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RESEARCH AND TECHNICAL NOTES

P.R. da CUNHA

A spontaneous conidial colour mutant

A spontaneous colour mutant was isolated from the strain pro 1 y; Acr 1 of the Sheffield stock. The conidial colour was autonomously controlled in heterokaryons

Meiotic analysis showed that the fawn phenotype was determined by mutation in a single gene which showed free recombination with y and w3. Significant deviation from free recombination was found only for fac A and fawn (24.5%)

Mitotic analysis confirmed that fawn (fw10) can be allocated to chromosome V The mutant allele fawn is recessive. Its combination with the alleles y, y^+ or w3, in a haploid, gave rise to colonies fawn, greenish and white yellowish respectively, showing that it interacts with them in such a way that the genotype could be deduced by inspection.

This fawn mutant retained the normal morphology and size of conidial heads as the strain from which it arose. Thus, fawn seems to be a useful colour marker, especially by its location on linkage group V whose nutritional markers lys 5 or fac A in master strains are not always ideal.

J.A. OSUNA

Auxotrophic mutants of Aspergillus rugulosus

Strain IZ 1523 of Aspergillus rugulosus was irradiated with ultraviolet light (5% survival) and from 3283 analysed colonies, 13 nutritional deficient mutants were isolated (about 0.4%). Four of these mutants were methionine requirers, two required lysine, three required arginine, one required proline, one required biotin, one required nicotinic acid and one required hypoxanthine. The proline mutant could grow only in proline but not in ornithine, citruline or arginine. From the three arginine mutants, two can grow when arginine or ornithine is added to minimal medium but not when proline or citruline is added The third arginine mutant responds only to arginine and not to proline, ornithine or citruline.

Mitotic analysis was carried out with six mutants (three arginine, one methionine, one hypoxanthine and one

nicotinic acid mutant) and only in one case (arginine-methionine) linkage was detected, but meiotic analysis indicated that the two genes are not meiotically linked.

I.R. BARACHO

Conidiation and perithecial formation in A. nidulans

The strain of A nidulans pro 1, paba 6, y; w 3 of the Glasgow stock, that we have in our laboratory, have only scanty conidiation and no perithecial production. This strain when grown at 37°C shows fluffy micelia that die out in a few days. We examined 684 colonies of this strain and all these colonies showed these characteristics.

Heterokaryons were established between this strain and strain su 1 ad 20, y, ad 20, paba 1; Acr 1; lys 5; cha (which presents perithecial production and abundant conidiation). From one out of three heterokaryons, pro 1, paba 6, y; w 3 reisolated showed abundant conidiation and perithecial production. Also some of the isolates showed fluffy micelia that did not die out in a few days.

The phenomenon suggests the participation of cytoplasmic factor in the conidiation, in the perithecial production, and in the persistence of fluffy micelia of A. nidulans.

I. PRASAD

White mutants in Aspergillus niger

In the process of selecting coloured mutants in Aspergillus niger after UV light irradiation, two white mutants were picked up. In both these mutants the conidial chains were adhered together and did not disperse in any mounting liquid. The mutants survived for a week and then died. Several isolations were made while they were alive, transferred to complete medium and minimal medium and incubated at different temperatures; still they did not propagate. They also failed to make heterokaryons with other mutants and therefore even their nuclei were not preserved. Probably in these mutants some sort of lethal-factor was associated with white pigmentation.

C. BALL

The Genetics of Penicillium chrysogenum

The emergence of a clearer understanding of Aspergillus nidulans in the last ten years and the work of Sermonti et al and Macdonald et al on Penicillium chrysogenum has provided a background against which a more detailed genetic analysis of the latter organism can be made

Prior to our present work, no genetic map existed. One reason for this may be that relatively few people have worked with the organism. However, a more convincing explanation is that the work that has been carried out has been empirically orientated, e.g. cross strain A to strain B and examine the penicillin productivity of any segregant. The production of strains A and B from a common ancestor is usually achieved by using a variety of mutagens such that, although it might not be intended, coincident chromosome aberration is produced Since such aberration can restrict recombinant recovery (e.g. by mitotic crossing over in the case of inversion or translocation, and by haploidisation in the case of translocation) it is difficult to assess segregation data.

We have adopted two approaches to overcome this problem, namely (a) synthesis of master strains, reference to which will enable detection of chromosome translocation. Also as an aid to this approach we are screening to assess the best non-translocating mutagen (b) treatment of diploids with mutagens that are known to increase the frequency of double mitotic exchange

In addition, we have obtained two findings that have not previously been recorded for Penicillium chrysogenum

(a) a modified pFA technique has been shown to induce haploidisation.

(b) spore colour mutants brown, yellow and bright green are "allelic" judging by complementation and recombination data.

Furthermore it can be said that we have analysed an instability system like the one described by Backus and Stauffer (1954) in strain Q176 but unanalysed genetically by them. Our findings indicate that the instability we are studying is chromosomal in origin.

A. PUTRAMENT

Osmotic-remedial mutants in Aspergillus nidulans

A total of 135 auxotrophic mutants were tested on minimal medium supplemented with KCl or KNO_3 or acetate or glucose to a final concentration of 1M, and in normal, 37°C or lowered, 25°C temperature (cf. D.C. Hawthorne and J Friis, Genetics 50:825, 1964). It was found that:

1. All pro, paba and ad mutants which were leaky when plated on MM and incubated in 37°C, showed considerably better growth in 25°C, or on the medium which increased osmotic pressure, particularly on glucose which even in 1M concentration only slightly inhibited growth of the wild-type strains. On the medium with increased osmotic pressure and in 25°C these leaky mutants grew as fast as wild-type strains.
2. adl and pabal1 mutants non leaky in normal plating conditions, showed good growth on 1M glucose medium, in 25°C, although they did not conidiate.
3. s and thio mutants are usually leaky; however, 16 such mutants tested showed poorer growth on 1M glucose or KCl medium, as compared with the standard MM. None of 52 meth mutants tested showed positive growth response to the raised osmotic pressure of the medium, or lowered incubation temperature, although 17 of them are leaky.

It would be of interest to see how osmotic conditions influence mutant enzyme activity in vitro

ZSOLT HARSANYI AND GORDON DORN

Purification and characterization of the acidphosphatases in A. nidulans: a preliminary report.

Five electrophoretically distinct acid phosphomonoesterases have been identified in mycelial extracts of Aspergillus nidulans, grown under limiting phosphate conditions. They have been designated P3 through P7, as shown in the accompanying diagram. Ammonium sulfate fractionation of chloroform-treated crude extracts (in 0.025 M. veronal buffer, pH 7.4) separates P7 (0-33% ppt.) from P3 through P6 (45-65% ppt.). Fractionation of the latter on Bio-Rad DEAE-cellulose allows rough separation of P5 (0.11 M. NaCl elution), P3 (0.15 M.), P4 (0.20 M.) and

P6 (0.25M.). P7 binds so tightly to DEAE columns that salt elution is ineffective.

The pH optima of P3, P4 and P5 all appear to be 6.1, but differ in the broadness of the pH-activity profile. P5 is the narrowest, P4 broadest and P3 intermediate with none showing appreciable activity above 7.0.

P5 has been purified about a hundredfold by following the ammonium sulfate treatment with: Sephadex G75, DEAE stepwise elution, Bio-Rad P60, DEAE gradient elution and Sephadex G200. It appears to be homogenous on Sephadex, ultracentrifugation, starch and acrylaminde gel electrophoresis.

P3 and P4 have been freed of contaminating phosphatases using similar steps, but the preparations still exhibit two to three protein bands after acrylamide electrophoresis.

P3 and P5 are both sensitive to fluoride (50% inhibition of both at 3×10^{-2} M.) and to molybdate (100% inhibition of both at 3×10^{-4} M.). Dialysis or EDTA treatment (0.01 M.) has little effect on activity, suggesting that neither cations nor cofactors are essential for activity.

Both P3 and P5 are more sensitive to temperature inactivation than P1, an alkaline phosphatase recently purified by Dorn (1968). Approximately, 50% of original activity remains after heating at 54°C. for one minute and 45 sec., respectively, at pH 7.4.

Elution from Sephadex, using known marker proteins, yield molecular weights of approximately 100,000 for P3 and P5, indicating that they are smaller than either alkaline phosphatase (Dorn, 1967).

Interest in this enzyme system stems from the fact that to date twenty distinct loci have been found to alter the activity of the alkaline and acid phosphatases, either singly or in combination. Some of the suppressor mutations that restore alkaline phosphatase activity have been found to be acid phosphatase mutants themselves Genetic data suggest that the phosphatases may be heteromultimeric proteins, having in common certain subunits (Dorn, 1965). Purification and biochemical characterization of the phosphatases is a necessary prerequisite to the elucidation of this genetically and biochemically complex system.

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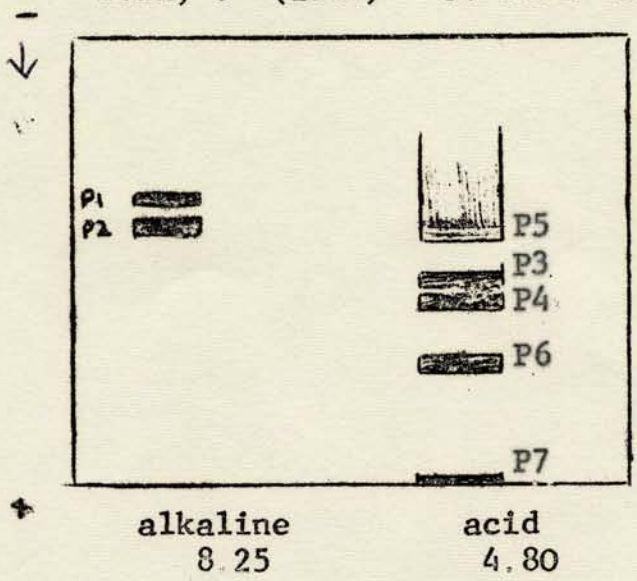


W. GAJEWSKI AND J. LITWINSKA

Lactose negative mutants of Aspergillus nidulans

After UV treatment 29 lactose negative mutants were obtained. They fall into 7 complementation groups including two already known Lac 1 and Lac 3. 5 new Lac$^-$ loci were provisionally designed Lac A to Lac E Lac B, Lac D and Lac E are in chromosome I (Lac D between pa^{b}a and y) whereas Lac A and Lac C are in chromosome VII. There is no close linkage between any two Lac loci.

