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J.L. AZEVEDO, Department of Genetics, The University, Sheffield S10 2TN, England.

A Pleiotrophic Effect of Lysine Mutants of *Aspergillus nidulans*

Strains of *A. nidulans* without *bi* markers have a requirement for biotin (Strigini and Morpurgo, A.N.L. No.2 and Nature 190 (1960) : 557). Such requirement is generally not noticed since chemicals and glassware probably carry biotin as contaminant. Roper (A.N.L. No. 6) has also reported that at 42°C the biotin requirement is very exacting mainly in relation to diploid strains. In addition, we have found that *lys1* and *lys5* mutants (Strains used; MSD and FGSC ≠ 178, FGSC ≠ 179 and FGSC ≠ 38 kindly supplied by Dr. Ogata) have at 37°C a requirement for biotin which is very exacting when no more than 10^4 conidia are spread onto MM without biotin In the same conditions strains MSE and *pro1, paba6, y; w3* used as controls did not require biotin. Full growth is restored at 25°C without biotin or at 37°C on MM + biotin. Mitotic and meiotic analysis have shown that the biotin requirement is always associated to lysine deficiency. It seems therefore that *lys1* and *lys5* markers besides the expected requirement for lysine also determine (at 37°C but not at 25°C) a more pronounced requirement for biotin than that normally found in *A. nidulans* strains.

C.E. CATEN, Department of Genetics, University of Birmingham, Birmingham 15, England.

Cleistothecial Colour Mutants in Aspergillus amstelodami

The cleistothecia of *Aspergillus amstelodami* are bright yellow in colour due to pigmentation of the wall, and on media encouraging sexual reproduction give the colonies a characteristic yellow colour. Following UV irradiation mutants with white (pigmentless) and red/orange cleistothecia were isolated. Crosses between the mutants and wild type, indicated that each differs from wild type at a single mutant site, designated *wc1* and *rc1* for white and redcleistothecia respectively. These two mutant sites are very closely linked (to date no wild type recombinant have been recovered in over 1000 progeny from repulsion crosses), but show no linkage or epistasis with the conidial colour loci, white (*whi*) and brown (*brw*).

Heterokaryons between each mutant and wild type and between the two mutants produce a mosaic of the parental and intermediate cleistothecia, indicating that cleistothecial colour is determined autonomously. As a result it is possible to use the mutants to visibly select for hybrid cleistothecia in heterokaryons and mixtures. The selection achieved is not complete, but gives a significant enrichment.

The autonomous determination of cleistothecial colour necessitated the use of diploids to investigate the dominance and complementation properties of the mutants. In diploids, wc1 and rc1 are recessive and dominant to their wild type alleles, respectively. As might be anticipated from this result, the trans heterozygote $\frac{wc1\ rc1^+}{wc1^+\ rc1}$ has red and not wild type cleistothecia. Attempts are presently being made to isolate the double mutant by irradiation of the $wc1^+\ rc1$ strain, with a view to determining the phenotype of the cis heterozygote.

A.J. CLUTTERBUCK, Institute of Genetics, The University, Glasgow W.2, Scotland.

Mitotic recombination in linkage group II of A. nidulans

Three diploids have been constructed from multiply marked strains suitable for analysis of linkage group II by mitotic recombination and selection of recombinants by the spore colour markers ygA6 (yellow-green) and $wA7^+$ (leaky white, allowing distinction of wA7 ygA6 from wA7 yg^+).

Diploid A: biA1; AcrA1 wA7 riboE6 thiA4 abA1 cnxE16 ygA6 + pantoB100 (G711)

Diploid B: biA1; AcrA1 wA7 palcA1 riboE6 thiA4 abA1 cnxE16 ygA6 (G238) + pantoB100 (G711)

Diploid C: AcrA1 wA7 palcA1 riboE6 thiA4 ygA6 + biA1 (G051)

These diploids were plated at low density on CM + riboflavin and aminobutyric acid, and from the resulting colonies, the diploid sectors shown in the following table were picked up and classified.

	Number of sectors from diploids: A	B	C	cross-over interval		a AcrA	wA	b.c palcA	riboE	d thiA	e abA	f cnxE	g ygA
white sectors	39	13	10	a	r	-	+		+	+	+	+	+
		8	12	b	r	-	-		+	+	+	+	+
yellow-green sectors	3	9		g	r/s	+	+		+	+	+	+	-
	3	2	2	f	r/s	+	+		+	+	+	-	-
	3	1		e	r/s	+	+		+	+	-	-	-
	16	14	12	d	r/s	+	+		+	-	-	-	-
	4	9	9	c	r/s	+	+		-	-	-	-	-
exceptional sectors	x.1	0	0	a/b,c,d?	r	-			-	+	+	+	+
	y.0	1	0	a,f	r	-	+		+	+	+	-	-
	z.0	0	1	a,c	r	-	+		-	-			-

These results confirm the order of markers in linkage group II and indicate that the centromere lies between palcA and riboE. The exceptional sectors y. and z. can be explained by coincidental crossing over in both arms. Exceptional sector x. can either be explained by triple crossing over, or can be seen as evidence for a pericentric inversion or diffuse centromere in whichcase the sector could be the result of a single crossover as discussed by Käfer 1966 (ANL 7.14).

The mitotic map given by this data is summarised in the following table in which distances are given as percentages of the total recombinants for that arm.

	wA	palcA	riboE	thiA	abA	cnxE	ygA
Interval:	a	b	c	d	e	f	g
Meiotic distance (percentage recombinants):	19	18	>50	36	12	36	
Mitotic distance (% of total for arm):							
Diploids B + C	55	45					
Diploids A + B			21	46	6	9	18
Diploid C			42	50		8	
Käfer 1958			80.2		10.5		9.3

Figures for the left arm from diploids B & C are homogeneous and have been combined, similarly with the right arm figures from diploids A & B. However the latter figures do differ significantly from the right arm data from diploid C and from that given by Käfer 1958 (Adv.Genet. 9, p139). This difference is probably due to selection against diploids homozygous for abA which would lead to a relative increase in the recovery of diploids with crossing over in regions f and g.

Comparison with the meiotic map shows that as with linkage group I (Pontecorvo & Käfer Adv.Genet. 9.71) mitotic recombination is relatively frequent in regions close to the centromere.

A.J. CLUTTERBUCK

Glasgow stocks of A. nidulans

The strains in the list published in ANL.10.30 have now been tested and are stored in silica gel. The testing revealed some strains that did not correspond to their labels: these should be scored out from the stocklist. Other changes are also listed below.

Strains lost

02,017,032,037,038,039,065,0107,0120,0155,0156,0218,0221,0233, 023 6,14,17,117,120,138,157,226,31,32,36,38,41,43,44,46,51,73, 74,86,88,822,824,827,99,913.147

Additions

No.	T/F	FGSC	mutant	chromosome	origin
0259,60	T		pabaA8,9	I	UV-biA1;wA3
0262,3	T		pabaA14,17	I	UV-biA1;wA3
0264		216	adA1	I	FGSC,UV-wild-type(ve)

recombinants:

168-173	pabaA10,12,13,15,16,18 all biA1
175-185	pabaA1,5,6,9,11,12,13,15,16,18,19 all yA2 adF9
234	biA1;drkB5
236	biA1;wA1
237	biA1;wA2
238	biA1;AcrA1 wA7 palcA1 riboE6 thiA4 abA1 cnxE16 ygA6
320	yA2;ivoA1;pyroA4 (ivo = ivory = colourless conidiophores)(Clutterbuck 1969. Genetics 63.317).

Change of number (on ascertainment of recombinant origin of imported strains)

053 becomes 831, 056 becomes 832, 060 becomes 713, 061 becomes 614, 0127 becomes 833, 0218 becomes 834.

Addition of locus-letter A to unique loci

In accordance with a suggestion of Dr. E. Käfer-Boothroyd, all mutants for which allelism tests have been made should be given a locus letter, only untested mutants retaining a hyphen. This means that all unique loci now receive the letter A.

