

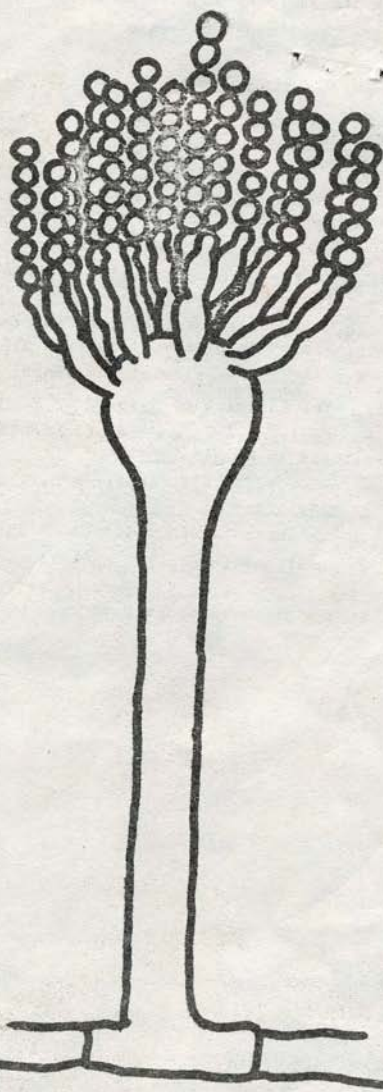
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E.I.C.



ASPERGILLUS NEWS LETTER

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Dear Colleagues

I would first like to repeat my thanks, on behalf of all *Aspergillus* workers to Professor J.A. Roper for the service he has performed in compiling the first eleven issues of this Newsletter.

Secondly I must apologize for the long delay since I received material for this issue. I asked for these contributions at very short notice last summer in the hope that I could produce an issue before the start of the academic year. However I failed in this and have only now been able to get down to it.

In order to avoid a repetition of this problem, I would like to ask for material for the next issue now: to reach me by July 1st 1975. I will use the present mailing list, so please send:
additions and alterations to the mailing list,
publication lists for *Aspergillus* and related organisms,
material for publication.

As in the present issue, material will be reproduced photographically, so it should be typed in final form - this time in columns 7½ ins (19cm) wide and tables and diagrams should also fit this width and be suitable for photographic reproduction. Layout of headings should be as in this issue.

John Clutterbuck

GENE SYMBOLS

I am taking over the services of the clearing house for gene symbols for *Aspergillus nidulans* as an adjunct to the Newsletter. If workers therefore send me their proposed symbols before publication I will try to maintain a running list and look out for conflicts or ambiguities.

I have published my proposed system for gene symbols tried out in ANL 9: A.J. Clutterbuck, 1973 Gene symbols in *Aspergillus nidulans*. Genet. Res. 21: 291-296.

Lists of loci and maps have also appeared in:
Clutterbuck, A.J. & D.J. Cove 1974 Linkage map of *Aspergillus nidulans*. in Handbook of Microbiology IV Microbial metabolism, genetics and immunology pp. 665-676, ed. A.I. Laskin & H.A. Lechevalier, C.R.C. Press Ohio.

A later version is also found in:
Clutterbuck, A.J. 1974 *Aspergillus nidulans*. in Handbook of genetics I. pp.447-510, ed. R.C. King, Plenum Press, New York.

A.J.C.

PUBLICATIONS

I have recently published what is intended to be a guide to the bibliography of A. nidulans: "Aspergillus nidulans" in Handbook of genetics I. pp.447-510. Ed. R.C. King, Plenum Press, New York 1974.

I hope to be able to keep this bibliography up to date by publishing supplements in the Newsletter. For this reason I have divided the following publications list into three sections: 1. New A. nidulans publications not in the Handbook.

2. Submitted A. nidulans publications which are in the Handbook.

3. Publications which concern related organisms.

Due to the length of these lists I have not included submitted articles on organisms not closely related to Aspergillus - I apologize to their authors.

(Incidentally, I have just 25 reprints of the Handbook article and am willing to send single copies to laboratories actually working on A. nidulans.)

A.J.C.

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RESEARCH NOTES

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Cytoplasmically determined barrage reaction in *A. nidulans*.

Wild-type and mutant haploid strains of *A. nidulans* maintained as agar slope cultures are often seen to segregate for a genetical difference which controls the presence or absence of a gap or 'barrage' (often pigmented) about 1 m.m. wide at the junction between two colonies which are allowed to grow into each other. Mycelia with the same factor grow imperceptibly into each other. Those carrying the alternative factors form the 'barrage'. Within any one heterokaryon compatibility group of isolates two alternative factors appear to exist but because of difficulties in scoring this character between pairs of strains in different heterokaryon compatibility groups it is not certain if it is the same two alternative factors which occur in all groups. Both of the factors are relatively stable but each may revert to the alternative form with about the same probability, especially after storage on slope cultures.

