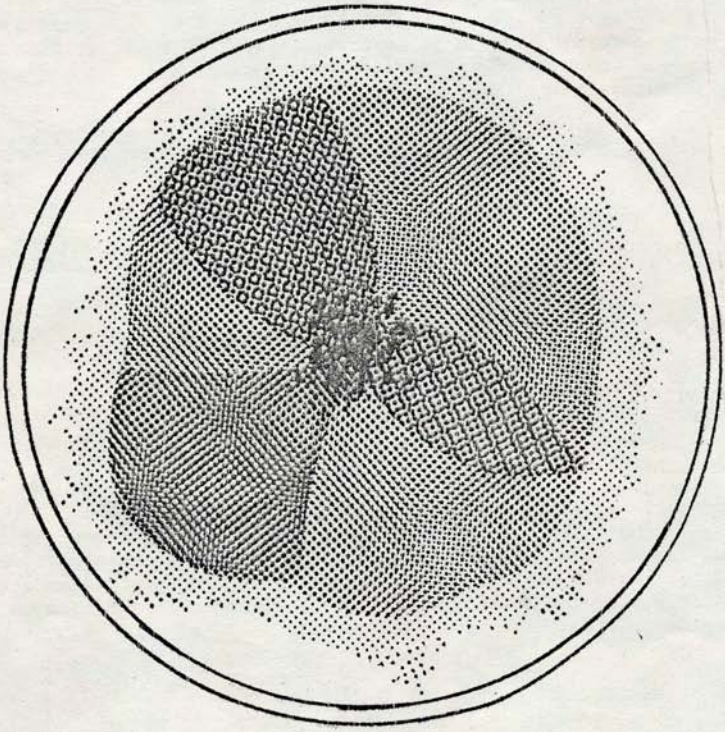
ASPERGILLUS NEWS LETTER

1978 NUMBER 14



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Dear Colleagues

I am again grateful to the following for help with the costs of this issue:

Glaxo Research Ltd.
Imperial Chemical Industries
Pfizer Ltd.

I also, once again, have to apologize for the long delay in production of this issue and also for its quality. We have been deprived of the print reduction system previously available and may for the next issue be forced to return to a large format.

That next issue I hope, despite all previous experience to produce in the summer of 1979, therefore all contributions arriving before the end of June 1979 will be welcome.

I would like to include in ANL 15 an updated list of gene symbols, I would be grateful, therefore if Aspergillus workers would draw my attention to gene symbols which have been used since the publication of the list in Handbook of Genetics 1974.

A.J. Clutterbuck

Fungal Genetics Stock Centre
Curator W.N. Ogata. FGSC. Humboldt State University Foundation, Arcata, California 95521, U.S.A.

Correction to stocklist in ANL 13:
FGSC strain 275: fpaA1 adG14 pabaA1 yA2.

Deposition of stocks

The stock Centre solicits deposition of cultures on which genetic or biochemical information is published or in press. Kindly write for deposition sheets prior to sending cultures. Additions or corrections to linkage data, genetic background, references etc. are welcomed,as are reprints concerned with strains in stock. Additional information on many stocks can be provided upon specific request.

ASPERGILLUS NIDULANS

Bibliography supplement 3

A basic bibliography was published in "Aspergillus nidulans", A.J.Clutterbuck 1974 in Handbook of Genetics I, 447-510, ed. R.C.King, Plenum Press, New York. Supplements 1 and 2 were in ANL 12 and 13 respectively.

I am grateful for the following omissions and errors that have been pointed out to me (corrected region underlined):

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Azevedo, J.L. & J.A.Roper 1970. Mitotic non-conformity in Aspergillus: successive....
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ANL 12:

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Omissions (before 1973):

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International Microbial Genetics Commission

At a meeting of the Executive Board of the International Association of Microbiological Societies (IAMS) held in Tokyo in 1974, it was decided to establish a Genetics Commission of the IAMS. This would bring together three bodies of the IAMS currently interested in Genetics, namely the Microbial Breeding Group, the Plasmids Group and the Genetic Engineering Committee.

This Commission is now functioning as the International Microbial Genetics Commission (IMGC). The composition is:

		Representing
Chairman	Professor S.W. Glover (U.K.)	Genetic Engineering Committee
Vice-Chairman	Professor E. Wollman (France)	" " "
Secretary	Professor B.W. Holloway (Australia)	IAMS Bacteriology Section
	Professor E.S. Anderson (U.K.)	Plasmid Commission
	Dr T. Arai (Japan)	" "
	Dr L.S. Baron (U.S.A.)	Genetic Engineering Committee
	Professor H. Heslot (France)	Microbial Breeding Subcommission
	Professor Sermonti (Italy)	" " "
	Professor G. Hirst (U.S.A.)	IAMS Virology Section
	Dr V. Vonka (Czechoslovakia)	" " "
	Dr Y. Hirota (Japan)	IAMS Bacteriology Section
	Prof. Dr R. Vanbreuseghem (Belgium)	IAMS Mycology Section
	Dr A. Stenderup (Denmark)	" " "
	Professor L. Alfoldi (Hungary)	Co-opted Member
	Professor G. Bertani (Sweden)	" "
	Professor A. Rorsch (Holland)	" "
	Professor Dr P. Starlinger (Germany)	" "
	Dr J. Puig (Venezuela)	" "
	Dr D. Goldfarb (U.S.S.R.)	" "
	Dr N. Notani (India)	" "

.../2

The objectives of IMGC are as follows:

(a) To maintain contact with laboratories, institutions and societies throughout the world dealing wholly or in part with microbial Genetics. This contact shall be within the general organization of the International Association of Microbiological Societies (IAMS).

(b) To initiate and maintain subcommissions and working groups as may be appropriate to encourage international co-operation and agreement.