This applies to the following symbols in the stock list:

abaA14	frA1	puA1-7	wA1,2,3,7,8,9	stuA1
abA1	luA1,2	pyroA1-9	ygA6	yA2,102
biA1-4	smA1	sbA3	brlA42	
blA1-6	malA1	sucA1	IodA1	
bwA1	nirA14,63	telA1	sulA1	
choA1,2	coA1	thiA4	dilA1	
fwA1	pA2,3	wetA6	medA15	

For the following mutants however there is no record of allelism tests, so the hyphen is retained in the symbol:

ad-42 to 44	meth-4 to 7	paba-23	s-16
an-3 to 7	nic-12 to 15	pro-12	thi-2,3,5 to 12
arg-9, 10	orn-2,5,6,10,11	pyro-11,12,13	ths-3,5

Addition of FGSC number

Glasgow number	= FGSC	Glasgow number	= FGSC	Glasgow number	= FGSC	Glasgow number	= FGSC
025	261	0105	55	0242	27	612	230
080	255	0111	34	124	275	89	302
091	286	0112	272	22	239	818	273
093	60	0131	302	27	254	821	256
095	291	0140	29	28	123	93	68
096	61	0170	39	216	268	94	288
097	62	0205	3	412	220	95	283
0104	292	0210	15	414	24		

Corrections

strain 050: cha-2 becomes chaA3 (to avoid clash with Montreal chA2)
strain 0243: w-4 becomes wA9 (" " " " " w-4)
strains 0239 & 0240: tyrA7 becomes fpaA58
strain 0213 contains pyroA4 and not biA1
strain 134 contains yA2
strain 229 contains pyroA4
strain 59 contains biA1 and not yA2
strain 98 does not contain sB3
ChromosomeIII heading: phanA should read phenA
other strains: delete 91- under palA, add under phenA
Chromosome VII: other strains 22, 91- should be under nicB and not palD.

J. CYBIS & P. WEGLENSKI, Department of Genetics, University of Warsaw, Poland.

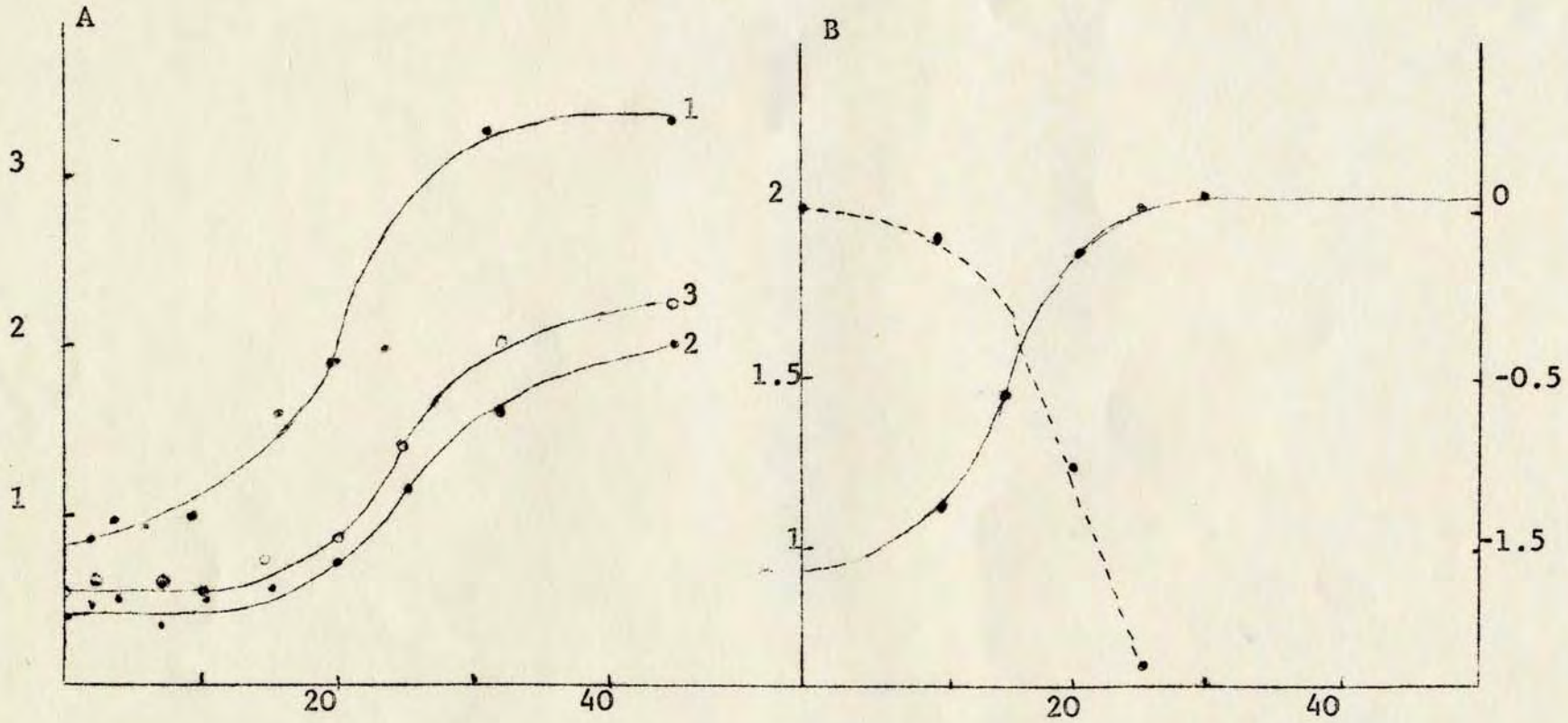
Kinetics of arginase inducation in Aspergillus nidulans

In the course of studies on the regulation of arginine metabolism in Aspergillus we have performed some experiments on the kinetics of arginase induction. We were able to determine the time necessary for the expression of the arginase gene and the half life of arginase messenger.

The strain bi-1 was cultured in a rotary shaker at 30°C for 16-18 hours in 2 l. erlenmayer flasks with 400 ml of standard minimal medium /MM/. At time „0" l-arginine HCL was added /10 ml 0.1 M/ to the culture and subsequently 2 0 ml samples were withdrawn with pipette. Mycelia were washed with water and transferred to fresh MM or MM + cycloheximide /50 μg/ ml/. Parallel samples were blotted dry and put to the deep freeze. The mycelium was kept in fresh medium for 1 h and was then collected, washed and frozen. The typical outcome of such an experiment is shown on the graph A.

Curve /1/ represents the activity of arginase in samples which were transferred to MM where the expression of produced mRNA may be completed. Curves /2/ and /3/ represent activities of the enzyme in mycelia transferred to MM + cycloheximide and frozen, respectively. Both curves are similar which indicates that cycloheximide stops the induced enzyme synthesis immediately. The observed ca 12 min lag in induction/curves 2 and 3/represents the time necessary for completion of all events leading to the expression of the arginase gene. Further experiments, now in progress, should help to determine the duration of some of them.

The half life of arginase mRNA was obtained from the experiment in which after 10 min of induction the mycelium was collected, washed with prewarmed medium, transferred to MM and sampled within 40 min for enzyme determinations. Graph B shows the arginase activity and the logarithm of the remaining synthesising ability/dashed line/plotted against time. The half life of arginase mRNA may be approximated as the time required for reduction of the remaining synthesising ability to one half of the initial value /A.Kepes, Cold Spring Harbor Symp.Quant.Biol. 28, 1963/. It is 4.7 min in the conditions employed in this experiment.


A
3
2
1
1
3
2
20
40
B
2
1.5
1
0
-0.5
-1.5
20
40

P.A.J. GORIN & D.E. EVELEIGH, National Research Council of Canada, Prairie Regional Laboratory, Saskatoon, Sask.

The Extracellular 2-Acetamido-2-Deoxy-D-Galacto-D-Galactan from *Aspergillus nidulans*

ABSTRACT: An extracellular polysaccharide from *Aspergillus nidulans* is a linear molecule containing 4-O-substituted α-D-galactopyroanosyl and 4-O-substituted 2'-acetamido-2'-deoxy-α-D-galactopyranosyl units in an approximately 2:1 ratio. A Smith degradation yielded threitol (23%), 2-O-(2'-acetamido-2'-deoxy-α-D-galactopyranosyl)-D-threitol, two hydroxyethylidene acetals in a 71% combined yield and a higher molecular weight material (5% - molar basis). The different sugar units are distributed evenly throughout the chain, consecutive N-acetyl-galactosamino-units not being detected in appreciable amounts. 3,4,6-Tri-O-methyl-D-galactose was prepared for use of its methyl glycoside as a gas liquid chromatographic standard.

(Biochemistry (U.S.)
Submitted for publication.

J.A. HOUGHTON, Department of Microbiology, University College, Galway, Ireland.

A technique for complementation tests using a new mutant of *A. nidulans*

The carbon-source utilisation (csu) mutants isolated from *A. nidulans* (Houghton, 1970a) grow slowly on glucose as sole carbon source, producing only a scanty mycelium with few conidial heads. They are unable to utilise tricarboxylic acid cycle acids due to their impaired ability to take them up across the hyphal wall (Houghton, 1970b). The mutants also displayed an unusual characteristic which could be used in complementation tests. When a mutant was grown on minimal or complete medium it never produced any perithecia. However, when two mutants of different functional groups, as determined by the standard technique of stable trans-heterokaryon synthesis, were cross-inoculated on the same plate, normal numbers of perithecia were produced. Mutants from the same group, when cross-inoculated, never formed perithecia. Newly isolated mutants could be quickly assigned to cistron by the following technique.