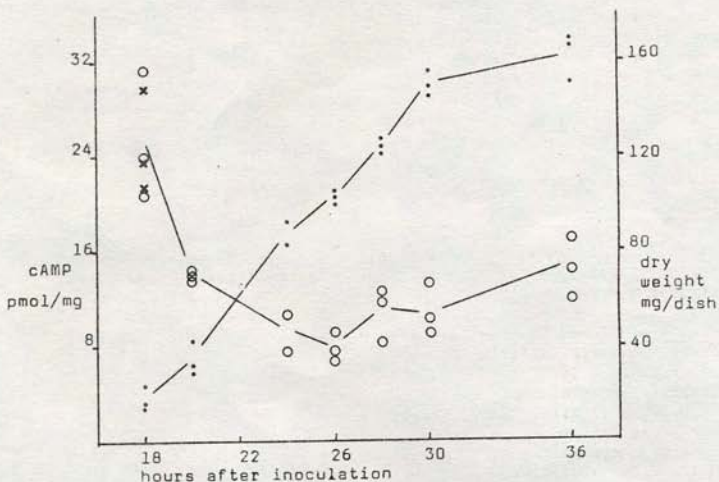
There is evidence that the location of the factor is extranuclear. For the purpose of illustration let us call the alternative factors α_1 and α_2 . Heterokaryotic conidial heads formed from strains which were green spored α_1 and yellow spored α_2 (and reciprocal combinations) gave rise to colonies which were all α_1 regardless of conidial colour. Similarly hybrid cleistothecia between these or similar strains gave ascospore progenies all of which were either α_1 or α_2 .

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Cyclic AMP levels during growth and conidiation

In order to see whether cyclic AMP levels vary markedly during conidiation, the protein binding assay of Gilman et al. (Proc. Nat. Acad. Sci., 1970 67:305-312) was applied to petri-dish cultures of the *bia1* strain (Glasgow strain G051) of *A. nidulans*.

Cultures were grown from 10^5 conidia per dish in 4ml of liquid complete medium over 20ml of complete agar. They were incubated on a level shelf at 37°. The whole mycelium from each dish was extracted with cold trichloroacetic acid, the remaining pellet being used for dry weight measurements. The trichloroacetic acid was removed by ether extraction and cyclic AMP estimated by competition with ^3H labelled cyclic AMP for binding to Sigma protein kinase.



Cyclic AMP and dry weight in surface culture. Each point represents one estimate from a single dish. Dry weight: .—., cyclic AMP: o—o and x.

The results shown in the graph demonstrate an initial high level, falling sharply before conidiation starts around 20 hours, a slight rise then follows.

In a second experiment, the estimates for 18 hours were repeated (x on the graph) and compared with estimates from shaken liquid cultures. The latter were grown in liquid complete medium, 120ml in one litre round bottomed flasks shaken at 100rpm on a gyrotory shaker at 30°. The inoculum was 1.2×10^6 conidia per flask. The results were as follows:

Hours growth	Dry weight per flask (mg)	Cyclic AMP (pmol/mg)	
10	38	39	The cyclic AMP values are the mean of two estimations on single cultures.
12.5	69	18	
14.25	120	25	
20	275	10	

Although somewhat erratic, these results appear to indicate that the decline in cyclic AMP level also occurs during exponential growth.

In a final experiment, extracts from 18 hour cultures were purified by passage through Bioigel 50W-X4. There was no evidence of significant quantities of any interfering material.

From these results it is apparent that no striking changes in cyclic AMP levels occur during conidiation, although the initial fall could play a part in leading to competence to conidiate.

Voichick et al. (J. Bact., 1973 115:68) have obtained very similar results for Tetrahymena. They conclude that in this organism cyclic AMP stimulates glycogenolysis but does not affect gluconeogenesis. High levels of cyclic AMP are therefore to be expected during periods when reserves are being mobilised (e.g. during germination), but not when such reserves are being synthesised. Such reasoning could also apply to Aspergillus.

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Benlate-induced haploidisation in diploid strains of *Aspergillus nidulans* and *Penicillium chrysogenum*.

Hastie (1970) induced haploidisation in *Aspergillus nidulans* by using the fungicide benlate (methyl 1-(butyl carbamoyl)-2-benzimidazole carbamate). We have also experimented with this compound and have examined haploids induced by para-fluorophenylalanine (PFA) and benlate in a test diploid synthesized from strain G38 (suA1adE20 ya2 adE20; acrA1; galA1; pyroA4; facA303; sB3 npeA02; nicB8; riboB2) and GH23 (biA1; cnxH). The final concentration of benlate and PFA in complete medium agar (CM) was 1.6ppm and 70ppm respectively. Diploid conidia of *A. nidulans* were stab-inoculated into the test agar.

Segregants appeared during vegetative growth. These were purified on CM and the haploids tested for joint segregation of markers. Segregation patterns were similar after treatment with benlate and PFA. In addition, the results with both provided evidence for a I;III chromosomal translocation in strain G38.

