable by U.V. and not by HNO_2. Moreover a big majority is suppressible both by U.V. and HNO_2.

HNO_2 induced mutations are generally not inducible to back mutate through the action of both U.V. and HNO_2.

Among the HNO_2 induced mutations two have been found to exibit a remarkable pattern of retromutation. The rate of retromutation is some ten times higher that the rate of the forward mutation in the whole cistron which determines the PFP resistance.

The rate of retromutation is not enhanced by treatment with U.V., HNO_2 or nitrogen mustard. The presence of mutator gene has been excluded.

The behaviour of these mutants is inexplainable on the basis of the simple models of Freese.

This research was partially supported by a grant from Consiglio Nazionale delle Ricerche.

ETTA KÄFER-BOOTHROYD.

Tests for Translocations by Mitotic Linkage in Heterozygous Diploids

Over the years it has become apparent that a few translocations are present in many Aspergillus strains. Mitotic recombination has been used to detect these and has permitted a complete tracing and, therefore, elimination of all translocations from common stocks. Certified, translocation free strains have been back crossed to various extents and are available at the Fungal Genetics Stock Center (FGSC, Dartmouth College, Hanover, N.H., U.S.A.)

Since the list of Aspergillus strains deposited at the Fungal Genetic Stock Center is now being published (BARRATT, JOHNSON and OGATA, 1965; Genetics, in press) it has been decided to make available and publish as a companion paper the more relevant information on translocations in Aspergillus stock strains. (KÄFER, 1965; Genetics, in press). As the details of the tests for translocations, like genotype of test diploid, number of haploids tested, segregation of markers on chromosomes not involved in aberrations etc., are only of interest to workers in the field, most of the Tables have been excluded from the main paper and are given here. To test for translocations two 8th generation back cross strains have been used as "standard" reference strains (Table 1, Nos. M 635 and 640). The "tester strains" were checked against these or are descendants of translocation free tested strains (see Table 1 for genotypes and stock numbers). All tester strains are available at the FGSC and the origin is indicated in Figs. 1 and 2 of BARRATT et al. (1965).

The tested strains have been arranged in six pedigree charts containing their strains or crosses of origin (Figs. 2-7 KÄFER, 1965). For identification of the crosses the cross-symbols and numbers in use in Montreal have been given in the Figures and Tables (capital letters indicate crosses carried out by Pontecorvo, Roper and co-workers, who kindly provided a large number of the tested strains). The details of the tests are presented in condensed form in the corresponding Tables (Table 2 in KÄFER 1965, and Tables 3-7 given here). Reprints of the two papers appearing in "Genetics" will be sent to all those obtaining ANL as soon as they are available (photographic copies of the figures are now available on request).

TABLE 1: Genotype, origin and stock numbers of tester strains.