(c) The planning and support of international and regional meetings, symposia and seminars to ensure rapid and effective dissemination of new and relevant knowledge of microbial genetics and to encourage personal and organizational contacts within the field.

(d) To maintain contact through IAMS with relevant UN organizations.

(e) To encourage research in microbial genetics and its applications to problems affecting the welfare of all people.

(f) To encourage the development of microbial genetics in countries where it is not strongly represented.

The Commission (through the Secretary) would be pleased to hear of any specific activities in which it could become involved to help achieve these objectives.

B. W. Holloway,
Secretary, International Microbial
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A table for Calculation of the LD_{95}

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The term LD_{95} refers to the least dosage that should be expected to kill 95% of the individuals that received it.

For certain mutagenic agents, as ultraviolet, the LD_{95} is considered a convenient dose to obtain mutants in fungi (Bernett, 1975).

Frequently the determination of LD_{95} in Aspergillus is based on a graphical method that is convenient for many pratical purposes. However it may be often suitable to determine this letal dose by an algebraic method. In this case, table 1, that permit us to determine the LD_{95} by interpolation, may be used. This table has been calculated according to formulae presented by Piedrabuena (1975).

The general formula for calculation of LD_{95}, using table 1, is

$$LD_{95} = D_1 + (D_2 - D_1)\, r \qquad (1)$$

where D_1 and D_2 ($D_1 < D_2$) are the doses, used in the calculation, r, the values in the table, for the proportion of survivals P_1 and P_2, that correspond to D_1 and D_2 respectively.

Exemple - Conidia of Aspergillus niger were irradiated with ultraviolet and the following survivals resulted,

Dose (minutes)	Percentage of survivals
10	40,0
20	4,0

From these data we have $D_1 = 10$, $D_2 = 20$, and from table 1, $r = 0.907$, since $P_1 = 0.40$ and $P_2 = 0.04$.

Now if we substitute these values into formula (1), we have

$$LD_{95} = 10 + (20 - 10)\ 0.907$$
$$= 19.07$$

19.07 minutes of irradiation is the dosage that should be expected to kill 95% of the conidia that received it.

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Table 1 - Values of r

P_1 \ P_2	0.10	0.09	0.08	0.07	0.06	0.05	0.04	0.03	0.02	0.01
0.40	1.499	1.388	1.284	1.186	1.091	1.000	0.907	0.813	0.711	0.589
0.39	1.512	1.397	1.290	1.189	1.093	1.000	0.906	0.811	0.708	0.585
0.38	1.526	1.407	1.296	1.193	1.094	1.000	0.904	0.808	0.705	0.582
0.37	1.541	1.418	1.303	1.197	1.096	1.000	0.903	0.806	0.702	0.578
0.36	1.558	1.429	1.311	1.201	1.098	1.000	0.902	0.803	0.699	0.574
0.35	1.576	1.442	1.319	1.206	1.100	1.000	0.900	0.801	0.695	0.570
0.34	1.597	1.455	1.328	1.211	1.102	1.000	0.898	0.797	0.691	0.566
0.33	1.619	1.470	1.338	1.216	1.104	1.000	0.896	0.795	0.686	0.562
0.32	1.645	1.487	1.348	1.222	1.107	1.000	0.894	0.791	0.683	0.558
0.31	1.673	1.506	1.359	1.229	1.110	1.000	0.892	0.788	0.679	0.553
0.30	1.706	1.527	1.372	1.236	1.113	1.000	0.890	0.784	0.674	0.547
0.29	1.744	1.555	1.387	1.244	1.116	1.000	0.887	0.780	0.669	0.542
0.28	1.789	1.577	1.403	1.253	1.119	1.000	0.885	0.776	0.664	0.537
0.27	1.842	1.610	1.422	1.262	1.123	1.000	0.882	0.771	0.658	0.531
0.26	1.904	1.647	1.442	1.274	1.128	1.000	0.880	0.766	0.652	0.524
0.25	1.983	1.691	1.467	1.286	1.132	1.000	0.876	0.761	0.646	0.518
0.24	2.085	1.745	1.496	1.300	1.138	1.000	0.872	0.755	0.638	0.510
0.23		1.812	1.530	1.317	1.144	1.000	0.868	0.749	0.631	0.502
0.22		1.899	1.573	1.336	1.151	1.000	0.864	0.742	0.623	0.493
0.21			1.626	1.360	1.160	1.000	0.858	0.734	0.614	0.484
0.20				1.389	1.170	1.000	0.853	0.725	0.604	0.475
0.19				1.426	1.182	1.000	0.847	0.715	0.593	0.464
0.18					1.196	1.000	0.839	0.705	0.581	0.452
0.17					1.214	1.000	0.830	0.693	0.567	0.439
0.16						1.000	0.820	0.678	0.552	0.425
0.15							0.808	0.661	0.535	0.409
0.14							0.793	0.642	0.515	0.391
0.13								0.619	0.492	0.371
0.12								0.591	0.465	0.348
0.11								0.556	0.433	0.322
0.10									0.395	0.290

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Chromosome II of Aspergillus nidulans: a new translocation, meiotic linkage and possible multiple mitotic crossing-over

Previous mitotic analyses (KÄfer 1958, Clutterbuck 1970) suggested the following gene order for chromosome II:

AcrA wA palcA riboE thiA puA abA cnxE ygA acrB

These analyses were based on selection, from heterozygotes, of homozygotes for acrB (KÄfer) and ygA (Clutterbuck). From diploids heterozygous for a duplicate segment of I, terminally attached to IIR, Case (1975) selected segregants which had lost by mitotic crossing-over, the translocated duplicate segment. This approach has the merit of selecting the products of crossing over within the whole arm. The results suggested a revision for the IIR gene order: riboE acrB thiA; recent meiotic results support this suggestion.

