Phenotypes of Double Conidiation Mutants of *Aspergillus nidulans*

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A series of strains, doubly mutant at conidiation loci, have been made. The phenotypes of these strains reflected the epistasy of earlier blocking mutants over later ones and confirmed the order of gene sequence predicted from the phenotypes of single mutants. Oligosporogenous mutants gave complex interactions, especially between *brl* and *med* mutants.

These results indicated that (i) gene action overlapped in time, (ii) several parts of the conidial apparatus were interchangeable and (iii) nuclei leaving the vesicle were not irreversibly programmed. Structures produced by mutants were reminiscent of the conidial apparatus of other *Aspergillus* species and of related genera.

INTRODUCTION

Several characterized conidiation mutants isolated by A. J. Clutterbuck and others (Clutterbuck, 1969, 1976, 1977) were used to construct double mutant strains. Although the phenotype of the double mutants could be mainly predicted from the phenotypes of the single mutants, interesting epistatic interactions were expected which could add to knowledge of the gene functions and more precisely define the order of gene action. In metabolic pathways, mutations in genes acting early are epistatic to those in genes acting later in the pathway, hence double mutants may on superficial examination resemble the earlier blocked mutant. The same effect was expected in pathways of morphological development and has already been shown for spore formation in *Bacillus subtilis* (Coote & Mandelstam, 1973). Double mutants have also been used, for example, in the elucidation of T4 phage maturation and assembly (Levine, 1969).

METHODS

General methods were those of Pontecorvo *et al.* (1953).

Organisms. Conidiation mutants were kindly supplied by A. J. Clutterbuck, Glasgow, and are described in Table 1.

Media. Minimal and complete solid media and their supplements were those described by Pontecorvo *et al.* (1953) as modified by Cove (1966).

Characterization of double mutants. In crosses where one mutation was completely epistatic over another, a progeny ratio M1:M2:wild-type of 2:1:1 was obtained (where M1 is the morphology of the epistatic mutant and M2 is the morphology of the hypostatic mutant). Since the double mutants were necessarily indistinguishable from one of the single mutants, a selection of six M1-type progeny was out-crossed to the wild-type to identify double mutant progeny. In other crosses, double mutant progeny were clearly different from either parent giving an overall ratio M1:M2:M1M2:wild-type of 1:1:1:1. In these, out-crossing of progeny was performed as a check.

The double mutants were characterized for colony morphology and pigmentation after growth at 37 °C (2 d) and in some cases also at 25 °C (5 d). Conidial head morphology was examined and photographed after
Table 1. Properties of conidiation mutants

Only type mutations cited in the text are described.

<table>
<thead>
<tr>
<th>Mutation</th>
<th>Morphological phenotype</th>
<th>Conidiation* at 37 °C</th>
<th>Colour of conidiophore†</th>
</tr>
</thead>
<tbody>
<tr>
<td>stuAI</td>
<td>(stunted). Short conidiophores. Poor subsequent development</td>
<td>Oli</td>
<td>Brown</td>
</tr>
<tr>
<td>brlAI</td>
<td>(bristle). Conidiophore growth continues without developing into a vesicle, etc.</td>
<td>Asp</td>
<td>White</td>
</tr>
<tr>
<td>apsA6</td>
<td>Anucleate primary sterigmata or metulae which develop no further. A few receive nuclei and are normal</td>
<td>Oli</td>
<td>Brown</td>
</tr>
<tr>
<td>apsB8</td>
<td>(ivory). Unpigmented conidiophores, metulae, phialides</td>
<td>Normal</td>
<td>White</td>
</tr>
<tr>
<td>ivoA51</td>
<td>(abacus). Proliferation of metulae before phialide formation</td>
<td>Oli</td>
<td>White</td>
</tr>
<tr>
<td>ivoB63</td>
<td>Yellow conidia, lacks p-diphenol oxidase</td>
<td>Oli</td>
<td>Brown</td>
</tr>
</tbody>
</table>

* Oli, Oligosporogenous, i.e. fewer conidia than wild-type; Asp, asporogenous, i.e. no conidia.
† This pigmentation may be masked in conidiating strains by coloured conidia.
‡ Illustrated in Fig. 2(j).

staining with lacto-phenol cotton blue, or in water-mounts. The underlying mycelia were grown in a thin layer on coverslips.