Ball (1976) has published a technique for inducing haploidization in industrial strains of *Penicillium chrysogenum* using PFA. We have synthesized diploids from genetically-labelled strains derived from the wild-type isolate of *P. chrysogenum*, strain NRRL 1951 and have demonstrated successful haploidization with benlate. Using a stab-inoculation method a suitable final concentration of benlate in CM was found to be 0.15 ppm. Alternatively, approximately 100 diploid conidia were spread on the surface of CM containing a reduced level of benlate (0.1 ppm). With the latter method 50-60% survival was found as estimated by the number of conidia forming colonies.

Many diploids in both *A. nidulans* and *P. chrysogenum* heterozygous for different genes controlling conidial colour have been synthesized. After growth on medium containing benlate coloured sectors were found to be haploid in all cases. Moreover, the coloured sectors were more easily distinguished on benlate than on PFA.

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Benzamide inhibited mutants of A. nidulans

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In the course of isolation of mutants unable to utilize benzamide as the sole nitrogen source (Hynes, in preparation) a series of mutants that are inhibited by benzamide have been found. The preliminary characterization of these is described here.

All mutants were isolated after N.G. treatment of conidia and replica-plating from glucose - 10mM NH_4^+ medium to glucose - 10mM benzamide medium. The mutants all show a similar pleiotropic phenotype - inhibition by benzamide and some other aromatic compounds (phenylacetamide, nicotinamide), inhibition by 10mM lysine and extremely weak growth on acetate as the sole carbon source. In addition, most mutants have a small abnormal morphology on all media (including complete) and there is poor recovery of mutants from crosses and from haploidization experiments. The pleiotropic effects segregate together in crosses and a spontaneous revertant of one mutant has been found to be revertant for all phenotypes. The mutants are recessive and have been provisionally designated bin mutants. Table 1 summarizes the genetic characterization of these. Not all mutants have been assigned to specific loci as comprehensive tests for allelism have not been carried out. It has been reported that lacD27 has pleiotropic effects on the utilization of carbon sources,

Table 1: Genetic characterization of bin mutants.

<u>Mutant no.</u>	<u>Designation</u>	<u>Linkage group</u>
TB-1	<u>binA1</u>	VI
TB-4	<u>bin-4</u>	I - close linkage with <u>yA1</u>
TB-6	<u>binB6</u>	I - approx. 5cM from <u>lacD27</u> - between <u>lacD</u> and <u>pabaA</u>
TB-11	<u>binC11</u>	I - not linked to <u>binB6</u>
TB-15	<u>binD15</u>	I - approx. 6% recombination with <u>binB6</u>
TB-21	<u>binE21</u>	VIII - not linked to <u>riboB2</u>
TB-24	<u>binF24</u>	VII - linkage with <u>nicB8</u> detected
TB-28	<u>bin-28</u>	I - approx. 6% recombination with <u>binB6</u>
TB-37	<u>bin-37</u>	I - close linkage with <u>yA1</u>

including acetate (Gajewski et al, Molec. Gen. Genet. 116: 99-106. lacD27 (obtained from Prof. W. Gajewski) was not found to be inhibited by benzamide.

It is tentatively suggested that these mutants may be altered in respiration functions. In this connection it should be noted that salicylhydroxamate inhibits a pathway of respiration in Newospora^{u-} (e.g. Edwards et al, J. Biol. Chem. 249: 3551-3556) and high concentrations of benzamide (0.5%) and low concentrations of salicylamide (< 1mg per ml) inhibit wildtype strains. These bin strains are available from this laboratory.

Caesium chloride sensitivity and ammonium
repression in A. nidulans

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The properties of two mutants, originally called amdR⁻18 and amdR⁻19, have been described previously (Hynes and Pateman, Molec. Gen. Genetics 108: 107-116). These were isolated as fluoroacetamide resistant mutants of strain amdR^C6 and acetamidase assays indicated that these strains were abnormally sensitive to ammonium repression of this enzyme. Investigations of the toxic effects of caesium chloride has led to the finding that these two strains are complex and that super-sensitivity to ammonium repression cannot be attributed to the amdR locus. The conclusions regarding the amdR⁻44 and amdR⁻11 mutants remains unchanged.

The properties of these two strains and a previously undescribed mutant are summarized here. It should be noted that, for unknown reasons, the medium at Flinders University led to a particularly leaky phenotype for s mutants (thiosulphate requiring).

Strain 1818 (formerly amdR⁻18) has been found to be abnormally sensitive to caesium chloride (10 - 200mM) only in the absence of a source of sulphur such as thiosulphate or methionine. In addition, this strain has a very leaky s lesion - probably allelic to s^C on linkage group III. The leaky thiosulphate requirement and CsCl sensitivity

segregate together in crosses. Spontaneous revertants resistant to caesium chloride have been isolated from a strain containing this lesion and two classes have been found. One is revertant for caesium chloride sensitivity ~~sensitivity~~ only and the other is revertant for both caesium chloride sensitivity and the thiosulphate requirement. In addition it has been found that 1818 has lost the original amdR^{C6} lesion.

Strain 1819 (formerly amdR⁻¹⁹) has not been investigated in detail. It has been found however to have an extremely leaky thiosulphate requirement and is sensitive to caesium chloride, but this sensitivity is not reversed by thiosulphate or other sulphur sources. The original amdR^{C6} lesion has been lost.