Most of these meiotic data were obtained using a new translocation strain, produced by exposing a chromosomally-standard strain, proA1 biA1, to a dose of gamma irradiation (75kr from a Co^{60} source). The survivors were plated on MM + pro + bi and subjected to mitotic and meiotic analysis. One of the morphologically normal survivors was shown, by mitotic analysis, to carry a translocation involving chromosomes II and III. Crosses of the TII-III strain and several standard strains gave a morphological segregation of 2 normal 1 abnormal and showed linkage of the wA locus and the determinant of abnormal morphology. These abnormal colonies behaved in a similar manner to duplication strains producing improved sectors on further incubation (Bainbridge and Roper, 1966, Nga and Roper, 1968). Analyses with two standard strains yA2; adH23 wA3 acrB2; and AcrA1 wA7 riboE6 thiA1 abA1 cnxE16 ygA6; (G 233) showed that the TII-III was of a non-reciprocal nature with a substantial portion of IIR attached to III. Duplication progeny heterozygous for acrB, ygA, cnxE, abA and thiA were obtained; these gave, by the loss of chromosomal material, sectors which revealed the recessive alleles. The linkage relationships of these alleles to the translocation breakpoint were determined using the Bainbridge technique (1970). A small sample of normal progeny were also analysed from one of these crosses.

A routine cross of G 233 to a standard TIIII→VIII translocation strain also yielded some data of interest. Only the III-VIII duplication progeny were analysed from this cross. Here a slight complication occurred because these duplication strains were found to be hypersensitive to acriflavin even when carrying the AcrA1 allele. However, the presence or absence of this allele could be tested by using medium containing 1/5 th of the normal concentration of acriflavin.

The results from all these crosses are summarised below:

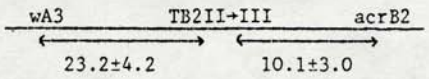
Cross A proA1 biA1; T2II→III; x MSE

wA3 TB2II III

←——→

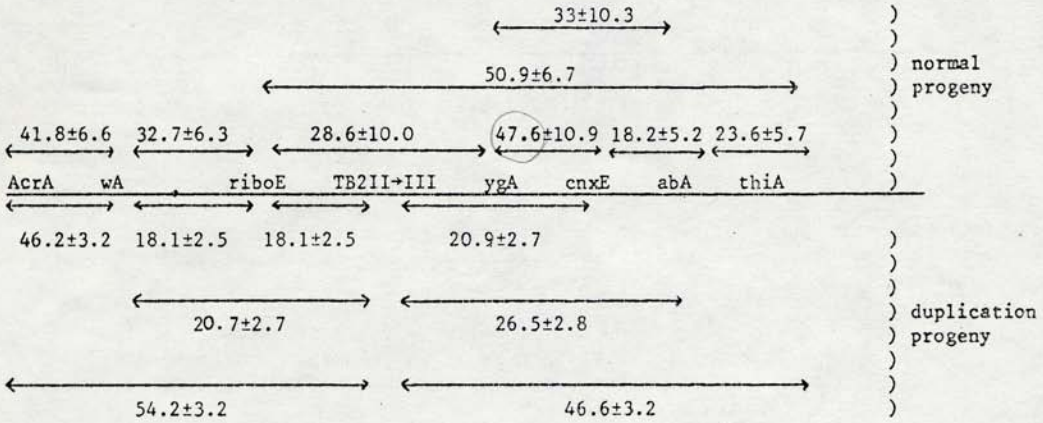
23.4±5.3

Cross B proA1 biA1; T2II→III; x yA2; adH23 wA3 acrB2;



Cross C

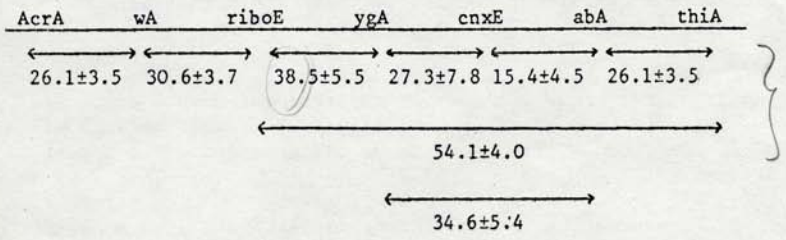
proA1 biA1; T2II→III; x AcrA1 wA7 riboE6 thiA4 abA1 cnxE16 ygA6;



proA1 → biA1 26.1±5.9 normal progeny
proA1 → biA1 23±2.7 duplication progeny

Cross D

proA1 biA1; cnxH3 sC12; T1III→VIII; x AcrA1 wA7 riboE6 thiA4 abA1 cnxE16 ygA6;



39% from 50

54 from 50

proA → biA 28±3.6

Distinguishing between wA7 ygA$^+$ and wA7 ygA$^-$ especially in the duplication strains was often difficult so wA7 was treated as a normal white allele. Also, the II→III duplication strains produce yellow-green heads in their colony centres due to their instability making it impossible to classify the colour accurately. Because of this, the linkage of ygA to the breakpoint could not be calculated.

Even though the data from crosses A, B and C may be disturbed due to the presence of the translocation, the overall data seems to confirm the suggestion (Case 1975) that the loci flanked by thiA and acrB should be in reverse order. The corrected order would then be:-

AcrA wA riboE TB2II→III acrB ygA cnxE abA thiA

It could be argued that the T2II→III segment had been inverted during the translocation process. However if the II→III duplication strains heterozygous for the ygA6 allele are grown at 42°C, they produce some yellow-green sectors that have no growth advantage. These sectors have a high level of instability similar to that of the parental duplication strain and are due to mitotic crossing-over between the duplicate segments. This behaviour would not be expected for a duplication strain carrying one segment in the normal and one in the inverted orientation.

Accepting the above gene order, there is still the problem of the mitotic data. It is possible that there is an inversion on chromosome II but since one of the strains used by Clutterbuck (1970) was G 233, this would not explain all of the data.