Conidia of the unknown strain were streaked down the vertical axis of a plate of minimal medium, conidia from representative members of the known groups were streaked in horizontal rows across the vertical streak from left to right. The plates were then sealed and incubated at 37°C for 14 days. The portions of the horizontal streaks to the left of the vertical streak were homokaryotic and did not produce perithecia. The portions of the right contained conidia from the tester strain

and from the unknown strain, carried over with the horizontal streak. The mycelia were thus intermingled and transient heterokaryotic regions could develop. If the two strains complemented, normal perithecia containing viable, parental and recombinant ascospores were produced. If the unknown and a tester strain were members of the same group they did not complement and perithecia were absent. Unknown strains which produced perithecia with all the testers were regarded as belonging to a new cistron and were tested further by standard techniques.

The 24 mutants fell into five cistrons and mitotic haploidisation revealed that these mapped on different linkage groups.

This mutant characteristic is potentially a useful genetic marker and may also be of value in complementation studies. By cross-inoculating representatives of each of the five cistrons together on one plate a complementation pattern is produced which makes good student-class demonstration material. It is intended to study this phenomenon further and in particular to investigate more thoroughly the site of breakdown in perithecial production.

Houghton, J.A.1970a. Carbon-source utilisation mutants of Aspergillus nidulans. I. Mutant isolation, growth tests and genetic analysis. In Preparation.

Houghton, J.A. 1970b. Carbon-source utilisation mutants of A. nidulans. II. Biochemistry. In Preparation.

P.LHOAS, Department of Biochemistry, Imperial College, London.

Use of heterokaryosis to infect virus-free strains of Aspergillus niger

One strain of A. foetidus and one strain of A. niger have been found recently to contain double-stranded RNA virus-like particles (G.T. Banks et al., Nature, in press). Auxotrophic and visible mutants of the A. niger strain were obtained by NTG treatment and heterokaryons with virus-free strains visibly and biochemically marked were snythesized. The virus-free parental strain recovered from the heterokaryotic heads were tested by electron microscopy for the presence of virus-like particles and several subcultures were made to test for virus replication in the new host: the concentration of virus particles was considerably higher than what would have been expected if no virus multiplication had taken place (the details will be published elsewhere).

The same method is now being used also with other strains of moulds as an introduction to the study of the genetics of the host-virus relationships.

A.M. MILLINGTON-WARD, J.A.M. REUSER & J.Y. SCHEELE,
Genetisch Laboratorium der Rijksuniversiteit,
Leiden, Nederland.

Localisation of hisB

A histidine locus (hisB) was shown (Berlyn 1967, Genetics 57: 561) to be on linkage-group I (see also Dorn 1967, Genetics 56: 619). A locus in the Leiden collection, termed hisFL was also known to be on linkage-group I (Pees per. comm.). A number of newly isolated histidine mutants have now been shown not to complement either hisB or hisFL and all three map in the same region of linkage-group I. It seems likely, therefore, that hisB, hisFL, and the new his mutants are allelic, and for uniformity with Yale, the latter are now termed hisB. From the localising results (Tables 1, 2 & 3) it is concluded that hisB is on the right arm of linkage-group I, as follows: pro-1 ←—9—→ hisB ——1——→ paba-1.

I am indebted to Dr. R. Gravel, Yale University, for providing me with the culture of Dr. Berlyn's strain: Yale No. 39, (hisB), bi-1; Acr-1; w-3; nic-8.

TABLE 1. Localization of hisB. Preliminary crosses (perithecium analyses), showing the location of hisFL. The "sample" column shows respectively number of colonies classified and number of perithecia samples in each of the four crosses. (Strains of heterogeneous background)

	Cross	Viab.%	% crossing over				Sample
			pro	his	paba	y	bi	
1	+ paba-2 y + / hisFL + + bi-1	48		4.1	15.1	4.5	1553-2
2	+ paba-2 y + + / hisFL + + bi-1 s-3	48		5.8	17.3	5.6	497-1
3	pro-1 + + + bi-1 / + hisFL paba-2 y +	49	8.7	1.1	14.3	5.9	818-2
4	+ + paba-2 y + / pro-1 hisFL + + bi-1	47	6.9	1.4	11.7	6.5	811-2

TABLE 2. Localization of hisB. Preliminary crosses (selective plating from hybrid perithecia), showing the location of hisFL. In the calculation (method of Roper, in Pontecorvo et al. 1953, Adv. Genet.) the values used were II = 15%; III = 6% (Strains of heterogeneous background)

Cross	Selected cross-over	Spores plated	Colonies	Recombination% his - paba
I II III 1 + paba-2 y + hisFL + + bi-1	I	92,000	24	using II = 1.1
	II	34,500	120	" III = 2.6
	III	46,000	28	
I II III 2 + paba-2 y + + hisFL + + bi-1 s-3	I	9n	5,960	using II = 0.89
	II	0.15n	1,670	" III = 1.18
	III	0.15n	503	

TABLE 3. Control crosses. Results from perithecium analyses. The "sample" column shows respectively number of colonies classified and hybrid perithecia samples in each cross. Compare with Tables 1 & 2. The hisB mutants in this table were newly induced (UV), either in strain paba2,y or in strain pro1,bi1. These two were crossed to each other (1st cross) to see that the markers already present segregated normally. Viability was measured from the hybrid perithecia.

Cross	Viab.%	Distance %				Sample
+ + paba y + pro + + + bi	52		12.1	16.8	6.9	979-2
		pro-1	hisB	paba-2	y	bi-1
+ his-1 paba y + pro + + + bi	52	11.2	1.2	15.1	4.3	829-2
+ his-2 paba y + pro + + + bi	50	7.9	0.7	14.1	5.4	895-2
+ his-3 paba y + pro + + + bi	48	9.1	1.2	17.1	7.5	850-2
+ his-4 paba y + pro + + + bi	44	11.4	1.1	16.8	6.7	772-2
+ his-5 paba y + pro + + + bi	53	8.4	0.7	15.3	7.4	865-2

TABLE 3 continued

pro / +	his-6 / +	+ / paba	+ / y	bi / +	53	8.8	1.7	13.5	5.2	884-2
pro / +	his-9 / +	+ / paba	+ / y	bi / +	48	8.5	1.2	12.6	2.5	446-1

MILENA BANDIERA & GIORGIO MORPURGO, Istituto Superiore di Sanita, Roma.

Mitotic Intragenic Recombination and Segregation of Distal Markers

We have previously reported[1] that mitotic recombination in the PFP gene in the left arm of the first chromosome of *Aspergillus nidulans* never produces the segregation of the distal marker su 1 ad 20 while segregation of the more proximal marker ribol occurs regularly. A point of discontinuity lying distally to ribol was therefore postulated. Some evidence was also found of the existence of a second point of discontinuity in the right arm of the same chromosome lying between pro1 and pabal. This find has been now confirmed. Intragenic recombinants were selected in the pro and paba genes in two sets of diploids of the general composition:

0---pro x + y +--- 0---paba x + y +---
+ pro y + bio + paba y + bio

and the segregation of the distal markers y and bio was studied. Among 323 pro$^+$ recombinants selected in 4 different diploids no segregants were found for the distal markers. Controls were done to exclude explanations based on retromutation. On the contrary 24 out of 64 recombinants selected in the paba gene showed segregation of the distal markers. This experiment confirms the existence of the barrier between pro 1 and paba 1.

(1) M. Bandiera, G. Morpurgo & V. Volterra (1970). Barriers in intragenic mitotic crossing-over. Mutation Res. 9, 213-217.

K.E. PAPA, LAURIE LEAICH & ERNEST CHILDS, Dept. of Plant Pathology and Plant Genetics, The University of Georgia College of Agriculture.

Mutants of *Aspergillus flavus*

Morphological and auxotrophic mutants of *Aspergillus flavus* have been recovered following exposure of conidia to ultraviolet light. The original isolate, covered from pecans by Dr. R.T. Hanlin, produces relatively high levels of aflatoxin B_1 and B_2 but no G_1 or G_2. Morphological variants include white, pale green and brown conidial mutants. Auxotrophs have been induced in all of the colour mutants, as well as in the wild-type isolate. These are summarized

as follows: methionine 10, arginine 5, lysine 4, leucine 2, serine 2, histidine 1, adenine 3, and one each for biotin, pyridoxin, p-aminobenzoic acid and thiamine.