Strain 1848 (not previously described) was isolated in the same experiment as the other strains. This strain has a reasonably stringent thiosulphate requirement and this is due to a lesion in the sC locus. In addition, the mutant is sensitive to caesium chloride, but the sensitivity is not relieved by thiosulphate or methionine. Haploidization analysis indicates that the lesion leading to caesium chloride sensitivity is located on linkage group VI. Strain 1848 has lost the amdR^{C6} lesion. No enzyme assays have been carried out on this strain.

Association between caesium chloride sensitivity and supersensitivity of the acetamidase to ammonium repression has not been investigated. The role of the alkali metal ions in ammonium effects is at present puzzling (Arst and Page, Molec. Gen. Genet. 121: 239-245; Arst and Cove, Molec. Gen. Genet. 126: 111-141; Hynes,

Molec. Gen. Genet. 132: 147-152). The properties of strain 1818 indicate a further complexity involving sulphur metabolism. It is clear however that there is no evidence for an involvement of amdR in ammonium regulation of the acetamidase.

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The order of some loci and the position of the centromere on chromosome III of *Aspergillus nidulans*.

To determine the order of some loci and the position of the centromere on chromosome III of *Aspergillus nidulans*, we synthesized a diploid strain of the following genotype:

+	<i>cnxH4</i>	<i>sC12</i>	<i>dilA1</i>	<i>galA1</i>	<i>phenA2</i>	+
<u><i>yA2</i></u>	+	+	+	+	+	<u><i>pyrA4</i></u>

The markers *y*, *cnx*, *s*, *dil*, *gal*, *phen* and *pyr* are recessive and respectively confer yellow conidial colour, nitrite requirement, sulphite requirement, dilute conidial colour, inability to use galactose as a carbon source, phenylalanine requirement and pyridoxine requirement. The *y* and the *pyr* locus belong to chromosome I and IV respectively. The other loci are known to be located on chromosome III, which we confirmed by selecting haploid segregants from the diploid strain mentioned.

Making use of the fact that the *s* marker confers selenate resistance in addition to a requirement for reduced sulphur (H.N. Arst, Nature 219, 268-270, 1968), we selected selenate resistant segregants from the diploid strain mentioned. This was done by growing diploid colonies on a glucose minimal medium supplemented with sodium nitrite, D-methionine (which at a concentration of $2 \cdot 10^{-4}$ M is a good source of reduced sulphur and does not reverse the toxicity of selenate), phenylalanine, pyridoxine, and sodium selenate ($1 \cdot 10^{-3}$ M). Out of 247 independently arisen diploid segregants 147 were phenotypically *cnx s*, 42 *cnx s dil*, 35 *cnx s dil gal*, and 23 *cnx s dil gal phen* (one of which was *y*, and one *y pyr*). The first three classes of segregants must have arisen by mitotic crossing-over, the *cnx s dil gal phen* class could either result from crossing-over or from nondisjunction. Our data show that the order of the loci on chromosome III is as indicated, and that the centromere is located either between *gal* and *phen* or to the right of *phen*.

Following a modified filtration-concentration procedure, we isolated some phenylalanine requiring segregants from the diploid strain mentioned. Out of seven independently arisen diploid segregants only one was phenotypically *cnx s dil gal phen*, whereas all of the remaining six segregants were *cnx⁺ s⁺ gal⁺ phen* (and probably *dil⁺*, which was not always unequivocally scorable). This result indicates that the centromere lies between *gal* and *phen*, which is in agreement with data published by B.W. Bainbridge (Genetical Research 15, 317-326, 1970). Our data do not however confirm Bainbridge's conclusion that the *cnxH* and the *sC* locus are on the same chromosome arm as the *phenA* locus.

7 *phen* — 1
6

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Aspergillus strains at the Fungal Genetics Stock Centre (Arcata, Calif.)

With the termination of the project on translocations and genetic mapping, a large number of improved strains will be sent to the stock centre (ca. 150). These will include the best simple strain for each mutant and each translocation, as well as meiotic mapping strains (many markers of one linkage group) and mitotic mapping strains (one marker in each group). These will be able to replace many of the previously deposited strains, especially for genetic work. The strains which are considered dispensable are listed below, so that they can be acquired by anyone who might need them. Also, for two groups of strains discarding is debatable, namely, for 1) original mutants with translocations or other undesirable mutations and 2) mitotic mapping strains with 7 groups marked. Either of these will be retained if there is sufficient interest in their maintenance. An up to date list of all strains available at the Fungal Genetics Stock Centre is planned for the following year.

