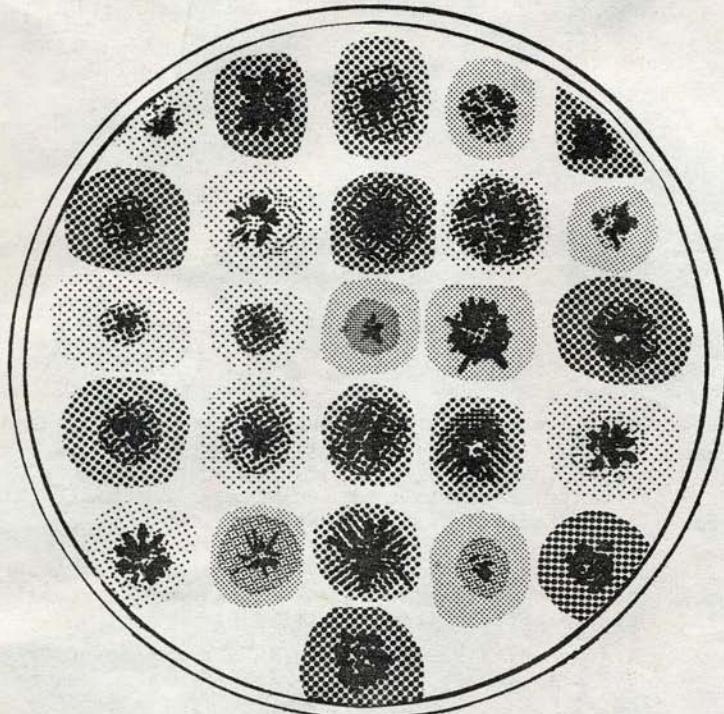


ASPERGILLUS NEWS LETTER

summer 1975

number 13



CONTENTS

Page

- 1 Editors note
Publications - Aspergillus nidulans bibliography supplement 2.
- 4 Publications - related organisms.
- 5 J.L. Azevedo, E. Pacheco Santana & R. Bonatelli Jr.
Effects of fungicides and ethidium bromide in a duplication strain of Aspergillus nidulans.
- 6 J.L. Azevedo & E. Pacheco Santana.
The use of chloroneb to obtain haploid segregants from heterozygous diploids of Aspergillus nidulans.
- 7 J.A. Birkett & J.A. Roper.
A temperature sensitive phenotype associated with the TVI-VII translocation.
- 8 B.L. Case & J.A. Roper.
Genetic imbalance and mitotic non-conformity in Aspergillus nidulans.
- 11 B.L. Case & J.A. Roper.
Possible revision of the right arm of linkage group II of Aspergillus nidulans.
- 13 A.J. Clutterbuck.
Ascospore maturation and germination.
- 14 B. Giddings & A. Upshall.
Spontaneous mapping of Aspergillus terreus and Aspergillus nidulans.
- 17 S. Gross & A.J. Clutterbuck.
"Fluffy" colonies obtained from Aspergillus conidia after ageing and other treatments.
- 19 L. Handley & C.E. Caten.
Effect of vegetative incompatibility on cytoplasmic infection in Aspergillus amstelodami.
- 20 K. Mohan Namasivayam & E.R.B. Shanmugasundaram.
Isolation of aminoacyl acceptor-RNA from a wild strain of Aspergillus nidulans.
- 23 R.W. Barratt, W.N. Ogata & Etta Käfer.
Aspergillus stock list (Fungal Genetics Stock Centre) - 1st revision.

1

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Dear Colleague

I again gratefully acknowledge the help of:

Glaxo Research Ltd.

Imperial Chemical Industries

Pfizer Ltd.

- in contributing to the production and mailing costs of this issue.

I hope to compile ANL 14 in summer 1976 and will send requests for updating the mailing lists next spring. Material for ANL 14 is welcome any time.

John Clutterbuck

ASPERGILLUS NIDULANS

Bibliography supplement 2

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Effects of fungicides and ethidium bromide in a duplication strain of
Aspergillus nidulans.

Strains of A.nidulans with a chromosome segment in duplicate are unstable at mitosis. They produce sectors of various kinds which can almost always be scored unequivocally by their characteristic morphology. The system was used successfully to test the effects of agents such as trypan blue to induce deletion in duplication strains (Cooke *et al*, Nature, 226: 276-277, 1970).

In order to test the effects of two fungicides, 1,4-dichloro-2,5-dimethoxibenzene (chloroneb) and 2,3-dihidro-5-carboxanilido-6-methyl-2,4-oxatiin (vitavax) and of ethidium bromide (EB) on mitotic non-conformity, conidia of duplication strain A (Nga and Roper, Genetics 58:193-209, 1968) were inoculated at the centre of 10cm dishes of complete medium containing these agents. Sectors were scored after 7 days at 37°C. In the concentrations used there was only a slight reduction of the growth rate of the colonies. The results (Table 1) show that chloroneb increases the frequency of sectors; vitavax and ethidium bromide reduce instability.

Table 1: Sectors produced by control and treated colonies of strain B.

Treatment	Nº of dishes	Mean nº of yellow sectors	Mean nº of green sectors	Mean nº of* other sectors	Total nº of sectors
Control	29	2.48	0.76	0.10	3.34
Chloroneb (5µg/ml)	10	3.30	1.00	0.70	5.00
Vitavax (5µg/ml)	10	0.20	0.10	0.00	0.30
EB (1.0µg/ml)	39	0.33	0.08	0.15	0.56
EB (1.5µg/ml)	38	0.45	0.00	0.05	0.50

* - Includes heterokaryotic and deteriorated sectors.

Pacheco Santana (M.S. thesis, Univ. of São Paulo, 1974) did show that chloroneb increases the frequency of haploidization in diploid strains and that vitavax reduces the frequency of mitotic crossing-over and haploidization. Since the same tendency was shown regarding instability in duplication strains it could be suggested that the mechanism which controls instability in duplication strains is related to that of mitotic crossing-over and haploidization. Ethidium bromide is known to affect extra-chromosomal elements in bacteria. In the present case it did not reduce the frequency of deteriorated sectors but did reduce the number of improved sectors, acting as a stabilizing agent for unstable duplication strains.

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The use of chloroneb to obtain haploid segregants from heterozygous diploids of *Aspergillus nidulans*.

Several agents including p-fluorophenylalanine, arsenate, benlate and acridine yellow are used in order to obtain haploid sectors from heterozygous diploids of *A.nidulans* (Clutterbuck, Handbook of Genetics 1: 447-510, 1974). We did find that other agent, the fungicide chloroneb (1,4-dichloro-2,5-dimethoxybenzene) have the same effect. Diploid conidia were stabbed into complete medium plus 10 μ g/ml chloroneb and after 4-6 days incubation at 37°C many haploid sectors could be obtained and easily purified. Haploid sectors were very conspicuous and no inhibition of several auxotrophic and morphological markers use was observed.

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A temperature sensitive phenotype associated with the TVI-VII translocation
During a routine cross:-

1 pro Al bi Al; gal E9 cnx H3 sc12; TIII-VIII (non-reciprocal) X y Al ribo Al;
nic B8 (translocation free)

it was found that the III-VIII duplication strains generated had a different morphology from the laboratory stock III-VIII duplication strain. This difference was only noticeable after the strains had been grown on complete medium for seven days from a central inoculum. The two morphologies are:-

M1 - morphology of the III-VIII duplication strains from cross 1 - crinkled morphology (Bainbridge and Roper 1966) - produce few improved sectors.

M2 - morphology of the stock III-VIII duplication strain - again a crinkled morphology but many apparently improved sectors are produced. The majority of these sectors do not resemble those produced by the M1 morphology strains.

The strain with the M2 morphology was known to carry the TVI-VII reciprocal translocation often found in stock strains carrying the TIII-VIII (Käfer 1965). No TVI-VII was present in either of the strains used in cross 1. It was suspected that the morphologies M1 and M2 were determined by the absence or presence of the TVI-VII in the respective strains. Normal haploids with or without the TVI-VII show no morphological differences.

In a further cross:-

2 pyro B12 bi Al; wA3; TIII-VIII, TVI-VII X yAl; gal E9 cnx H3 sc12; nic B8;
(translocation free)

of the six duplication strains isolated for further study three had the M1 morphology and three the M2 morphology. One of each morphological type was taken and analysed via the parasexual cycle. It was found that the morphologies corresponded with the presence of absence of the TVI-VII as above.

The III-VIII duplication strains carrying the TVI-VII produce many early sectors which show a slight reduction in conidiation and an apparent increase in growth rate as their only phenotypic change. When these sectors are subcultured via their conidia or leading hyphae the resulting colonies show the same phenotype as the parent duplication strain. That is, these 'sectors' are a feature of the colonial morphology and do not represent a class of sectors caused by deletion of duplicate material. (Bainbridge and Roper, 1966; Nga and Roper, 1968). The analysis shows this morphology to result from a combination of the III-VIII duplication with the TVI-VII or less likely, with a locus or loci linked to either or both breakpoints of the TVI-VII.

These pseudosectors are produced when the strains are grown at 37°C but not at 42°C. The morphology is more compact at 42°C but the complete lack of pseudosectors would seem to indicate a temperature sensitive phenotype. This phenotype was described by M.M. Lieber A.N.L. 12 26. However he interpreted the data to mean that there was an increase in chromosome instability with a reduction in temperature (36°C compared to 42°C). This new data may suggest an alternative interpretation.

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Genetic imbalance and mitotic non-conformity in Aspergillus nidulans

CASE, B.L. and ROPER, J.A.

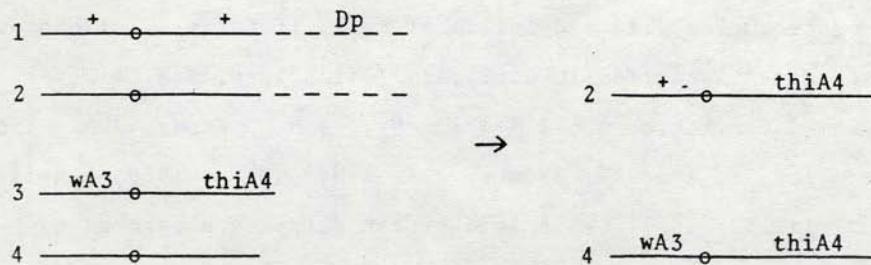
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Strains of Aspergillus nidulans with a chromosome segment in duplicate (one in normal position and one translocated to another chromosome) show a characteristic instability at mitosis, and produce nuclei with a deletion of most, if not all, of one of the segments carried in duplicate (Bainbridge, B.W. & Roper, J.A., 1966, J.Gen.Micro. 42, 417-424; Nga, B.H. & Roper, J.A., 1968, Genetics 58, 193-209; Roper, J.A. & Nga, B.H., 1969, Genetical Res. 14, 127-136). A haploid strain carrying a segment of

chromosome I in duplicate has been studied extensively and shows deletions predominantly from the translocated segment and only rarely from the non-translocated segment. In studies of this duplication in a diploid, a considerably increased frequency of loss from the translocated segment was observed. Nga and Roper (1969, Genetical Res. 14, 63-70) suggested that this could be due to the genetic imbalance in these strains, although it is interesting to note that a duplication diploid is relatively less unbalanced than the corresponding duplication haploid.

The products of instability have now been examined from diploids carrying markers on the right arms of chromosomes II and VIII for the I-II and III-VIII duplications respectively. The I-II Dp diploid produced many diploid sectors which no longer carried the duplicate segment. These were assumed by Nga and Roper to arise from "simple" deletion. In fact, at least 50% arose by mitotic crossing-over, as shown by their homozygosity for thiA4 but not for other markers (Figure 1).

Figure 1



Mitotic crossing-over distal to thiA4 could also eliminate the duplicate segment but would have been indistinguishable from loss by deletion. Mitotic crossing-over was also involved in the instability of the III-VIII Dp diploid. These findings make it likely that the frequency of deletion of the translocated segment in diploids was little different from that seen in the duplication haploid. Deletions from the duplicate segments in normal position were also demonstrated and occurred at a frequency greater than that detected in the duplication haploid.

A diploid homozygous for the I-II duplication was synthesized and was shown to be very unstable although, in this strain, mitotic crossing-over could not eliminate either duplicate segment to yield products with a relative growth advantage. Analysis of a sample of sectors showed a) loss from either translocated duplicate segment, at a frequency similar to that which was occurring in the unbalanced haploid and b) frequent loss from both of the corresponding non-translocated segments. This pattern of instability was shown initially by the loss of appropriately arranged markers and confirmed by extensive genetic analysis.