Illustrations of conidiation mutants not given here are described in detail by Clutterbuck (1969, 1976, 1977). Diagrams of conidiation of the wild-type and further details of some mutants are given by Oliver (1972). Structures previously referred to by Clutterbuck (1969) as primary and secondary sterigmata are here referred to as metulae and phialides, respectively.

A summary of the properties of conidiation mutants is given in Table 1 and they are illustrated in Fig. 1.

RESULTS

Phenotypes of double mutants containing the asporogenous mutation brlAI

The brlAI (bristle) mutation is completely unleaky. Double mutant strains constructed with this mutation and mutations presumed to block later stages in the developmental pathway, i.e. apsA6, apsB8, medAI5, abaAI, wetA6, were like brlAI strains in every respect. brlAI was thus epistatic to all these mutations and the brl gene presumably acted before them.

Phenotypes of double mutants containing the oligosporogenous mutations stuAI, brlA42, apsA6, apsB8, medAI5 and wetA6 and asporogenous abaAI

Each of the oligosporogenous mutants allows some later development culminating in most cases in the production of some viable conidia. In most of these crosses, the double mutant progeny were clearly recognized.

stuAI is the type mutation in the stu gene (stunted) and apparently produces conidia directly on the vesicles. After repeated attempts only two other mutations were successfully introduced into stuAI strains; these were medAI5, the medusa type-mutation, and brlA42, a temperature-sensitive mutation in the brl gene (Fig. 1, Table 2). The morphology of the progeny from these two crosses showed the ratio 2 stunted: 1 medusa or bristle: 1 wild-type. Microscopically, it was possible to identify stuAI, brlA42 double mutants by their stunted bristles but stuAI, medAI5 strains were only identified by out-crossing and thus resembled stuAI strains. stuAI modified the growth of bristles produced by the brlA42 mutation by altering the normally forked bristle of brlA42 to a simple bristle, like that produced by
Conidiation mutants

Fig. 1. Diagrams of single conidiation mutants of *Aspergillus nidulans*. All are drawn to the same scale, except *brlA1* which should be seven times the normal conidiophore length and old *medA26* which is reduced.
brlAI. stuAI was epistatic to medAI15 and, by inference, to all mutations in genes acting later in the pathway, but stuAI, medAI15 strains were unpigmented like single medusa mutants.

apsA6 and apsB8 (anuclear primary sterigmata or metulae), the type mutations at the two aps loci, are affected in nuclear division and/or migration. Only some of the metulae on the conidial vesicle receive nuclei and can develop further to form phialides and conidia (Clutterbuck, 1977). In crosses with abaAI (abacus) or wetA6 (wet-white) (Fig. 2j), the double mutant progeny colonies were not clearly distinguishable from those of apsA or apsB strains until examined microscopically. The progeny ratios were 1 aps:1 double mutant:1 abacus or wet:1 wild-type. In crosses with medAI15, the double mutants were identified by their pale colour and aps appearance, contrasted with the brown aps colonies and white medusa colonies; hence an overall ratio of 1:1:1:1 was obtained. Microscopically, conidial heads from wetA6, apsA/apsB colonies looked mostly like aps except where enough conidia were produced to see a slimy aggregation of conidia from several adjacent chains. However, in medAI15, apsA/apsB and abaAI, apsA/apsB strains, both phenotypes were clearly visible, the poor aps-like conidial heads bearing a few chains of metulae as in medusa strains or bearing abacus-like chains. Hence the oligosporogenous mutations apsB8 and apsA6 allowed partial expression of genes whose activity was expressed later in the pathway, i.e. abaAI, medAI5 and wetA6.

apsA6 and apsB8 mutants were crossed giving a progeny ratio of 3 aps:1 wild-type. Closer examination revealed a ratio of 2 apsA:1 apsB:1 wild-type morphology. The double mutant resembled the rather more extreme apsA6 mutant (Fig. 1).