Stock No. M**	FGSC	Genotype – Linkage groups I	II	III	IV	V	VI	VII	VIII	Cross of origin No	see Fig.
a) Recombinants from translocation-free crosses											
143	160	su pro paba ad20	Acr w2	---	pyro4	lys5	s3	nic8	---	63	6
300	163	su y ad20	Acr	phen2	pyro4	lys5	s3	nic8	co		
301	104	y ad20	Acr	phen2	pyro4	lys5	s3	nic8	co		
302	103	su y ad20	Acr	phen2	pyro4	lys5	s3	--	co	282	4
664	197	su paba y ad20 bi	Acr	---	pyro4	lys5	s3	nic8	co		
767	132	ad20 bi	Acr	phen2	pyro4	--	s3	cho	---	365	--
1058	178	su paba y ad20	Acr	---	---	lys5	--	--	cha		
915	70	su ad20 bi	Acr	phen2	pyro4	lys5	s3	nic8	cha		
935	198	ad20 bi	---	---	---	--	s3	nic8	ribo2	434	[2]
1061	180	ad20 bi	---	phen2	---	--	s3	--	cha		
881	169	---	Acr	---	pyro4	--	s3	nic8	ribo2		
947	173	su paba y ad20	---	phen2	---	--	--	nic8	---	461	[2]
1040	176	y ad20	---	phen2	---	lys5	s3	nic8	ribo2		
1041	177	y ad20	Acr	phen2	---	lys5	s3	nic8	ribo2(ve+)		
913	207	su paba y ad20	Acr	---	pyro4	p2	s3	nic8	ribo2		
914	171	su paba y ad20	Acr	phen2	---	p2	s3	nic8	ribo2	468	[2]
925	145	y ad20	---	phen2	pyro4	p2	s3	--	ribo2		
946	172	su ad14 bi	---	phen2	---	p2	--	nic8	ribo2		
794	167	su y ad20	Acr	---	---	lys5	s3	cho	co ---		
857	44	su ad20 bi	Acr	phen2	pyro4	lys5	s3	cho	co cha		
878	168	su ad20 bi	---	---	---	--	s3	cho	cha		
883	170	su paba y ad20 bi	w2	phen2	pyro4	lys5	s3	cho	---		
956	174	su paba y ad20	Acr	phen2	pyro4	--	s3	cho	cha		
1025	175	su paba y ad20	w2	phen2	---	lys5	s3	cho	co cha	474	[2]
1026	199	su ad20 bi	Acr	---	pyro4	lys5	s3	cho	cha		
1056	155	su paba y ad20	Acr	phen2	pyro4	lys5	s3	cho	co cha		
1059	179	su ad20	Acr	phen2	---	lys5	s3	cho	---		
1062	181	su paba y ad20	---	---	pyro4	lys5	s3	--	---		
1064	182	su paba y ad20	---	phen2	---	lys5	--	cho	---		
635	17	ad14	---	---	---	--	--	--	(ve)	BC	[1]
640	18	ad14 y	---	---	---	--	--	--	(ve)	VIII	
911	68	su y ad20	Acr	phen2	pyro4	lys5	s3	nic8	ribo2	000	--
173	161	ad20	Acr w2	phen2	pyro4	lys5	s3	nic8	---	142	--
179	162	su ad20	---	---	pyro4	lys5	s3	nic8	---	214	--
391	75	su ad20 bi	w2	---	---	--	--	cho	cha	362	2
1043	154	ad20 bi	w2 ni3	so	meth	nic2	lac	cho	cha	500	--
b) Tested strains without T(VI;VII) from crosses heterozygous for T(VI;VII)											
276	105	bi	Acr w3	phen2	pyro4	lys5	s3	nic8	co	280	4
330	98	su paba y ad20	Acr w3	---	pyro4	lys5	s3	nic8	ribo2	283	4
1295	159	su paba y ad20	Acr	phen2	pyro4	lys5	s3	nic8	ribo2		
626	79	su paba y ad20	ni3	s0	pyro4	nic2	s3	cho	cha	400	4
c) Tested strains with T(VI;VII) from crosses heterozygous for T(VI;VII)											
275	131	bi	Acr w3	phen2	pyro4	lys5	s3	nic8	ribo2	280	4
721	166	bi	Acr w3	---	pyro4	lys5	s3	nic8	co		
676	165	paba y ad20	Acr	phen2	---	lys5	s3	--	ribo2	420	4
d)* Lost strain, likely without translocations											
250*	--	y ad20	Acr w3	---	---	--	--	--	ribo2	165	--

* Strain lost, not tested, parents without translocations

** Stock number in use at McGill University, Montreal

TABLE 3

Test for deviations from random segregation of markers of different linkage groups in mitotic haploids selected from heterozygous test diploids containing strains of Fig.3 (allele number 1 omitted in all cases).