The susceptibility of the translocated segment to deletion and its suggested increase in diploids must, in the light of these findings, be reassessed. The frequency of loss by deletion from the translocated segment appeared to be similar in the duplication haploid, the single duplication diploid and the double duplication diploid. However, the frequency

of loss from the non-translocated segments increased from the duplication haploid, through the single duplication diploid, to the double duplication diploid.

It is difficult, at this stage, to interpret these results in terms of a mechanism for deletion but clearly the different chromosomal positions of the duplicate segments affects their instability at different doses of the duplication. The translocated segments appeared to be unaffected by dosage whereas the non-translocated regions were greatly influenced by the dosage of the segments concerned.

Possible revision of the right arm of linkage group II
of Aspergillus nidulans

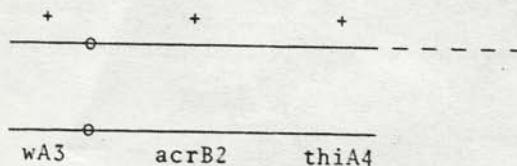
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As reported elsewhere in this issue, it is easy to detect mitotic crossing-over in diploids which carry an extra chromosome segment in translocated position. Crossing-over between the centromere and the point of attachment of the duplication can yield a balanced diploid with a growth advantage over its unbalanced parent. This selective technique has been used to study gene order on II R.

Sectors were taken from a diploid heterozygous for wA3, acrB2, thiA4 and the terminally attached I-II Dp. Of 184 sectors which had lost the terminal duplication, 100 definitely arose by mitotic crossing-over. All 100 were homozygous for thiA4 and 67 were homozygous also for acrB2. The remaining 84 sectors arose either by "simple" deletion or mitotic crossing-over between the most distal marker and the point of attachment of the duplication. The presence of wA3 allowed distinction between mitotic crossing-over and non-disjunction as a cause of elimination of the duplicate segment. These findings are best explained by the gene order shown in Figure 1.

Figure 1



no. of events 67 33 84

The linkage relationships of mutant alleles between thiA4 and acrB2 are extensively reported (Dorn, G.L., 1967, Genetics 56, 619-631) but there is no good evidence for meiotic linkage of this region to any centromere linked genes. The order of these markers relative to the centromere had been determined by selection for either acriflavine resistance, (Käfer, E., 1958, Adv. in Genetics 9, 105-145) or yellow-green conidia

(Clutterbuck, A.J., 1970, *Aspergillus Newsletter* 11, 2-3) on the assumption that the selected marker was, in each case, the most distal. In fact, earlier results are compatible with the presently suggested order if allowance is made for double crossover events. Conversely the duplication diploid data, in terms of the accepted order of markers, would have required exceptionally improbable multiple events.

Investigation of this phenomenon is continuing using additional markers but selection of mitotic crossing-over by the elimination of terminally attached duplications clearly offers an excellent method for mitotic mapping.

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Ascospore maturation and germination.

Inconsistent germination frequencies of stored ascospores led to the following investigation. Cleistothecia of the cross adF17 pabaA1 yA2 X proA1 biA1;acrA1; pyroA4 (cross d, experiment 36 of Pritchard in "Experiments in microbial Genetics" ed. Clowes and Hayes, Blackwell 1968) were harvested at intervals after inoculation of the cross plates. Ascospore suspensions were made in the usual way, counted with a haemocytometer and stored at 4°. They were then sampled periodically by plating on CM + adenine + sodium deoxycholate. The original cross was set up by mixing conidia of the parent strains on a small area at the centre of a thick MM plate and germination was started by the addition of a drop of liquid CM. The cross plate was sealed with cellulose tape and incubated at 37°.

The results in the table show that while very young ascospores have poor germination rates and these decline on storage, germination rates of ascospores harvested at 2-3 weeks improve on storage. This confirms the suspicions which led to these experiments.

Percentage germination of ascospores

		Cleistothecium												
		a	b	c	d	e	f	g	h	i	j	k	l	m
Days after cross set up	7	1	2											
	9	-	-	5	11									
	11	10	3	4	7	4								
	14	1	7	2	4	2	5	10						
	18	-	-	1	3	4	7	11	13	39				
	22	-	-	-	2	26	3	8	39	37	22	44		
	33	-	-	-	-	-	25	28	43	44	45	60	100	77
	37	0	0	1	0	11	54	63	44	52	44	78	100	77

The underlined figure indicates the plating made the day the cleistothecium was harvested from the cross plate.

A proportion of the ascospores examined at 7 and 9 days were not pigmented, but all older ascospores were the normal dark red. Germination of ascospores from cleistothecia harvested at 7 and 9 days was spread over five or more days, even with the stored ascospores, but ascospores from the cleistothecia harvested later appeared to germinate in two days or not at all. All cleistothecia were hybrid and the products apparently normal, although the cross was not analysed in detail.

It can be concluded that ascospores go through a definite maturation process, some steps of which can be completed in stored suspensions at 4°. Plating, however must interrupt this process and lead to death since delayed germination of immature ascospores was not observed.

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Spontaneous mapping of *Aspergillus terreus* and *Aspergillus nidulans*.

It is now well documented both from academic and industrial circles that the mutagenic treatments used for the recovery of gene mutations can lead to chromosome damage. Provided there are adequate systems for the recognition

of such aberrations the problems are not insurmountable. However, imperfect fungi are limited in these systems resulting in the difficulties experienced in many laboratories when attempting to recombine highly mutagenised strains. Over the last few years powerful new selective techniques have emerged which enable the researcher to select spontaneous gene mutations and hence in theory at least allow the construction of a basic genetic map before mutagenic treatments are employed. The results to date of such an empirical approach to A.terreus and A.nidulans are presented below and we believe illustrate the suitability of the method to other organisms.

Aspergillus terreus.

This fungus was chosen for the following reasons

- Its' growth morphology and nutritional requirements are extremely similar to those of A.nidulans so that no new techniques or media were required.
- The vegetative cycles are identical with the production of uninucleate conidia.
- It has a commercial value being used in the production of itaconic acid.
- Nothing is published concerning the genetics of the organism.

The following classes of mutation have been recovered:-

(1) Spore colour These were easily obtained by microscopical screening of a dense plating followed by selecting the deviant colour from the brown background. Two such mutants have been isolated and designated whiA1 and creA1 (white and cream respectively). These are complementary, a heterozygous diploid having wild-type spore colour, albeit somewhat lighter brown than the wild-type haploid.

(2) Auxotrophs

- Easily recovered were auxotrophs for sulphate metabolism isolated as resistors to selenate (Arst 1968). Two complementation groups have been identified, one of which is additionally resistant to chromate. As per A.nidulans this mutation has been designated sB, the nonchromate resistor sC.
- Similarly, chlorate selection (Arst and Cove 1973) has yielded a spectrum of auxotrophs for nitrogen metabolism. Initially these experiments were carried out using arginine as the sole nitrogen source. In A.nidulans this gives rise almost exclusively to niaD mutations (Arst pers comm). Similarly, in A.terreus the vast majority of mutants recovered are non-complementary and hence we have classified these as niaD mutations. Additionally we have recovered five other complementing groups of auxotrophs the nomenclature for which we have not yet finalised.
- A problem with both the sulphate and nitrate auxotrophs is leakiness which creates difficulties when attempting to synthesise heterokaryons and the subsequent isolation of heterozygous diploids. To make such procedures easier non leaky auxotrophs were screened for, using the nystatin selective

technique of Ditchburn and MacDonald (1971). This was modified by incorporating sodium desoxycholate into the basal and overlaying agars and by the velvet replication of the resultant colonies directly on to minimal medium. Three clean auxotrophs have been isolated, one requiring adenine (adeA1) and two complementing methionine auxotrophs (metA1 and metB1).

(3) Drug resistors To date we have isolated spontaneous mutations resistant to acriflavine, 8-azaguanine, sulphanilamide, p-fluorophenylalanine and benlate. Detailed alleleism analyses are in progress but of interest are those resistant to sulphanilamide. Three classes have been identified resistant to increasing levels of concentration. Those resistant to the highest level (7.5 mg/ml) are somatically unstable and have an abnormal morphology when grown on sulphanilamide free medium.

Genetic analysis.

Parasexual analysis was initially inhibited by the fungus showing tolerance to p-fluorophenylalanine which nullified attempts at haploidisation analysis. However this drug was eventually more than adequately substituted by benlate which has proved a highly efficient haploidising agent. Results to date have identified five freely recombining linkage groups with only two genes showing association namely adeA1 and acriflavine resistance. Other selective procedures have been a) the utilisation of acriflavine for somatic crossing over. Few haploids are isolated from heterozygous diploids grown on the drug. b) the utilisation of both selenate (for sB) and chlorate (for nialD) as agents for both haploid and diploid recovery.

Technical problems:- The first was the recognition of diploidy, especially following selection for white or cream spore colour. Measurement of conidia is neither convenient nor accurate since the conidial diameter is small (about half the size of those of A.nidulans). The problem has been overcome by utilising benlate whence haploid colonies prove stable and diploids show a sectoring growth pattern on medium containing the fungicide.

The second problem concerns the presence on the mycelium of large hyaline cells which are produced in abundance and which are capable of germinating to produce colonies hence problems in attempting to use conidial suspensions. We have solved this by passing spore suspensions through millipore filters before use.

For control and repeatability purposes we have followed the same basic procedure in A.nidulans and have isolated the following mutant strains.

Spore colour - yellow; white; pale green (this is probably an allele at the white locus since no recombinants have been recovered from over a thousand meiotic progeny)

Auxotrophs:- alleles of sC and sB via selenate toxicity. Alleles of niaD niaA nirA cnxE cnxG and strains which complement all three closely linked cnx A B and C loci, via chlorate toxicity. Additionally via the modified nystatin technique, an allele of the pabaA locus (linked by 10 meiotic units to the yellow mutation spontaneously isolated) one adenine requiring and one methionine requiring strain. Allelism tests for the two latter strains are not yet complete.

Resistors to acriflavine; actidione; p-fluorophenylalanine; 8-azaguanine; methylammonium and benlate.

In none of the strains so far tested has a translocation type aberration been detected.

References

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"Fluffy" colonies obtained from Aspergillus conidia after ageing and other treatments

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The spontaneous appearance of "fluffy" mutants is a common finding in Aspergillus cultures. We have investigated some factors which we thought might affect the mutation rate. Fluffies were defined as all colonies with excessive aerial mycelium; most were not tested further.

1. Heat treatment. An experience of Dr. B.L. Cohen's in which large numbers of fluffies appeared in cultures from spores of some rather old stocks carried across the U.S.A. in the boot of a car in summer suggested that heat treatment may induce fluffies. Slants of the biAl strain (G051) were heated at 51° or 60° for various times. Conidia from these slants were then plated out and incubated at 37°. At 51° the decline in viability in 24 hours was slight and there were no fluffies in a total of 10^4 colonies. At 60°, on the other hand, viability declined rapidly to 10^{-6} or 10^{-7} after 24 hours and only 66 colonies were recovered in all. None of them were fluffy. It may be significant however, that subculture of one of the normal colonies obtained from this treatment did yield a fluffy colony.

2. Ageing. Samples of conidia were plated from slants of various strains stored for up to 18 weeks at 4° or room temperature. During this period viability changed only slightly, apparently rising from 7% at 10 days to 23% and 19% at 55 and 127 days respectively. Samples of 10^4 colonies per strain taken from 10, 16 and 65 day old slants gave only one fluffy in all. However, 622 fluffies were obtained from 2.17×10^5 colonies in platings from 113-127 day old slants. In each test, separate platings were made from seven slants of each strain; variation between these slants was very high, e.g. from 1 to 192 fluffies per 4.5×10^4 colonies in the most extreme case. This variation was sufficient to obscure differences between strains and storage temperatures. The strains used were: biAl (G051), biAl; methG1 (G0110), biAl; yoA6 (G0247), MSD (G93) and MSE (G94). All these gave rise to at least some fluffies (contrary to Dorn, Martin & Purnell, Life Sciences 6;629-633).

The biAl; methG1 strain was used in control experiments in which spontaneous mutation frequency to meth reversion was tested in the 127 day old cultures. No revertants were obtained from 2×10^6 viable conidia plated on MM + biotin. Alderson & Clark (Nature 210;593) quote reversion frequencies of methG1 from 1 to 16×10^{-6} so we have no evidence of a general rise in mutation frequencies due to ageing.

3. Heavy metals. Preliminary experiments had suggested that copper might induce fluffies, however, experiments in which fresh conidia of the biAl strain were inoculated onto CM plates in which sterilized copper coins were embedded gave no fluffies. Since in the earlier experiments fluffies had invaded the inhibition ring around the coin, this suggests that heavy metals may select for pre-existing mutants rather than induce them.