Fungal Genetics Stock Centre Strains To Keep

(Not listed in Barratt et al , Genetics 52:233-246, 1965)

FGSC NO.	Genotype	Translocations	Origin
27	pabaA1 b1A1; wa3	T1(VI;VII)	X-ray & spont.
29	pabaA1 b1A1; coA1	T1(VI;VII)	" "
38	wa1; lysA1	T? Ab VI	X-ray of wa1
40	b1A1; sa1	T1(V;VI)	UV of 26
55	luA1 b1A1	none	UV of 26
68	suA1adE20 ya2 adE20;AcrA1;phenA2; pyroA4;lysB5;sB3;nicB8;riboB2	} none	Forbes MSD
121	suA1adE20 riboA1 proA1 pabaA1 adE20; chaA1	none	Käfer cross
122	riboA1 ya2; nicB8	none	" "
129	pabaA1 ya2 adE20; choA1	T1(I;VII)	" "
140	suA1adE20 adE20 b1A1;AcrA1;phenA2; pyroA4;lysB5;sB3;choA1;coA1 chaA1	} none	" "
146	pabaA1; AcrA1; phenA2; pyroA4; lysB5;sB3;nicB8; riboB2 chaA1	} none	" "

218	ad55yA2 b1A1; AcrA1; riboB2	T?	J.W. Foley
219	b1A1; methG1 (replaces 67)	none	UV of 26
220	yA2; pyroA4	not likely	early Glasgow cross
221	adG14; AcrA1 wA3	none	KHfer cross
222	AcrA1 wA3; lysB5; chaA1	none	" "
223	wA3; lysB5; chaA1	none	" "

Published Strains to Discard

FGSC No.	Reason for discarding or replacement genotype
9	X - rays, unmapped
11	T?, replace with M 162: pabaA1 yA2 adE20; AcrA1; nic 10 (\pm T)
12	T, no use
42	adB, poor strain, replacing with M 958: adB8 b1A1 (\pm T)
49	only 7 marked groups
53	" " " "
56	poor strain, sending M 966: b1A1; ornB9 riboB2 (\pm T)
58	lac, has mycelial colour mutant on VIII, replacing with: M 917 adE20 b1A1; lacA1; choA1; chaA1 (+ ^d) (496.3.15)
59	fruct, has 2 morphological mutants, replacing with: M 2219: M 2219 proA1 b1A1; methG1 fra1; chaA1 T1(IV;VIII)(1929.1.1)
63	Has 2 Translocations, sending M 23: riboA1 b1A1; wA3 thia4 cnxR16 (noT)
65	Has 2 " , sending M 2298: pabaA1 yA2 adE20; nicA2; sbA3 (noT)
67	replaced by FGSC # 219
73	only 7 marked groups
74	sending M 799: adG14 proA1 pabaA1 yA2; wA3; ve ⁺ (BG TX)
76	" "
80 - 82	" "
77	sending M 1282: yA2; bwa1; chaA1 (T ⁺) " M 2209: suA1adE20 pabaA1 yA2 adE20; lacA1 bwa1 sb3 sbA3; malA1 (H ⁺)
84	replaced by # 23 pabaA1 yA2; chaA1 ve
86	like # 72
89	poor strain, being replaced by argB2 - recombinant
98	only 7 marked groups

Published Strains to Discard

FGSC No.	Reason for discarding or replacement genotype
99	replaced by teaching strains:
100	{ M2234 <i>SulA1</i> <i>adE20</i> ; <i>AcrA1</i> ; <i>ActA1</i> ; <i>choA1</i> ; <i>chaA1</i> M 2235: <i>fpaB37</i> <i>galD5</i> <i>suA1adE20</i> <i>riboA1</i> <i>ana1</i> <i>ya2</i> <i>bia1</i> ; <i>sD85</i> <i>fwa2</i> { M 2243: <i>galD5</i> <i>suA1adE20</i> <i>riboA1</i> <i>ana1</i> <i>pabA1</i> <i>ya2</i> <i>adE20</i> <i>bia1</i> M 2239: <i>SulA1</i> <i>adE20</i> ; <i>AcrA1</i> <i>wa3</i> ; <i>ActA1</i> ; <i>lysB5</i> ; <i>chaA1</i>
101	
102	only 7 marked groups
103	" " " "
106, 107	are <i>sC</i> <i>methH</i> , <u><i>SuB4pro</i></u> Replaced with <i>sC</i> <i>meth</i> ⁺ <i>SuB4pro</i>
112 - 118	keep or discard ?
108	<i>sC</i> <i>methH</i> , 7 groups
123	replaced by M 1089: <i>bia1</i> ; <i>wa3</i> (standard strain)
128	some isolates contain <i>sC</i>
131	has T, no use
132 - 139	only 6 or 7 groups marked
144, 145	only 7 marked groups
149	only 4 groups
151-153	only 6 and 7 groups marked
156	" 7 " "
157	replaced by M 799 (BC IX)
160	only 6 groups marked
161	" 7 " "
162	" 5 " "
164	" 7 " "
165	" 6 " "
166	" 7 " "
167	" 6 " "
169	" 5 " "
170	" 7 " "
171	" 7 " "
172	" 5 " "
174 - 179	4 - 7 groups marked only
181	} <i>lysB</i> strains replaced by: # 205 and M 1239 <i>AcrA1</i> ; <i>lysB5</i> ; <i>chaA1</i>
182	
185, 190	= <i>ve</i> ⁺
195 - 197	} 6 - 7 groups marked
199	