Tested strain: Origin	Tested strain: Genotype	Stock no. M**	Tester-strain stock no. M**	Unmarked linkage groups in test diploid	No. of selected haploids	Complete linkage trans-location $\chi^2 > 10$	No. of χ^2 tests not influenced by viability or selection P: > .05	.05-.01	<.01
Induced and spontaneous mutants									
spontaneous in paba bi	paba bi; w3	805	300	-	31	T(VI;VII)	20	1	-
			911	-	26		15	2	-
	paba bi; co	609	911	-	25	T(VI;VII)	20	1	-
spontaneous in paba y; co	paba y; Acr; co	822	878	III,IV,V	16	T(VI;VII)	4	-	1
UV of bi;Acr w3	bi; Acr w3; ribo2	149	300		22	no T	26	1	1
Recombinants									
c†	paba y; co	872	915	-	7	T(VI;VII)	21	-	-
b	y; Acr ad3; co	908	915	-	21	T(VI;VII)	15	-	1
			1043	-	44		19	1	-
y	an pro bi; w3; s0; pyro4	45	914	-	58	no T	26	1	1
KKK	su ribo pro ad20 bi; Acr	846	1041	II,IV	37	no T	12	1	-
KK	su paba y ad20; Acr	66	1043	-	45	no T	27	-	1
	su ribo paba y ad20; Acr w2	59	301	-	65	no T	26	1	-
38	pro bi; Acr; pyro4	1148	914	II	35	no T	20	-	1
	an pro bi; Acr; s0; pyro4	43	914	II	22	no T	21	-	-
	pro bi; Acr ad3	1157	956	V	16	no T	17	1	1
			1056		8		19	1	-
40	ribo y bi; Acr; phen2; s3; nic8	889	635	IV,V,VIII	11	no T	10	-	-
51	pro bi; Acr; meth pyro4	41	626	-	39	no T	25	-	1
234	su pro paba y ad20; Acr	181	1043	-	37	no T	20	2	1
235	ad20 bi; w3; meth pyro4	180	914	-	73	no T	26	1	-
							389	14	9

** Stock number in use at McGill University, Montreal, P.Q.

† Cross symbol or number in use at McGill University, Montreal, P.Q.

TABLE 4.

Test for deviation from random segregation of markers of different linkage groups in mitotic haploids selected from heterozygous test diploids containing strains of Fig. 4 (allele number 1 omitted in all cases).

Tested strain: Origin	Tested strain: Genotype	Tested strain: Stock no. M**	Tester-strain stock no. M**	Unmarked linkage groups in test diploid	No. of selected haploids	Complete linkage translocation P << .01	No. of χ^2 - tests not influenced by viability or selection P: > .05	.05-.01	< .01
232[†]	paba y; Acr w3; co ribo2	158	915	-	25	T(VI;VII)	20	-	-
	bi; Acr w3; co ribo2	157	626	-	13	T(VI;VII)	21	-	-
	paba y; Acr; co ribo2	159	1043	-	38	T(VI;VII)	19	2	-
280	paba y; Acr; phen2; ribo2	716	721[‡]	-	14	T(VI;VII)[‡]	25	3	-
	bi; Acr w3; pyro4; lys5; s3; nic8; co	721	640	III	24	T(VI;VII)	12	-	-
	bi; Acr w3; phen2; pyro4; lys5; s3; nic8; co	276	-[*]	-		no T			
	bi; Acr w3; phen2; pyro4; lys5; s3; nic8; ribo2	275	640	-	14	T(VI;VII)	17	-	1
281	su y ad20; Acr; ribo2	424	1043	-	42	T(VI;VII)	21	-	-
	paba y ad20; Acr; co	286	275[‡]	-	26	no T	21	-	-
283	su paba y ad20; Acr; phen2; pyro4; lys5; s3; nic8; ribo2	1295	635	-	59	no T	27	-	-
	su y ad20; Acr; lys5; nic8; ribo2	333	767	II	41	T(VI;VII)	14	2	1
			1061	IV	38		17	2	-
352	su pro y ad20; Acr; cho; cha	416	925	-	19	no T	27	-	-
	su ribo pro paba y ad20; nic8 cho; cha	367	676[‡]	IV	65	T(VI;VII)[‡]	19	1	-
400	su paba y ad20; ni3; s0; pyro4; nic2; s3; cho; cha	626	635	-	87	no T	20	2	1
	ad20 bi; Acr;phen2; pyro4;lys5;s3;cho nic8;ribo2; cha[*]	627	640	-	17	T(VI;VII)	21	-	-
420	paba y ad20; Acr; phen2; pyro4; lys5; s3	671	391	-	42	no T	20	-	-
	paba y ad20; Acr; phen2; lys5; s3; ribo2	676	391	-	36	T(VI;VII)	13	1	-
380	pro y ad20; Acr; phen2 Su4pro s0; cho; cha	447	143	-	33	no T	20	2	-
381	su pro paba ad20; Acr; pyro4; lys5; nic8; ribo2	446	1061	-	13	T(VI;VII)	20	1	-
382	su pro y ad20; Acr; phen2 s0; pyro4; lys5; cho; cha	520	935	-	18	T(VI;VII)	18	-	-
	pro paba ad20; Acr;phen2 Su4pro s0; cho nic8;ribo2 cha	457	1062	-	45	no T	26	1	-

* Tester strain and original data lost.