4. UV irradiation. This is clearly an efficient agent for the induction of fluffies. A ten minute irradiation (survival 7.0×10^{-3}) gave 78 fluffies: a frequency of 3.9×10^{-4} (biAl strain).

In conclusion, it seems likely that much of the "fluffy menace" results from the subculturing of old stocks. Although we have not specifically tested this, it is not our experience that conidia stored on silica gel (Roberts ANL 10;29) produce fluffies in any numbers, so the value of this storage method is re-emphasised.

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Effect of vegetative incompatibility on cytoplasmic infection in *Aspergillus amstelodami*

The infectious spread of the cytoplasmically inherited condition "ragged" from donor to recipient mycelia of *A.amstelodami* is restricted where donor and recipient are unable to form heterokaryons, i.e. they show vegetative/heterokaryon incompatibility. This restriction is complete where the incompatibility is determined by several genes as is the case between most independent wild isolates, but appeared only partial when a single heterokaryon incompatibility gene, hetA, was involved, suggesting a degree of additivity in the action of the genes (Caten, J.gen.Microbiol. 72, 221-229, 1972). The effect of three other known incompatibility genes hetB, hetC and hetE has recently been investigated using the same experimental system (Table 1).

Table 1. Effect of differences at single heterokaryon incompatibility loci on the transfer of "ragged" from donor to recipient strains.

<u>Locus</u>	<u>Compatibility</u>	<u>Number of mixtures showing</u>	
		<u>Transfer</u>	<u>No Transfer</u>
<u>hetA</u>	compatible	46	0
	incompatible	9	33
<u>hetB</u>	compatible	17	0
	incompatible	0	13
<u>hetC</u>	compatible	19	0
	incompatible	2	13
<u>hetE</u>	compatible	15	0
	incompatible	12	0

Compatible controls included in each experiment always showed transfer of the infectious cytoplasmic factor. The effect of the single incompatibility genes varied with the particular gene involved; hetB completely blocked transfer while hetE had no effect, transfer occurring in all cases as in the compatible controls. Genes hetA and hetC were intermediate, showing a marked but not complete inhibition of transfer. Since a difference at a single heterokaryon incompatibility locus, hetB, completely blocks cytoplasmic infection, it is not necessary to invoke additivity of the het genes to account for the similar reaction observed between incompatible wild isolates. The differences in the strength of inhibition of

infection are correlated with the quantitative effects of the individual genes on heterokaryon formation and it seems likely that the occasional instances of incompatible transfer involving hetA and hetC reflect restricted heterokaryosis.

Although the available evidence suggests that the "ragged" determinant is not a mycovirus, it may be considered as a model of infectious, cytoplasmic genetic elements, as far as its transmission between mycelia is concerned, and it is suggested that the incompatibility genes studied would have similar effects on the transmission of mycoviruses.

ISOLATION OF AMINOACYL ACCEPTOR-RNA FROM A WILD STRAIN OF
Aspergillus nidulans

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A wild strain of *Aspergillus nidulans* (Green) was grown in minimal medium at 37°C (Pontecorvo et al.. Advances in Genetics, 5, p.141, 1953). The mycelia obtained after 7 days' growth were washed with water, dried between folds of filter paper and then kept in cold room until it is worked out.

A.nidulans sRNA was prepared by the procedure of Elinor F.Brunngraber (Biochem. Biophys. Res.Commun. Vol.8, p. 1 - 3). About 30 gms of dry mycelium of *Aspergillus nidulans* were homogenized in a waring blender with 40 ml of phenol saturated with water (Mallincckrodt, Analytical Reagent) and 30 ml of 1.0 M

sodium chloride, 0.005 N EDTA in 0.1 M Tris-HCl buffer (pH 7.5) for 3 to 5 minutes. The homogenate was spun down in an International refrigerated centrifuge for 10 minutes at 4000 r.p.m. The supernatant was carefully decanted off. To the aqueous residual solution, 3 volumes of 95% ethanol were added and kept overnight in the cold room. The precipitate was removed by centrifugation and dissolved into solution using 100 ml of 0.1 M Tris-HCl buffer (pH 7.5). The solution was chromatographed on a column of DEAE cellulose (2 gms filled in a column of 2 x 10 cm) previously equilibrated with cold 0.1 M Tris-HCl buffer of pH 7.5. The column was washed with one litre of the same buffer and the aminoacyl acceptor RNA was eluted with 1.0 M sodium chloride in 0.1 M HCl buffer of pH 7.5. Sufficient effluent was collected until the optical density of the effluent was less than 260 nm. The solution was extracted with equal volume of phenol saturated with water and then twice with ether. To the aqueous solution containing the sRNA, 3 volumes of 95% ethanol were added and kept in the cold room. The precipitate of sRNA was spun down and washed with 80% ethanol and then twice with 90% ethanol and dried in a vacuum.

The absorbancy at 260 nm of a 0.1 mg/ml of solution was 2.0, which corresponds to sRNA.

The phosphate was detected as follows:

Less than a mg of sRNA was kept in muffle furnace for 3 hours. The ashes were dissolved in 1N HCl. To this solution, molybdate and ANSA were added. After 10 minutes, a blue colour

was obtained. The riboside was tested as follows: The sRNA was dissolved in 0.5 ml of 5 mM NaOH. To this solution, 1 ml of (0.5 N) HClO_4 was added, and put in 70°C for 20 min. To this mixture, 5 ml ferric chloride and 1 ml resorcinol in alcohol were added and kept in water bath for 10 minutes and cooled in tap water. Green colour indicates the presence of ribose.

The nitrogenous bases were detected as follows: The sRNA was hydrolysed with 0.5 N HCl at 120°C for 2 hours or 110°C for 2 hours, and an aliquot was spotted on Whatman No.1 filter paper and chromatographed using butanol:water (7:1) as solvent. The bases were detected under UV light and identified using standards. All the bases were detected.

Further work on the molecular weight determination, activity of sRNA using radioactive amino acids and the amino-acyl RNA synthetase are in progress.

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Aspergillus Stock List (1st Revision, July 1975)*

This listing supersedes the original listing in *Genetics* (1965) 52: 233-246 (Barratt, Johnson and Ogata) and the supplements in *Genetics* (1967) 56: 619-631 (Dorn) and in *Aspergillus News Letter* (1970) #11: 23-24 (Barratt) and represents a complete revision of the stocks and the listing.

The list consists of eight sections as follows:

- Section I. Alphabetical Listing of Genetic Loci and Alleles
- Section II. Aberration Stocks
- Section III. Wild Type Stocks and Related Strains
- Section IV. Mitotic and Meiotic Mapping Strains
- Section V. Special Purpose Strains
- Section VI. Numerical Listing of Stocks
- Section VII. Nomenclature: uvs and lys Terminology
- Section VIII. Key to Symbols

Gene symbols employed are those given in Clutterbuck, (1973) *Genet. Res.* 21: 291-296.

Stocks are available free of charge for research and teaching. A charge of \$25.00 per culture plus \$5.00 service fee is made to commercial concerns. Write directly to FGSC.

*Supported by grant GB-30487 from the National Science Foundation (U.S.A.).

Section I. Alphabetical Listing of Genetic Loci and Alleles*

The term "no T" signifies tested in diploids; the term "no T?" signifies tested in crosses, or not tested but derived from translocation free descendants.

Fungal Genetics Stock Center										
symbol	locus and allele	linkage group and arm	genotype	strain of origin FGSC#	translocation	mode of origin cations	new or induced translocation	original strain or untested descendant	best available strains likely to translocate free	numbers
ALLANTOIC ACID UTILIZATION	ααX1	VI	γA2;pyroΔ4	220	no T	?	279	none		
ACRIFLAVINE RESISTANT	AcrA1	III	pabaA1 γA2;coA1	--	T1(VI;VII)	S	none	36	204, 205, 221, 222, 36[
	AcrA3	III	biA1;adC12;	259	no T	S	none?	346	none	
			pyroA4					338	none	
ACTIDIONE RESISTANT	acrB2	IIR								
Act	ActA1	III	riboA1 γA1;nicB8	122	no T	UV	none	231	412, 511	
ADENINE REQUIRING	adA55	IL	prototroph	--	no T?	UV	?	216, 218	none	
	adB57	VIII	prototroph	--	no T?	UV	?	217	none	
	adC1	IIR	γA2 (veA ⁺)	--	T1(III;IV;VIII)	X	?	--	259, 319, 344	

*For loci not in FGSC collection see end of Section I

Section I. (continued)

symbol	locus and allele	linkage group and arm	strain of origin FGSC#	mode of translocation	Fungal Genetics Stock Center numbers	
					original strain or untested cations	descendant free
ADENINE REQUIRING (continued):						
ad	adD3	TIR	biA1	26	no T	UV
	adE8	IR	biA1	26	no T	UV
	adE20	IR	biA1	26	no T	T1 (II;VII) -- + other T?
	adF15	IR	biA1	26	no T	UV
	adF17	IR	biA1	26	no T	UV
	adG14	IL	biA1	26	no T	UV
	adH23	IL	biA1	26	no T	UV
	adI50	III	biA1	26	no T	UV
am	see mir					none
ANEURIN REQUIRING						
an	anA1	IL	biA1	26	no T	UV
	anB2	II(R?)	biA1;AcrA1;wA3	123	no T	UV
					Aberration?	31
						261,382
ARGININE REQUIRING						
arg	argA1	V1				230
	argB2	III	biA1	26	no T	UV
	argC3	VIIIR				?
						89
						256
BIOTIN REQUIRING						
bi	biA1 (veA ⁺)	IR	wild type (veA ⁺)	4	no T	X
						none
						26
BLUE ASCOSPORES						
bl	blA1	IIIR				268
						none
						26, 194, 357, 391, 394

bge	see fawn (fw)										
BROWN CONIDIA bwA1	VIR	[2n]proA1 γA2;wA3// adG14 pabaA1 biA1	--	{T1(III → VII) {T1(VI;VII)?	S	none	lost	366	112, 113, 114, 115, 366		
CHARTREUSE CONIDIA cha chaA1 chaA2	VIIIR VIIIR	biA1;choA1 biA1;sA2	1 316	T1(I;VII) T1(V;VIII)	S	none none	lost	372	23, 204, 360, 416 none		
CHOLINE REQUIRING cho choA1	VII	biA1	26	no T	UV	T1(I;VII)	1		75, 168, 342, 469, 511		
COLOURLESS ASCOSPORES cl clA6	IL				280				none		
NITRATE AND HYPOXANTHINE UTILIZATION cnx cnxB2* cnxB50** cnxE16***	VIIIR VIIIR IIR	AcrA1 wA3;niceB8 biA1 biA1;wA3	-- 26 --	no T no T {T1(III → VII) {T1(VI;VII)	?	UV UV	none none?	-- --	449 266 337, 462		
cnxE16	III								451, 490		
COMPACT MORPHOLOGY co coA1	VIIIR	pabaA1;biA1		T1(VI;VII)	S	none	29		120, 449		
DILUTE CONIDIAL COLOUR di diA1	III	γA2;pyroA4	220?	no T	UV	?	--		489, 503		

* also called ni21/1

** also called ni50

*** also called ni3

Section I. (continued)

Fungal Genetics Stock Center									
symbol	locus and allele	linkage group and arm	strain of origin genotype	strain of origin		mode of translocation	new or induced translocation	original strain or untested descendant	best available strains likely to translocate free
				FGSC#	translocation				
FLUOROACETATE	RESISTANT	ACETATE	NON-UTILIZATION						
fac	facA303	VR	wA3;pyroA4	--	T1(VI;VII)	S	?	--	502
	facB101	VIIIR	wA3;pyroA4	--	T1(VI;VII)	S	?	--	409,420
	facC102	VIIIR	wA3;pyroA4	--	T1(VI;VII)	S	?	--	427
FLUFFY	MORPHOLOGY	VII	biA1;methG1	219	no T			326	none
fl	flA1								
FLUOROPHENYLALANINE	RESISTANT	(also called fp)							
fpa	fpaA1	IL							
	fpaA91	IL	proA11 yA2;pyroA4	--	T?	NG	?	275	none
	fpaB37	IL	rib0A1 biA1	158	no T	S	none	387	390,439
	fpaC43	VIII	rib0A1 biA1	158	no T	S	none	422	507
	fpaD11¢	III	ødF17 pabaA1 yA2	--	T?	S	?	286	
								273	none
FRUCTOSE	NON-UTILIZATION								
fr	frA1	IVR	yA2;pyroA4	220	no T				
FAWN	CONIDIA	(also called bge)							
fw	fwA1	VIIIR	biA1		UV	T1(TV;VIII)	470,276,435	none	379
	fwA2	VIIIR	AcrA1;lysB5	26	no T	S	none	378	440,469
				205	no T	S	none	371	