In crosses between medAI15 and abaAI or wetA6, superficial phenotypic ratios of 2 medusa:1 abacus or wet:1 wild-type were obtained, reflecting the partial epistasy of medAI15 over the other mutations. Double mutants, however, developed no conidial colour since either abaAI or wetA6 alone prevented the expression of conidial pigment and medAI5 prevented the expression of brown-grey conidiophore pigment. Conidial heads of medAI15, abaAI strains were interesting since they bore long chains of metulae followed by intermediate cells then abacus chains, clearly showing the expression of both mutations (Fig. 2d). The med gene clearly expressed itself before the aba gene. medAI15, wetA6 double mutants also showed medusa conidial heads, but with normal chains of conidia replaced by an autolysing sticky mass of white conidia. The wetA6 mutation was presumably expressed later than medAI15.

Double mutants synthesized from the asporogenous mutation abaAI and wetA6 were indistinguishable from abaAI strains hence abaAI was epistatic over wetA6, and the aba gene acted prior to the wet gene.

**Interactions between several brl and med alleles**

Even in maturity, all known medusa mutants have pale or unpigmented conidiophores, vesicles, metulae and phialides. Since double mutants of medAI15 with either abaAI or aps or stuAI mutations also lacked this pigment, it was interesting to look at the interaction between four med alleles and four brl alleles including leaky brl alleles which normally make pigment. In addition, the morphology of leaky brl and med mutants is so similar that medusa was originally thought to represent another type of bristle mutation (A. J. Clutterbuck, personal communication).

Each of four brl alleles, brlA1, 9, 7, 42, was crossed with each of four med alleles, medAI15, 26, 27, 30. The bristle mutants represent a series of modifications ranging from unleaky brlA1 to very leaky brlA42 (Clutterbuck, 1969). The four medusa mutants are all similar. In some cases, double mutants were identified by their unique morphology and in all cases by out-crossing the putative double mutant to the wild-type. These double mutants have been classified for colony morphology, pigment and conidial head structure. The results are summarized in Table 2.
Table 2. Phenotypes of *brl* and *med* mutants and of double mutant strains

The presence or absence of structures was scored, not the quantity (but see †).

<table>
<thead>
<tr>
<th>Strain</th>
<th>Growth temperature (°C)</th>
<th>Colour of conidiophores etc.</th>
<th>Secondary conidiophores with vesicles</th>
<th>Secondary bristles (with septa, s)</th>
<th>Long metulae or short bristles*</th>
<th>Normal-sized metulae†</th>
<th>Conidia</th>
<th>Summary of phenotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wild-type</td>
<td>37</td>
<td>Brown</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>Wild-type</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>Brown</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>Wild-type</td>
</tr>
<tr>
<td>Any medA allele</td>
<td>37</td>
<td>White</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Medusa</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>White</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>More extreme medusa expression</td>
</tr>
<tr>
<td>brlA1</td>
<td>37</td>
<td>White</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>Unleaky bristle</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>White</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>Unleaky bristle</td>
</tr>
<tr>
<td>brlA9</td>
<td>37</td>
<td>Brown</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>Leaky bristle</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>Brown</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>Leaky bristle</td>
</tr>
<tr>
<td>brlA7</td>
<td>37</td>
<td>Brown</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>Leaky bristle</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>Brown</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>Leaky bristle</td>
</tr>
<tr>
<td>brlA1, with medA alleles</td>
<td>37</td>
<td>White</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>Unleaky bristle</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>White</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>Unleaky bristle</td>
</tr>
<tr>
<td>brlA9, with medA alleles</td>
<td>25</td>
<td>Pale brown</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>Unleaky bristle‡</td>
</tr>
<tr>
<td>brlA7, medA15 or medA30</td>
<td>37</td>
<td>Almost white</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>Bristle and medusa, i.e. weak medA15, equal medA26, medA27, strong medA30</td>
</tr>
<tr>
<td>brlA7, medA26 or medA27</td>
<td>37</td>
<td>Almost white</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>Leaky bristle with weak medusa expression</td>
</tr>
<tr>
<td>brlA7, medA26 or medA30 or medA27§</td>
<td>25</td>
<td>Almost white</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>Leaky bristle with strong medusa expression</td>
</tr>
<tr>
<td>brlA42 with medA alleles</td>
<td>37</td>
<td>White</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>Bristle and medusa</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>White</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>−</td>
<td>Weak bristle and very strong medusa</td>
</tr>
</tbody>
</table>

Fig. 2(c)  Fig. 2(g)  Fig. 2(h)  Fig. 2(a, b, e, f, k, i)

* It is difficult to distinguish between the two possibilities unless the initial cell gives rise to another one either by budding (metula) or by septation (bristle).