200 an y;selfs
 203 replaced by M 1279:ya2;AcrA1;lysB5;chaA1
 207 only 7 groups marked
 209 galC7, difficult to test, 2 T's
 210 2 T's replaced by # 214
 212 } galD5 with T, replaced by
 213 } M 2277:galD5 suA1edE20 riboA1 ya2 adB5; AcrA1 (noT)

EGSC Strains to discard (Dorn, 1967)

224 & 225 duplicates of 101, 137
 240 identical to 26
 249 has T, morphological mutation, replaced by:
 M 1500: bia1; lysB5; sd50 T2(I;VIII)
 253 T1(III;VIII), replaced by M2274:riboA1 ya91;IodA1 (noT)
 255 T1(VI;VII), replaced by M 2027:pabaA1;AcrA1;sdB5;facC102noT
 258 KERP but is + ⁺facA
 265 (is not UV of bia1) T1(VI;VII) replaced by:
 M 426:suA1adE20 proA1 pabaA1 adE20;AcrA1 pua1;
 choA1 nicB8; chaA1 (noT)
 267 fl, infected, replaced by M 1990:tpaB37 riboA1 bia1
 269 KERP, but is not galC7
 271 has T(VI;VII) replaced by:
 M 2162:SulA1 anA1 pabaA1; wa3; chaA1
 M 2271:riboA1 bia1; dila1; choA1
 274 T? replace with M 1824:l i ya2 (backcross, noT)

Strains to discard from ANL 11: 1970

289 same as 68
 290 is not His D
 302 contains galC7; better strain available.
 M 1371:fwA1 ornB7 (va⁺)
 321 same as 63, 2 T's?

Unlisted Strains to Discard

PGSC No.

Genotype

3	bia1; wa3; sa6; T(VI;VII); T(III → VIII)	
6	nicA2; ve ⁺ X-ray of wildtype	T?; replaced by T-free
7	ya2; thiA1; nic-3; ve ⁺ T(III;IV;VIII)	nicA2
8	ya2; thiA1; nic-4; ve ⁺ T(III;IV;VIII)	
10		
13	ya2; thiA1; pantoA1; T(III;IV?;VII;VIII)	
14	ya2; thiA1; T(III;IV;VIII)	
15	bia1; wa3; cys2 = sc13 T(III;VIII); T(VI;VII)	
19	bia1; wa3; nicB8	from duplication cross
20		
21	} replaced by 183 - 194	
22		
25	ya2; wa2 adC1; ve ⁺ T(III;IV;VIII)	
30	ya2; ad-2; ve ⁺ T(III;IV;VIII)	
48	ya2 bia1; thiA1; lanose	± T
51	bia1; wa3; T(III;VIII); T(VI;VII)	
54	bia1; ab = ile3; fl-mutant	
60	bia1; wa3; galA1 T(III;VIII); T(VI;VII)	
61	galD5 bia1; wa3	" "
62	bia1; wa3; gal-2	" "
88	ya2; veA1 (origin uncertain)	(± T)
109	suA1adE20 proA1 pabaA1 ya2 adE20; AcrA1 pu ; phenA2 sa4; lysA1 sb3; choA1 nicB8; chaA	(± Ab VI)
110	ya2 bia1(± T)	replaced by # 194
119	pabaA1 ya2 adE20; AcrA1	(± T(VI;VII))
124	bia1; AcrA1; phenA2; pyroA4; lysB5; sb3; nicB8	from
127		T(III → VIII) pedigree
130	pabaA1 ya2 adE20; nicB8 choA1; T(I;VII); T(I;VIII)	

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The Location of *pyro 12* in *Aspergillus nidulans*.

In studies with unstable strains of *Aspergillus nidulans*, it was of interest to determine the location of *pyro 12*, a marker whose location has been hitherto unknown. A strain, carrying the *y* and *pyro 12* alleles was combined in a diploid with a strain carrying the *bi1* allele. *y* and *bi1* are both located on chromosome (linkage group) I. The diploid was haploidized and the haploid segregants were subject to analysis. It was found that *pyro 12* is in definite linkage with the *y* and *bi1* loci. It was thus indicated that *pyro 12* is on linkage group I. In order to determine the position of *pyro 12* in relation to other markers on linkage group I, a cross was made between a strain carrying *pyro 12* and a yellow strain carrying the markers *ribo 1* and *ad 14*, the latter two markers being on the left arm of chromosome I. Analysis of the cross showed the following: the recombination frequency (R.F.) between the *pyro 12* and *ad 14*⁺ loci was 33%, the R.F. between the *ad 14* and *ribo 1* loci was 20%, and the R.F. between the *pyro 12* and *ribo 1*⁺ loci was 15%. These results clearly place the *pyro 12* locus approximately 15 units distal to the *ribo 1* locus, with the *ribo 1* locus between the *pyro 12* and *ad 14* loci. In summary, *pyro 12* is located on the left arm of chromosome I, approximately 15 units distal to the *ribo 1* locus.