GALACTOSE NON-UTILIZATION									
gal galA1	III	biA1;wA3	--	{ T1(III → VIII)	UV	?	--	--	211
galB3	II	biA1;wA3	--	{ T1(VI;VII)	UV	?	215	none	
galC4	VIII	biA1;wA3	--	{ T1(III → VIII)	UV	?	291	none	
galD5	IL	biA1;wA3	--	{ T1(VI;VII)	UV	?	--	494	
galE9	III	biA1;wA3	--	{ T1(III → VIII)	UV	?	--	214	
HISTIDINE REQUIRING									
his hisA10	IV(R?)	γA2 biA1;AcraA1;ribob2	--	no T (?)	UV	?	277	none	
hisC38	VIIIR	biA1;AcraA1 wA3;nicB8	--	no T	UV	?	257	none	
hisH13	VIII	γA2;biA1;AcraA1;ribob2	--	no T	UV	?	278	none	
hisJ122	VII	biA1;sB3	--	no T (?)	NA	?	299	none	
HYPOXANTHINE NON-UTILIZATION									
hx hxA1	VR	biA1	26	no T	DES	?	--	258, 491	
ISOLEUCINE REQUIRING									
ile ileA1	IIR	biA1;sB3	41	no T (?)	NA	none?	295	none	
ileA3 = ab1	IIR	biA1	26	no T	UV	?	--	505	
INDOACETATE RESISTANT									
iod iodA1	III	biA1;wA3;nicB8	--	T1(III → VII)	S	none?	--	492	
LACTOSE NON-UTILIZATION									
lac lacA1	VIR	γA2;pyroA4	220	no T	UV	none	--	349, 455	
lacB3	IIR	biA1;wA3	--	(T1(III → VIII) (T1(VI;VII))	UV	none	292	none	
LEUCINE REQUIRING									
lu luA1	IL	biA1	26	no T	UV	none	55	406	

C_{semi-dominant}

Section I. (continued)

Fungal Genetics Stock Center

symbol	locus and allele	linkage group and arm	genotype	strain of origin		mode of origin	new or induced translocations	original strain or untested descendant	best available strains likely translocation free
				FGSC#	translocation				
LYSINE REQUIRING									
lys	lysA1	VIR	wA1 (veA ⁺)	--	none	X	AbVI	38	350*
	lysB5	V	biA1	26	no T	UV	none	66 (smA1)	205,360,365
	lysD7	VII	biA1;bs3	41	no T(?)	NA	?	300	none
	lysD18	VII	biA1;bs3	41	no T(?)	NA	?	294	none
	lysD20	VII	biA1;bs3	41	no T(?)	UV	T1(III;VII)	395,418	none
	lysE13	V	biA1;bs3	41	no T(?)	NA	?	?	none
	lysE14	V	biA1;bs3	41	no T(?)	NA	?	296	none
	lysE23	V	biA1;bs3	41	no T(?)	NA	?	298	none
	lysF51	IR	biA1;bs3	41	no T(?)	NA	?	301	none
	lysF88	IR	biA1;bs3	41	no T(?)	NA	?	297	none
					no T(?)	NA	none?	293	376
MALTPOSE NON-UTILIZATION									
mal	malA1	VIR	yA2;pyroA4	220	no T	UV	?	57	437
METHYLLAMMONIUM RESISTANT									
	near R	meaB6	III					—	451,496
MELANIN FORMATION									
	mel	mel						332	none
METHIONINE REQUIRING									
meth	methB3	VI	yA2;pyroA4	220	no T	UV	?	272	none
	methG1	IVL	biA1	26	no T	UV	none	219	486,500
	methH2	III	biA1	26	no T(likely)	UV	none?	34	503

MORPHOLOGICALLY ABNORMAL		P	232	none
mo	moC96	III		
NITRATE REDUCTASE (also called nia and nii)				
ni	ni3 ni21 ni50 ni51	see cnxE16 see cnxB2 see cnxB50 see nirA14	-- -- 266 260	337,462 449 none 472
NICOTINAMIDE REQUIRING				
nic	nicA2 nicB8 nicC10	V VIIR VI	wild type (veA ⁺) biA1 biA1;AcrA1 wA3	4 26 no T no T
NITRATE PATHWAY REGULATOR				
nir	nirA14	VIIIR	biA1;phenA3	--
	r	see uvrs	T1 (I;IV)	S
DOLIGOMYCIN RESISTANT				
oli	oliA2	VIIR	pabaA1 yA2	187
DORNITHINE REQUIRING				
orn	ornA4 ornB7 ornB9 ornB20	IVR VIIIR VIIIR VIIIR	biA1	26
FALE CONIDIA				
p	pA2	VR	adG14 biA1	37

* contains "Ab VI" = Inversion?

PHENYLALANINE REQUIRING											
phen	phenA2	IIIR	biA1	26	no T	UV	T1(I;VIII)	304	150, 173, 180, 461		
	phenA3	IIIR	biA1	26	no T	S	T1(II;IV)	260	402		
	phenB6	VIR	biA1	26	no T	UV	?	394	499		
PROLINE REQUIRING											
pro	pro-94	IL	wild type ($\vee\wedge A^+$)	4	no T	UV	?	386	393		
	proA1	I	biA1	26	no T	UV	none	32	85, 343, 367, 411		
	proA2	I	biA1	26	no T	UV	T1(II;III)	111	364, 415		
	proA5	I	biA1	26	no T	UV	?	45	none		
PUTRESCINE REQUIRING											
pu	puA1	IIR	biA1;wA3	--	T1(III \rightarrow VIII); T1(VI;VII)	UV	T1(V;III) or (I;VIII)	--	341		
PYRIDOXINE REQUIRING											
pyro	pyroA4	IVR	biA1	26	no T	UV	none	33	220, 426		
RIBOFLAVIN REQUIRING											
ribo	riboA1	IL	biA1	26	no T	UV	none	158	188, 356, 361		
	riboB2	VIIIR	biA1;AcrA1	waA3	--	UV	?	--	198, 410		
	riboD5	VR	biA1	26	no T	UV	?	--	258		
SULPHATE NON-UTILIZATION											
s	sA1	IIIIL	biA1	26	no T	UV	T1(V;VI)	40	417		
	sA2	IIIIL	biA1	26	no T	UV	T1(V;VIII)	316	368		
	sA4	IIIIL	biA1	26	no T	UV	none?	2	339		
	sA49	IIIIL	biA1	26	no T	NG	none?	--	404		
	sA91	IIIIL	biA1	26	no T	NG	none?	--	405		
	sB3	VIR	biA1	26	no T	UV	none?	41	147, 369		
	sB25	VIR	riboA1;AcrA1, chaA1	--	no T	NG	?	383	none		
sC12 (=s0)		IIIIL	wild type ($\vee\wedge A^+$)	4	no T	NM	?	24	488		
sC13		III	biA1;wA3	--	(T1(III \rightarrow VII) { T1(VI;VII)}	UV	?	15	none		
sC22		III	riboA1 yA2	--	no T	NG	?	384	none		

Section I. (continued)

		Fungal Genetics Stock Center numbers			
symbol	locus and allele	linkage group and arm	genotype	strain of origin FGSC#	translocation
\$	sD50 sD85 sD157 sE15 sF211	NON-UTILIZATION VIIIR VIIIR VIIIR VIIIR VIIIR	biA1 biA1 biA1 biA1 biA1;pyroA4	26 26 26 26 33	no T no T no T no T no T
SULPHATE		(continued)			
				UV NG NG NG NG	T2(I;VIII) none? ? none? none?
					398,431,434 419 420 399 403
					none strains likely translocation free
SORBITOL	sbA3	NON-UTILIZATION VIR	biA1;wA3	-- T1(III→VIII) T1(VI;VII)	UV UV
					T2(I;VII)? --
					500,510
SMALL COLONIAL SIZE	smA1	III.	biA1	26	no T
SUPPRESSOR	suadE20 suC11adE20	IL IVR	adE20;pyroA4 pabaA1 yaA2 adE20 Acra1;cnxB2;coA1	227 --	T(?) no T
sumeth	see sup				
suornB9					
supabab22	suA1pa1B7 suB2pa1B7 suCpa1F15 suD2pa1A1 suB4pro	VIIIR VI V I III	biA1;pa1B7 biA1;pa1B7 biA1;pa1F15 biA1;pa1A1 proA5 biA1	244 244 247 243 45	no T(?) no T(?) no T(?) T(?) ?
supal					
supro					

SULPHANILAMIDE RESISTANT	sulA1	IL	$\gamma\text{A}2,\text{pyroA}4$	220	no T	UV	?	--	452, 511
THIAZOLE REQUIRING	thiA4	IIR	biA1	26	no T	UV	?	52	337
TRYPTOPHAN REQUIRING	tryP	I(R?)	pabaA1 $\gamma\text{A}2$	234	no T	UV	?	235	none
tryP	tryPA69	I(L?)	pabaA1 $\gamma\text{A}2$	234	no T	UV	?	236	none
tryP	tryPB403	VIII	pabaA1 $\gamma\text{A}2$	234	no T	UV	?	237	none
tryP	tryPC801	I(R?)	pabaA1 $\gamma\text{A}2$	234	no T	UV	?	238	none
tryP	tryPD432		pabaA1 $\gamma\text{A}2$	234	no T	UV	?		
TEMPERATURE-SENSITIVE						269			
ts	tsD15	VIII							
UREA NON-UTILIZATION	uY5	VII	biA1	26	no T	DES	262	none	
URIC ACID NON-UTILIZATION	uX10	VI	biA1	26	no T	DES	264	none	
ULTRAVIOLET SENSITIVE	uvS	uvS-4	addE20, biA1, wA3, methG1, pyroA4	--	no T	UV	?	329	none
	uvS-5		addE20, biA1, wA3, methG1, pyroA4	--	no T	UV	?	327	none
	uvS-77		addE20, biA1, wA3, methG1, pyroA4	--	no T	UV	?	330	none
	uvSAl							328	none

Section I. (continued)

Fungal Genetics Stock Center

35

symbol	locus and allele	linkage group and arm	genotype	strain of origin		mode of origin cations	new or induced translocation cations	original strain or untested descendant	best available strains likely to translocate free	
				FGSC#	translocation				original	original
ULTRAVIOLET SENSITIVE (continued)										
uvs	uvsB10	IV	pabaA108 biA1	--		UV	?	333	none	
	uvsC14	VIII	pabaA108 biA1	--		UV	?	334	none	
	uvsD153	V	proA1, pabaA125, biA1;pyroA4	--		UV	?	335	none	
	uvsE182	V	proA1, pabaA125, biA1;pyroA4	--		UV	?	336	none	
	uvsF201	IL	riboA1 ya2	361	no T	UV	none	--	389	
VELVET	MORPHOLOGY	VIII	wild type (veA ⁺)	--	no T	X	none	26(biA1)	all strains if not labelled (veA ⁺)	
ve	veA1									
WHITE CONIDIA										
w	wa1	III	wild type of Yui1(1936)	--	no T	S	none	38	none	
	wa2	III	ya2 (veA ⁺)	--	T1(III;IV;VII)	X	none	--	75,344	
	wa3	III	pabaA1, biA1	--	T1(VI;VII)	S	none	27	72,78*, 188, 191, 357, 359, 408	
	wa4	III	biA1;smA1;lysB5	66	no T	S	none	69	436	
WET WHITE CONIDIA										
Y	yA2	IR	wild type (veA ⁺)	4	no T	X	T1(III;IV; VIII)	lost, 23	71, 83, 187, 194, 361	
	y ^a A91 (olive)	IR	biA1;phenA2	498	no T?	S	none	425	445	
YELLOW-GREEN CONIDIA	ygA1	IR	biA1	26	no T	S			270	270

LOCI NOT AVAILABLE FROM FGSC:

	sul-req		
aaau	ppa	fan	mec
ab	ppp	flu	med
aba	prn	fmd	mg
ac	pur	gam	gdh
aco	pyc	glu	mol
al	pyr	azg	nia
alc	r	bga	nii
ald	uap	ben	npe
alp	uru	bri	ota
amrd	uvr	cre	exp
amrr	v	csu	pcnb
ani	wet	drk	pdh
anth	xpr	est	pen
			mas

*78 --- may carry suA1 adE20, wA2 and/or chaA1.