The analysis of 39 isolates obtained following irradiation indicated a wide range of variability in aflatoxin levels. Negligible amounts of aflatoxins B_1 and B_2 were recorded in some isolates, whereas others exhibited a 30 fold increase over the original wild-type. Two isolates produced small amounts of B_1, no B_2 and small amounts of G_1. One of the isolates producing G_1 required arginine.

These and other aflatoxin variants, along with morphological auxotrophic mutants to serve as genetic markers, should aid in the elucidation of the genetics of aflatoxin production.

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ANDRZ E. J. PASZEWSKI Department of General Genetics Warsaw

The use of sodium selenate containing media for selecting mutants of *Aspergillus nidulans*.

Sodium selenate ($Na_2SeO_4 \cdot 9H_2O$) inhibits the uptake and reduction of sulphate (Shrift, Fed.Proc. 1961). 0 1mM selenate completely inhibits the growth of *Aspergillus nidulans* strain bi_1. Mutants incapable of reducing sulphate (s_1, s_3, s_{12}) are resistant to selenate (Arst, Nature 1968). These are leaky mutants and they have the same rate of growth on MM as on MM plus 2mM selenate.

Mutants resistant to selenate appear spontaneously with the frequency of $2 \cdot 10^{-6}$. All these mutants grow poorly on MM and well on MM supplemented with sulphite, thiosulphate, sulphide or cysteine. They may be divided into two groups:

a) resistant to 10mM selenate
b) resistant only to 2mM selenate.

The reversion of selenate toxicity by various sulphur amino acids was studied. Cystein, homocystein and methionine but not cystathionine could reverse selenate toxicity.

Selection has been started for mutants incapable of using methionine as a source of sulphur on medium containing selenate. They could possibly be deficient in the uptake of methionine or in the conversion of methionine to cysteine. Some mutants of this type have already been obtained.

The genetical and biochemical investigation on these mutants is in progress.

M. PIOTROWSKA, P. WEGLENSKI, Department of Genetics,
The University, Warsaw, Poland.

The Common Control of Anabolic and Catabolic Pathways of Arginine Biosynthesis in *Aspergillus nidulans*

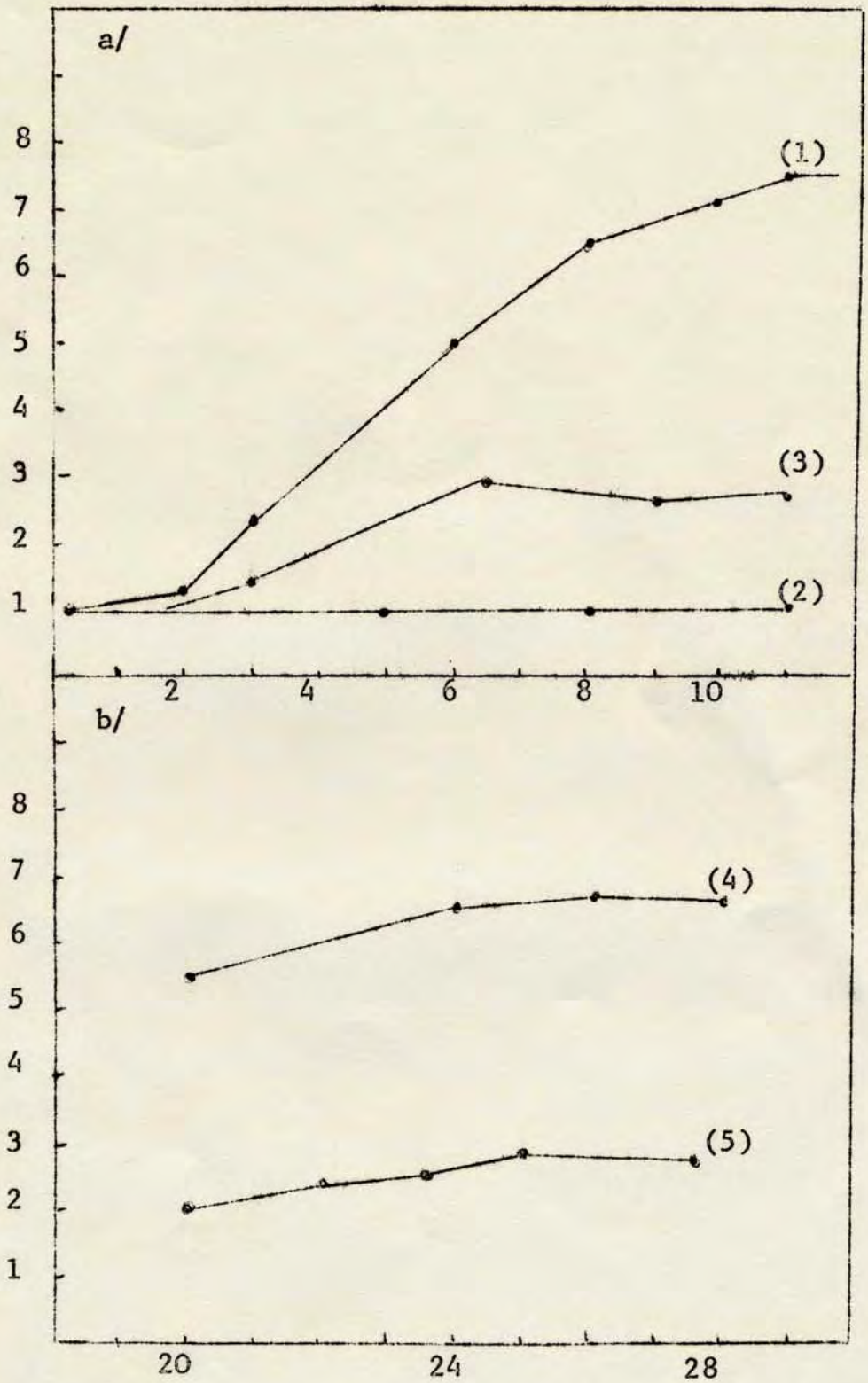
Several regulatory mutants of arginine catabolism obtained previously show the constitutive synthesis of arginase and ornithine-δ-transaminase (Weglenski 1967). They were tested for the pattern of synthesis of ornithine-δ-transcarbamylase (OTCase), an anabolic enzyme. One of them, su-19 causes OTCase synthesis to be not repressed by exogenous arginine in su-19 arg-I strain.

Another effect of su-19 was revealed when su-19 ota-I strain was constructed. Strain ota-I blocked in ornithine-δ-transaminase accumulates endogenous ornithine, which results in a high level of OTCase (to be published). With su-19 ota-I strain this effect of ornithine accumulation is not observed (see accompanying graph).

Besides su-19, mutants with constitutively synthesised arginase and ornithine-δ-transaminase so far tested, display unchanged OTCase repressibility.

References:

Weglenski, P., J. gen. Microbiol. (1967). 47, 77-85.


a/
8
7
6
5
4
3
2
1
(1)
(3)
(2)
2
4
6
8
10
b/
8
7
6
5
4
3
2
1
(4)
(5)
20
24
28

Ordinate: Activity of OTCase μmoles citrulline/min mg protein.

Abcissa: a) Time (in hours). At 0 time an 18 hr old culture was transferred to arginine free or arginine supplemented medium, as indicated below.

b) Age (in hours) of culture grown in presence or absence of arginine.

Curve (1) represents the derepressed level of OTCase in an arg-1 auxotroph transferred to arginine free medium, while curve (2) represents OTCase in arg-1 in the presence of exogenous arginine. Curve (3) represents OTCase in su-19 arg-1 strain - the levels being the same in presence and absence of arginine.
Curves (4) and (5) represent the OTCase levels in ota-1 and su-19 ota-1 strains respectively.

ALEXANDRA PUTRAMENT, Institute of Biochemistry & Biophysics, Polish Academy of Sciences, Department of Genetics, Warsaw, Poland.

Osmotic-remedial revertants of some *A. nidulans* mutants

Reversions of adF9, adF33, adG14 and adH23 were tested. Ultraviolet-irradiated conidia (viability 90-100%) were divided into two equal samples one of which being plated on a standard adenine-less medium, while the other - on the same medium supplemented with KCl to a final concentration of 0.5M. The induced reversion frequency of adF9 was 1×10^{-7}, that of adG14 - 2×10^{-7}, irrespective of the plating medium. The induced reversion frequency of adF33 was 12.6×10^{-7} on standard medium, and 22.4×10^{-7} on KCl-containing medium. The respective values for adH23 were 2.8×10^{-7} and 8.6×10^{-7}. As could be expected, some of the revertants obtained on KCl-containing medium did not grow, or showed only traces of growth on standard medium.