If the data presented by Käfer (1958) and Clutterbuck (1970) is considered in terms of double mitotic events as suggested by Case (1975) then the results are more in line with other mitotic data. For double events, the chance of a second cross-over occurring at the same time as the selected event decreases with increasing distance from the centromere. This would explain why the previous data and order suggested that mitotic crossing-over increased close to the centromere, decreased further out and then increased again towards the end of the chromosome arm.

In other cases of mitotic mapping, the position of the distal marker selected for has been confirmed by meiotic analysis. In these cases the selection procedure requires the occurrence of an odd number of cross-overs as did the method used by Case (1975) for chromosome II.

It would seem that mitotic crossing-over may be a more frequent event than first anticipated. Double events on the same chromosome arm would appear to be fairly common if only on the meiotically long chromosome arms of Aspergillus nidulans, while triple events on the same arm would appear to be much less frequent.

It would be interesting to know if any workers have used a non-distal marker for selective purposes on chromosome VIII and found similar results.

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Conidiospore size in duplication strains of *Aspergillus nidulans*

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Strains of *Aspergillus nidulans*, which carry translocated duplications of chromosome segments, are unstable at mitosis and produce chromosomally balanced sectors, which have suffered deletions from the segments carried in excess. The instability of two duplication strains (carrying duplicate segments of chromosomes I and III on the right arms of chromosomes II and VIII respectively) have been extensively studied and show loss of genetic markers carried on the excess segments (Bainbridge, B.W. & Roper, J.A., 1966, J. Gen. Micro. 42, 417; Nga, B.H. & Roper, J.A., 1968, Genetics 58, 193; Roper, J.A. & Nga, B.H., 1969, Genetical Res. 14, 127).

The diameters of conidiospores from standard and duplication strains were determined microscopically using a calibrated micrometer eye-piece. The I-II duplication produced a significant increase in the conidiospore diameter of both haploid and diploid strains and a diploid with two I-II duplicate segments had a conidiospore size that was even greater than those with a single duplicate segment. These increases in size could be attributed to the duplication because, as shown in Table 1, the residual

genotype had little effect on conidiospore diameter and because a yellow sector from strain A (which was stable and likely to have a quantitatively standard or near standard genome) had a conidiospore diameter approaching that of a standard haploid.

The III-VIII duplication haploid showed an increase in conidiospore diameter which was less than that produced by the I-II duplication and the double duplication haploid had a conidiospore diameter comparable with that of a single I-II duplication haploid.

These definitive differences in conidiospore sizes encouraged the investigation of the quantitative relationship of cytoplasm to nucleus, in other phases of growth, to see whether the differences between duplication and standard strains were maintained.

Germinating conidiospores at the two, four and eight nuclei stages of growth as well as hyphal tips were examined by fluorescenc microscopy using acridine orange stain, having grown the cultures on microscope slides covered with a thin layer of agar medium (Case, B.L., 1976, Ph.D. thesis, University of Sheffield, adapted from the method of Clutterbuck, A.J. & Roper J.A., 1966, Genetical Res. 7, 185). From photographs of the various stages, projected areas were determined using a planimeter and these were converted to volumes.

At the two nuclei stages in conidiospore germination there was a larger mean volume per nucleus in strain A (40.5 μm^3) compared with biA1 (35.1 μm^3). However, no increase was found at any of the later stages examined.

Table 1. Conidiospore diameter measurements in standard and duplication strains

STRAIN	SAMPLE SIZE	MEAN DIAMETER, μm.
Standard haploid		
biA1.	100	3.5
M.S.E.	100	3.3
yA;wA3 thiA4 acrB2;pyroA4; facA303;sB3;riboB2.	50	3.5
III-VIII duplication haploid		
biA1;sC12;dpIII-VIIIsC12$^+$.	100	3.7
I-II and III-VIII double duplication haploid		
proA1 pabaA6 yA adE20$^+$ biA1$^+$;dpI-IIyA$^+$ adE20 biA1;sC12$^+$;dpIII-VIIIsC12.	100	3.9
I-II duplication haploid		
proA1 pabaA6 yA adE20$^+$ biA1$^+$; dpI-II yA$^+$ adE20 biA1.(strain A)	100	3.9
riboA1 yA biA1;dpI-II yA biA1$^+$; nicB8.(strain E)	50	3.9
proA1 pabaA6 yA$^+$ adE20 biA1; dpI-II yA adE20$^+$ biA1$^+$.	100	3.9
yA adE20$^+$ biA1;thiA4 dpI-II yA$^+$ adE20 biA1.	50	4.0
Standard diploid		
biA1 // M.S.E.	100	4.4
I-II duplication diploid		
strain A // M.S.E.	50	4.5
pabaA6 yA adE20$^+$ biA1$^+$ niiA$^+$; dpI-II yA$^+$ adE20 biA1 niiA // M.S.E.	100	4.7
pabaA6 yA adE20$^+$ biA1$^+$ niiA; dpI-II yA$^+$ adE20 biA1 niiA$^+$ // M.S.E.	100	4.7
I-II double duplication diploid		
strain E // yA adE20$^+$ biA1;thiA4 dpI-II yA$^+$ adE20 biA1;sA1;pyroA4.	50	4.9

Clutterbuck (1969, J. Gen. Micro. 55, 291) has shown that the mean volume of cytoplasm, in hyphae with haploid or diploid nuclei, is ploidy related; this must reflect an effect of ploidy on a mitotic trigger, possibly by controlling the initiation of DNA replication. In duplication haploids, which are "diploid" for a part of their genome, an alteration in cytoplasmic volume might be predicted as a compensation for the excess genetic material. The finding of a volume increase in conidiospores might be significant although in many ways the conidia are specialized cells. The lack of an increase in later stages of growth from germinating spores may indicate that replication of the duplicate segments is under less than optimal control, and this could have a role in its instability.