Section II. Aberration StocksReciprocal Translocation Strains

T1(I;IV)	:	260	T1(II;VII)	:	429
T1(I;VII)	:	1,129,354,430,438	T1(III;VII)	:	395,418,447
T2(I;VII)	:	355	T1(IV;VIII)	:	250,276,435,470
T1(I;VIII)	:	125,304,352	T1(V;VI)	:	40,370,373,518,521,522
T2(I;VIII)	:	398,431,434	T1(V;VII)	:	316,372,413
T1(II;III)	:	111,414	T1(VI;VII)	:	27,29,36,482,519,520
T1(II;IV)	:	345			

Other Aberration Strains

T1(VI;VII)	T1(III → VII) — strains (\pm UV treatment)	:	15,215,291,292
various intrachromosomal aberrations		:	38,228,229,261,382
T1(III → VII;VII)		:	340
T(I;VI;VII) possibly related to T2(I;VII)		:	347

Section III. Wild Types and Related Strains

FGSC NO.	Origin	References
4	Glasgow wild type (ve ⁺)	Yull (1939) Yull (1950)
90	Grindle wild isolate #2	Grindle (1963)
92	Grindle wild isolate #26	Grindle (1963)
94	Grindle wild isolate #36	Grindle (1963)
96	Grindle wild isolate #44	Grindle (1963)
251	Aspergillus heterothallicus A #WB5096	Raper
252	Aspergillus heterothallicus a #WB5097	Raper

Section IV. Mitotic and Meiotic Mapping Strains

Mitotic Mapping Strains

44	suA1adE20	adE20 biA1; AcrA1; phenA2; pyroA4; lysB5; sb3; choA1; coA1	choA1
68	suA1adE20	ya2 dfE20; AcrA1; phenA2; pyroA4; lysB5; sb3; nicB8; riboB2 (=MSD*)	
70	suA1adE20	adE20 biA1; AcrA1; phenA2; pyroA4; lysB5; sb3; nicB8; choA1	
79	suA1adE20	pabaA1 ya2 adE20; cnxE16; sC12; pyroA4; nicA2; sb3; choA1; choA1	
104	ya2 adE20; AcrA1; phenA2; pyroA4; lysB5; sb3; nicB8; coA1		
105	biA1; AcrA1 wa3; phenA2; pyroA4; lysB5; sb3; nicB8; coA1		
146	pabaA1; AcrA1; phenA2; pyroA4; lysB5; sb3; nicB8; riboB2 choA1		
154	adE20 biA1; wa2 cnxE15; sC12; methG1; nicA2; lacA1; choA1; choA1		
155	suA1adE20 pabaA1 ya2 adE20; AcrA1; phenA2; pyroA4; lysB5; sb3; choA1; coA1 choA1		
159	suA1adE20 pabaA1 ya2 adE20; AcrA1; phenA2; pyroA4; lysB5; sb3; nicB8; riboB2		
163	suA1adE20 ya2 adE20; AcrA1; phenA2; pyroA4; lysB5; sb3; nicB8; coA1		
283	suA1adE20 ya2 adE20; AcrA1; galA1; pyroA4; facA303; sb3; nicB8; riboB2 (=MSF*)		
288	suA1adE20 ya2 adE20; wa3; galA1; pyroA4; facA303; sb3; nicB8; riboB2 (=MSE*)		
374	suA1adE20 ya2 adE20; AcrA1; sa4; pyroA4; pa2; lacA1; nicB8; riboB2		
375	suA1adE20 adE20 biA1; AcrA1; sa4; pyroA4; pa2; lacA1; nicB8; riboB2		
407	adE20 biA1; AcrA1; phenA2; pyroA4; lysB5; lacA1; riboB2; choA1		
446	suA1adE20 ya2 adE20; AcrA1; galA1; pyroA4; facA303; lacA1 sb3; choA1; choA1		
465	suA1; AcrA1; galA1; pyroA4; facA303; lacA1; choA1; riboB2 choA1		
468	suA1; AcrA1 wa3; galA1; pyroA4; facA303; lacA1 sb3; choA1; riboB2 choA1		
473	suA1 adE20; AcrA1 wa3; ActA1; pyroA4; facA303; lacA1 sb3; choA1; riboB2 choA1		
477	suA1 adE20; AcrA1 wa3; ActA1; pyroA4; facA303; lacA1 vsB5; sb3; choA1; choA1		

. Glasgow Master strains

Section IV. (continued)

Mitotic Mapping Strains (continued):

- 478 *sulA1 adE20; AcrA1 wA3; ActA1; pyroA4; lysB5; sB3; choA1; riboB2 chaA1*
 480 *proA1 y^oA91; IodA1; phenA2; methG1; nicA2; sbA3; malA1; riboB2*
 487 *sulA1; AcrA1; galA1; PyroA4; facA303; lacA1 sB3; choA1 nicB8; riboB2 chaA1*
 513 *sulA1 adE20; AcrA1; ActA1; pyroA4; facA303; lacA1 sB3; choA1 chaA1*

Meiotic Mapping Strains (usually 4 or more markers)

- Linkage Group I 71, 87, 121, 193, 208, 275, 362, 363, 376, 432, 439, 463, 466, 475, 479, 483, 494, 507, 515
 Linkage Group II 239, 254, 268, 338, 505
 Linkage Group III 423, 441, 448, 456, 457, 458, 471, 490, 496, 516
 Linkage Group IV no T: 512, 517 [with T1 (IV;VIII): 250, 276, 435]
 Linkage Group V 258, 491, 495, 504, 508
 Linkage Group VI 459, 464
 Linkage Group VII 424, 444, 467, 481, 506
 Linkage Group VIII 269, 380, 401, 442, 484, 516

Main Meiotic Standards: 187, 356, 360, 408

Section V. Special Purpose Strains

- 1) Combinations of colour mutants; $\gamma A2$, $wA3$, $chaA1$, $bwA1$ (prototroph, no T): #78, 84, 112, -118, 366 (set of 10 strains)
- 2) Pairs of strains for diploids with all homologues marked: 159/154 or 68/154 or 283/154 465/480 or 477/480 or 513/480
- 3) Strains for Teaching or "mutagenesis" diploids: 514/515; 475/477 or 475/513
- 4) Set of strains for control, $T/+$ and T/T strains: a) T1(I;VII) : 424 (no T) 430 (T) 438 (T) 439 (no T)
b) T2(I;VIII) : 432 (no T) 431 (T) 434 (T) 433 (no T)
- 5) Strain for Triploid : 475//473/480
- 6) Strains for diploids to map centromeres:

Linkage Group I	Linkage Group II	Linkage Group III	Linkage Group IV	Linkage Group V, T1(V;VI)/T	Linkage Group VI	Linkage Group VII, T1(VI;VII)/T:
:	475/474 or 513	:	466/474	479/476 or 474	363/477 or 154	338/480
:	:	457/458	:	517/476	:	464/377
:	:	:	518/370 or 521	:	:	519/520

- 7) Strains for teaching meiotic recombination; w , cha and green strains $\pm AcrA1$: 189, 205, 221, 222, 356, 359, 360, 452 standard in Montreal
- 8) Strains for mutant selection by biotin starvation with $biA1$: 26, 194, 357, 378, 391, 392'
- 9) Back cross strains:
 - a) (veA⁺) BC VIII 17, 18, 83, 85, 126
BC IX 343
 - b) (veA1) BC V 360, 365
(veA1) BC IV 204, 205
 - c) (wA3; ± chaA1) BC V 221, 222, 223

Section VI. Numerical Listing of Stocks

This revised stock list was prepared in conjunction with FGSC by Dr. Eita Käfer-Boothroyd, McGill University, Montreal, as a revision from the original list by Barratt et al., 1965 (*Genetics* 52: 233-246 and [1] and [2] refer to Figures therein), the supplement thereto (Barratt, *Aspergillus Newsletter* 11: 23-24), and that of Dorn, 1967 (*Genetics* 56: 619-631) updating the cultures at the Fungal Genetics Stock Center, Humboldt State University Foundation, Arcata, CA 95521, U.S.A. Numbers in parentheses refer to Figures in Käfer, 1965 (*Genetics* 52: 217-232). Underline refers to the unique characteristic(s) of strain. Number sign (#) in origin column refers to FGSC stock number.

FGSC#	genotypes	Montreal	
		origin or Montreal cross nos.	stock number or depositor
1	<u>biA1;choA1</u>	T1(V _I ;VII)	M 7
2	<u>biA1;sA4</u>	no T(?)	M 27
4	Glasgow wild type (veA ⁺)	UV of #26	
11	<u>biA1;AcrA1 wA3;nicC10</u>	UV of #26	
15	<u>bi1;wA3;sC13 = cys2</u>	Yull, 1950 ^δ	M 139
		UV of #123 [†]	M 231
		UV of #51 [†]	M 422
		(T _I III → V _{III}) (T _I IV;V _I ;VII)	
17	adG14; (veA ⁺)	no T	BC VIII [1]
18	<u>adG14</u> yA2; (veA ⁺)	no T	BC VIII [1]
23	<u>pabA1</u> YA2;chaA1	no T(?)	Cross 507
24	sC12;(veA ⁺)	T(?)	NM of #40
26	<u>biA1</u>	no T	X of #4
27	<u>pabA6</u> biA1;wA3	T1(V _I ;VII)	X of #26 and S
28	<u>pabA6</u> biA1	T(?)	UV of #26
29	<u>pabA7</u> biA1;coA1	T1(V _I ;VII)	X of #26 and S
31	<u>anA1</u> biA1*	no T	UV of #26
32	<u>proA1</u> biA1	no T	UV of #26
33	<u>biA1;pyroA4</u>	no T	UV of #26
34	biA1;methH2*	no T (likely)	UV of #26
35	<u>adG14</u> biA1;paA2	S in #37	M 818
36	<u>pabA1</u> yA2;AcrA1;coA1	S in pabaA1	M 820
		yA2;coA1;	M 822
		T1(V _I ;VII)	
37	<u>adG14</u> biA1	UV of #26	M 823
38	<u>wA1;lysA1</u> (veA ⁺)	X of wa1?	M 829

40	<u>biA1;sa1</u>	M 837
41	<u>biA1;sb3</u>	M 834
42	<u>adE8 biA1*</u>	M 851
43	<u>biA1;ornA4</u>	M 232
44	Mitotic Mapping Strain	
45	<u>proA5 biA1</u>	T(?)
46	<u>adF15 biA1</u>	T(?)
47	<u>suA1;adE20 adE20;pyroA4</u>	S in #227
52	<u>biA1;thiA4</u>	T(?)
55	<u>luA1 biA1</u>	T(?)
57	<u>ya2 pyroA4;malA1</u>	T(?)
66	<u>biA1;smA1;ysB5</u>	T(?)
68	Mitotic Mapping Strain	
69	<u>biA1;waA4;smA1;ysB5</u>	T(?)
70	Mitotic Mapping Strain	
71 ^Δ	<u>suA1;adE20 riboA1 pabaA1 ya2;adE20;Acra1 wa2</u>	Cross of Forbes; MSD
72	<u>adG14 proA1 pabaA1 ya2;waA3</u>	S in #66
75	<u>suA1;adE20 biA1;waA2;choA1;chaA1</u>	C 434 [2]
78 [¢]	<u>wa3</u>	C KK (3)
79	Mitotic Mapping Strain	
83	<u>ya2;(veA⁺)</u>	BC VII [1]
84 ^Y	<u>ya2;chaA1</u>	C 419 [1]
85	<u>adG14 proA1 pabaA1 ya2;wa3 (veA⁺)</u>	BC VII [1]
87 ^Δ	<u>riboA1 adG14 proA1 pabaA1 ya2</u>	C X (2)
89	<u>biA1;argB2*</u>	UV of #26
90	Grindle wild isolate 2	M 894
91	white spores	MG

⁶ See Pontecorvo et al., 1953 (*Adv. Genetics* 5: 141-238).