Lac C, Lac 1 and Lac 3 mutants are leaky on lactose, the degree of leakiness increasing from Lac C to Lac 3. Lac B mutants are slightly leaky on lactose and are poorly growing on acetate. All these mutants do grow on melibiose. Mutants from Lac A and Lac B loci do not grow at all on lactose and melibiose. They do not grow also on acetate, succinate, pyruvate, citrate and fumaric acid.

Mutations from Lac D locus are slightly leaky on lactose and do not grow on melibiose and acetate.

Preliminary determinations of β-galactosidase activity in some mutants after lactose induction were carried out. All mutants tested show lack or lowered to different degrees the β-galactosidase activity except mutant No 5 from Lac B locus which has the same activity level as the wild type. We intend

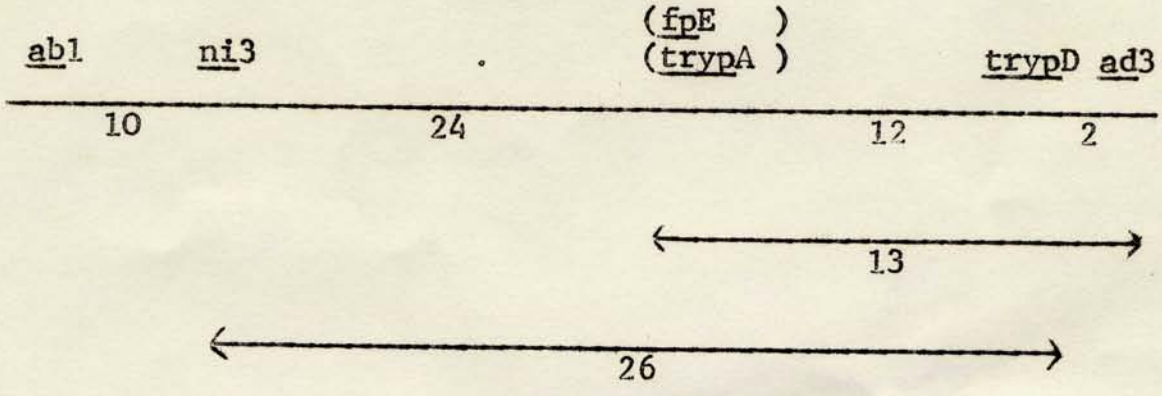
to determine now the induction of β-galactosidase and β-galactosidase permease activities in all mutants and to study the regulation of lactose utilization in Aspergillus nidulans.

C.F. ROBERTS

Mapping of trypA and trypD loci in A nidulans.

Mutants at the trypA locus are defective in anthranilate synthestase. The pfE series of mutants are resistant to p-fluorophenylalanine, have a partial growth requirement for tryptophan, replaceable by anthranilic acid and are allelic to trypA mutants (Sinha, 1967 and personal communication). The trypD mutants lack phosphoribosyl transferase, the second enzyme in the tryptophan series.

A cross between Sinha's strain w3 abl ni3 fpE48 ad3 and pabal y; trypD 432 has been analysed. The location of fpE (trypA) is confirmed and also trypD mapped in linkage group IIR



It was possible to distinguish fpE, which has a partial tryptophan requirement, from trypD which has a total requirement. Selection of tryp$^+$ recombinants in the cross is consistent with the above map. The segregation of the outside markers was:-

ni try$^+$ ad	31
ni try$^+$ ad$^+$	2
ni$^+$ try$^+$ ad	159
ni$^+$ try$^+$ ad$^+$	2

I am indebted to Dr Sinha for the fpE48 strain and information about the allelism of fpE and trypA mutants.

C.F. ROBERTS

Isolation of multiple aromatic amino-acid mutants in A. nidulans.

Great difficulty was experienced in early attempts to isolate multiple requiring mutants, and only a few were recovered after exhaustive efforts with strain A160. Suspensions of conidia were exposed to UV light (95% kill) and then incubated in liquid MM to enrich for auxotrophs by the filtration procedure. The final filtrates were plated in MM supplemented with paba (0.5 ml); L-phenylalanine (phen), L-tryptophan (tryp) and L-tyrosine (tyr) all at 50 γ/ml. Auxotrophs were identified by replica-plating.

Many auxotrophs with single requirement for either paba, phen or tryp were isolated. The few multiply deficient mutants were used in reconstruction experiments to test procedures and particularly to find suitable medium

The difficulties proved to be in the plating medium. The mutants requiring paba and all three amino-acids are very sensitive to the ratio of the amino acids supplied. The ratio of tyr to phen and tryp is critical. There is no growth when the ratio phen:tryp:tyr is 2:2:1 (or less) and very poor growth when 1:1:1. Best growth was obtained at 1:1:5 and subsequently the amino acids supplied at 100: 100: 500 γ/ml.

It was also found that the plating efficiency of the mutant conidia is very low on MM supplemented as above (2-20% depending upon the mutant), and that the colonies conidiate very poorly. The plating efficiency was dramatically improved if 1% peptone was added to the medium and the conidiation was also enhanced though poor compared to wild type. Difco Neo- or Bacto-peptones were most effective

Further filtration experiments done with R44 and R46 yielded many multiple aromatic mutants which, in the case of the yellow strain, could be identified by their poor conidiation. The mutants were classified for growth requirements, and were tested for functional identity by heterokaryon complementation. The results are shown in the Table.

The bulk of the mutants fall into complementation group A; some mutants in this group do not complement in any combination but many complement others within the group. This group probably corresponds to the cluster of five genes controlling five (number 2-6) of the seven enzymes involved in chorismate

synthesis described in Neurospora by Giles and his associates (Giles et al, 1967, P.N.A.S., 58, 1453)

Fewer mutants fall in complementation group B, within which only three of 26 combinations tested complemented. It is most likely that this group corresponds to the arom3 locus in Neurospora controlling enzyme chorismic acid synthetase. Within groups A and B a small number of 'leaky' mutants occur which have no apparent requirement for paba or tryptophan. Growth tests for response to quinic or shikimic acids were mainly negative, and while some weak responses to quinic and/or shikimic could be detected if phen and tyr were included in the test media, these were inconclusive and, generally involved the 'leaky' mutants.

A small number of phen-tyr mutants (aroC) were recovered and presumably are defective in the synthesis of the common precursor (prephenic acid) from chorismic acid. They complemented phenA2 and all other phen or tyr mutants. These last two classes are probably alleles of phenB or tyrA (Sinha, 1967, Genetical Research, 10, 261) and indicate the separate identity of aroC which is confirmed by mapping.

Table 1 Multiple aromatic amino acid mutants

Complementation Group	Phenotype(s)	A160	R44	R46
	Requirement for:-			
aroA	pab phen try tyr	2	55	45
	phen try tyr	0	5	6
aroB	paba phen try tyr	2	12	7
	phen try tyr	0	1	0
	phen tyr	0	3	0
aroC	phen tyr	3	3	1
phenA2	phen	0	7	7
Not located:				
	phen	0	2*	0
	tyr	0	0	2**
Not tested:				
	pab phen try tyr	0	0	79
	phen try tyr	0	0	2
Totals:		7	88	149

* complement phenA2; probably alleles at the phenB locus.

** probably alleles at the tyrA locus.

A160 = bil; Acr1 w3; nic8

R44 = prol y; pyro4

R46 = ribol ad14; w3.

GILLIAN ZAUDY

The location of some multiple aromatic mutants in Aspergillus nidulans.

The mutants were allocated to linkage groups by mitotic haploidisation:

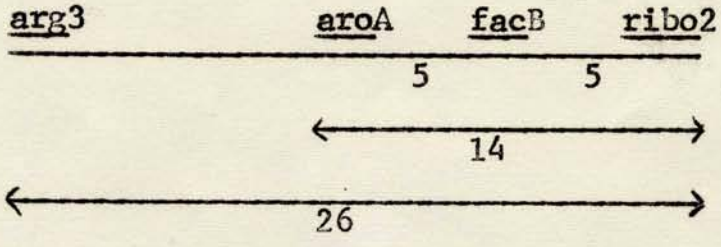
aroA (R44 aroA 1233) Linkage Group VIII

aroB (R44 aroB 1185) Linkage Group V

aroC (R44 aroC 1248) Linkage Group I

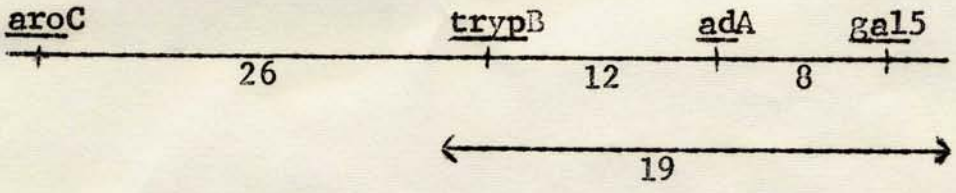
aroA 1233 VIII R

Two crosses involving the markers orn7 arg3 facB101 and ribo2 were analysed. The fac mutants were not distinguishable in the presence of aromatic amino acids, therefore only the aro$^+$ progeny were scored with respect to acetate utilisation.



aroC 1248 and trypB 26 I L

Three crosses involving the markers aroC, trypB, adA74 gal5, ribo1 and ad14 were analysed. It was necessary to supplement the plating medium with 100 γ/ml adenine and 50 γ/ml histidine to recover the adA mutant. The markers aroC and trypB recombined freely with ribo1 and ad14. The location of aroC and trypB distal to adA lengthens the left arm of linkage group I by some 30 units.