† Wild-type has one row of metulae (designated +); some leaky bristle strains produce more than one row of normal-sized metulae without becoming medusoid (+ +); medusa expression is recognized by many branching chains of metulae ( + + +).

‡ *brlA9* is structurally identical with *brlA1* at 37 °C, therefore unleaky, but it produces pigmentation characteristic of most leaky bristle mutants.

§ *brlA7, medA15* was not tested at 25 °C.
Double mutants containing brlA1 and any of the four medusa alleles were all identical with brlA1 strains in producing stiff, white, elongated bristles instead of conidiophores, etc. brlA1 was completely epistatic to all the med alleles tested.

The situation was much more complicated in the case of leaky brl mutations. All the latter when present as single mutants produced a brown pigment in their conidial apparatus thus colouring the whole colony brown. When combined with any med allele, the colonies and conidial heads had little or no pigmentation. This reduction in pigment was always visible after growth at 25 °C, but in some cases the colonies were intermediate in colour at 37 °C. Since lack of pigment is a characteristic of med mutations, med alleles were epistatic over leaky brl alleles in this respect.

The conidial apparatus seen on colonies of leaky bristle type was very variable, hence it is not possible to illustrate typical brlA9, 7, 42 structures (but see Table 2). Each brl strain and each double mutant was examined after growth at 37 and 25 °C. The strains were scored for the presence or absence of normal conidiophores, vesicles, metulae, phialides and conidia and for various abnormal structures such as bristles (Fig. 1), secondary conidiophores (Fig. 2e), secondary bristles (Fig. 2g), medusa-like proliferation of short cells (Figs 2f, i, k) and various types of longer cell (Fig. 2h) or short bristle. In several cases, more than one structure arose from one vesicle. Seldom were the cells growing from the vesicle recognizable as metulae and phialides.

All single and double mutant strains carrying the brlA9 mutation were identical in having long brown bristles with no further development at 37 °C, but double mutants were paler brown. The expression of med alleles was visible in double mutants with brlA7 or brlA42 grown at 37 °C since either a medusa-like chain of cells or secondary bristles divided by cross-walls at frequent intervals were visible (Fig. 2e). The overall morphology strongly resembled that of the respective brl allele, but colonies were pale or unpigmented reflecting med action.

The expression of med mutations was more obvious at 25 than 37 °C and increased with the leakiness of the brl alleles. The brlA9 mutation was leakier at 25 °C than at 37 °C and produced vesicles, secondary conidiophores, secondary bristles and several rows of normal-looking metulae (Fig. 2i). Double brlA9, med mutants possessed these characters at 25 °C and, in addition, either medusa-like chains of cells or divided bristles, characteristic of med expression (Fig. 2k).

The brlA7 mutation was leakier than brlA9 at both temperatures since brlA7 strains bore vesicles with secondary conidiophores (Fig. 2e) and secondary bristles divided by cross walls. Cells resembling short bristles or long metulae were also produced (Fig. 2h). At 25 °C, some normal metulae and phialides were formed (Fig. 2i). med mutations barely influenced the development of brlA7 strains at 37 °C except for the possession of a few medusa-like chains of cells in the double mutants. At 25 °C, med mutations exercised greater influence by producing obvious medusa-like chains of cells from vesicles, in addition to some structures characteristic of brlA.

The brlA42 mutation was very leaky at 37 °C and gave rise to wild-type morphology at 25 °C. While having typical leaky bristle structures, it also displayed slight medusa character at 37 °C. The addition of med mutations to these strains was very dramatically expressed after growth at 25 °C when conidial heads were almost completely medusoid (Fig. 2f). At 37 °C, conidial heads expressed both mutations.

Since the appearance of colonies depended on the type of conidial apparatus present, colony morphology closely reflected the pattern of mutant expression discussed above. At 37 °C, brlA9, med strains were indistinguishable from brlA9 strains, but at 25 °C appeared to have slightly modified leaky bristle morphology. brlA7, med strains most closely resembled brlA7 strains at 37 °C but were intermediate between brlA7 and med strains at 25 °C. brlA42, med strains were obviously intermediate in morphology at 37 °C and rather medusa-like at 25 °C.
Conidiation mutants

All the med alleles used gave rise to similar morphological aberrations. At 37 °C, they could be classified quite early for their medusa character of producing several rows of metulae before normal phialide formation (Fig. 2c). At 25 °C, they all resembled a form intermediate between medusa at 37 °C and very leaky bristle (Fig. 2a). Conidiation was extremely poor at 25 °C compared with 37 °C. Some strains produced secondary conidiophores with medusoid metulae at 25 °C (Fig. 2b). There were no consistent differences in the effects of these alleles on the expression of brl mutations.