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Products of A. nidulans

No comprehensive list of products from A. nidulans appears in the literature. The following table was therefore compiled from published data. Twenty-two compounds are given in the table, but others probably exist. Production of many is probably strain-specific, as was shown with benzylpenicillin, for example (Holt, G. and Macdonald, K.D., 1968, Antonie van Leeuwenhoek 34, 409).

Product	Nature of compound	Biological activity* (where known)	First published report
	Products of A. nidulans var. echinulatus		
Echinocandin B	Polypeptide	F	Benz, F., Knusel, F., Nuesch, J., Treichler, H., Voser, W., Nyfeler, R. and Keller-Schierlein, W. (1974). Helv. Chim. Acta. 57, 2459.
Benzylpenicillin (from Birmingham isolate No. 12)	β-Lactam	GP	Merrick, M.J. and Caten, C.E. (1975). J. Gen. Microbiol. 86, 283.
Cordycepin (from Birmingham isolate No. 23 - this is given as A. nidulans var echinulatus by Grindle (1963) Heredity, 18, 191)	Nucleoside	GP, T	Merrick, M.J. (1973). Ph.D. Thesis, Birmingham University

Product	Nature of compound	Biological activity* (where known)	First published report
U-13,933 (Asperline)	δ-Lactone	GP, GN, F	Argoudelis, A.D., Coats, J.H. and Herr, R.R. (1965). Ant. Ag. Chemo., 801.
Sterigmatocystin	Xanthone	M	Holzapfel, C.W., Purchase, I. F.H., Steyn, P.S. and Gouws, L. (1966). S. African Med. J. 40, 1100.
Nidulotoxin	(Unknown)	M	Lafont, P., Lafont, J. and Frayssinet, C. (1970) Experientia 26, 61.
3'-amino-3'-deoxyadenosine	Nucleoside	T, F	Suhadolnik, R.J. (1970). "Nucleoside Antibiotics" New York, Wiley Interscience, p76.
Trisdechloro-nornidulin	Depsidone		Sierankiewicz, J. and Gatenbeck, S. (1972). Acta Chem. Scand. 26, 455.
Orsellinic acid	Hydroxybenzoic acid		(As for trisdechloro-nornidulin)
Emerin	(Analogous to the mycotoxin, xanthocillin X, from A. chevalieri)		Ishida, M., Hamasaki, T. and Hatsuda, Y. (1972). Agr. Biol Chem. 36, 1847.
Averufin	Anthraquinone		(As for emerin)
Versicolorin C	Difuroanthraquinone		(As for emerin)
Asperthecin	Anthraquinone		Laskin, A.I. and Lechevalier, H.A. (1973). "Handbook of Microbiology, Volume III". Cleveland, CRC Press, p214.
Emericellin	Xanthone		Ishida, M., Hamasaki, T., Hatsuda, Y., Fukuyama, K., Tsukihara, K. and Katsube, Y. (1975). Agr. Biol. Chem. 39, 291.
Antibiotic produced in Soyabean extract medium	(Unknown)	GP, F	Cole, D.S., Edwards, G.F.St.L Holt, G. and Macdonald, K.D. (1976). Proc. Vth Int. Ferm. Symp., p193.
Anti-Semliki Forest Virus substance	(Unknown)	V	Maheshwari, R.K. and Gupta, B.M. (1973). Ind. J. Med. Res. 61, 1292.

Product	Nature of compound	Biological activity* (where known)	First published report
Kojic acid	γ-Pyrone	GP, GN	Prescott, S.C. and Dunn, C.G. (1940) "Industrial Microbiology" New York, McGraw-Hill. p.405.
Nidulin**	Chlorine-containing depsidone	GP, F	Kurung, J.M. (1945) Science 102, 11.
Benzylpenicillin	β-Lactam	GP	Foster, J.W. and Karow, E.O. (1945) J. Bact. 49, 19.
Nornidulin (Ustin)	Chlorine-containing depsidone		Dean, F.M., Robertson, A., Roberts, J.C. and Raper, K.B. (1953) Nature 172, 344.
Dechloronornidulin	Depsidone		Dean, F.M., Erni, A.D.T. and Robertson, A. (1956) J. Chem. Soc., 3545.
Ferricrocin	Iron (III) trihydroxamate		Zahner, H., Keller-Schierlein, W., Hutter, R., Hess-Leisinger, K. and Deer, A. (1963). Arch. Mikrobiol. 45, 119.
Ferrirhodin	Iron (III) trihydroxamate		(As for ferricrocin)
Cordycepin (3'-deoxyadenosine)	Nucleoside	GP, T	Kackza, E.A., Dulaney, E.L., Gitterman, C.O., Boyd Woodruff, H. and Folkers, K. (1964). Biochem. Biophys. Res. Comm. 14, 452.

*GP = active against Gram-positive bacteria

GN = Active against Gram-negative bacteria

F = antifungal

T = antitumour

M = mycotoxin

V = antiviral

**Producing strain then considered to be A. ustus (see Dean et al, 1953, under nornidulin).

Barry Scott[+] and T. Alderson[*]

Conditions which do not effect the radiosensitivity of Aspergillus nidulans conidia

A number of additional observations on radiation-induced inactivation and mutation of inhibitor-depleted conidial suspensions of the haploid Glasgow meth GlbiA1 strain of Aspergillus nidulans are reported. These are as follows:

1. Cell concentration: The dose required to reduce the viability of the population by 50%, the LD_{50}, was independent of concentration of conidia in the range $3.5\ 10^2$ - 4.10^6 Tween 80 inhibitor-depleted conidia/ml at the time of UV or γ irradiation (Scott, Alderson and Papworth, 1972, Radiation Botany 12:29-30 for method of preparation). Over the same range of conidial concentration there was no change in the UV or γ ray induced reversion frequencies to methione independence for class A, B and C revertants (Lilly, 1965, Mutation Research 2:192-195).