Δ Meiotic mapping strain (see Section IV)

ξ_1, ξ_2 carry $suAladE20$, $\gamma A2$ and/or $chaA$

May carry suAladE20

* Poor strain, contains UV induced morphological mutants

J Hockenhull

*Discarded from collection; replaced by standard strain #357

Section VI. (continued)

FGSC#	genotypes	translocations		origin or Montreal cross nos.	Montreal stock number or depositor
92	Grindle wild isolate 26			UV of #92	MG
93	yellow spores			UV of #92	MG
94	Grindle wild isolate 36			UV of #94	MG
95	white spores			UV of #96	MG
96	Grindle wild isolate 44			UV of #96	M 301
97	yellow spores			C 282 (4)	M 276
104	Mitotic Mapping Strain			C 280 (4)	M 455
105	Mitotic Mapping Strain			C 349	M 233
108	adE20 biA1;wA2 cnxE16;sC12;methG1;nicA2;choA1;chaA1			UV of #26	M 545
111	proA2 biA1			<u>no T</u>	
112Y	YA2; <u>bwA1</u>			<u>no T</u>	
113Y	<u>bwA1</u>			<u>no T</u>	
114Y	<u>bwA1</u> ;chaA1			<u>no T</u>	
115¢	<u>wA3;bwA1</u>			<u>no T</u>	
116Y	YA2			<u>no T</u>	
117Y	prototroph, wild type colour (no veA ⁺)			<u>no T</u>	
118Y	chaA1			<u>no T</u>	
120	pabaA1 YA2 adE20;AcrA1;coA1			<u>no T</u>	
121Δ	suA1adE20 riboA1 proA1 adE20;chaA1			<u>no T</u>	
				(= cross d, K ["] afer 1962)	
122	riboA1 YA2;nicB8			<u>no T</u>	C 4
125	riboA1 biA1;AcrA1;pyroA4;sB3;nicB8			<u>no T</u>	C 62 (6)
126	pabaA1 YA2;wA3; (veA ⁺)			<u>no T</u>	BC VIII [1]
129	pabaA1 YA2 adE20;choA1			<u>no T</u>	C 103
				(= b 1, K ["] afer 1962)	
146	Mitotic Mapping Strain			<u>no T</u>	C 434 [2]
147	AcrA1;lysS5; <u>b3</u> ;chaA1			<u>no T</u>	C 434 [2]
148	suA1adE20 pabaA1 ya2 adE20;lysB5			<u>no T</u>	C 461 [2]
150	suA1adE20 adE20 biA1; <u>phen</u> 2;sB3;ni cB8			<u>no T</u>	C 434 [2]
					C 500
154	Mitotic Mapping Strain			<u>no T</u>	M 1043

155	Mitotic Mapping Strain		M 1056
158	<u>rib</u> <u>oA1</u> <u>biA1</u>	C 474 [2]	M 827
159	Mitotic Mapping Strain	UV of #26	M 1295
163	Mitotic Mapping Strain	C 283 (4)	C 283 (4)
168	<u>suA1</u> <u>adE20</u> <u>biA1</u> ; <u>b3</u> ; <u>choA1</u> ; <u>chaA1</u>	C 282 (4)	C 282 (4)
173	<u>suA1</u> <u>adE20</u> <u>pobaA1</u> <u>ya2</u> <u>adE20</u> ; <u>phenA2</u> ; <u>nicB8</u>	C 474 [2]	C 474 [2]
180	<u>adE20</u> <u>biA1</u> ; <u>phenA2</u> ; <u>b3</u> ; <u>chaA1</u>	C 461 [2]	M 947
183	<u>rib</u> <u>oA1</u> <u>pobaA1</u> <u>ya2</u> ; <u>wa3</u>	C 434 [2]	M 1061
184	<u>adG14</u> <u>ya2</u>	no T	no T
186	<u>rib</u> <u>oA1</u> <u>pobaA1</u> <u>biA1</u>	C 630 [1]	M 1072
187	<u>pobaA1</u> <u>ya2</u> (Meiotic Standard Strain)	C 630 [1]	M 1073
188	<u>rib</u> <u>oA1</u> ; <u>wa3</u>	C 630 [1]	M 1078
189	<u>adG14</u> ; <u>wa3</u>	C 630 [1]	M 1079
191	<u>wa3</u>	C 640 [1]	M 1085
192	<u>adG14</u>	no T	no T
193	<u>rib</u> <u>oA1</u> <u>adG14</u> <u>pobaA1</u> <u>ya2</u> ; <u>wa3</u>	C 640 [1]	M 1086
194	<u>ya2</u> <u>biA1</u>	C 640 [1]	M 1088
198	<u>adE20</u> <u>biA1</u> ; <u>bs3</u> ; <u>nicB8</u> ; <u>rib</u> <u>oB2</u>	no T	no T
201	<u>onA1</u>	C 639 [1]	M 1090
202	<u>rib</u> <u>oA1</u> <u>anA1</u>	C 637 [1]	M 1202
204	<u>AcrA1</u> ; <u>chaA1</u>	C 638 [1]	M 1205
205	<u>AcrA1</u> ; <u>lysB5</u>	C 434 [2]	M 935
206	<u>rib</u> <u>oA1</u> <u>adG14</u> <u>ya2</u>	C 758 [1]	M 1243
208 ^A	<u>suA1</u> <u>adE20</u> <u>rib</u> <u>oA1</u> <u>proA1</u> <u>pobaA1</u> <u>adE20</u> ; <u>choA1</u> ; <u>chaA1</u>	C 758 [1]	M 1244
211	<u>biA1</u> ; <u>wa3</u> ; <u>galA1</u>	C 751 [1]	M 1240
214	<u>biA1</u> ; <u>wa3</u> ; <u>gal</u> <u>E9</u>	C 751 [1]	M 1241
215	<u>biA1</u> ; <u>wa3</u> <u>gal</u> <u>B3</u>	C 758 [1]	M 1250
216	<u>adA55</u>	{ T1(III → VIII) T1(VI;VII) T(?) T(?) }	M 279
217	<u>adB57</u>	UV of green pro- totroph from cross #82++ x #34 (no T)	JMF JMF

May carry suA1 adE20

\$May carry sua lade20, y

Meiotic mapping strain (see Section IV)

Discarded from collection; replaced by standard strain #357

[†]Discarded from collection; see Barratt et al. *Genetics* 1965 for genotype

FGSC#	genotypes	translocations		origin or Montreal cross nos.	Montreal stock number or depositoir
218	<u>adA55</u> yA2 biA1;AcrA1;ribob2	T(?)	cross of #216 x yA2 JMF		
219	biA1;methG1	no T	biA1;AcrA1;ribob2	M 800	Montreal
220	yA2;pyroA4	no T	UV of #26	M 1105	stock number
221	adG14;AcrA1 wa3	no T	yA2;sC12 x #33	M 1248	or depositoir
222	AcrA1 wa3;lysB5;chaA1	no T	Cross 753 (no T)	M 1251	
223	wa3;lysB5;chaA1	no T	Cross 762 (no T)	M 1252	
227	adE20;pyroA4	no T	Cross 762 (no T)	RHP	
228	yA2;dp(adE20 biA1);pyroA4	T(?)	Cross AAA		
229	adE20 biA1;dp(adE20 biA1)	{ strains			
230	yA2;wa2;orgA1;(veA ⁺)	T(?)			
231	ribA1 YA2;ActA1;ni;cB8	no T	Cross of #344 x biA1;argA1 (T?)		
232	yA2;adD3;sA1 moC96	T(?)	UV of #122	BWB	
234	pabaA1 ya2	no T	P	BWB	
235	pabaA1 ya2;tryPA69	T(?)	from cross of JMF	C.F.R	
236	tryPB403 pobaA1 ya2	T(?)	UV of #234	CFR (M 1722)	
237	pabaA1 ya2;tryPC801	T(?)		CFR (M 1716)	
238	pabaA1 ya2;tryPD43/2	T(?)		CFR	
239 ^A	biA1;adH23 AcrA1 wa3;ni;cB8	no T	Cross 59	M 6	
241	biA1;pacA1	T(?)	recomb. as #243	GD	
242	biA1;pacC5	no T	UV of #26	GD (M 1541)	
243	biA1;pa AT	T(?)		GD (M 1484)	
244	biA1;pa B7	T(?)		GD (M 1654)	
245	biA1;pa DB8	T(?)		GD (M 1286)	
246	biA1;pa ET1	T(?)	*UV induced in rA1	GD (M 1653)	
247	biA1;pa F15	T(?)	biA1	GD (M 1287)	
248	biA1;pa CA1	T(?)		GD	
250 ^A	yA2;frA1 TI (IV;VII)	T(?)	from cross	GD	
251	Aspergillus heterothallicus				
252	(see Section III)				
254 ^A	biA1;AcrA1 wa3 ileA3 (= ab1)	T(IV;VIII)	Cross of E-Forbes		

256	pabaA1;wA3; <u>argC3</u> facB10T riboB2	T(?)	Cross of E. Forbes UV of no T strain	GD
257	biA1;AcrA1 waA3;ni ^r CB8;hisC8	T(?)	of J. Foley Cross of Darlington	MB
258 ^A	nicA2 hxA1 (not facA303) riboD5	no T	Cross R (2)	GD
259	biA1; <u>adCl</u> ;sC12;pyroA4	no T	S in biA1;phenA3;	GD (M 1579)
260	biA1; <u>phenA3</u> ;nirA14 (=ni51)	T1(I;IV)	T1(II;IV)	
261	biA1;AcrA1 wA3 anB2	no T, other oberration?	UV of #123 ^a	GD (M 1384)
262	biA1; <u>yY5</u>	T(?)	DES of #26	GD
263	biA1; <u>ornB20</u>	no T	Darlington	GD
264	biA1; <u>uaX10</u>	T(?)	UV of #26	GD (M 1378)
265	biA1;cnxB50 (= ni50)	no T	DES of #26	GD
266	ya2;wa2 <u>hiA4</u> cnxE16 (=ni3) <u>adD3</u> <u>biA1</u>	no T	Darlington	GD
268 ^A	paBA1; <u>tsD15</u> fwA2 facB10I riboB2	T(?)	UV of #26	GD
269 ^A	biA1; <u>ygA1</u>	T(?)	Cross of Apirion	GD
270	ya2;pyroA4; <u>methB3</u>	no T	Cross of Forbes	GD (M 1871)
272	ya2;pyroA4; <u>methB3</u>	T(?)	S of #26	GD (M 1386)
			UV of #220	GD
273	adF17 pabaA1 ya2; <u>fad11</u>	T(?)	mutant of Roberts	
275 ^A	F ^a A1 adG14 pabaA1 ya2	T(?)	mutant of Sinha	GD
276 ^A	ya2; <u>frA1</u> T1(IV;VIII) pabaB22 pyroA4	T(?)	from cross of McCully	GD
277	ya2 <u>biA1</u> ;Acra1;hisA10;riboB2	T(?)	T1(IV;VIII)	GD
278	ya2 <u>biA1</u> ;Acra1; <u>riboB2</u> hisH13	T(?)	{ Cross of Siddiqi his UV ind. by Berlyn	GD
279	ya2;pyroA4; <u>aaX1</u>	T(?)	{ in strain (no T) of JMF	GD
280	clA6 proA1 <u>pabaA1</u> ya2;palB7	T(?)	induced in #220 (?)	GD
281	biA1; <u>suA1</u> palB7;palB7	T(?)	from cross of Apirion	GD
		T(?)	• S in #244	GD

^AMeliotic mapping strain (see Section IV)

*Crossed to a variety of strains to separate from rA1 (e.g. x yA2;methH2 argB2 galA1 smA1 phenA2) #357

†Discarded from collection; replaced by standard strain #357

Section VI. (continued)

FGSC#	genotypes	Montreal stock number or depositor	
		origin or Montreal cross nos.	cross nos.
282	$b1A1;suC6;pa1;F15;pa1;F15$	T(?)	S in #247
283	Mitotic Mapping Strain	no T	MSF of McCully
284	$b1A1;suB2;pa1;B7;pa1;B7$	T(?)	S in #244
285	$b1A1;ad1;S0**$	no T	UV of #26
286	$riboA1;b1A1;fpA43$	T(?)	S in #158
287	$b1A1;suD2;pa1;pa1;A1$	T(?)	S in #243
288	Mitotic Mapping Strain	no T	MSF of McCully
291	$b1A1;wA3;2a1;C4$	UV in #51*	GD (M 1655)
292	$b1A1;wA3;locB3$		CFR
293	$lytF88;biA1;B3$	no T(?)	NA of #41, Pees
294	$biA1;B3;lytD18(BL*)$	T(?)	NA of #41, Pees
295	$biA1;leA1;B3$	no T(?)	NA of #41, Pees
296	$biA1;lytE13(EL* on V);sB3$	T(?)	NA of #41, Pees
297	$lytF51;biA1;B3$	T(?)	NA of #41, Pees
298	$biA1;lytE14(EL* on V);sB3$	T(?)	NA of #41, Pees
299	$biA1;B3;lytJ122(EL*)$	T(?)	NA of #41, Pees
300	$biA1;B3;lytD7(BL* on VII)$	T(?)	NA of #41, Pees
301	$biA1;lytE23(EL* on V);sB3$	T(?)	NA of #41, Pees
303	$proA1;ya2;AcrA1$	T(III → VIII)?	Cross 38 (3)
304	$proA1;pabaA1;ya2;phenA2$	T(II,VIII)	Cross 2 (6)
305	$proA2;pabaA18;biA1;phenA2;lytB5$	T(?)	RHP (M 1156)
306	$odf17;pabaA1;ya2$	T(?)	Cross of RHP?
307	$proA1;pabaA1;nicB8$	T(?)	Cross of RHP?
308	$pabaA18;biA1$	T(?)	RHP
309	$proA2;pabaA18;ya2$	T(?)	Cross of ?
310	$proA1;biA1;AcrA1;proA4$	T(III → VIII)?	RHP (M 1148)
311	$odf20;biA1;wA2;nicA2;ribod3$	T(?)	Cross of RHP?
312	$suA1;adE20;ya2;adE20;AcrA1;phenA2;lysS5$	T(?)	Cross of RHP?
314	$proA1;pabaA1;ya2;wA3;adC1(veA+)$	no T	Cross of Forbes, #13 × adC1 (veA+) strain, T(III → VII)