The location of aroC clearly distinguishes the locus from the tyrA (fpA) locus which is closely linked to ribo1 (Sinha, 1967, Genetical Research, 10, 261)

C.E. CATEN

Single Cleistothecia originating from three parental strains in Aspergillus heterocaryoticus

It was suggested on the basis of heterogeneous segregations for conidial colour markers that cleistothecia of Aspergillus heterocaryoticus frequently originate from more than one dikaryotic initial (Caten, ANL.9) Poor ascospore germination has prevented confirmation of this hypothesis by analysis of individual asci, as was possible for A. nidulans. An alternative test can be made, however, by examining the progeny from cleistothecia by dense mixtures of three differently marked strains. Of 53 cleistothecia sampled from two such mixtures, 8 segregated for markers derived from all three parental strains. Such complex segregations are expected if single cleistothecia contain two or more independent dikaryotic systems. Both (A x B) + (C x C) and (A x B) + (B x C) types were recovered among the 8 'anomalous' cleistothecia.

G.J.O. JANSEN

Location anddesignation of some uvs loci in Aspergillus nidulans.

Some years ago we isolated a number of UV-sensitive Aspergillus nidulans mutants, which we designated by uvs-1 - uvs-95. Actually we retained some forty uvs mutants for further investigation. The uvs mutations appear to be distributed over a large number of loci. So far we have found uvs loci on six of the eight chromosomes.

The mutant uvs-1 has already briefly been described (G.J.O. Jansen, ANL. 8, 20-21, 1967) and has been indicated on the recent genetic map of Aspergillus (G.L. Dorn, Genetics 56, 619-631, 1967). We would propose to designate this mutant henceforward by uvsA1. The uvsA locus lies between the pabaA locus and y locus on chromosome I.

Some of our uvs mutations affect mitotic recombination. These latter mutations are distributed over four loci which we would propose to designate by uvsB, uvsC, uvsD and uvsE, respectively. The uvsB locus lies very close to the hisA locus on chromosome IV, and the uvsC locus lies close to the ornB locus on chromosome VIII. The loci uvsD and uvsE are situated on chromosome V.

Data concerning the mutants uvsB10 and uvsC14 will soon be submitted for publication.

S.D. MARTINELLI

Phenol oxidases produced by mutant and wild type strains of Aspergillus nidulans.

In an attempt to elucidate the control of melanin synthesis in Aspergillus nidulans, mutants with abnormal melanin synthesis have been isolated following UV irradiation. One mutant (Mel^{x}A) produces excess melanin compared with BWB 224 (y; ve); the other produces no visible pigmentation (mel^{o}B).

Both abnormalities are due to recessive mutations within single genes. They complement each other in a doubly heterozygous diploid, giving a phenotype similar to BWB 224. Both gene loci have been assigned to linkage group VII by haploidisation. Mel^{x}A is unlinked to any of the markers already mapped on VII, whereas mel^{o}B has been provisionally located between mal 1 and cho.

Profiles of phenol oxidases have been compared in mutant and wild type strains, since some of them are involved in the early stages of melanin synthesis. Enzyme extracts from submerged batch culture have been applied to polyacrylamide gel electrophoresis columns using standard techniques. After electrophoresis, the enzymes were stained in situ with 0.2 M dihydroxyphenylalanine in 0.1 M phosphate buffer pH 7.0

A considerable degree of heterogeneity exists in strains of A. nidulans. BWB 224 has up to 4 phenol oxidases, mel^{o}B up to 5 and mel^{x}A up to 2. Various other wild types have up to 5 enzymes. In all strains so far studied, the number of phenol oxidases increases from a minimum number in the lag period to a maximum number in the late logarithmic or stationary phases. An intermediate number of enzymes is present during autolysis while melanin is being formed.

Differential centrifugation of BWB 224 extracts has shown that phenol oxidases are present in all fractions of the cell. One heat resistant enzyme is common to all of these fractions, whereas extra heat sensitive enzymes occur in the small particle fraction and final supernatant. A basic

pattern of distribution is found throughout the life cycle but with some variation.

Currently, a survey is being made of the variation in phenol oxidase profiles in a range of wild type isolates These profiles are being compared with those of mutant strains. The enzymes are being characterised by heattreatment, substrate and inhibitor specificity.

It is unknown which of these enzymes are involved in melanin synthesis It has also been claimed that tyrosinases can act as generators of oxidised NAD(P) in coupling with quinone reductions.

D.E. EVELEIGH AND P.A.J. GORIN

An extracellular polysaccharide containing N-acetylgalactosamine and galactose units from Aspergillus nidulans.

Initial studies on the cell wall of A. nidulans have shown that the organism produces a slimy extracellular polysaccharide containing N-acetylgalactosamine and galactose units. This material is produced by both strains tested to date (bil - FGSC 26/paba 1 y ad 20; Acr 1; Co - FGSC 120) when grown on Johnson's (1) or Vogel's minimal N (2) medium containing 1% glucose Greater polysaccharide yields are obtained using larger amounts of glucose e.g. 4%. The yield rises and falls over the growth cycle reaching a maximum of about 120 mg/l from 6-8 days growth (900 ml/ 2 l shake flask 30°C.). The polysaccharide is obtained from the culture medium by precipitation with ethanol (1:1, v/v). Mild acid hydrolysis of the polysaccharide (M H_2SO_4, 100°C 16 hr; 4 M HCl, 100°C, 2 hr) yields galactose and galactosamine, identified by paper chromatography using p-anisdine and alkaline silver nitrate as detection reagents D-galactose was isolated as crystals. The identity of galactosamine was confirmed by positive tests with ninhydrin and Elson-Morgan dimethylamino benzaldehyde reagent and also its degradation with ninhydrin to lyxose. These aldoses have also been obtained by hydrolysis using an autolytic crude enzyme from A. nidulans.

The polysaccharide contained 3.2% of nitrogen and had a specific rotation of +165° (10% aqueous NaOH) indicating a predominance of α-D-linkages. It consumed 0.97 mole of

sodium periodate per mole of anhydroaldose unit with production of 0.25 mole/mole of formic acid. Reduction of the periodate oxidized polysaccharide followed by acid hydrolysis gave threitol arising from 4-O-linked-D-galacto-pyranosyl units. A quantitative estimate of N-acetyl groups in the polysaccharide could not be made by proton magnetic resonance (p.m.r.) spectrophy since it gives viscous solutions. However acid degradation (0.1 M HCl, 30 min, 100°) gave a more amenable polymer which had 57% of its sugar units N-acetylated as indicated by the size of its T 7.39 p.m.r. signal compared with the H-1 signal (D_2O solution at 70°, tetramethylsilane external standard (3)).

References

1. M.J. Johnson (Personal communication - A highly buffered medium containing 19 g/l of phosphate)
2 H.J. Vogel. Neurospora Newsletter 10, P 34 (1965).
3 P.A.J. Gorin and J.F.T. Spencer. Can J Chem. 46, 2299 (1968)

A.J. CLUTTERBUCK

Further comments on gene symbols

The following are corrections and additions to my proposals in ANL. 9 (p.26):

Proposals 1 & 4: Gene symbols as well as locus-specific letters (but not isolation numbers) should be written in italics.

Proposal 5: (omitted in ANL.9): Individual mutants should be distinguished by serial isolation numbers following the gene symbol and locus-specific letter or hyphen.

Proposal 6: It is now clear that no unified strain numbering system is possible. The identity of strains should rather be described in publications by reference the F.G.S.C. or Glasgow collection strain numbers (see this issue).

Note 2: Dr. D.J. Cove has convinced me that the great variety of loci concerned with nitrate reduction requires a more complex nomenclature than that suggested in ANL.9. The Glasgow *ni* stocks have therefore been altered to conform to

the Cambridge nomenclature using the symbols nia, nii, cnx and nir (Pateman & Cove, 1969, J. Bact. 97, 1374). ni3 thus becomes cnxE16, ni7 becomes niaD15 and am1 is nir-14.
Note 4: Gajewski & Litwinska, 1969 (Mol. Gen. Genet. 102, 210) have now used the symbol methF for a locus other than meth1 as suggested in ANL 9, Professor Gajewski therefore suggests that meth1 and meth2 should become methG1 and methH2 respectively. meth3 is allelic with methB mutants.
In ANL.9 the last two ribo mutants should read riboE6 and riboF7.

In addition to the proposals in ANL.9 it has been suggested by Dr. J.G.O. Jansen (to whom I am also grateful for pointing out the omissions in proposals 1 and 5) that phenotypic symbols should be adopted in conformation with recommendation 8 in Demerec's proposals. This reads: "Phenotypic traits should be described in words, or by the use of abbreviations which are defined the first time they appear in a given paper". For example it is suggested that non-italicised three-letter phenotype symbols, the first letter being a capital, could correspond to the three-letter italic gene symbols.