2. Delayed Plating (liquid holding): When plating was delayed after γ irradiation of diethyl ether prepared inhibitor-depleted conidia (Scott and Alderson, 1974, Journal of General Microbiology 85:173-176 for method of preparation) there was no change in the degree of inactivation or the induced mutation frequency of any class of methionine suppressor mutation. However, with UV there was a positive delayed plating effect for both phenomena (Scott, Alderson and Papworth, 1975 Mutation Research - accepted for publication). This may indicated that the types of damage induced in the conidia of Aspergilus are different for the two types of radiation both for inactivation and for mutation.

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A salt sensitive mutant on chromosome VI of Aspergillus nidulans

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The mutant sltA, was a survivor among N-methyl-N-nitro-N-nitrosoguanidine treated conidia of the biotin requiring strain, (survival 12%). It was initially isolated, by velvet replication, as a probable osmotic mutant which grew feebly on medium supplemented with 5% NaCl.

Further tests however showed that unlike the Neurospora osm mutants (L. Livingston Mays, 1969, Genetics: 63, 781-794), it grows normally on media with extra glucose and that only high salt concentrations restrict its growth.

Almost any salt would do for this purpose, at concentrations usually ranging between 0.5-1.0 Molar, added to minimal or complete medium. Sodium, potassium, ammonium, magnesium chloride and sodium nitrate, sulphate, tartrate and phosphates were all effective. Ammonium tartrate was particularly potent, 0.1-0.2 M. in minimal medium being enough to restrict the mutant's growth almost to point inoculum.

The mutant has wild type internal ammonium pools. Tests for ammonium derepression (toxicity of chlorate, bromate, methylamine, aspartic hydroxamate, thiourea), gave wild type results.

It grows normally on nitrate, nitrite, ammonium, alanine, asparagine, arginine, urea, proline, glutamate, glutamine as nitrogen sources and glucose, fructose, sucrose, galactose, lactose, sorbitol, mannitol, mannose, ribose, arabinose, xylose, glycerol, ethanol, inositol as carbon sources. Sodium Acetate can be utilized as carbon source at concentrations up to 50 mM, but at higher concentrations it acts as a growth-restricting salt.

sltA is recessive in the diploid with Master Strain E. Haploidization of this diploid with p-fluorophenylalanine located the mutant on chromosome VI, and further mapping (in conjunction with J.R. Kinghorn) placed it at the end of a newly mapped group of mutants on this chromosome:

$$\text{-----pacC}\overset{15}{\text{----}}\text{uaX}\overset{33}{\text{----}}\text{molA}\overset{28}{\text{-----}}\text{tamA}\overset{18}{\text{-----}}\text{argA}\overset{12}{\text{-----}}\text{sltA-----}$$

In Neurospora, strains carrying the osmotic mutant gene form protoplasts at high hemicellulase and salt concentrations (S. Emerson and M.R. Emerson, 1958, P.N.A.S. 44, 668-671). Similar attempts in Aspergillus using the sltA strain did not produce any protoplasts, however 3% hemicellulase reverses to some extent the salt effect restoring growth and conidiation.

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Pleiotropic effects of imazalil resistant mutants in Aspergillus nidulans.

Imazalil (1-β-(allyloxy)-2,4diCl-phenetylimidazolephosphate) is a recently developed fungicide by Janssen Pharmaceutica, Beerse, Belgium. Mutant strains of Aspergillus nidulans have been isolated which show a low level of resistance to imazalil. The frequency with which imazalil resistant mutants occurred spontaneously was about one per 10 million conidia and after mutagenic treatment (UV-irradiation and NG treatment) about one per 100.000 surviving conidia. The level of resistance of the strains measured on complete medium as the minimal inhibitory concentration (mic) varied from 2·5-12 µg/ml imazalil compared with 1·2 µg/ml for the wild type strain(biA1, acrA1).

For genetic analysis 21 imazalil resistant mutants were selected. The mutant numbers 1-8 were obtained after UV-irradiation, 9-15 spontaneously and 16-21 after NG treatment. In these strains 8 different loci were determined, showing different pleiotropic effects (table 1). Eleven imaA mutations were in allelism tests located to linkage group VII. imaA was mapped 12 units from wetA6 and 42 from benC28 (see note in this issue). Four imaB mutations were located to linkage group V. These mutations, also leading to acriflavin, cycloheximide and neomycin hypersensitivity and chloramphenicol resistance, are found to be allelic with camD (Gunatilleke et al., Molec. gen. Genet. 137, 269-316, 1975), a chloramphenicol resistance with the same pleiotropic effects as imaB. From 202 isolated imazalil resistant strains, 113 were of the type imaA and 52 were of the type imaB. From the remaining 37, 17 lead to cycloheximide and neomycin hypersensitivity (imaD13 and imaF15) and 19 lead to cycloheximide and chloramphenicol resistance (imaC10, imaE14, imaG18, imaH19). In order to investigate the frequency of imazalil resistance in cycloheximide resistant mutants, 120 spontaneous and NG induced cycloheximide resistant strains were isolated, of which 90 also appeared to be imazalil resistant.

A cross between imaG and actA gave 400 colonies, only one of which was cycloheximide sensitive. This suggests that these two mutants may be allelic.

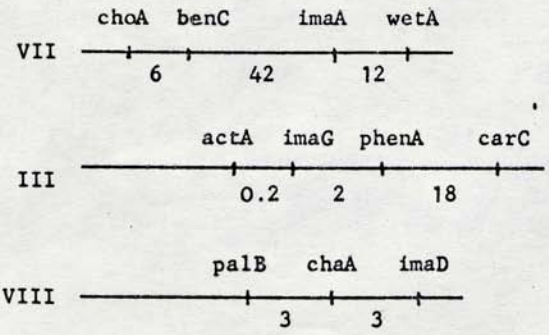
These results show that imazalil resistance in A. nidulans is based on a multigenic system. The different mutants showing a number of pleiotropic effects could give information about the mode of action of imazalil.