Teaching strains
Cloves and Hayes
1968

315	adG14 pobaA1 yA2	no T	Cross V (2) of G. Pontecarvo	M 825
316	biA1;sa2 anA1 ya2;wA3 adCl;sC12	#37 x pobaA1	ya2;coA1 T1(VI;VII)	
319	suA1 adE20 riboA1 proA1	UV of #26	UV of #26	M 845
320	biA1:metH G1;flA1	T(?)	Cross n, EKB	M 1128
326	adE20 biA1;wA3;metH G1	T(?)	Cross g, EKB	GD (M 1100)
327 ^a	biA1 ad-20;met-3;uvsA1 (probably allele of methG)	T(?)	mutant in #219?	JLA
328 ^a	adE20 biA1;wA3;metH G1	T(?)	UV of #139++	RWT
329 ^a	adE20 biA1;wA3;metH G1	T(?)	strain of Lanier	RWT
330 ^a	adE20 biA1;wA3;metH G1	T(?)	UV of #139++	RWT
332 ^a	meI (different wild type, from Birmingham)] 8-azaguanine	T(?)	UV of #139++	RWT
333 ^a	pobaA108 biA1;uvsB110	AZA	AZA	?
334 ^a	pobaA108 biA1;uvsC114	T(?)	UV of pobaA108 biA1	GJOJ
335	proA1 pobaA125 biA1;pyroA4;uvsD153	T(?)	UV of pobaA108 biA1	GJOJ
336	proA1 pobaA125 biA1;pyroA4;uvsE182	T(?)	UV of proA1	GJOJ
337	riboA1 biA1;wa3 thiA4 cnxE16 (= ni3)	T(?)	pobaA125 biA1;pyroA4	GJOJ
338 ^a	yA2;wA3 puA1 cnxE16 adC1 acrB2	no T	C 50	M 23
339	suA1 adE20 pobaA1 yA2 adE20;A4	T(?)	C 85	M 94
340	proA1 biA1;phenA2;pontoA1	no T	C 136	M 146
341	suA1 adE20 proA1 pobaA1 ya2 adE20;Acra1 puA1;choA1 nicB8;chaA1	T1 (III → VII)	C 243	M 347
342	suA1 adE20 biA1;Acra1;choA1;chaA1	no T	C 347	M 426
343	adG14 proA1 pobaA1 ya2;wa3; (ve ⁺)	no T	C 365	M 771
344	yA2;wA2 adCl;sC12;(ve ⁺)	no T	C 1620 (= BCIX)	M 799
345	biA1;sC12;pyroA4	T1 (II;IV)	C J (2)	M 814
346	biA1;Acra3 adCl;sC12;pyroA4	no T(?)	C s (2)	M 854
347	anA1 adE20 biA1;wa2;lysB5	T1 (I;V;VI;VIII)	S in #259	M 875
348	anA1 adE20 biA1;wa2;lysB5 [from cross of dupl. T1 (III → VIII)]	no T	S in #348	M 900
349	adE20 biA1;lacA1;choA1;chaA1	no T	C 512	M 921
350	biA1 phena2;lysA1 sb3	"Ab VI"=Inversion?	C 496	M 917
		"Ab VI"=Inversion?	C 346	M 918

^a

Meiotic mapping strain (see Section IV)

^o

See uvs --- terminology (Section VII)

*Leiden,

terminology of E. Pees

**adJ50 strains do not respond well on acetate

↑Discarded from collection; replaced by standard strain #357

+Discarded from collection; genotype adE20 biA1; waA3; methG1 pyroA4

Section VI. (continued)

FGSC#	genotypes	translocations		origin or Montreal cross nos.	Montreal stock number or depositor
		T1(I;VIII)	T(?)		
352	riboA1 adG14 yaA2;phenA2	C 2 (6)	M 962		
353	biA1;ornB9 riboB2	C 494	M 966		
354	AcrA1;lysB5	C 515	M 1014		
355	ana1 adE20 biA1;waA2;lysB5	C 542b	M 1054		
356	riboA1 biA1 (Meiotic standard strain) <u>biA1;wa3</u>	no T	C 630 [1]		
357	riboA1 adG14 (replaces #51)	no T	C 640 [1]		
358	riboA1 adG14 proA1 yaA2;ornA4	T(?)	C 3	M 1089	
359	pabaA1;waA3	no T	C 639 [1]	M 1101	
360	AcrA1;lysB5;chaA1 (Meiotic Standard Strain [5th BC])	no T	C 752	M 1231	
361	riboA1 ya2	no T	C 630 [1]	M 1239	
362 ^a	riboA1 ana1 adG14 proA1 pabaA1 yaA2	no T	C 773	M 1265	
363 ^a	suA ladE20 riboA1 ana1 luA1 pabaA1 yaA2 adE20 biA1;AcrA1	no T	C 792	M 1274	
364	suA ladE20 proA2 ya2 adE20;AcrA1 wa2	no T	Dipl. 629	M 1277	
365	ya2;AcrA1;lysB5;chaA1 (5th BC)	no T	C 752	M 1279	
366	yaA2;chaA1;bwA1	no T(?)	C 789	M 1282	
367	suA ladE20 proA1 yaA2 adE20;AcrA1 wa2	no T	Dipl. 628	M 1285	
368	riboA1 yaA2;sa2	no T	C 785	M 1301	
369	pabaA1 biA1;sb3	no T	C 788	M 1303	
370	adG14;sa1;pyroA4;chaA1	T1(V;VI)	C 838	M 1309	
371	AcrA1;lysB5;fwA2	no T	S in M 1124	M 1318	
372	biA1;sa2;chaA2	T1(V;VI)	S in #316	M 1319	
373	ana1;sa1;lysB5;chaA1	T1(V;VI)	C 838	M 1324	
374	Miotic Mapping Strain	no T	C 569	M 1335	
375	Miotic Mapping Strain	no T	C 569	M 1343	
376 ^a	suA ladE20 lysFB8 pabaA1 yaA2 adE20	no T(?)	C 841	M 1346	
377	riboA1 yaA2 adE20	no T	C 847	M 1368	
378	biA1;fwA1	no T	S in #26	M 1370	
379	ornB7 fwA1 (veA ⁺)	no T(?)	cross of AJC	M 1371	
380 ^a	lysB5;fwA2 cnxB2 facB101 psB7 chaA1	no T(?)	C 804	M 1373	
381	cnxB2 (=ni21/11) psB7	no T(?)	C 804	M 1376	
382	YA2;adH23 AcrA1 wa3 anB2	no T, other aberr.?	Cross of AJC	M 1383	

rib0A1;AcrA1;sB25;chaA1	T(?)	NG of M 1421 (lost) M 1398
rib0A1 ya2;sC22	T(?)	NG of M 1420 (lost) M 1400
biA1;sE15	no T(?)	NG of #26 M 1402
pro-94;(veA ⁺)	T(?)	UV of #4 M 1414
fpaA91 proA1 ya2;pynoA4	T(?)	NG of orig. proA1 strain
biA1;pyroA4;sF211	no T(?)	NG of #33 M 1433
uvsF201 pabaA1	no T(?)	C 915 M 1444
fpaA91 pabaA1 biA1;pyroA4	no T(?)	C 883 M 1447
biA1;chaA1	no T	C 843 M 1466
ya2 biA1;chaA1	no T	C 843 M 1469
suA1adE20 pro-94 ya2 adE20;AcrA1 wA3	no T(?)	Dipl. 860 M 1474
biA1;phenB6	T(?)	UV of #26 GD (M 1490)
biA1;sB3;lysD20 T1(III;VII)	T(?)	UV of #41 EP (M 1493)
pantoB100	no T(?)	UV?(BMR) M 1498
biA1;lysB5;sD50 T2(I;VIII)	T2(I;VIII)	C 980 M 1500
facB101 riboB2 sE15	no T	C 898 M 1503
ya2 adE20;AcrA1;sF211 palF15 choA1	no T(?)	C 2116 M 2281
facB101 riboB2 palB7 chaA1 sE15	no T	C 1932 M 1581 b
phenA3;palB7 chaA1 (ni51) = nirA14	no T(?)	C 1045 M 1626
pabaA1;wA3;pyroA4;sF211	no T(?)	C 966 M 1631
biA1;AcrA1 wA3;sA49;lysB5	no T(?)	C 1164 M 1641
pabaA1 ya2;sA91	no T(?)	C 1166 M 1643
luA1 ya2	no T	C 1605 M 1824
Mitotic Mapping Strain		C 1608 M 1832
pabaA1 ya2;wA3 (Meiotic Standard Strain)	no T	C 1621 M 1849
pabaA1;facB101 chaA1	no T(?)	C 1701 M 1854
pabaA1 biA1;riboB2 chaA1	no T	C 434 M 1869
proA1 pabaA1 yA2	no T	C 1709 M 1885
pabaA1 ya2;AcfA1	no T	C 1649 M 1897
pabaA1;sA2;chaA2	T1(V;VIII)	C 1708 M 1907
riboA1 proA2 ya2;ActA1	T1(I;III)	C 1760 M 1937
riboA1 proA2 biA1;ActA1	no T	C 1760 M 1940
riboA1;chaA1	no T	C 1754 M 1941

Δ Meiotic mapping strain (see Section IV)

Section VI. (continued)

FGSC#	genotypes	Montreal stock number or depositar	
		origin or Montreal cross nos.	translocations
417	pobaA1;sa1;chaA1	C 1754	M 1942
418	pobaA1 yA2;lysD20 T1 (III;VII)	C 1732	M 1944
419	biA1;D85	NG of #26	M 1951
420	pobaA1;sd1 57 riboA1;sD85	no T T1(III;VII) no T(?)	
421	riboA1;sD85	no T	
422	fpaB37 riboA1 biA1	no T	
423 ^A	riboA1 proA1 ya2;sC12 phenA2 suB4pro	no T	
424 ^A	sua1adE20 riboA1 proA1 pobaA1 ya2 adE20;AcrA1;pyroA4;phenB6	no T	
425	sf211 malA1 nicB8;chaA1 oA91 biA1;phenA2	no T(?)	
426	yA2;pobaB22 pyroA4	no T	
427	pobaA1;AcrA1;sD85 fa;cC102	no T	
428	pobaA1 adE20 biA1	no T	
429	pobaA1 adE20 biA1	no T	
430	sua1adE20 riboA1 proA1 pobaA1 ya2 adE20;pyroA4;phenB6 sf211 malA1;chaA1	T1(II;VII) T1(I;VII)	
431	galD5 suAladE20 riboA1 adE20 biA1;sD50 T2(I;VII) fwA2 facC102	T2(I;VII)	C1808
432	galD5 suAladE20 riboA1 adE20 biA1;fwA2 FacB101	no T	C1809
433	fpaB37 sulA1 anA1 pobaA1 ya2 adE20;pyroA4;cnxB2 chaA1	(±T1(VI;VII))	C1822
434	fpaB37 anA1 pobaA1 ya2 adE20;sD50 T2(I;VII) cnxB2 chaA1	T2(I;VII)[as #433]	C1822
435 ^A	fra1 T1 (IV;VIII) palC4 pobaB22 pyroA4;chaA1	T1(IV;VIII)	C1911
436	adE20 biA1;wA4;smA1;ysB5	no T	
437	proA1;wA3;nicA2;malA1	no T	
438	fpaA91 anA1 adE20 biA1;wa2;lysB5;choA1	T1(I;VII)	C1519
439 ^A	fpaA91 anA1 adE20 biA1;AcrA1 wa4;ActA1;choA1	no T	C1518
440	riboA1 ya2;AcrA1;fwA2	no T	
441 ^A	riboA1 proA1 biA1;wA3;sC12 galA1 ActA1 phenA2 suB4pro	no T	
442 ^A	facB101 riboB2 chaA1 sE15 nra14 (= ni51)	no T	
443	pobaA1 yA2 adE20;AcrA1;suC11adE20;coA1 cnxB2 (=ni21/11)	no T	
444 ^A	sua1adE20 yA2 adE20;AcrA1;phenB6 sfF211 malA1 choA1 nicB8	no T	