Editor's note: I suggest that in the light of these results *pyro-12* should be regarded as defining a new locus: *pyroB* ✓

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Environmental and Genetic Factors Affecting Instability at Mitosis in *Aspergillus nidulans*.

Previous work (Bainbridge and Roper, 1966; Nga and Roper, 1968) has shown that strains of *Aspergillus nidulans* with a chromosome segment in duplicate (one in normal position, one translocated to another chromosome) are unstable. Deletions occur from either duplicate segment. Present work has shown that most deletions occur from the translocated duplicate segment. Furthermore, it has been found that the overall frequency of deletions from a duplication is dependent upon the temperature of growth. The overall frequency of deletions from a chromosome III duplication is greatly enhanced by low temperatures, while the overall frequency of deletions from a chromosome I duplication is markedly enhanced by high temperatures. A temperature of 39.5°C

appears to enhance the overall frequency of deletions from the I duplication to the greatest extent. At respectively 39.5°C and 42°C a section of the I duplication is most susceptible to deletion during a particular period of growth and at 42°C, a particular section of the III duplication is most susceptible to deletion during a particular growth period.

When the I duplication and the III duplication are together in a haploid, deletions from the intact III duplication generally precede deletions from particular sections of the I duplication. Furthermore, the III duplication can enhance to some (but not major) extent the frequency of deletions from the I duplication. After the III duplication becomes reduced in size as a result of the loss of chromosomal material from the translocated duplicate III segment, such a reduced III duplication can greatly enhance the frequency of deletions from the I duplication. In other words, a III duplication of reduced size can promote far more deletions from the I duplication than can the intact III duplication. The major increase in the deletional instability of the I duplication as promoted by the reduced III duplication is confined to the translocated duplicate I segment. The reduced III duplication can induce deletions from a section of the translocated duplicate I segment in accord with a temporal programme. Furthermore, a particular region of the I duplication is far more under the mutagenic influence of the reduced III duplication than another region.

In cultures carrying single duplications, the underlying cause or regulation behind the induction of deletions from the single duplications in such cultures are the respective duplications themselves (Nga and Roper, 1969; Roper and Nga, 1969). In double-duplication cultures, on the other hand, where the reduced III duplication is present, the I duplication largely loses its ability to direct or control deletions from itself; it appears that the reduced III duplication takes over this function. That is, in a double-duplication strain where the reduced III duplication is present, it appears that the I duplication loses (to a large extent) its self-directing-mutagenic-capability, in so much as its capability of directing or provoking its own deletional instability becomes largely subsumed under the control of a second duplication, the reduced III duplication. It is as if such control represents some kind of bond or tie between the two duplications. What is of interest here in regards to these two duplications is that we have one genetic-entity on the chromosomal level (as opposed to a single gene) programming or determining or directing an extremely high frequency of deletions from a second genetic-entity on the chromosomal level, or from another point of view, we have one chromosomal imbalance causing or inducing a second chromosomal imbalance to become extremely instable.

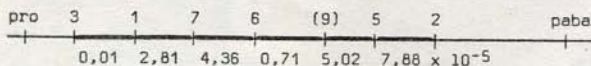
This and other information shows that a chromosomal imbalance either in the form of a duplication in a haploid or a triplicated chromosome segment in a diploid can cause mutations or deletions within chromosomal regions not linked to the chromosomal imbalance in question. In effect, a chromosomal imbalance can promote deletions or mutations outside itself.

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The hisB mutants of Aspergillus

The hisB locus is located on linkage-group I, between pro1 and paba1 (Millington-Ward et al, A.N. no.11). During fine-structure mapping it was observed that certain combinations of allelic crosses were highly infertile; that is they yielded few or no ascospores per plate. Three very infertile mutants are available (hisB2, hisB4 & hisB5), and one that is partly infertile (hisB6). The preliminary fine-structure map is:



hisB4 is the most infertile mutant, and does not revert. We have been unable to localize it yet. hisB2 and hisB5 revert, but segregate no suppressors, suggesting that the latter are tightly linked or intragenic. This conclusion is corroborated by allelic complementation studies: combinations with hisB2, hisB4 and hisB5 do not complement and by the fact that these are non leaky mutants.

Our working hypothesis is that hisB is either a bifunctional gene (controlling both histidine and fertility), or two adjacent genes. We suppose that hisB2 and hisB5 are frameshift mutants, and hisB4 a small deletion inactivating both functions. Encouragement for the hypotheses is further provided by Mahoney & Wilkie (Proc.R.Soc.1962) who also found a fertility gene in this location.

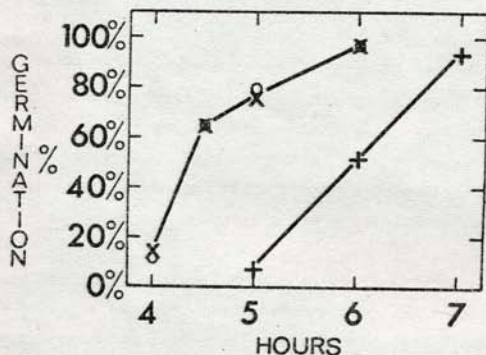
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An observation on conidial germination times

In the course of studies on radiation mutagenesis in Aspergillus, some experiments have been performed using time-lapse cine-photography to determine the germination times of unirradiated conidia in minimal medium. We were able to demonstrate that, under the particular conditions used, a constant and reproducible rate of germination is obtained after a period of "treatment" exceeding 400 mins. (at room temperature).