† Cross number

** Stock numbers in use at McGill University, Montreal, P.Q.

‡ Tester strain containing T(VI;VII); diploids homozygous for T(VI;VII) show no linkage [between] _s3_ and _nic8_ or _cho_.

TABLE 5

Test for deviation from random segregation of markers of different linkage groups in mitotic haploids selected from heterozygous test diploids containing strains of Fig. 5 (allele number 1 omitted in all cases).

Tested strain									
Origin	Genotype	Stock no. M**	Tester-strain stock no. M**	Unmarked linkage groups in test diploid	No. of selected haploids	Complete linkage; trans-location P << .01	No. of χ^2-values not influenced by viability or selection P: > .05	.05-.01	< .01
Induced mutants									
UV of bi; w3	bi; ad23 w3	858	1295	-	32	T(III;VIII) and T(VI;VII)	20	-	-
	bi; w3 pu	831	1295 626	- -	66 49	T(I;III;VIII) and T(VI;VII)	§		
Recombinants									
UU†	pro paba y; ad23 w3 (ve^+)	1151	915	-	50	no T‡	18	5	1
AA	bi; w3 pu ad	56	1295	-	15	T(VI;VII)	20	-	-
	w3 pu (ve^+)	853	626	-	27	T(VI;VII)	21	-	-
	pu ad	57	1056	-	12	T(VI;VII)	19	1	-
70	w3; meth pyro4	106	914	-	53	T(VI;VII)	27	-	-
	Acr w3; meth	182	1055 1056	-	31	T(VI;VII)	24	-	1
	pro paba y bi; pyro4	183	1043	-	46	no T	24	2	
50	ribo bi; w3 thi4 ni3	23	1056	-	28	no T	25	3	-
111	su paba y ad20; "cys2"*	128	25(*	IV,V,VI,VII	64	T(III;VIII)	(	-	-
			173	VIII	33	and T(VI;VII)‡	17	2	-
310	su paba y ad20; "cys2";cha	341	767	V	30	T(VI;VII)	15	-	-
337	paba y ad20; "cys2"; co ribo2 cha*	346	179	II	38	T(VI;VII)	18	-	1
351	su paba y ad20; pu; "cys2"; cha	357	767	V	36	T(VI;VII)	14	-	-
							262	13	3

* Strain lost

† Cross symbol or number

‡ Details in text, Käfer 1965

** Stocknumber in use at McGill University, Montreal

§ Complete results in Table 1, Käfer 1965

TABLE 6

Test for deviation from random segregation of markers of different linkage groups in mitotic haploids selected from heterozygous test diploids containing strains of Fig. 6 (allele number 1 omitted in all cases

Tested strain							No. of χ^2-values not influenced by viability or selection		
Origin	Genotype	Stock no. M**	Tester-strain stock no. M**	Unmarked linkage groups in test diploid	No. of selected haploids	Complete linkage; trans-location P << .01	P: > .05	.05-.01	< .01
Induced mutant									
UV of bi	bi; sm; lys5	905	626	-	31	no T‡	26	1	-
Recombinants									
2†	ribo ad14 y; phen2	962	1026	-	22	T(I;VIII)	24	3	-
6	bi; phen2; lys5	961	626	-	25	T(I;VIII)	26	1	-
	ribo ad14; bi; sm	1155	913	-	47	T(I;VIII)	26	-	-
18	bi; w2; pyro4 lys5; s3	888	947	VIII	14	T(I;VIII) §	20	-	1
	an bi; w2; phen2; pyro4; s3	136	1058	VII	49	no T	14	4	-
62	bi; Acr; phen2; pyro4; lys5; s3;nic8	65	640	-	44	no T	25	-	1
	ribo bi; Acr; pyro4; s3; nic8	78	640	III,V	15	T(I;VIII)	14	-	-
66	pro paba y; Acr; phen2	77	1026	II	37	T(I;VIII)	19	-	-
437	ribo bi; Acr; (ve⁺)	706	1295	II	40	T(I;VIII)	20	-	-
			300	II	54	T(I;III;VIII)‡	18	-	-
							232	9	2

† = Cross numbers

‡ = Details in text, Käfer 1965.