Table 1. Properties of the imazalil resistant mutants

Locus involved	allele numbers	linkage group	pleiotropic effects *	
imaA	1,2,3,4,5,6, 7,8,16,20,21	VII	slight neomycin slight acriflavin	} resistance
imaB	9,11,12,17	V	chloramphenicol resistance acriflavin cycloheximide neomycin	} hypersensitivity
imaC	10	II	chloramphenicol cycloheximide	} resistance
imaD	13	VIII	cycloheximide	} hypersensitivity
imaF	15	I	neomycin	
imaE	14	II	chloramphenicol	} resistance
imaG	18	III	cycloheximide	
imaH	19	III	neomycin	

* The cycloheximide hypersensitivity was measured in CM at 0,5 mg/ml, the mic of the wildtype is a factor 4 higher, resistance was tested at 2 mg/ml, the acriflavin sensitivity measured in SM is relativ: an acrA-1 mutant hypersensitive has a mic of a 0,5 mg/ml, the wildtype 1,5 mg/ml. The neomycin hypersensitivity was determined in SM at 1 mg/ml the mic of the wildtype is a factor 2 higher, of the slight resistant types a factor 4; neomycin resistance was tested at 4 mg/ml. The chloramphenicol resistance was determined in SM at 5 mg/ml.

Mapping data:



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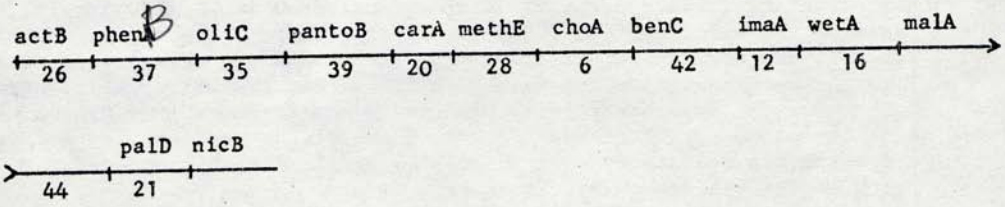
The location of 13 markers on linkage group VII in Aspergillus nidulans.

In studies on the genetic aspects of resistance to systemic fungicides (Van Tuyl et al., 1974, Neth. J. Pl. Path. 80, 165-168; Van Tuyl, 1975, Neth. J. Pl. Path. 81, 122-123; Van Tuyl, 1975, Meded. Fac. Landbouuw. Rijksuniv. Gent. 40: 691-697, five resistant mutants were allocated to linkage group VII. This linkage group is still very poorly mapped and so it seemed worthwhile to find linkages with the new markers. The markers are benC, carA, actB, imaA and oliC resistant mutants to benomyl, carboxin, cycloheximide, imazalil and oligomycin, respectively. All the mutants were obtained after UV-irradiation. From the Glasgow stock were obtained three strains with eight markers on linkage group VII. These markers were brought together with the resistant mutants in two strains. The results of the cross of these strains is given in a map of linkage group VII:



Mitotic analysis confirmed the position of choA and benC. In a diploid are actB, carA and imaA dominant; benC and oliC are semi-dominant, giving resistant segregants on a medium containing benomyl or oligomycin.

(Editor's note: The two strains carrying these markers are available from the Glasgow stocks: nos. G715 & G716.)

Biosynthesis of pyridoxine in *Aspergillus nidulans*

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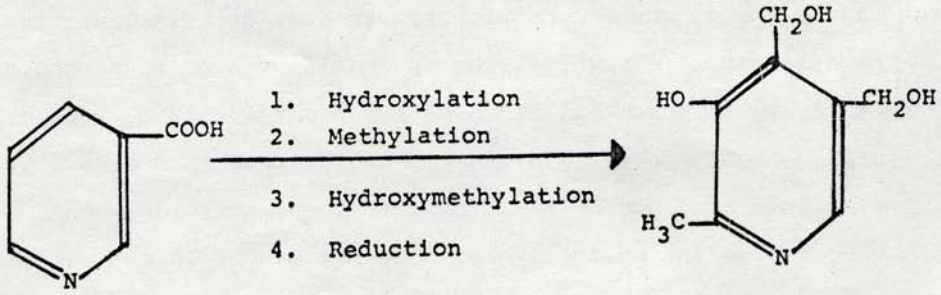
Seven non-allelic, temperature independent pyridoxineless mutants of *Aspergillus nidulans* designated as $\underline{w}_3;\underline{pyro}_5$, $\underline{w}_3;\underline{pyro}_6$, $\underline{w}_3;\underline{pyro}_7$, $\underline{y}_1;\underline{pyro}_8$, $\underline{w}_3;\underline{pyro}_9$, $\underline{w}_3;\underline{pyro}_{10}$ and $\underline{w}_3;\underline{pyro}_{12}$ were obtained by UV irradiation from the parent strain $\underline{y},\underline{ribo}_1$. These mutants were used to investigate the sequence in the biosynthetic pathway of the vitamin.

Plate tests were carried out to test the growth promoting activities of amino acids, sugar phosphates, 3-carbon compounds in glycolytic pathway and a few pyridine compounds to substitute pyridoxine in these mutants. 2- and 3-phosphoglycerates, 3-phosphoserine and pyruvate promoted the growth of $\underline{w}_3;\underline{pyro}_7$. The strain $\underline{w}_3;\underline{pyro}_9$ could grow in β-glycerophosphate, dihydroxy acetone phosphate, 2- and 3-phosphoglycerates. 3-phosphoserine promoted the growth of $\underline{w}_3;\underline{pyro}_{10}$. It is interesting to note that $\underline{y}_1;\underline{pyro}_8$ could grow with kynurenine, formyl kynurenine and nicotinic acid. However, the optimal levels of these compounds necessary for growth (700 µg of 2-phosphyglycerate, 750 µg of 3-phosphoglycerate, pyruvate and β-glycerophosphate, 800 µg of nicotinic acid per 10 ml of MM) are thousand folds when compared to the pyridoxine supplement (0.8 µg/10 ml) necessary for growth.