Thus by plating on a medium with increased osmotic pressure new classes of revertants can be obtained.

B.R. SCOTT & T. ALDERSON, MRC Radiobiology Unit, Harwell, Didcot, Berks.

Features of the Radiosensitivity and mutation of *Aspergillus nidulans*

A number of additional observations arising from a study of the effect of γ-irradiation on the inactivation and mutation of a conidial suspension of the Glasgow meth G1 bi$_1$ strain of Aspergillus are reported; they demonstrate features which may

produce variations in radiation experiments.

I. Holding conidia in buffer prior to irradiation

Regardless of whether the irradiation is carried out in oxygen or nitrogen, holding the suspension in neutral phosphate buffer prior to irradiation has drastic effects on the survival curve obtained (fig. I). The number of meth G1 suppressor mutations for each of the three phenotypically distinguishable classes (A, B and C) of revertants (Lilly Mut. Res. 192, 1965) also decreases with prior holding (Table 1), although there is no change in the distribution of the A, B and C classes ($X^2_4 = 0.06$ $p = 0.99$).

A volume measuring technique similar to that of the Coulter counter, but whose output information is monitored by a 100-channel pulse-height analyser which compares the sample to standard polystrene spheres, has been used to compare the conidial size of a freshly-prepared suspension and ones kept in buffer for 24 and 48 hours. The output information is punched onto tape, in each case, for computer analysis comparison using a programme which plots the volume distribution of the samples (in collaboration with P. Gray and D.G. Papworth). Examination of a freshly-prepared suspension, 24-hour and 48-hour, has shown that as the holding time increases so does the volume of the cell. This observation indicates that water is being progressively taken up by the conidia with the resultant effect of increased radio-sensitivity. This conclusion is supported by the observation that there is no change in sensitivity or mutation of the conidial suspension when it is held on a millipore filter in an enclosed contained for 24 hrs, and subsequently resuspended in buffer prior to irradiation.

II. Inactivation by nitrogen bubbling

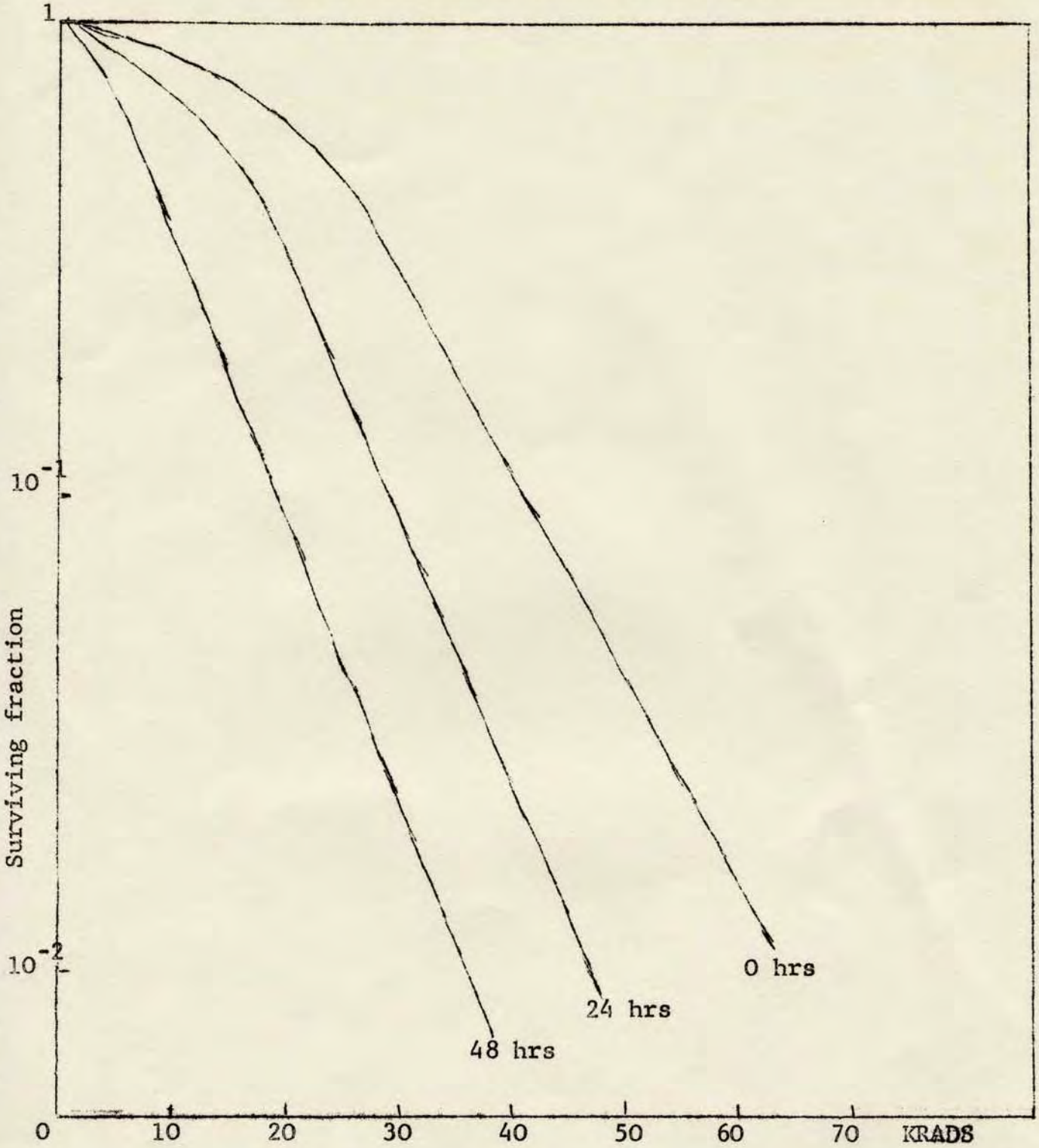
Bubbling oxygen through a conidial suspension had no effect on the viability or mutation rate of the Meth G1 bi_1 strain. Nitrogen bubbling (see fig. II), however, is found to decrease the survival whilst having no effect on the mutation rate. This result is not dependent on the time the condia are held in buffer. Furthermore, it is suggested that this might be a feature of strains containing the bi_1 marker, since Rao and Modi (Can. J. Microbiol. 14 (7) 818-825 1968) have reported that these strains have weakened mitochondria. To test this possibility a strain lacking the biotin marker (cnxF α 59-Cambridge) was used under the same conditions. This strain shows little or no inactivation, which tends to implicate the biotin marker as the cause for the loss of viability by nitrogen bubbling.

Table I.

No. of revertants induced per ml by γ-irradiation of 2×10^6 conidia per ml under oxygenated conditions after holding in buffer (48 Kr dose).