A second additional proposal is that, contrary to Demerec's recommendations, superscripts may be added to gene symbols in cases where mutants at a single locus give very different, in some cases, opposite, phenotypes. This has been found to be valuable both in bacteria (Curtis, 1968, Genetics 58, 9) and Aspergillus (Pateman, Rever & Cove, 1967, Biochem. J. 104, 103). It is particularly valuable for constitutive mutants, and it is suggested that the less specific superscript ^{x} might be used where mutants giving rise to excess of a feature are to be contrasted with the more usual loss mutants. If this system is used, it should be understood that a mutant should be recognisable by its symbol and allele number, irrespective of any superscript the symbol may carry in a particular context; e.g. the symbol xyz-5 should belong to only one mutant; the same mutant may be described on different occasions as xyz-5, xyz^{c}-5 or xyz^{x}-5.

Since a questionnaire is being organised on the choice of gene symbols, I would like to comment on those proposed by Professor G. Sermonti in ANL.9 p.24. In the bacterial system the symbol pyr stands for pyrimidine requirement; pdx is used for pyridoxine requirement. In bacterial nomenclature, ane and abt are replaced by thi and ile respectively. I would suggest that ple would be better than pal (already used for phosphatase mutants) for pale conidia. pal and pac might be retained for phosphatase mutants, with the addition of plc for the present palc, or alternatively all phosphatase mutants might be pho. Additional symbols are also required: acr, iod, sul and uvs could be retained from the present system, and I suggest tps for temperature sensitive mutants and mor for morphological ones

J.A. ROPER

Clearinghouse for allele numbers

Several workers have sent details of allele numbers which they propose to use. These will be included in a consolidated list when replies to the questionnaire are received.

Silica-gel stock cultures of Aspergillus nidulans

C.F. ROBERTS, Department of Genetics, University of Leicester.

This most convenient and time-saving technique has been successfully applied to the storage of strains of Aspergillus nidulans using the procedures described by Perkins and employed at the Fungal Genetics Stock Centre. Cultures set up by Dr Foley at Yale University in 1962-63 have retained excellent viability and not one stock has been lost in the several hundred subsequently prepared by Jean Foley or myself. These include many strains which do not conidiate very well. The procedure does not appear to be widely known in the U.K., hence the preparation of this note.

PERKINS, D.D. 1962 Preservation of Neurospora stock cultures with anhydrous silica-gel. Can. d. J. Microbiol. 8, 591-594.

OGATA, W.N. 1962 Neurospora News Letter, 1, 13.

BARRATT, R.W., G.B. JOHNSON & W.N. OGATA, 1965. Wild-type and mutant stocks of Aspergillus nidulans. Genetics, 52, 233-246.

Materials:

(1) Non-fat skim milk powder. (Marvel, Cadbury's from any chemist's shop.)

(2) Silica-gel purified (without indicator) 6-22 mesh (Hopkin & Williams, cat. no. 7553).

(3) Suitable screw-cap containers to allow repeated sampling of the gel. The cap liners should be of metallic foil; waxed paper or cork liners discolour the gel on heat sterilisation.

The following procedure is based on the use of screw-cap vials containing 4 g of gel. (2 dram vials series SNB 17X58 mm C94. Spec No.6/H/0001. Closure 39/H/2053/0. Johnsen and Jorgensen)

Procedure:

(1) Threequarters fill containers with gel. Sterilise for a minimum of 90 min. at 180°C. Store tubes in a dry atmosphere (such as 37° hot room). Resterilise if damp.

(2) Prepare 5% solution of milk in distilled water. Distribute 2.0 ml lots to small capped tubes. Autoclave 15 lbs/10 min. and store at 4°.

(3) Have fresh well conidiated slants of strains (17 x 150 mm tubes.)

(4) Stand silica-gel containers in an ice bath for at least 30 min. (Considerable heat is evolved when the gel is wetted, hence the necessity to cool the gel and use cold milk.)

(5) Tip milk onto slant and make a heavy suspension of conidia using a long sterile wire to scrape off the conidia. (Purists may prefer to use pipettes to transfer the milk and the conidial suspensions, but the pipettes are foul to clean)

(6) Tip conidial suspension (1.0 - 1.5 ml) onto cold gel, return container to ice-bath at once and keep it there for at least 15 min. Do not saturate the gel, only about threequarters should be wetted.

(7) Keep gels at room temperature until the crystals readily separate when shaken (about a week).

(8) Check a sample for viability.

(9) Screw cap down firmly. Store over indicator-gel (Hopkin & Williams 7554.5) in an airtight container at 4°C. Plastic 'freezer' boxes with a good seal are ideal. The indicator gel will require drying once or twice a year.

(10) Sub-cultures are taken by transferring a few crystals of gel to a suitable agar slant.

Note The gels can be kept at room teperatures for prolonged periods without any apparent loss of viability. It is convenient to mail cultures by sending a few crystals of gel in a small vial.

Stock List of *Aspergillus nidulans* strains held at the Department of Genetics, University of Glasgow

These stocks have been rearranged and sorted into mutants and recombinants, the latter being numbered according to the chromosome principally marked. It is proposed to discard some recombinants which do not appear to be of value either as chromosome marker strains or as master strains: anyone requiring any of these should ask for them as soon as possible.

The markers of strains retained are being checked as the strains are put on silica gel for storage. The strains and further information about them will be available from Mr. E. Forbes or myself.

A.J. Clutterbuck.

Mutants

All mutant strain numbers begin with 0... FGSC numbers are given where available, and in the same column, some Cambridge strain numbers are entered as (C...). T = translocation(s) present, F = translocation-free. Gene symbols are given in the form proposed by A.J. Clutterbuck, A.N.L. 9, 26, with modifications proposed in this issue. For simplicity, however, no attempt has been made to indicate italic script. Mutagens: UV = ultraviolet light, X = X-rays, N = nitrous acid, NTG = N-methyl-N'nitro-N- nitrosoguanidine, M = nitrogen mustard, D = diethyl sulphate, sp = spontaneous. Some strains originating from Cambridge (Camb) or from Dr. G.L. Dorn, New York (N.Y) are listed here as mutants although their actual origin has not been ascertained.

Glasgow No.	T/F	FGSC No.	Mutant	Chromosome	Origin
00	F	4	wild type	-	-
01			aba-14	VIII	N-bi-1
02			ac-1	-	UV-bi-1;AcrA1 w-3
03	T	36	AcrA1	II	sp-pabaA1 y-2;co-1
04			AcrA3	II	sp-bi-1;adC1;sC12;pyro-4
05			acrB2	II	sp-adF15 pabaA1 y-2
06		42	adE8	I	UV-bi-1
07	T(VIII;II)	50	adE20	I	UV-bi-1
08-011			adE10,11,19,22	I	UV-bi-1
012		46	adF15	I	UV-bi-1
013-015			adF9,13,17	I	UV-bi-1
016,017	T		adF32,33	I	UV-bi-1;w-3
018		37	adG14	I	UV-bi-1
019			adG18	I	UV-bi-1
020	T		adH23	II	UV-bi-1;w-3
021	F		ad-42	I	UV-bi-1
022,023			ad-43,44	-	NTG-bi-1
024			ab-1 → dese. not	II	UV-bi-1
025			anB2	II	UV-bi-1;AcrA1 w3 →
026			anB8	II	UV-bi-1
027			an-7	-	UV-bi-1
028-030	T		an-4,5,6	-	UV-bi-1;w-3
031,032			an-3,9	-	UV-y-2;pyro-4
033	F		argA1	VI	X-bi-1
034		89	argB2	III	UV-bi-1
035			argC3	VIII	UV-bi-1
036			argD11	V	UV-bi-1
037-039			arg-5,6,7	-	UV-bi-1;lysB5
040	T		arg-9	-	UV-bi-1;w-3
041			arg-10	-	UV-bi-1
042	T		bi-2	I	X-y-2;thi-1
043-048			bl-1,2,3,4,5,6	II	N-y-2;w-2;sC12
049			bw-1	-	sp-2n-proA1 y-1;w-3
050			cha-2 → 3	-	N-bi-1
051	F	26	bi-1	I	X-wild-type
052	T		bi-4	I	X-y-2
053		(C.150)	cnxA5	VIII	Camb. y-2;pyro-4
055			cnxB11	VIII	Camb. D-bi-1
056		(C.237)	cnxC3	VIII	Camb. y-2;pyro-4
058	T	63	cnxE16(= ni3)	II	UV-bi-1;w-3
059	F		cnxE14	II	Camb. D-bi-1
060		(C.147)	cnxF8	VII	Camb. y-2;pyro-4
061		(C.152)	cnxG4	VI	Camb. y-2;pyro-4
063	F		cnxH4	III	Camb. D-bi-1