445	ribosomal A1	<u>y^oA91</u>	Mitotic Mapping Strain		M 2136
446	cmA1	<u>yA2;wA3;adI50** galA1</u>	ActA1;sF211 lysD20	T1(III;VII)	M 2139
447	choA1;nicB8	proA1;AcrA1;galE9 sA1	adI50** ActA1;chaA1		M 2150
448 ^Δ	AcrA1;lysB5;coA1	<u>cnxB2</u>			
449	pabaA1	<u>yA2;oliA2</u>			
450	pabaA1;meaB6	<u>cnxH3</u>			
451	sulA1	<u>anA1 pabaA1;wA3;chaA1</u>			
452	<u>oliA2 pantob100</u>				
453	ribmA1	<u>proA1 yA2;ActA1</u>	<u>sub4pro</u>		
454	sulA1;AcrA1	<u>wA3;lacA1</u>	<u>sB3;choA1</u>		
455	proA1	<u>yA2;galE9 adI50**</u>	<u>methH2 diIA1</u>		
456 ^Δ	proA1	<u>biA1;galE9 sC12 diIA1</u>	<u>phenA2;choA1</u>		
457 ^Δ	ribmA1	<u>proA1 yA2;AcrA1</u>	<u>adI50** methH2 galA1</u>	<u>ActA1 subB4pro</u>	
458 ^Δ	sulA1;adE20	<u>pabaA1 yA2 adE20;AcrA1;lacA1</u>	<u>sbwA1 sbA3;sF211</u>		
459 ^Δ	AcrA1;lysB5	<u>PA2</u>			
460	proA1	<u>biA1;phenA2;malA1;ribob2</u>			
461	pobaA1	<u>y^oA91;lodA1</u>	<u>cnxE16</u>		
462	sulA1;adE20	<u>pro-94 lysF88</u>	<u>pabaA1 yA2 adE20;wA2</u>		
463 ^Δ	sulA1;adE20	<u>pabaA1 yA2 adE20;AcrA1;lacA1</u>	<u>bwA1 sb3 sbA3</u>		
464 ^Δ	ribmA1	<u>adE20;lacA1;phenB6</u>	<u>oliA2 sF211 palF15 malA1 choA1</u>	<u>nicB8</u>	
465	Mitotic Mapping Strain				
466 ^Δ	galD5	<u>suA1;adE20 riboA1 anA1 pro-94 lysF88</u>	<u>pabaA1 yA2 adE20</u>		
467 ^Δ	yA2	<u>adE20;lacA1;phenB6 oliA2 sF211</u>	<u>palF15 malA1 choA1</u>	<u>nicB8</u>	
468	Mitotic Mapping Strain				
469	AcrA1;lysB5;sD85	<u>fwA2</u>			
470	proA1	<u>biA1;methG1 frA1</u>	<u>T1(IV;VIII);chaA1</u>		
471 ^Δ	proA1	<u>yA2;meaB6 sC12 methH2</u>	<u>diIA1</u>		
472	ribob2	<u>nirA14</u>			
473	Mitotic Mapping Strain				
474	suA1	<u>adE20;AcrA1;ActA1;choA1</u>	<u>chaA1</u>		
475 ^Δ	fpaB37	<u>galD5 suA1 adE20 riboA1 anA1</u>	<u>pabaA1 yA2</u>		
	adE20	<u>biA1;sD85 fwA2</u>			

Δ Mitotic mapping strain (see Section IV)
 ** adI50 strains do not respond well on acetate

Section VI. (continued)

FGSC#	genotypes	Montreal	
		stock number or depositor	origin or Montreal cross nos.
476	suA1 adE20;AcrA1 wA3;ActA1;lysB5;choA1; (chaA1?)	no T	C 2112
477	Mitotic Mapping Strain	no T	C 2125
478	Mitotic Mapping Strain	no T	C 2125
479Δ	galD5 suA1adE20 riboA1 anA1 pabaA1 γA2 adE20 biA1	no T	C 1523
480	Mitotic Mapping Strain	no T	C 1974
481Δ	ya2 adE20;AcrA1;phenB6 oliA2 sF211 malA1 choA1 nicB8	no T	C 2120
482	bwA1 sbA3;phenB6 oliA2;ribob2	no T	C 2121
483Δ	fpaB37 galD5 suA1adE20 suA1 riboA1 anA1 pabaA1 γA2 adE20 biA1	no T	C 1523
484Δ	AcrA1;lysB5;sD85 fwA2 facC102	no T	C 1983
485	riboA1 adE20;fwA2	no T	C 2105
486	riboA1 <u>yA2 adE20;methG1 sucC11adE20</u>	no T	C 2134
487	Mitotic Mapping Strain	no T	C 2134
488	pabaA1 ya2;sC12	no T	C 1987
489	riboA1 biA1; <u>dilA1</u> ;choA1	no T	C 1762
490Δ	proA1;meab6 cnxH3 adJ50** dilA1	no T	C 1967
491Δ	AcrA1;lysB5 pA2 facA303 hxA1 riboD5	no T(?)	C 2108
492	pabaA1 y ^o A91;lodA1	no T	C 2133
493	PabaB22	no T(?)	C 1978
494Δ	galD5 suA1adE20 riboA1 γA2 adE20;AcrA1	no T	C 1907
495Δ	lysB5 nicA2 pA2	no T	C 1951
496Δ	pabaA1 yA2;meab6 adJ50** dilA1	no T(?)	C 2127
497	AcrA1;palC4 pabaB22;chaA1	no T	C 2108
498	biA1;phenA2	T(?)	C 1911
499	suA1 adE20 pabaA1 γA2 adE20;phenB6 sf211	no T	C 1632
500	biA1;methG1;nicA2;sba3;malA1	no T	C 1951
501	adE20 biA1;smA1 ActA1	no T	C 1977
502	suA1;AcrA1;facA303 lysB5;sB3;chaA1	no T	C 1935
503	proA1 yA2;methH2 dilA1	no T	C 2125
504Δ	AcrA1;lysB5 pA2 riboD5	no T(?)	C 1969
		no T(?)	C 2127

505 ^A	pabaA1 (yA2?);AcrA1 wa3 ileA3	no T(?)
506 ^A	AcrA1;phenB6 oliA2 sF211 matA1 choA1 niaC8	no T
507 ^A	fPab37 sulA1 anA1 pabaA1	no T
508 ^A	AcrA1;nicA2 facA303;hxA1 riboD5;sB3;chaA1	no T
509	riboA1 adE20	no T
510	pobaA1 ya2 adE20;AcrA1;nicA2;sB3;A3	no T
511	sulA1;ActA1;choA1;chaA1	no T
512	AcrA1;pa C4 pabaB22 pyroA4;chaA1	no T(?)
513	Mitotic Mapping Strain	no T
514	anA1 adE20 biA1;AcrA1;ActA1;sB3;fwA2	no T
515 ^A	fPab37 galD5 suA1adE20 riboA1 ya2 adE20;pyroA4;facA303;chaA1	no T
516	galE9 meaB6 adI50** ActA1;chaA1 sE15 niaA14 (= n151)	no T?
517 ^A	riboA1 ya2 adE20;methG1 suC11adE20 pa C4 pabaB22 pyroA4	no T
518	AcrA1;lysB5 facA303 hxA1;lacA1 sb3 sbA3	T1(V;VI)
519	suA1adE20 riboA1 proA1 pabaA1 ya2 adE20;wA2;oliA2 malA1	T1(V;VI)
	choA1 nicB8	Dipl. 1995
520	pabaA1;AcrA1;sB3 sbA3;phenB6;riboB2	T1(VI;VII)
521	suA1adE20;AcrA1;galA1;pyroA4;pA2 facA303 riboD5;sB3	T1(V;VI)
	pobaA1 ya2 adE20;AcrA1;facA303;lacA1 sB3	T1(V;VI)
		C 2131
		M 2322

Δ Meiotic mapping strain (see Section IV)
** adI50 strains do not respond well on acetate

Section VII. Nomenclature: uvs and lys Terminology

1) uvs of Tuveson

FGSC#

328

Residual Genotype

bi1 ad20; met-3 (met-3 probably allelic to meth1, induced by Lanier).

Origin: cross of Glasgow strains with new mutants induced in these by Lanier (Bot. Gazette, 128, p.16, 1976).

Caution: ad20 according to Lanier gives 3-8% recombination with γ.

<u>Utrecht</u>	<u>Jansen</u>	<u>FGSC#</u>	<u>Strain of origin</u>
uvsA101		333	UT408 = pabaA108 biA1
B110		334	"
C114		335	"
D153			UT439 = proA1 pabaA125 biA1;pyroA4
E157			"
E180			"
E182		336	"
C190			"
B191			"
C194			"

2) uvs - mutants -change of isolation numbers (Kafér, Jansen and Pateman)

FGSC#

Strain of origin

<u>Utrecht</u>	<u>Jansen</u>	<u>FGSC#</u>	<u>Strain of origin</u>	<u>Linkage Group</u>
uvsA101		333	UT408 = pabaA108 biA1	IR
B110		334	"	IV
C114		335	"	VII
D153			UT439 = proA1 pabaA125 biA1;pyroA4	V
E157			"	V
E180			"	V
E182		336	"	VIII
C190			"	IV
B191			"	VII
C194			"	

unmapped mutants

uvs-111 and uvs-115 in UT408

28 mutants from uvs-116 to uvs-195 in UT439

3)	<u>Montreal</u>	<u>F GSC#</u>		<u>Strain of origin</u>	<u>Linkage Group</u>
	uvsF201	389 (F ₁)		M 1265 (riboA1 γA2)	IL
	F202			M 1421 (riboA1; AcrA1; chaa1)	VIII
	uvsG211				with T (III;IV?)
	<u>unmapped:</u>				
	3 uvs mutants (uvs-208, -209 and -228) all in			M 1423 (pabaA1 γA2; AcrA1)	

- 4) Pateman (Wright and P., Mut. Res. 9, 579, 1970) has agreed to add 300 to all isolation numbers. Resulting published mutants:
 uvs-304, -311 and -313 on IV,
 uvs-308 on V,
 uvs-302 on VII
- unmapped --- uvs-303, uvs-307, uvs-309

Section VII. (continued)5) Lys-gene terminology (lysC and D recombine freely, also lysB and E)

<u>Glasgow</u>	<u>Linkage Group</u>	<u>Leiden</u> -- (internal designations, used by Pees)
<u>old</u>		
lysI	VI	lysAL
lysA	V	lysCL
lysB	VII	lysDL
lysC	VII	(alleles 6, 15) lysBL
lysD		" 18 = FGSC #294 (?)
		" 7 = FGSC #300
		" 20 = FGSC #395
		(alleles 13 = FGSC #296)
	T(III;VII)	" 14 = FGSC #298
	V	" 231 = FGSC #301
		(alleles 51 = FGSC #297)
<u>new*</u>		
lysE	IR	lysEL
lysF		lysFL

*To be used for publications (according to Millington-Ward).

Section VIII. Key to Symbols

1) Symbols used are as follows:

Ab	Aberration	MSE	master strain E; excellent tester strain
BC	back cross	MSF	master strain F; excellent tester strain
C	cross	T	translocation (reciprocal or other Aberration strains)
MSD	master strain D		

2) Symbols used to indicate Montreal numbers or depositor are as follows:

AJC	A.J. Clutterbuck	GD	Gordon Dorn	MB	M. Berlyn
BMR	B.M. Revers	GJOJ	G.J.O. Jansen	MG	M. Grindle
BWB	B.W. Bainbridge	JLA	J.L. de Azevedo	RHP	R.H. Pritchard
CFR	C.F. Roberts	JMF	J.M. Foley	RTR	R.T. Rowlands
EP	E. Pees	M	Montreal	RWT	R.W. Tuveson

3) Symbols used in mode of origin are as follows:

AZA	azaguanine	P	β -propiolactone
DES	diethyl sulfate	S	spontaneous
NA	nitrous acid	UV	ultraviolet light
NG	nitrosoguanidine	X	x-rays
NM	nitrogen mustard		