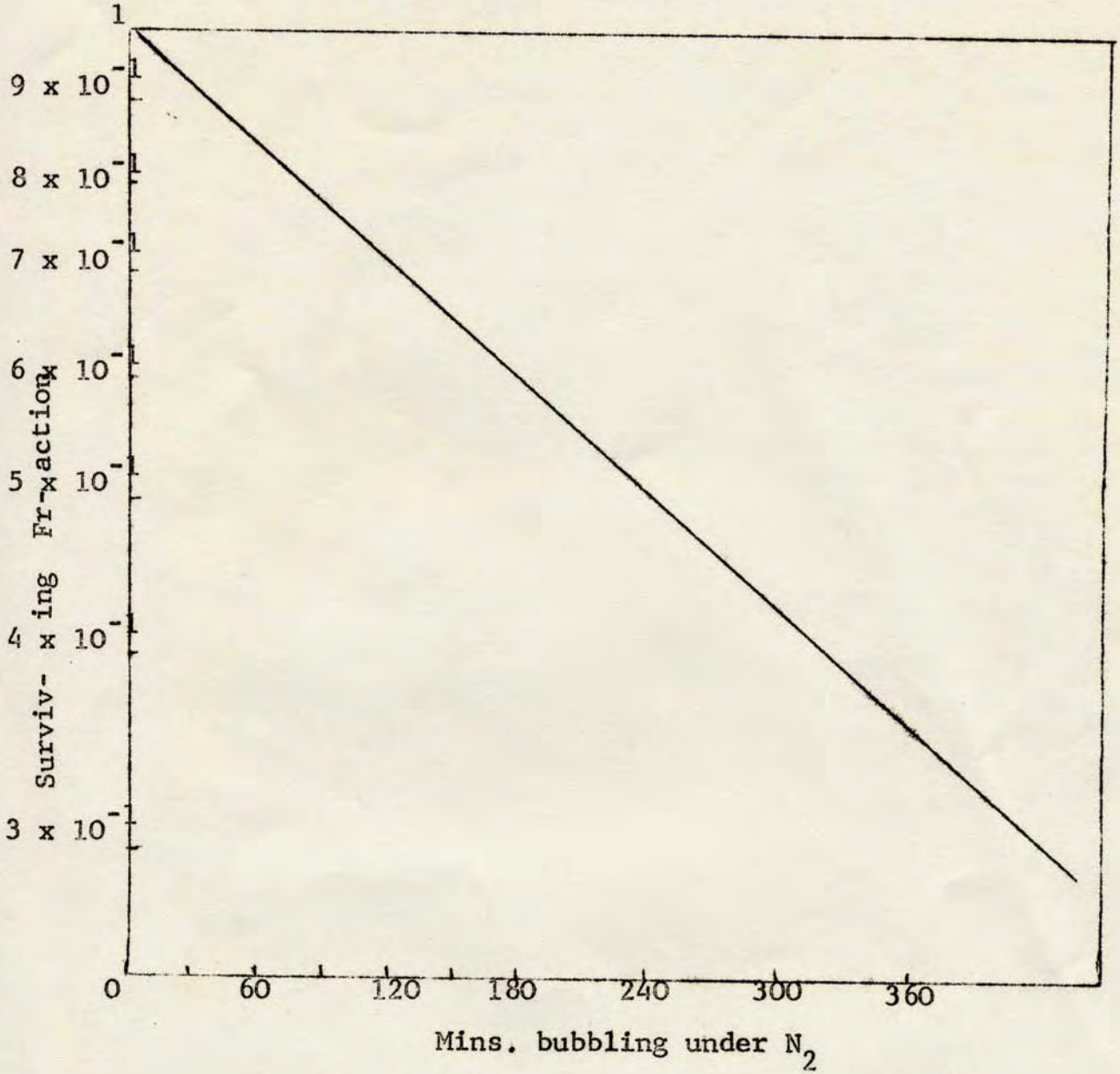
Time	Class A	Class B	Class C	Total
0	56	69	32	154
24 hrs	12	15	7	34
48 hrs	5	5	2	12

Fig. I. The effect of holding the conidial suspension in neutral phosphate buffer prior to irradiating under oxygenated conditions with γ rays from a Co^{60} source.



KRADS γ-irradiation Co^{60} source

Fig. II. Loss in viability of the Glasgow meth G1 bi_1 strain due to nitrogen bubbling



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Changes in the hexosamine content of hyphal walls of *Aspergillus nidulans* induced by pentachloronitrobenzene (PCNB)

The hexosamine and hexose content of cell wall hydrolysates of wild-type and PCNB resistant (*pcnb-2*) strains (Threlfall, R.J., J.gen.Micro., 52, 35. 1968) grown in liquid complete medium with or without PCNB has been measured. Observations have been made on the cytology of these strains on solid media.

The hexosamine content of the wild-type and *pcnb-2* is increased by a third in the presence of PCNB. That of the mutant is less than that of the wild type but it increased in the presence of PCNB to that of the wild-type control. The hexose content showed less alteration. Sodium deoxycholate which also restricts growth is without effect on the hexose or hexosamine content of either strain. PCNB inhibits the production of extracellular galactose-galactosamine polymer (Everleigh and Gorin, A.N.L. 10, 25) by the wild-type but is without effect on that produced by the resistant mutant. Deoxycholate enhances the production of polymer by both strains. The galactosamine content of the wild-type is drastically reduced in the presence of PCNB; the effect on the mutant is less marked. The striking effects on hyphal morphology may indicate that galactosamine plays a special role in determining the form of the hyphal wall. Efforts are now being made to find out if PCNB inhibits an enzyme (eg. UDP-N-acetyl D glucosamine-4-epimerase) involved in the synthesis of acetyl-galactosamine.

M.D. HILL, Department of Biology, University of Lancaster

Temperature Sensitivity in a wild-type isolate of *Aspergillus nidulans*

According to Pontecorvo et al., *A. nidulans* grows within a wide range of temperatures and has a high optimum, near 40°C. This would appear to be the case for most isolates that have been used in experiments (Upshall, Croft, unpublished). An exception is Birmingham wild-type isolate 3 where this high temperature is distinctly unfavourable to germination, normal hyphal growth and conidiation. At 35°C, colonies are abnormal in phenotype and conidial germination is reduced.

Parental Colony (Grown at 25°C)	Incubation temperature 25°C Total No. Colonies	25°C % Abnormal	35°C Total No. Colonies	35°C % Abnormal
1	177	4.1	25	100
2	129	5.6	11	100
3	278	1.9	50	100
4	143	3.9	31	100
5	220	3.3	20	100

From the table it can be seen that with a parental colony originally grown at 25°C (which is normal in phenotype), when conidia are incubated on minimal medium at 25°C germination is normal. On average 96% of the colonies are phenotypically normal while the remaining 4% are abnormal. When incubated at 35°C germination is markedly reduced, i.e. fewer colonies grow, and all that do so are abnormal in phenotype.

The abnormals are of the same phenotype at both temperatures and are also somatically unstable. Current experiments are attempting to determine the controlling mechanism of both the instability and the temperature sensitivity.

Reference

Pontecorvo, G. et al. 1953. Advances in Genetics. Vol. 5.

QUESTIONNAIRE ON GENE SYMBOLS (sent out with ANL.10)

32 completed questionnaires wererecieved, all of which expressed approval of the clearing-house system operated by Professor Roper and agreed to the adoption of a uniform nomenclature system for all strains of *Aspergillus nidulans* originating from the Glasgow wild-type.

The following table shows the distribution of replies with respect to the nomenclature system preferred and the fraction of research using *A. nidulans*.

Nomenclature system preferred	Research use of A.nidulans			
	small	major	all	total
Sermonti's proposals ANL.9.24	4	2	2	8
Ditto, if agreed to by all fungal geneticists	6	1	0	7
Clutterbuck's proposals ANL.9.26	4	7	6	17
				32

From this it appears that Dr. Clutterbuck's proposals receive an overall majority, and particular support from workers whose main interest is in A. nidulans. Not surprisingly, workers for whom A. nidulans provides only a small fraction of their research material are the major supporters of a unified fungal system.

The conclusion from the questionnaire therefore, is that Dr. Clutterbuck's proposals should be adopted. The possibility of future cooperation with other fungal systems may, perhaps, be left open, although at present nomenclature proposals for Neurospora (Neurospora Newsletter 8.23 (1965)) and yeast (Supplement to Microbial Genetics Bulletin 31 (1969)) differ considerably from each other and from the Aspergillus system. The basic points of Dr. Clutterbuck's proposals can be summarised by the following example:

xyzA2

Three letter gene symbol (established symbol retained even if not three-letter).	italicised Locus-specific letter; replaced by a hyphen in mutants whose allelism is untested.	Serial mutant isolation number.

Wild-type alleles are indicated by a superscript $^{+}$. Phenotype symbols such as three letter capitals corresponding to the gene sybols may be used. Local strain collection may be numbered serially with a distinguishing initial letter or letters.

Although not specifically tested in the questionnaire, the locus letters proposed by Clutterbuck (ANL.9.26) seem to be generally accepted and are now appearing in publications. In addition, it now seems sensible that all loci should be lettered therefore where there is only one locus with a particular symbol, this should now carry the letter A e.g. biA1. Only mutants which have not been tested for allelism should retain a hyphen.

HELEN M. PALMER AND J.A. ROPER, Department of Genetics, University of Sheffield.

Induced deletions in duplication strains of *Aspergillus nidulans*

Strains with a chromosome segment in duplicate are unstable at mitosis. Among other variants, they give types which have lost, by deletion, part or all of either duplicate segment. The strain studied most intensively is:

I ———————— y + +

II - - - - - - - - - - - - y^+ ad bi

Sectors arising following deletions are generally scored easily by their improved growth rate. In some, the position and approximate extent of the deletions are revealed by the uncovering of recessives. Our particular interest is in the processes of frequent deletion and it is easy to study the influence of chemical agents (Nature (1970), 226, 276). The system can be used also to test for deletion-producing agents. Positive results need cautious interpretation (especially when colonies are grown for long periods on an agent) because of possible selection effects; furthermore, because of special features of the system, positive results may not indicate a general deletion-inducing ability.

We report briefly on some of the agents screened recently:

Caffeine. Colonies grown on caffeine (0.25 to 2 mg/ml of complete medium) show a greatly enhanced frequency of sectors arising by deletion, either from the translocated or non-translocated segment. Under these conditions there was no increase in gene mutations as tested by the methl system. We are investigating whether caffeine actively induces breaks or whether it prevents repair of spontaneous lesions, most of which would normally not give deletions.