Glasgow No.	T/F	FGSC No.	Mutant	Chromosome	Origin
064,065			clB1,3	I	N-y-2;w-2;sC12
066,067			clB5,6	I	UV-y-2;w-2;sC12
068			clA4	IV	N-y-2;w-2;sC12
069	F		cho-2	VII	UV-bi-1
070	F		drkA1	VII	N-bi-1
071	F		fw-1	VIII	sp-bi-1
072	T		facA3	V	sp-w-3;pyro-4
073-075			facA303,305,306	V	sp-bi-1
076	T		facB101	VIII	sp-w-3;pyro-4
077-079			facB302,308,309	VIII	sp-bi-1
080	T		facC102	VIII	sp-w-3;pyro-4
081,082			facC301,307	VIII	sp-bi-1
083			fanA3	V	sp-bi-1;facA303
084			fanB52	VII	sp-bi-1;facA303
085			fanD151	VIII	sp-bi-1;facB302
086			fanE7	VI	sp-bi-1
087			fpaA1	I	sp-bi-1
088			fpaA12	I	sp-riboA1 bi-1
089	F		fpaB37	I	sp-riboA1 bi-1
090	F		fpaD11	VIII	sp-riboA1 bi-1
091			fpaD43(=fpaC43)	VIII	sp-riboA1 bi-1
092		59	fr-1	IV	UV-y-2;pyro-4
093	T		galA1	III	UV-bi-1;w-3
094	T	215	galB3	II	UV-bi-1;w-3
095	T		galC4	VIII	UV-bi-1;w-3
096	T		galD5	I	UV-bi-1;w-3
097	T		gal 2	-	UV-bi-1;w-3
098,099	T		glu-1,2	-	UV-bi-1;w-3
0100	F		hxA1	V	Camb. D-bi-1
0101			inoA1	II	UV-bi-1
0102	F		inoB2,w-5	IV,II	NTG-bi-1
0103		58	lacA1	VI	UV-y-2;pyro-4
0104	T		lacB3	II	UV-bi-1;w-3
0105			lu-1	I	UV-bi-1
0106			lu-2	I	NTG-bi-1
0107			lys-4	-	UV-bi-1
0108	F	66	lysB5,sm-1	V,III	UV-bi-1
0109		57	mal-1	VII	UV-y-2;pyro-4
0110		67→219	methG1	IV	UV-bi-1
0111		34	methH2	III	UV-bi-1
0112			methB3	VI	UV-y-2;pyro-4
0113	T		meth-5	-	UV-bi-1;w-3
0114,0115	F		meth-6,7	VII	UV-bi-1
0116			meth-4	-	UV-y-2;pyro-4
0117			nicA2	V	X-wild-type
0118			nicB8	VII	UV-bi-1
0119		11	nicC10	VI	UV-bi-1;AcrA1 w-3
0120			nic-11	-	UV-bi-1
0121,0122			nic-14,15	-	NTG-bi-1
0123			nic-12	-	UV-y-2;pyro-4
0124	T		nic-13	-	UV-bi-1;w-3
0125			niaD15 (= ni7)	VIII	N-bi-1
0126	F		niaD17	VIII	Camb. D-bi-1
0127		(C.222)	niiA4	VIII	Camb. y-2;pyro-4
0128		(C.153)	nir-1	VIII	Camb. y-2;pyro-4
0129			nir-14 (=am) = ni51	VIII	N.Y. bi-1;phenA3
0130		43	ornA4	IV	UV-bi-1
0131,0132			ornB7,8	VIII	UV-bi-1
0133		56	ornB9	VIII	UV-bi-1
0134			orn-2	-	X-bi-1
0135-0138			orn-5,6,10,11	-	UV-bi-1
0139			partial reversion(om)	VIII	sp-bi-1;ornB9
0140	T		pabaA1, co-1	I, VIII	X-bi-1
0141,0142			pabaA2,3	I	UV-bi-1
0143,0144			pabaA4,5	I	X-bi-1
0145		28	pabaA6	I	UV-bi-1
0146			pabaA7	I	UV-y-2;pyro-4
0147,0148			pabaB21,22	IV	UV-bi-1
0149			paba-23	-	N-bi-1;methG1

Glasgow No.	T/F	FGSC No.	Mutant	Chromosome	Origin
0150	F	35	p-2	V	FGSC. sp-adG14 bi-1
0151,0152			phenA4,5	III	UV-y-2;pyro-4
0153,0154			phenA7,8	III	NTG-bi-1;w-6
0155			ppa-1	-	N-bi-1
0156			pacC5	VI	UV-bi-1
0157		32	proA1	I	UV-bi-1
0158		111	proA2	I	UV-bi-1
0159		45	proA5	I	UV-bi-1
0160-0162			proA6,7,8	I	UV-bi-1
0163,0164			proA10,11	I	UV-y-2;pyro-4
0165			proB3	I	UV-bi-1
0166			proB13	I	NTG-bi-1
0167	T		proB4	I	UV-bi-1;w-3
0168			proB9	I	UV-y-2;pyro-4
0169	T		pro-12	-	UV-bi-1;w-3
0170	T		pu-1	II	UV-bi-1;w-3
0171,0172			pu-2,3	II	N-bi-1
0173-0176			pu-4,5,6,7	II	UV-bi-1
0177	F	33	pyro-4	IV	UV-bi-1
0178,0179			pyro-1,2	IV	X-bi-1
0180-0183			pyro-5,6,7,8	IV	UV-bi-1
0184,0185			pyro-9,10	IV	UV-bi-1;~~AcrA1 w-3~~
0186-0188	T		pyro-11,12,13	-	UV-bi-1;w-3
0189		158	riboA1	I	UV-bi-1
0190	F	5	riboB2	VIII	UV-bi-1;AcrA1 w-3
0191			riboB4	VIII	UV-y-2;pyro-4
0192			riboC3	V	UV-y-2;pyro-4
0193			riboD5	V	UV-y-2;pyro-4
0194	T	64	riboE6	II	UV-bi-1;w-3
0195	T		ribo-7	-	UV-bi-1;w-3
0196			ribo-8	-	N-bi-1
0197	T	65	sb-3	VI	UV-bi-1;w-3
0198		41	sB3 → dense	VI	UV-bi-1
0199,0200	T		sB10,11	VI	UV-bi-1;w-3
0201		40	sA1	III	UV-bi-1
0202			sA2	III	UV-bi-1
0203		2	sA4 → dense	III	UV-bi-1
0204-0207	T		sA5,6,8,9	III	UV-bi-1;w-3
0208		24	sC12	III	M-wild-type
0209,0210	T		sC7,13	III	UV-bi-1;w-3
0211			s-16	-	UV-y-2;pyro-4
0212	T		suc-1	-	UV-bi-1;w-3
0213		47	su1adE20	I	sp-adE20 bi-1
0214			su2adE20	-	sp-adE20 bi-1
0215-0217			su6,7,8 methH2	-	sp-bi-1;methH2
0218			su1ornB9	II	sp-pabaA1 adE20 bi-1; w-3 thi-4;ornB9
0219			su1,pabaB22	IV	sp-bi-1;pabaB22
0220			su4 pabaB22	-	sp-bi-1;pabaB22
0221			su1 proA1	III	sp-proA1 pabaA1 bi-1
0222			suB2 palB7	VI	sp-bi-1;palB7
0223	F		tel-1	VII	sp-bi-1
0224			tsC17	II	UV-bi-1
0225		52	thi-4	II	UV-bi-1
0226-0233			thi-2,3,5,6,7,8,9,13	-	UV-bi-1
0234,0235			thi-10,11	-	UV-y-2;pyro-4
0236			thi-12	-	UV-bi-1;w-3
0237,0238			ths-3,5	-	UV-bi-1
0239	F		tyrA7	I	NTG-bi-1;phenA3
0240	F		tyrB1	III	NTG-tyrA7 bi-1
0241	F		wet-6	VII	UV-bi-1
0242	T		w-3	II	sp-pabaA1 bi-1
0243	F		w-4	II	UV-bi-1
0244			w-7	II	sp-bi-1
0245			w-8	II	NTG-2n-bi-1
0246	F		p-3	V	N-bi-1
0247	F		yg-6	II	N-bi-1
0248	F		brl-42	VIII	N-bi-1
0249	T		nir-63	VIII	UV-bi-1;w-3

Glasgow No.	T/F	FGSC No.	Mutant	Chromosome	Origin
0250		217	adB1	VIII	FGSC wild-type
0251	$T_2(I;VIII)$	249	sD50	VIII	FGSC bi-1
0252		237	try C801	VIII	FGSC pabaA1 y-2
0253	T	253	iod-1	II	FGSC bi-1;w-3;nicB8
0254		271	sul-1;dil-1	I,III	FGSC adD3
0255	F		med-15	I	N-bi-1
0256	F		stu-1	I	N-bi-1
0257	T		y-2	I	X-wild-type
0258			y-102	I	Camb. D-bi-1

Standard symbols will be found in Barratt, Johnson & Ogata 1965 (Genetics 52,233) or Dorn 1967 (Genetics 56, 619). Additional symbols are given below.

Symbol	Phenotype	Reference
ac	acetate non-utilization	—
glu	glutamate requirement	Sneath, unpublished
inoA	inositol requirement	Forbes, do.
inoB	do. do.	Clutterbuck, do.
ppa	phenylpyruvic acid requirement	Sinha, do.
suc	succinate non-utilization	Luig, do.
tel	slow growth	Clutterbuck, do.
ths	thiosulphate requirement	Macdonald, do.
tyrA	partial tyrosine requirement	Sinha, 1967 Genet. Res. 10, 261
tyrB	tyrosine requirement (with tyrA)	do. do.
cnx, nia, nii	nitrate and nitrite non-utilization	Pateman & Cove 1969 J. Bact. 97, 1374
nir		
aba	"abacus" morphology	Clutterbuck, Genetics (in press)
brl	"bristle" morphology	do. do.
drk	dark conidia	do. do.
med	"medusa" morphology	do. do.
stu	stunted conidiophores	do. do.
wet	wet-white conidia	do. do.

Recombinants : These are arranged according to the chromosome principally marked, with an additional list of other strains carrying useful markers. Abbreviations are as for mutants. The figures in the tables are allele numbers of the markers present.