** = Stock number in use at McGill University, Montreal, P.Q.

§ = T(I;VIII) deduced from absence of yellow diploid segregants

TABLE 7

Test for deviation from random segregation of markers of different linkage groups in mitotic haploids selected from heterozygous test diploids containing strains of Fig. 7 (allele number 1 omitted in all cases).

Tested strain: Origin	Tested strain: Genotype	Stock no. M**	Tester-strain stock no. M**	Unmarked linkage groups in test diploid	No. of selected haploids	Complete linkage; trans-location P << .01	No. of χ^2-values not influenced by viability or selection P: > .05	.05-.01	< .01
Recombinants									
383†	pro paba ad20; Acr thi; phen2 Su4pro s0 panto	482	664	-	41	T(III;VIII)	24	-	-
	ad20; Acr thi; (±Su4pro) panto; nic8 cho; ribo2 (ve+)	491	302	-	39	T(III;VII;VIII)	18	-	-
	pro paba ad20; thi; phen2 Su4pro; cho; ribo2 cha *	496	664	-	52	no T‡	26	1	-
	pro paba ad20; phen2 Su4pro s0; ribo2 cha*	498	664	-	66	no T	21	3	-
39	su ad20 bi; panto; pyro4	165	1049		17	T(III;VII;VIII)‡	13	-	-
			1041	-	21		21	2	-
240	(±su) pro paba y; panto	267	883	-	7	T(III;VII;VIII)	15	3	-
	su ad20 bi; panto	405	301	-	49	T(III;VII;VIII)	13	1	-
243	pro bi; phen2 panto	347	330	-	56	T(III;VII;VIII)	14	-	1
	pro paba y; Acr; phen2 Su4pro s0	345	721	-	30	no T	22	-	-
248	an bi; w2; pyro4; lys s3	348	1064	VIII	27	no T	15	2	-
350	an bi; phen2 panto; lys s3	363	330	-	39	T(III;VIII)	18	1	-
							220	13	1

† = Cross numbers

‡ = Details in text, Käfer 1965

** = Stock number in use at McGill University, Montreal, P.Q.

P.C. MCMAHON.

Cytochrome patterns in A. nidulans.

Some abnormal strains of A. nidulans were found to have cytochrome absorption bands of greater intensity than their normal counterparts. Most of the strains examined were mycelial in type (Roper, 1958). Where a strain exhibited a high cytochrome c value, cytochromes b and a were also high in value. Unlike the petites in yeast, no strains were found, either normal or abnormal, where one of the cytochrome bands was absent.

The majority of normal strains had cytochrome c values between 100 and 200 (arbitrary cytochrome units) and abnormal strains between 300 - 450. Some normal and abnormal strains overlapped in the 200 - 300 region. Approximately 25% of abnormals (of the fluffy variety), induced by U.V., had high cytochrome values. Cytochrome absorption readings varied during the growth of a culture, being highest during the early log phase and lowest towards the end of growth.

So far only one cross has been examined. The abnormal parent had a cytochrome c value of 338.6 ± 39.6 (i.e. $\pm$ 2 std. dev.) and the normal parent 139.2 ± 61.5. The morphological abnormality segrated in a 1 : 1 ratio. The cytochrome c values for the abnormal segregants ranged from 176 to 386 and the normal segregants ranged from 82 to 281. Approximately half of these abnormal segregants fell within the range of the abnormal parent and half of the normal segregants fell within the range of the normal parent. The remaining halves of the abnormal and normal segregants overlapped one another in the 180 - 300 range. Exactly the same pattern was obtained for cytochromes b and a.

Roper, J.A. Cold Spring Harbour Symp. on Quant. Biol XXIII, 1958.