Atebrin increases specifically the frequency of yellow sectors which arise by deletions from the translocated segment. It is active in complete medium (50 μg/ml) but shows little or no activity in minimal medium supplemented with necessary nutrients.

Malachite green (0.4 μg/ml in complete medium) increases yellow sectors occurring after 8 days' incubation at 37°.

Compounds which do not significantly increase sector frequency, at the concentrations tested, include: cyclo-heximide, 6 aminonicotinamide, urethane ,acriflavine and crystal violet.

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Mitotic non-conformity in *Aspergillus nidulans*: Derivatives from deteriorated variants

Duplication strains of *Aspergillus nidulans* are unstable and produce, during vegetative growth, both improved and deteriorated variants. Deteriorated variants generally have enhanced instability; they give derivatives which may be morphologically improved or more deteriorated than the parental strains. From a strain with a segment of linkage group I in duplicate (attached terminally to linkage group II), 31 independent, deteriorated variants were isolated; from these a representative sample of 50 mitotic derivatives were analysed. The causes of improvement or further morphological deterioration were classified as follows:

1. Phenotypic improvement. a) By a deletion producing a quantitatively haploid or near haploid condition, but without loss of the general characteristics of deteriorated morphology: Deletion in translocated segment (9 derivatives); deletion in linkage group I. (5).
b) Deletion as in 1a)but also simultaneous loss of the determinant of deterioration (4).
c) Suppression of the deteriorated phenotype without loss of the determinant of deterioration: By deletion in linkage group I (4); by further mutation (2).
d) By loss or reversion of the determinant of deterioration without any detectable deletion: Loss associated with increased instability (2); loss not associated with increased instability (3).

2. Phenotypic deterioration. a) By further mutations which increase the degree of morphological abnormality (8).
b) By a possible translocation of the determinant of deterioration from one linkage group to another (1).
c) By aneuploidy: Apparently simple aneuploidy (2); aneuploidy and other changes (2).

In 8 derivatives the causes of improvement or deterioration were not determined precisely, mainly because of extreme instability of the variant or the derivative. In at least two of the cases there was a strong suggestion of transposition from one linkage group to another, of the determinant.

_____X_____X_____X_____X____

HSIN-KAN WU, Department of Genetics, University of Sheffield.

Microcolony Technique

A year ago we were in need of a technique for dealing with some problems posed by unstable strains of *A. nidulans* and by certain other situations involving instability. A satisfactory technique has been developed and used extensively by the group for, among other things:

1) growing a required number of colonies (including one), each from one spore, in determined arrangement on a dish.
2) purifying or recovering extremely unstable strains.
3) recovering, for example, the p-fluorophenylalanine-induced breakdown products of diploids, especially from mixed or unstable sectors.
4) classifying segregants from meiotic and mitotic analyses on the basis of early morphology (16 hr. growth) and, later, the corresponding gross colony morphology. Certain aneuploids and duplication types can be distinguished from each other and from wild type at 16 hr.

The basic steps in the technique are

1) Place the smallest possible inoculum of conidia at the centre of a 9 cm dish of medium. In the case of ascospores, release spores from a cleaned perithecium at the agar centre using a pair of finely pointed Pasteur pipettes and binocular microscope; remove perithecium wall.

2) Drop 0.25 ml saline on top of the spores and also on each of two or three dishes of medium (depending on final density of colonies desired).

3) Spread spores evenly with a glass spreader. Without sterilising the spreader, proceed to spread on remaining dishes so that spreading and dilution are accomplished at the same time.

4) Incubate at 37° for 16 hr.

5) Examine by transmitted light (30-60X magnification) and determine early morphology. Transfer well separated microcolonies using a fine inoculating needle with bent cutting end.

F.G.S.C.

Additional strains in stock. Reported by the Director, Dr. R.W. Barratt.

FGSC No.	Mutant	linkage group	complete genotype	origin (X = cross)
280	c16	IL	c16,pro1,paba1,pal B7	X(c16,pro1,paba1,y) x (bi1;palB7)
281	suA1palB7	VIIIR	bi1;suA1palB7,palB7	(spont) bi1;palB7
282	suC6palF15	V	bi1;suC6palF15;palF15	(spont)bi1;palF15
283	multi:		su1ad20,y,ad20;Acr1; gal1;pyro4;facA303;s3; nic8;ribo2	
284	suB2palB7	VI	bi1;suB2palB7;palB7	(spont)bi1;palB7
285	ad50	IIIL	bi1;ad50	(UV)bi1
286	fpC43	VIII	ribo-1,bi1;fpC43	(spont)ribo-1,bi1
287	suD2palA1	I	bi1,suD2palA1;palA1	(spont) bi1;palA1
288	multi:		suad20?y w3;gal1;pyro4; facA303;s3;nic8;ribo2	
289 disc	multi		suad20,y,ad20;Acr1;phen2; pyro4;lys5;s3;nic8;ribo2	#68
290	hisD34	VIIIR	bi1;Acr1,w3;nic8;hisD34	(UV) bi1;Acr1,w3 nic8;vc ?
291	gal4	VIII	bi1;w3;gal4 T(VI-VII)(III-VIII)	
292	lac3	IIR	bi1;w3,lac3 T(VI-VII)(III-VIII)	(UV)bi1;w3 T(VI;VII) T(III;VIII)
293	lys88	IR	lys88,bi1;s3(FL)	(NA)bi1;s3
294	lys18(BL) (allelic with lys7)	VII	bi1;s3;lys18(BL)	"
295	ile1(AL)	VII	bi1;s3;ile1(AL)	"
296	lys13(EL) (allelic with lys10 & 14)	V	bi1;lys13(EL);s3	"
297	lys51(FL)	IR	bi1,lys51(FL);s3	"
298	lys14(EL) (allelic with lys10)	V	bi1;lys14(EL);s3	"
299	his-122(EL)	VII	bi1;s3;hisEL	"
300	lys7(BL) (allele of lys18)	VII	bi1;s3;lys7(BL)	"

alleles

alleles

alleles

lys 5 = B

301	lys231(EL) (allele of lys10)	V	bil;lys23(EL);s3	(NA)bil;s3 ± T
302	orn7	VIIIR	pabal;w3;orn7;facB101 ribo2,gal7 T(III→VIII)?	X(bil;w3;gal7)x (pabal;orn7;facB101, ribo2) T(III;VIII) T(VI;VII)
303	Teaching strain		pro1,y;Acr1	P123 25% T(VI;VII)
304	"		pro1,pabal,y;phen2	P97 T_1 (I;VIII)
305	"		pro2,paba18,bil;phen2; lys5	P138
306	"		ad17,pabal,y	P112
307	"		pro1,pabal,nic8	P87
308	"		pabal,bil	P115
309	"		pro2,paba18,y	P117
310	"		pro1,bil,Acr1;pyro4	P254
311	"		ad20,bil;w2,ribo3,nic2	P12
312	"		sul ad20,y,ad20;Acr1; phen2;lys5	P20
313	multi:		sulad20,pabal,y,ad20; Acr 1;phen2,lys5;s3; nic8;co	M [=303 co?] noT (EKB?)
314	multi:		pro1,pabal,y;w3,ad1;ve+	(QQ) noT (EKB)
315	multi:		ad14,pabal,y T free	(cross V) " "
316	s2	III	bil;s2 (allelic with s1 by heterokaryon)	(UV)bil (+ test) T(V - ?)
317	nic8	VII	bil;nic8 (multiple)	(UV)bil
319	multi:		an1,y;w3,ad1;s12 T free	(from cross (n) EKB)
320	multi:		sul ad20,ribo1,pro1,ad20, bil;pyro4	(Dom, from cross ribo, pro, paba y bi x su ad20)
321	Teaching strain		bil;w3,ni3	T(VI; VII) T(III; VIII)

= #63

CLEARINGHOUSE FOR GENE SYMBOLS, LOCUS LETTERS AND ALLELE NUMBERS. - J.A. Roper.

At present there is some confusion which will not be removed in one step. I am grateful to Dr. A.J. Clutterbuck for producing the list below. In view of the labour involved and the anomalies and difficulties which exist it would not be surprising if, as he says, the list is possibly incomplete and has some errors.

I should like to suggest the following future procedure so that the role of ANL is, as it should be, entirely passive. It might be simplest if workers interested in the same loci communicate among themselves and either reach agreement or outline their disagreement so that others are informed. A note will then be included in ANL: if there is urgency a prompt circular can be sent from here to all those likely to be concerned. Where conflict or confusion is unlikely, claims for symbols, letters and numbers will be recorded in the following issue of ANL. In due course a list of the following kind, appropriately modified, can be produced.