Chromosome I

Glas-gow No.	T/F FGSC	Other markers	fpaB	suaE20	rA	med	galD	sul	riboA	cilB	fpeA	anA	adG	lu	stu	proB	proA	adF	pabaA	y	adE	bi	No.
		Mutants	089	0213		0255	096	0254	0189	064 -067	087 088 0239		018 019	0105 0106	0256	0165 -0168	0157 to 0164	012 to 017	0140 to 0146	0244 0245	06 -011	042 051 052	
		Other strains:		78 91-					many			314 316 42	many	311			many	220 818 819	many	many	78 91- 231	many	
11		–		37		15														2			11
12		–		37														17	1	2			12
13		–			1				1					1						2	20		13
14		pyro-4			1				1								1				20	1	14
15	F 71	AcrA1 w-2			1				1										1	2	20		15
16		AcrA1 w-2			1							1					1		1		20	1	16
17		w-2			1												1		1		20	1	17
18		–			1												1		1		20		18
19		–			(?)	1		5				1		1						2			19
110		–			(?)			5				1		1								1	110
111		w-2;pyro-4							1			1	14				1					1	111
112		w-2;pyro-4							1			1					1					1	112
113		pyro-4							1			1							1	2	20		113
114		methG1							1			1							1	2			114
115		–							1				14	1						2			115
116	F ?	–							1				14				1		1	2			116
117		–							1				14				1		1			1	117
118		–							1				14				1			2			118
119	F ?	–							1				14									1	119
120		glu-1							1				14							2			120
121		w-3							1								1		1		20	1	121
122		pyro-4							1								1			2	20		122
123		–								6		1	14						1	2			123
124		–									1		14						1	2			124
125		–									12							17	1	2			125
126		–									1								1	2			126
127		w-3 adC1;sC12										1					1		1			1	127
128		w-2										1								2	20		128
129		–											14		1				1	2			129
130		w-3											14				1		1			1	130
131	F? 72	w-3;ve+											14				1		1	2			131
132	F	–											14						1	2			132
133		–											14						1			1	133
134		–												1	1		1						134
135		adD3												1						2			135
136		w-3														4			1	2			136
137		–														4			1			1	137
138		w-3;adC1														4				(+?)		1	138
139		–															1	17	1	2			139
140		w-3															1	15	1	2			140
141		–															1	9	1			1	141
142		–															1	17				1	142
143		w-2;pyro-4															1		1	2	20		143
144		–															1		1	2		1	144
145		–															1		6	2			145
146	F	ve+															1		1	2			146
147		w-3															1			2			147
148		–																13	1	2			148
149		–																9	1	2			149
150		–																32	1	2			150
151		w-3																33	1	2			151
152		–																17	1	2		1	152
153		w-3																17	1	(+?)		1	153
154		–																	1	2	20	1	154
155		nicA2																	1	2	11		155
156		pyro-4																	1	2	8		156
157		–																	1	2		1	157
158		pyro-4																	1	2			158
159		–																	1			1	159
160		–																	20			1	160
161		–																		2		3	161
162		(light)																		2		1	162
163		–																		2		1	163
164		–																				3	164

Chromosome II

Glas-gow No.	T/F FGSC	Other markers	iod	adH	AcrA	w	palcA	anB	riboE	thi	pu	ab	cnxE	tsC	adC	adD	yg	lacB	acrB	bl	suornB	inoA	galB	No.
		Mutants	0253	020	03 04	0242- 0245		025 026	0194	0225	0170 -0176	024	058 059	0224			0247	0104	05	043 -048	0218	0101	094	
		Other strains:		824 828 829 830	15 16 41 91-	many				43					127 138 910 914	135 315								
21		y-2		23	1	3	2																	21
22		bi-1; nicB8		23	1	3																		22
23		proA1 pabaA1 y-2;ve+		23		3																		23
24		pabaA1 y-2		23																				24
25		riboA1 bi-1			1	3				4			16			3								25
26		bi-1;pyro-4			1	3					1					3								26
27		bi-1			1	3						1	16			3								27
28		bi-1			1	3																		28
29		riboA1 bi-1			1					4		1	16			3								29
210		proA1 bi-1			1											3								210
211		proA1 y-2			1																			211
212		bi-1			1	3	1																	212
213		bi-1				3			6		1													213
214		–				3					1				1									214
215		bi-1;pyro-4				3					1				1									215
216		y-2				2				4			16			3				1				216
217		riboA1 bi-1				3				4			16											217
218		bi-1				3										3								218
219		proA1 pabaA1 y-2;ve+				3									1									219
220		adF33 pabaA1 y-2				3													2					220
221		proA1(y+?)bi-1				3													2					221
222		y-2					2		6	4						3				1				222
223		–					?		6	4							6			?				223
224		bi-1							6		1													224
225		y-2;pyro-4													1									225
226		bi-1;pyro-4													1				2					226
227		pabaA1 y-2																		4				227
228		pabaA1 y-2; ornB9																			1			228
229	(C335)	y-2											14											229
230		y-2 bi-1								1														230
231		adE20 bi-1;sC12;pyro-4			3																			231
232		proA1 y-2																	2					232
233		–			1	7			6	4		1	16				6							233

Chromosome III

Glasgow Number	FGSC T/F	Other markers	palcB	methH	argB	palA	galA	sm	phanA	sC	sA	cnxH	suproA	dil	tyrB	No.
		Mutants		0111	034		093	0108	0151 to 0154	0208 to 0210	0201 to 0207	063	0221	0254	0240	
		Other strains:				91-	59 94- 96			127 231 45 58 813 98-						
31		riboA1 adG14 y-2	3													31
32		y-2		2	2	1										32
33		y-2;w-3		2	2		1									33
34		y-2		2	2											34
35		y-2		2					2							35
36		(y-2?);w-3;facA303			2		1									36
37		riboA1 adG14 y-2			2											37
38		bi-1				1	1	1	2							38
39		y-2;lysB5						1	2							39
310	F	bi-1							3							310
311	F	lu-1 bi-1							3							311
312	F	y-2;w-2;ve^{+}								12						312
313	F	y-2;w-2;pyro-4								12						313
314		anA1 y-2;w-2;pyro-4									2					314
315		y-2;adD3									1					315
316		anA1 y-2;pyro-4									1					316
317		bi-1;w-2;pyro-4									1					317
318		bi-1;w-2;pyro-4									2					318
319	(C333)	y-2;pyro-4										4				319

Chromosome IV

Glasgow Number	T/F FGSC	Other markers	methG	fr	palC	inoB	pabaB	pyro	ornA	pacA	clA	su pabaB22	No.
		Mutants	0110	092		0102	0147 0148	0177- 0185	0130		068	0219	
		Other strains:	114 910					many	911				
41		proA1 bi-1;AcrA1	1					4					41
42		riboA1 anA1 adG14 pabaA1 y-2	1					4					42
43		riboA1 pabaA1 y-2; thi-4	1						4				43
44	#250	y-2		1	4		22	4					44
45		y-2; sC12		1			22	4			4		45
46		y-2		1			22	4					46
47		-		1			22	4					47
48		bi-1						7	4				48
49		bi-1						6	4				49
410		adG14 proA1 bi-1						4	4				410
411		riboA1 bi-1						8	4				411
412	F	y-2						4					412
413		riboA1 pabaA1 y-2							4				413
414		bi-1								1			414

T(IV;VIII)

Chromosome V

Glasgow number	T/F FGSC	Other markers	lysB	nicA	p	facA	hxA	riboD	argD	fanA	No.
		Mutants	0108	0117	0150 0246	072- 075	0100	0192 0193	036	083	
		Other strains:	39 91-	155 98 911		36 94- 96					
51		bi-1	5					5			51
52		adG14 y-2	5								52
53		y-2;methB3		2	3	303		5			53
54		y-2		2		303		5			54
55		y-2		2				5			55
56		(y^{+}?)bi-1		2				5			56
57		bi-1;w-3		2				5			57
58		y-2;w-2;sC12		2							58
59		y-2;w-3;galA1				303					59

Chromosome VI

Glasgow Number	T/F FGSC	Other markers	sB	lysA	lacA	tsB	nicC	methB	argA	sb	cnxG	pacC	subpalB7	fanE	No.
Mutants			0198-0200		0103		0119	0112	033	0197	061	0156	0222	086	
Other strains:			92-					53							
61		y-2	3		1		10			3					61
62		w-3	3			5				3					62
63		y-3	3			5									63
64		w-3	3			5									64
65		-		1											65
66		y-2			1	5				3					66
67		bi-1			1	5									67
68		y-2;pyro-4			1	5									68
69		bi-1;pyro-4			1	5									69
610		y-2				5				3					610
611		bi-1					10								611
612		y-2;w-2;ve+							1						612
613		pabaA1 y-2							1						613

lysD20

Chromosome VII

Glasgow Number	T/F FGSC	Other markers	palD	nicB	wet	mal	palF	cho	drkA	phenB	tel	pantoB	meth	cnxF	fanB	No.
Mutants				0118	0241	0109		069	070		0223		0114	060	084	
Other strains:			22 91-													
71		y-2	8	8		1					1					71
72		riboA1 bi-1	8	8												72
73		y-2		8			15				1					73
74		riboA1 y-2		8			15									74
75		(suladE20?) y-2		8				1	1							75
76		riboA1 y-2		8												76
77		proA1 pabaA1		8												77
78	75	suladE20 adE20 bi-1;w-2;cha-1						1								78
79		bi-1								6						79
710		-										100	6			710
711		-										100				711
712		y-2	8	8	6	1										712