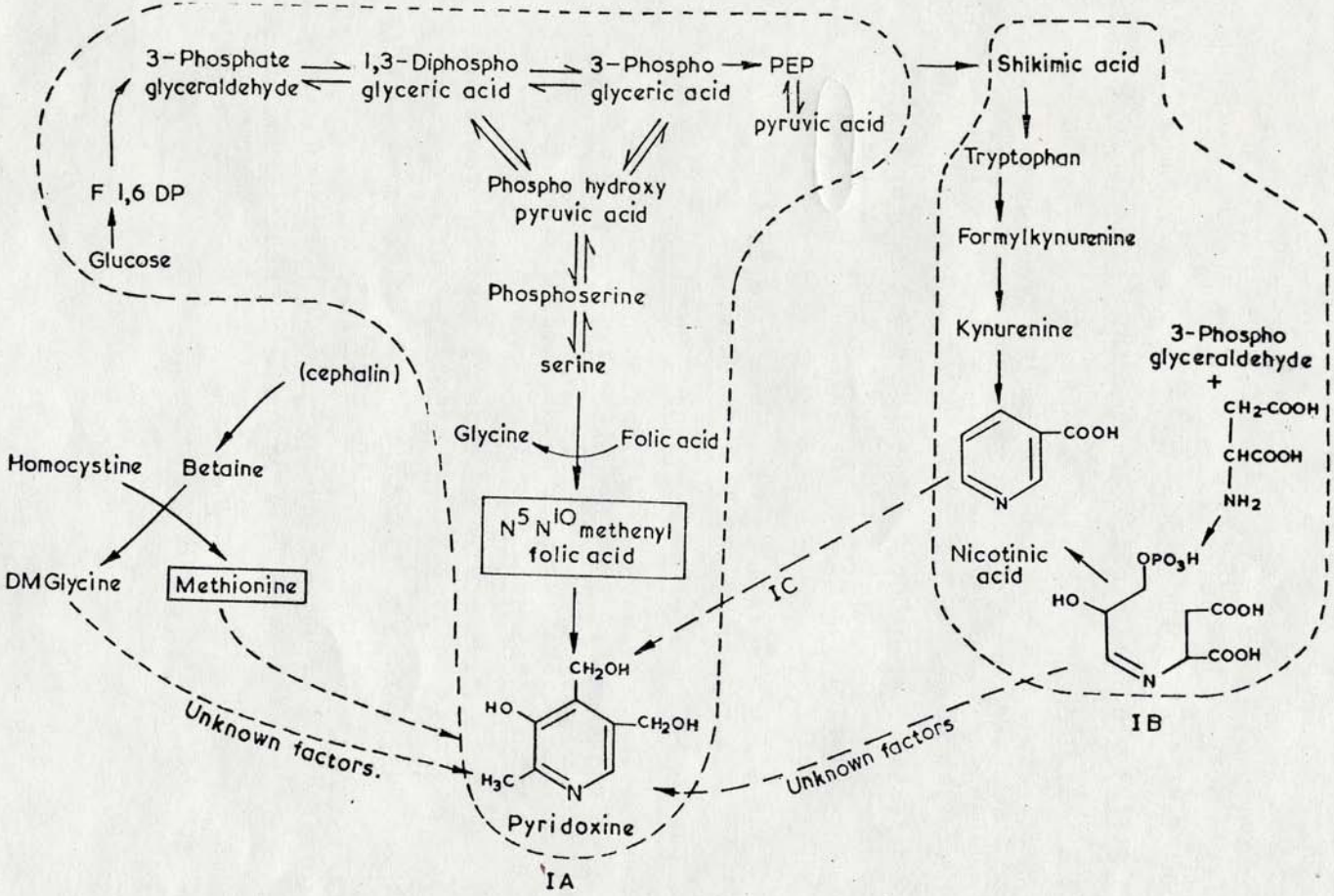
In vivo studies carried out to find out the amount of pyridoxine synthesized with the above mentioned supplements as compared with pyridoxine supplement (as control) showed that pyridoxine was synthesized at eighty per cent level.

In vitro studies by the cell free extracts of the pyridoxineless mutants for the formation of pyridoxine from nicotinic acid with three carbon compounds as carbon donors were carried out, with many inhibitors and activators. The strains $\underline{w}_3;\underline{pyro}_7$, $\underline{Y}_1;\underline{pyro}_8$, $\underline{w}_3;\underline{pyro}_9$ and $\underline{w}_3;\underline{pyro}_{10}$ could synthesize pyridoxine from nicotinic acid or from one of the three carbon compounds tested. The synthesis of pyridoxine was inhibited by aminopterin, barbiturate, cyanide, lactoflavin, sorboflavin, mercaptoethanol, αα'-dipyridyl and pyridine-3-sulfonic acid. The formation of pyridoxine was activated by folic acid, ATP, FAD and methionine. From all these observations a probable sequence of pyridoxine biosynthesis from nicotinic acid which involves methylation, hydroxylation, hydroxymethylation and reduction probably mediated by folate coenzymes, flavoproteins and pyridine nucleotides has been suggested.



Nicotinic acid

Pyridoxine



PROBABLE BIOSYNTHETIC PATHWAY OF PYRIDOXINE IN ASPERGILLUS NIDULANS FROM OUR EXPERIMENTAL OBSERVATIONS.