"Treatment" of the meth G1 b1A1 strains involved collecting conidia from a 5-6 day old complete medium plate, filtering to remove mycelial debris, and, after washing, resuspending at a density of approximately 1×10^7 conidia per ml. in 30 mls of phosphate buffer (pH7) containing 0.01% "Tween 80" in a 20 oz. medical flat. The enclosed suspension was continuously rotated at 42 rpm, and, at the appropriate time, added to 100 mls of melted minimal medium containing methionine, biotin, glucose and tartrate. After dispersal of the conidia throughout the medium, 1 ml of the suspension was distributed over the surface of a 8 cm diameter Carrel flask which was positioned in the photographic equipment (at 37°C). Typical examples of the analysis of the photographs obtained are given in fig.1.

Prior to 400 mins the position of the germination curve is variable and dependent upon the time period the conidia are in buffer containing Tween. However, after this period a constant position and rate of germination is obtained. Efforts are now being made to find out how this observation affects radiation sensitivity and mutation.



Time in minimal medium at 37%

Fig. 1. The germination of unirradiated "Tween 80" prepared conidia, samples 45 min (+), 455 (o) and 535 (X) min after the start of rotation.

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Mutations increasing non-disjunction in *Aspergillus nidulans*

The relatively recent identification of the $n + VIII$ disomic condition (Kafer and Upshall, J. Hered. 64, p35, 1973) makes a study of chromosomal non-disjunction feasible since all possible disomics can now be phenotypically differentiated.

Phenotypically normal survivors from a U.V. treatment of Glasgow standard strain biAl were isolated and screened for enhanced meiotic and mitotic non-disjunction. This was recognised as an increase in the frequency of aneuploid progeny when compared with the control value of 0.2%. Three of the survivors have proved to have deviant non-disjunction patterns.

- (1) U.V.146 showed increased non-disjunction in both selfed (16% abnormal) and hybrid (3% abnormal) meioses and in mitosis (7% abnormal). The aneuploids were almost exclusively multiple segregating types.
- (2) U.V. 192 showed inflated aneuploid frequency only amongst selfed (homozygous) meiotic progeny, at a level of 1%. This suggests a recessive mutation active in the first meiotic division.

For both of these strains translocations (which are known to affect heterozygous meioses) have not been eliminated as possible causes although the pattern of aneuploid recovery makes this unlikely.

- (3) U.V.157 inflated non-disjunction to 0.6% in meioses and 0.8% in mitosis. Parasexual analysis has ruled out a translocation and located the mutation to linkage group I (1 recombinant in 62 haploid progeny tested). Meiotic analysis (based on a sample of 125) has tentatively located the mutation 27 units distal to *bi* from the centromere. Conidial samples of heterozygous diploids indicate recessiveness of the mutation which has been called *hfa* 1 where *hfa* designates high frequency of aneuploids.

A summary of the results of a detailed analysis of mitotic non-disjunction is shown in table 1. It can be seen that all 8 linkage groups undergo more frequent mis-segregation both individually and in multiple associations. Additionally there appears to be variation in the factor of increase over the control ranging from 3x for l.g. III to 13x for l.g. VIII.

Table 1

Table 1. Comparison of frequency of aneuploid types ($\times 10^5$) recovered from *hfa* and Δ *hfa* strains.

	Disomics n+1 for linkage groups									
	I	II	III	IV	V	VI	VII	VIII	n+1	others
mutant(23)	55	72	55	178	21	136	102	89	68	68
non mutant(74)	7	11	19	17	7	26	25	7	19	9
Factor of increase	8	7	3	10	3	5	4	13	4	8

numbers in brackets refers to total number of relevant strains tested.

Work is continuing (1) to identify the nature of the changes in strains U.V.146 and U.V.192 and (2) to isolate additional mutations of this type as part of an investigation into the genetic control of chromosome segregation in *Aspergillus*.

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Properties and chromosomal locations of two mannose mutants

Gene symbol	Properties	Chromosomal Location
<u>mnr</u> - 455 ^{ts}	Fails to grow on minimal med. at 40°C. Produces balloon- shaped structures at hyphal tip when transferred from 25° to 40°C. Grows on minimal med. plus 1% mannose as sole carbon source. (<u>mannose</u> relief) Reduced activity of phospho-mannose mutase.	Linkage group V <u>nicA- mnr- pA</u> <u>37.2 19.1</u> <u>± 4.3 ± 3.1</u>
<u>manA1</u>	Inability to use mannose as sole carbon source. Reduced activity of phospho-mannose isomerase.	Linkage group VIII <u>manA1</u> 0.85 ± 0.85 ? ?

riboA

is on chromosome I