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Chromosome VIII

Glasgow No.	T/F FGSC	Other markers	ve	fw	ornB	co	sD	facC	cnxB	niaD	niiA	adB	brl	fpaD	palcC	argC	facB	riboB	tryC	hisC	nir	pacB	palB	cha	palE	aba	tsD	galC	fanD	No.
Mutants				071	0131 to 0133 0139	0140	0251	080 to 082	055 053 056	0125 0126	0127	0250	0248	090 091		035	076 to 079	0190 0191	0252		0249 0128 0129			050		01		095	085	
Other strains: many					228													92 - 96						78						
		-	+	1	7												101	2									15	7	81
		pabaA1		1													101	2										7	82
		pabaA1		1																									83
		bi-1			10	1																							84
		pabaA1 y-2			8	1																							85
		pabaA1			7												101	2									15	7	86
		pabaA1;w-3			7												101	2									15	7	87
		pabaA1			7												101	2					7					7	88
		pabaA1;w-3			7												101	2										7	89
		bi-1			7													2											810
		adG14				1																							811
		adG14 y-2				1																							812
		y-2;sC12						307																					813
		y-2							11					43															814
		pabaA1							11															1					815
		y-2								15			42	43															816
		y-2;pyro-4								17																			817
		adF17 pabaA1 y-2												11 43															818
		adF17 pabaA1 y-2													4													7	819
		pabaA1 y-2														3	101	2											820
		pabaA1;w-3															101	2					7	1			15	7	821
		pabaA1															101	2					7	1				7	822
		pabaA1															101					4		1					823
		adH23																		801 38									824
		pabaA1 bi-1																		38									825
		pabaA1 bi-1																					7						826
		bi-1																						1	11	14			827
		adH23																						1	11				828
		adH23																						1		14			829
		adH23																											830

Master Strains

Linkage Groups

Glasgow Number	T/F	Designation	I	II	III	IV	V	VI	VII	VIII
91		M.S.A.	suladE20 y-2 adE20	AcrA1	phenA2	pyro-4	lysB5		nicB8	
92		M.S.C.	suladE20 y-2 adE20	AcrA1				sB3		riboB2
93		M.S.D.	suladE20 y-2 adE20	AcrA1	phenA2	pyro-4	lysB5	sB3	nicB8	riboB2
94	F	M.S.E.	suladE20 y-2 adE20	w-3	galA1	pyro-4	facA303	sB3	nicB8	riboB2
95	F	M.S.F.	suladE20 y-2 adE20	AcrA1	galA1	pyro-4	facA303	sB3	nicB8	riboB2
96	F	M.S.G.	(suladE20?)		galA1	pyro-4	facA303	sB3	nicB8	riboB2
97	F?		riboA1 y-2 bi-1	AcrA1	phenA2			sB3	nicB8	
98			y-2		sC12	pyro-4	nicA2	sB3		
99			bi-1	AcrA1	phenA2		lysB5	sB3	nicB8	
910			bi-1	adC1	sC12	methG1				
911			bi-1	w-2	sC12	ornA4	nicA2			
912	F		bi-1	w-2	phenA2		lysB5	sB3		
913	F		bi-1	AcrA1	phenA2		lysB5	sB3		
914	F?		bi-1	adC1	sC12	pyro-4				
915			suladE20 adE20 bi-1	AcrA1	sC12	pyro-4			nicB8	
916			(y-2?)	AcrA1 w-2	phenA2		lysB5	sB3		

Strains to be discarded:

adD3
pabaA1 adE8
adF17
riboA1 adG14 proA1 pabaA1
riboA1 proA1 pabaA1
pu-1
bi-1;phenA2;lysB5;sB3;nicB8
proA1(pabaA1?) adF15 bi-1
anA1 proA1 bi-1; AcrA1;sC12
riboA1 adG14 bi-1;thi-4;ornA4
adF17 pabaA1 bi-1
riboA1 adG14 bi-1;sm-1
proA1 adF15 bi-1
anA1 proA1 bi-1;pyro-4
anA1 proA1 bi-1;AcrA1;pyro-4
adF17 pabaA6 bi-1
anA1 bi-1;sA2;pyro-4
anA1 proA1 bi-1;AcrA1;sC12;pyro-4
suladE20 riboA1 adE20 bi-1
suladE20 riboA1 proA1 adE20 bi-1;AcrA1
riboA1 proA1 bi-1;AcrA1 thi-4 cnxE16 adD3
bi-1;AcrA1 adD3
proA1 adF9 bi-1
proA1 pabaA6 bi-1
riboA1 bi-1;thi-4;ornA4
bi-1;thi-4;ornA4
bi-1;ornB10 riboB2
bi-1;sC12;pyro-4
bi-1;pu-1;sC12;pyro-4
bi-1;pu-1
bi-1;bl-5
bi-1(recombinant FGSC 240)
clB5 bi-1
bi-1;phenA2;lysB5
bi-1;phenA2;pyro-4;lysB5;sB3
proA1 bi-1;AcrA1;pyro-4
riboA1 bi-1;thi-4
riboA1 proA1 bi-1
suladE20 y-2 adE20;AcrA1;riboB2
pabaA1 y-2;adD3;ornA4
riboA1 adG14 proA1 y-2;ornA4
y-2;adD3
riboA1 adG14 y-2;phenA2
suladE20 y-2 adE20;AcrA1;pyro-4
suladE20 y-2 adE20;AcrA1;sB3
adG14 pabaA1 y-2;ornB9(partial reversion)
adF17 pabaA1 y-2
pabaA1 y-2 adE20
anA1 y-2;AcrA1;sC12;pyro-4
suladE20 pabaA1 y-2 adE20;AcrA1
riboA1 adG14 pabaA1 y-2
y-2;AcrA1 adD3;sC12;pyro-4
adF17 y-2
y-2 adE8
y-2;AcrA1 adD3
anA1 y-2;AcrA1 adD3;pyro-4
adG14 pabaA1 y-2;methG1
riboA1 adG14 pabaA1 y-2;methH2 phenA2
riboA1 adG14 y-2;sm-1 phenA2;lysB5
suladE20 y-2 adE20;AcrA1;pyro-4;riboB2
y-2;AcrA1 adD3;co-1
pabaA1 y-2;ornB10 co-1
pabaA1 y-2;ornB9(partial reversion)
pabaA6 y-2;nicA2
pabaA1 y-2;co-1
proA1 adF15 pabaA1 y-2
proA1 pabaA1 y-2 adE20;adC1
y-2;sC12;pyro-4
proA1 pabaA1 y-2;phenA2
clB6 proA1 pabaA1 y-2
w-3 pu-1
bi-1;w-3 pu-1
bi-1;w-3 pu-1 adC1
bi-1;AcrA1 w-3 adD3
pabaA1 bi-1;w-3 pu-1 adC1;pyro-4
proA1 y-2;AcrA1;pyro-4;nicA2
proA1 pabaA1 y-2 bi-1;AcrA1;methG1
proA1 adF17 pabaA1 y-2 bi-1
riboA1 proA1 pabaA1 y-2 bi-1
proB4 pabaA1 y-2 bi-1
proA1 pabaA1 y-2 bi-1;pyro-4
proA1 pabaA1 y-2 bi-1;pyro-4 methG1
anA1 y-2 adE20 bi-1;sC12
suladE20 pabaA1 y-2 adE20 bi-1;AcrA1
anA1 y-2 bi-1;AcrA1 adD3;sC12
anA1 y-2 adE20 bi-1
adG14 pabaA1 y-2 bi-1
y-2 bi-1;sC12
y-2 bi-2
bi-1;w-3
pabaA1 y-2;w-3
anA1 y-2;w-2;pyro-4
proA1 y-2;w-3 adC1
pabaA1 y-2;w-3 adC1
adF17 pabaA1 y-2;w-3
adF15 pabaA1 y-2
adF15 pabaA1 y-2;w-3
y-2 adE20;w-2;fpaD11
proA1 pabaA1 y-2;w-3 adC1;sC12;methG1
pabaA1 y-2 bi-1;w-3
pabaA1 y-2;AcrA1 w-3
w-3 adD3
anA1 pabaA1 y-2 adE20;w-2;pyro-4
anA1 pabaA1 y-2 adE20;w-2;sC12
anA1 pabaA1 y-2 adE20;w-2;sC12;pyro-4
anA1 y-2;w-3 adC1;sC12
suladE20 riboA1 pabaA1 y-2 adE20;w-2
adF15 pabaA1 y-2;w-3 acrB2
adG14 pabaA1 y-2;w-3;methG1
riboA1 anA1 pabaA1 y-2;w-2;methG1
riboA1 adG14 pabaA1 y-2;w-2;methG1
riboA1 adG14 proA1;w-2
riboA1 anA1 proA1 adE20 bi-1;AcrA1 w-2
anA1 proA1 bi-1;w-3;sC12;pyro-4
anA1 bi-1;w-3;sC12;pyro-4
anA1 y-2 adE20 bi-1;w-2;pyro-4
anA1 bi-1;w-2;pyro-4;sB3
adG14 bi-1;w-3
adG14 proA1 y-2;w-3;methG1
adG14 proA1;w-3
adG14 proA1 pabaA1 y-2;w-3 (FGSC86)
proA1 pabaA1 bi-1;w-3
proA1 pabaA6 bi-1;w-3
proA1 pabaA1 y-2;w-3;methG1
proA1 pabaA6 y-2;w-3
proA1 pabaA1 bi-1;w-3 adC1
proA1 y-2 (adE20?);w-3 adC1;sC12
proA1 y-2;w-3;pyro-4
proA1 adF17 y-2;AcrA1 w-3
adF17 pabaA1(y-2?);AcrA1 w-3
adF17 bi-1;AcrA1 w-3
w-3;pyro-4
bi-1;w-3;pyro-4
clB3;w-2 bl-2;sC12
pabaA1;w-2 bl-2
pabaA1;w-2 bl-3;sC12
pabaA1;w-3 sulornB9;ornB9 riboB2
bi-1;AcrA1 w-3;ornB8 riboB2
bi-1;AcrA1 w-3;ornB10 riboB2
bi-1;w-3 sulornB9 riboB2 ornB9
bi-1;phenA2;lysB5 riboD5
y-2;w-2;phenA2;lysB5;sB3
bi-1;w-2;pyro-4;lysB5;sB3
bi-1 adE20;w-2;sC12;ornA4;nicA2
bi-1;w-3 thi-4 cnxE16;ornA4
pabaA1 bi-1;adH23 w-3

List of gene symbols, locus letters and allele numbers which have been used or suggested in Aspergillus nidulans up to May 1970.