The following prefixes have been used for stocks from laboratories or centres.

FGSC - Fungal Genetics Stock Centre.
G - Dept. of Genetics, University of Glasgow.
M - Dept. of Genetics, McGill University, Montreal.
BWB - B.W. Bainbridge, Queen Elizabeth College, London.
C - this prefix has sometimes been used for strains from the Dept. of Genetics, University of Cambridge.

1970
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LIST OF GENE SYMBOLS, LOCUS LETTERS AND ALLELE NUMBERS WHICH HAVE BEEN USED OR SUGGESTED IN ASPERGILLUS NIDULANS UP TO MAY 1970.

Not all these symbols are in current use but obsolete symbols are included to avoid future confusion. The locus letter A has been added to all previously unlettered single loci likely to be in current use; similarly, allele number 1 has been added to single mutants. References are given to Dorn 1967 whenever possible and further references should be found there.

The list is necessarily incomplete and inaccurate, amendments will be welcomed.

Symbol	Phenotype	Locus Letters	Allele Numbers	References
aa	allantoic acid util.	X	?	Cove request
ab	aminobutyric acid req.	A	1	1
aba	abacus-aconidial	A	1-66	3
ac	acetate util.	A	1	2
Acr,acr	acriflavine res.	A,B	1-3	1
Act	actidione res.	A	1	1
ad	adenine req.	A-I	1-44,50, 51	1
al	allantoin util.	X	?	Cove request
alp	allantoin permease	A?	?	Cove request
am	= niiB	-	-	2
amd	acetamidase	(A),R	?	Pateman request

an	aneurin req.	A,B	1-8	1,2
ap	aminopterin res.	A	1	Apirion,Dorn & Forbes ANL 4,11.
apl	allpurinol + purine res.	A	?	Cove request
arg	arginine req.	A-D	1-11, 100-114	1,2,Weglenski request
aro	aromatics req.	A-C	1233,1185, 1248	Roberts,ANL 10,19 Zaudy,ANL 10,22
azg	azaguanine res.	A?	?	Cove request
bge	=fwa2	-	-	Käfer-Boothroyd
bi	biotin req.	A	1-4	1
bl	blue ascospores	A	1-6	1
br	= br1A	-	-	14
br1	bristle-aconidial	A	1-43	3
Bw	brown aconidial	A	1	5
cha	chartreuse conidia	A	1-3	1,Käfer-Boothroyd, Clutterbuck requests
cho	choline req.	A	1,2	1,2
cl	colourless asco-spores	A,B	1-6	1
cnx	nitrite req. purine util.	A-H	1-16?	4

co	compact growth	A	1	1
cys	= $_{s}$C13	-	2	5
dil	dilute conidial colour	A	1	1
drk	dark conidial colour	A,B	1-5	3
est	esterases	A	1,50,51	Dorn & Rivera ANL 6,13
f	= fac	-	-	Apirion et al ANL 4,11.
fac	fluoroacetate res. acetate non-util.	A-C	2-4,101, 102, 301-309	1
fan	fluoroacetate res.	A-E	1-16,52, 54,60, 101,151	1
fl	fluffy	A	1	Ball & Azevedo ANL 5,9.
fmd	formamidase	(A)	?	Pateman request
fp	= fpa	-	-	1
fpa	fluorophenylalanine res.	A-D	1-58	13,14
fr	fructose non-util	A	1-3	1
fw	fawn conidia	A(B)	1,2,10	1,da Cunha, ANL 10,12 Käfer-Boothroyd request
gal	galactose non-util.	A-D	1-36	1
gam	galactose non-util. molybdenum res.	A-D	?	Cove request

glu	glutamate req.	A	1,2	2
his	histidine req.	A-J A(L)-F (L)	1,10,13,38,? 2,8,14,100, 115,122	1 16 Pees ANL 7,11.
hx	hypoxanthine non-util.	A,B	1,13 +?	1
ile	isoleucine req.	A	1	1
ino	inositol req.	A,B	1,2	2
Iod	iodoacetate res.	A	1	1
ivo	ivory conidiophores	A,B	1-119	3
lac	lactose non-util.	A-E	1-7 +?	1, Gajewski & Litwinska ANL 10,17.
lu	leucine req.	A	1,2	1,2
lys	lysine req.	A(L)-F (L)	1-10,16,51	Pees, ANL 7,11
m	mycelial growth	?	1-6	15,Kwiatkowski ANL 3,1
mal	maltose non-util.	A	1,2	1
mau	ethylamine non-util.	A ?	?	Cove request
mea^R	methylammonium res.	A ?	?	Cove request
med	Medusa-morphological	A	1-3	3
mel	melanin formation	A,B	1,2	Martinelli ANL 10, 24
meth	methionine req.	A-H	1-66, 101-106	1,2,6,11

mg	malachite green res.	A	1	12
mo	morphological	A-D	1,9,50 87,89,96	Bainbridge ANL 4,17; ANL 7, 19.
mol	molybdate res.	A,B	?	Cove request
ni	= niaD or cnx	-	3,7,50,51	1
nia	nitrate reductase	D	1-68	4
nic	nicotinamide req.	A-C	1-15	1,2
nii	nitrate reductase	A	?	7
nir	nitrate path regulator	A	?	7
nr	= uvs	-	1,2	8
orn	ornithine req.	A,D	1-11,20,21 100-114	1,2 Dorn request Weglenski "
ota	ornithine trans-carbamylase	A	1	Piotrowska ANL 9, 18.
p	pale conidia	A	1-3,12	1,Clutterbuck ANL 9,14 Dorn request
paba	p-aminobenzoic acid req.	A,B	1-23 101-126	1 Jansen request
pac	acid phosphatase	A-C	1-5	1
pal	alkaline phosphatase	A-F	1-15	1
palc	acid & alk. phosphatases	A-C	1-4	1
panto	pantothenic acid req.	A,B	1,100	1,2

pcnb	pentachloro-nitrobenzene res.	A(B?)	1-3	9
pf	p-fluorophenyl-alanine res.	-	21,22	12
pfp	" " "	-	1-104 ?	dePalma & Morpurgo ANL 4,7.Petrelli et al. ANL 5,7
phen	phenylalanine req.	A,B	1-11,1481	1,13 Dorn request
ppa	phenylpyruvate req.	A	1	2
pro	proline req.	A,B	1-16	1,10
pu	putrescine req.	A	1-7	1,2
pyro	pyridoxine req.	A	1-13	1,2
r	enhanced phosphatase	A-C	1-3,50,51	1
ribo	riboflavin req.	A-F	1-8	1,2
s	sulphite req.	A-D 205 E15	1-16,50	1
sb	sorbitol non-util.	A	1-9	1,Roberts ANL 2,13
sor	sorbose res.	A,B	?	Cove request
suadE20	suppressor	A	1,2	1,2
sumeth	"	-	1-68	2,6
suornB9	"	A	1	2
supabaB22	"	A	1-4	1
supalB7	"	A,B	1,2	1
supalF15	"	C	1,6	1
supalA1	"	D	1,2,5	1
supro	"	A,B(D?)	1-68	1,10
suc	succinate non-util	A	1	2
Sul	sulphanilamide res.	A	1	1

sup	suppressor(of meth)	A?	101 110	11
stu	stunted conidiophores	A	1-4	3
tcnb	tetrachloro-nitro benzene	A	1	9
te	teoquil res.	A	6	1
tel	tel (mound)	A	1	2
thi	thiazole req.	A	1-4	1
ths	thiosulphate req.	-	3,5	2
tryp	tryptophan req.	A-D	48,69,26 801,432, 403,+?	1
ts	temperature-sensitive growth	A-D	1,5,17, 25,15	1,2
tyr	tyrosine req.	A,B	1,7,1403	13,Dorn request
u	urea non-util.	X-Z	5 +?	Cove request
ua	uric acid non-util.	X-Z	1-12	1,Cove request
uap	uric acid permease	A ?	?	Cove request
uvs	ultra violet sensitive	A,B - A-E -	1,2 1,4,5,7,77 1-95 1-14	8 17 Tuveson request Jansen request 18
ve	velvet morphology	A	1	1
w	white conidia	A	1-12	1,2 Käfer - Boothroyd request
wet	wet white conidia	A	1-6	3

y	yellow conidia	A	1-39,102	1,Clutterbuck request
yg	yellow-green conidia	A	1-6	1,3

Phenotypes are given only briefly. For full phenotypes the original references should be consulted.

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