Martinelli, I

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

The list is necessarily incomplete and inaccurate and ammendments will be welcomed.

Symbol	Phenotype	Locus letters	Allele numbers	References
as	allantoic acid util.	X	?	D.J. 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
ad	adenine req.	A-I	1-44,50,51	1
al	allantoin util.	X	?	D.J. Cove, request
alp	allantoin permease	A?	?	D.J. Cove, request
am	~~ammonium req~~ = niiB or niiA	-	-	2 ~~(obsolete)~~
an	aneurin req.	A,B	1-8	1,2
ap	aminopterin res.	A	1	Apirion, Dorn & Forbes ANL 4.15
apl	allpurinol + purine res.	A	?	D.J. 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?	?	D.J.Cove request
bge	=fwA2	-	-	(Wood & Käfer) ~~unknown~~
bi	biotin req.	A	1-4	1
bl	blue ascospores	A	1-6	1
br	=brlA	-	-	xxxxxxxxxxxxxxxx 14
brl	bristle - aconidial	A	1-43	3
Bw	brown conidia	A	1	5
cha	chartreuse conidia	A	1-3	1, Käfer, Clutterbuck requests
cho	choline req.	A	1,2	1,2
cl	colourless ascospores	A,B	1-6	1
cnx	nitrite req.purine util.	A-H	1-16?	4
co	compact growth	A	1	1
cys 2	=sC13	-	2	x 5
dil	dilute conidial colour	A	1	1
drk	dark conidial colour	A,B	1-5	3
est	esterases	A	1,50,51	G.Dorn & W.Rivera ANL6.1
f	=fac	-	-	Apirion et al ANL4.15
fac	fluoroacetate res. acetate non-util.	A-C	2-4,101,102 301-309	1
fan	fluoroacteate res.	A-E	1-16,52,54,60,101,151	1
fl	fluffy	A	1	C.Ball & D'Azevedo ANL5.
fp	xxxxxxxxxxxxxxxxxxxxxxxx =fpa	-	-	1
fpa	fluorophenylalanine res.	A-D	1-58	13,14
fr	fructose non-util.	A	1-3	1

fw	fawn conidia	A,(B) xxx 1,2,10		1,Käfer,request,P.R.da Cunha ANL10.12
gal	galactose non-util.	A-D	1-36	1
gam	galactose non-util., molybdenum res.	A-D	?	D.J. Cove, request
glu	glutamate req.	A	1,2	2
his	histidine req.	A-J	1,10,13,38 +?	1,16
		AL-FL	2,8,14,100,115,122	E.Pees,ANL 7.11
hx	hypoxanthine 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?	1,E.Pees ANL 7.11
m	mycelial growth	?	1-6	15, Kwiatowski ANL 3.3
mal	maltose non-util.	A	1,2	1
mau	methylamine non-util.	A?	?	D.J. Cove, request
mea	methylammonium res.	A?	?	" " "
med	medusa - morphological	A	1-30	3
mel	melanin formation	A,B	1,2	S.D.Martinelli ANL 10.24
meth	methionine req.	A-H	1-66,101-106	1,2,6,11
mg	malachite green res.	A	1	~~J.R.Warr ANL 4.22~~ 12
mo	morphological	A-C	1,9,50,87,89,96	1 B.W.Bainbridge ANL4.20; 7.19
mol	molybdate res.	A,B	?	D.J.Cove, request
ni	xxx =nia or cnx	-	3,7,50,51	1
nia	nitrate reductase	D	15,17 +?	4
nic	nicotinamide req.	A-C	1-15	1,2
nii	nitrite reductase	B	?	7
nir	nitrate path regulator	A	?	7
nr	=nuvs	-	1,2	8
orn	ornithine req.	A,B	1-11,20,21	1,2,G.L.Dorn,request
ota	ornithine transcarbamylase	A	1	Piotrowska ANL 9.18
p	pale conidia	A	1-3,12	1,A.J.Clutterbuck ANL 9.14, G.L.Dorn,request
paba	p-aminobenzoic acid req.	A,B	1-23,101-126	1,G.J.O.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	pantothenate req.	A,B	1,100	1,2
pcnb	pentachloronitrobenzene res.	A,(B?)	1-3	9
pf	flucrophenylalanine res.	-	21,22	12
pfp	" " " "	-	1-104?	C.de Palma & G. Morpurgo ANL 4.11, M.G.Petrelli et al ANL 5.7
phen	phenylalanine req.	A,B	1-11,1481	1,13,G.L.Dorn, request.
ppa	phenylpyruvate req.	A	1	2
pro	proline req.	A,B	1-16	xx 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	1-16,50	1
sb	sorbitol non-util.	A	1-9	1, Roberts ANL 2.13
sor	sorbose res.	A,B	?	D.J.Cove request
su..adE20	suppressors	A	1,2	1,2
su..meth		-	1-68	6,2
su..orn89		A	1	2
su..paba822		A	1-4	1
su..pal87		A,B	1,2	1
su..palF15		C	1,6	1
su..palA1		D	1,2,5	1
su..pro		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
tcnb	tetrachloronitrobenzene res.	A	1	9
ts	teoquil res.	A	1 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, 403	13;Dorn request.
u	urea utilization	X-Z	5+?	1; Cove request
ua	urate util.	X	1	1
uap	uric acid permease	A?	?	Cove request
uvs	ultra-violet sensitive	A?	1,4,5,7,77+?	17, Tuveson request
		B-F?	101-195?	1,Jansen request
		?	201-?	Käfer request
ve	velvet morphology	A	1	1
w	white conidia	A	1-12	1,2,Käfer 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

Addendum

Act	actidione res.	A	1	1
amd	acetamidase	(A),R	?	Pateman request
fmd	formamidase	(A)	?	" " " "
1476 sta	stunted conidiophores	A	1-4	3

References: 1 G.L.Dorn Genetics 56.619 (1967)
2 Stocklist ANL 10.30 (1969)
3 A.J.Clutterbuck Genetics 63.317 (1969)
4 J.A.Pateman et al Nature 201,58 (1964)
5 Käfer,E. Adv. Genetics 9.105 (1958)
6 W.Gajewski & J.Litwinska,Mol. Gen. Genetics 102.210 (1958)
7 J.A.Pateman & Cove,D.J. J.Bact. 97.1374 (1969)
8 R.Threlfall, J.Gen. Microbiol. 52.35 (1968)
10 P.Weglenski, Genet. Res. 8.311 (1966)
11 P.Ayling, Genet. Res. 14.275 (1969)
12 J.R.Warr & J.A.Roper, J.Gen.Microbiol. 40.273 (1965)
13 U.Sinha Genet.Res. 10.261 (1967)
14 U.Sinha Genetics 62.495 (1969)
15 J.A.Roper CSHSQB 23.151 (1958)
16 M.Berlyn Genetics57.561 (1967)
17 Lanier & Tuveson Mut. Res. 5.23 (1969)

1969 Sent out with ANL 10

The following questionnaire has been drawn up by several Aspergillus workers. I should be grateful if people engaged in research with Aspergillus would kindly complete this and return it to me as soon as is convenient.

J. A. Roper.

In 1956/57 Professor Pontecorvo and co-workers decided on a system of gene symbols for Aspergillus nidulans which would facilitate typing and printing, especially of diploids (i.e. no hyphen, first mapped isolation number becomes gene number etc.) This system was then used in the papers of Advanc. Genet. 9: 71-145, which include a list of all mutants mapped at that time . It has by now become clear that in certain respects this system is unsatisfactory and certain modifications have already appeared in print. In an attempt to keep a unified system of nomenclature, two suggestions have been presented: 1) by Dr. Sermonti (ANL 9: 24-26) with modifications and additions by Dr. Clutterbuck in ANL 10: 26-29); 2) by Dr. Clutterbuck (ANL 9: 26-29). Since both of these have their merit and adoption of either one would be an improvement, as long as all Aspergillus workers adopt the same one, we would like to find out the general preferences with the following questionnaire.

1) Do you agree that modification of the nomenclature system of Aspergillus should be kept uniform for all publications in which strains derived from the Glasgow wild type strain have been used.? Yes:_____ No:_____

2) Would you agree to use the system which emerges as the preferred one from this questionnaire ? Yes:_____ No:_____

3) What fraction of your research uses A. nidulans ?
Small_____ Major_____ All_____

4) Dr. Sermonti's proposal suggests adoption of the bacterial nomenclature system. Do you favour:

_____ a) its adoption for Aspergillus independent of decisions about nomenclature in other fungi or

_____ b) adoption for Aspergillus if all fungal geneticists can agree to this system or

_____ c) do you prefer the system suggested by Dr. Clutterbuck, which adopts the major features of the bacterial system but retains existing abbreviations ?

If you know of any omissions in either system, please send your information in with this questionnaire.

5) To avoid confusion in the assignment of gene symbols and isolation (or allele) numbers Professor Roper has agreed to operate a clearing house through ANL.

Do you think this is a good idea and would you be prepared to make use of it (also retrospectively): Yes:_____ No:_____

If yes, please will you fill in below a list of symbols of new loci and of isolation numbers of all mutants in use in your laboratory which are not represented in the FGSC (Barratt et al. 1965, Genetics 52: 233; Dorn 1967, Genetics 56: 619 and ANL 10) or in the Glasgow stocks listed in this issue.