In summary, double mutants most closely resembled their brl mutation in expression at 37 °C, but at 25 °C medusa characters were seen increasingly clearly with progressive leakiness of the brl allele. There was, therefore, no clear-cut epistasy of one group of mutants over the other.

DISCUSSION

The epistasy of the asporogenous mutant brlA1 over apsA6, apsB8, abaA1, wetA6 and four med alleles and that of asporogenous abaA1 over wetA6 is consistent with the predicted order of gene expression in this developmental pathway, and confirms the usefulness of this approach. Within the crosses performed in this study, complete epistasy of brl, aba and wet mutations over conidial colour mutations cha, fwn, w, y and yg was also observed. The order of expression is brl, aps and med, aba, wet.

The time of expression of genes which give oligosporogenous mutations is more difficult to interpret. stuA1 strains have shortened conidiophores, but their form is determined by the brl allele. The stuA gene must therefore act over the same period as the brlA gene; however, brlA42 is not phenotypically leaky in a stuA1 background but produces simple bristles like brlA1. This could imply an overlap in gene function. stuA1 is epistatic over medA15 and by inference over apsA, apsB, abaA and wetA, but not over colour mutations. Mutations in genes apsA and apsB also give rise to an oligosporogenous phenotype. Since med, aba and wet mutations appear to alter events occurring later than the formation of the initial metulae, their expression in cells produced from the few nucleated metulae of aps strains is only to be expected. The intermediate phenotypes illustrate the action of aps before that of med, aba or wet.

The most complex series of phenotypes was observed in strains constructed from one of four medA alleles which are oligosporogenous and one of three leaky brlA alleles. Generally, the double mutants most strongly resembled the bristle parent at 37 °C and had medusoid morphology at 25 °C. The exceptions to this were brlA9, medA strains in which brlA9 was epistatic over medA alleles for structural phenotype. These results are consistent with the leaky bristle mutants being more extreme at 37 °C and the medusa mutants at 25 °C. The complex phenotypes are found because neither mutant completely blocks development and growth, and because the action of the two genes must overlap in time since both are concerned with metula and phialide production. The overall effect of medA alleles on brlA alleles is that of increasing septation either in secondary bristles or by encouraging metula proliferation. The overall shape of the brlA conidial apparatus is, however, maintained. Clearly, brlA action begins before that of medA and provides a basic structure for med alleles to modify. The possession of multiseriate metulae by leaky bristle strains and that of secondary conidiophores by medusa strains indicates a large degree of overlap in gene function, as well as time of action. Indeed, Clutterbuck (1969) originally expected medusa mutations to map at the bristle locus. The more variable results obtained with combinations of brlA7 and medA is probably a reflection of the delicate balance in gene expression between two oligosporogenous mutants. Repeated observation ruled out the possibility of experimental error in this matter.

Interactions of conidiation mutants also produced an interesting effect on pigmentation. The introduction of any medA allele into a strain carrying any apsA, apsB, abaA, stuA or leaky brlA mutation inhibited pigmentation of these mutants. All normally form a grey-
Conidiation mutants

Fig. 2. Characteristic structures produced by some single and double conidiation mutants of *A. nidulans*. (a) medA26 at 25 °C showing medusoid proliferation of metulae. (b) medA26 at 25 °C showing secondary conidiophores. (c) medA30 at 37 °C. (d) abaA1, medA15 at 37 °C showing medusa and abacus features. (e) brlA7, medA27 at 37 °C showing secondary conidiophores and medusoid character. (f) brlA42, medA27 at 25 °C showing strong medusoid expression. (g) brlA9, medA15 at 25 °C showing metulae–bristle transitions. (h) brlA7, medA30 at 37 °C showing long metulae or short bristles. (i) brlA7 at 25 °C showing bristle proliferation of metulae. (j) wetA6 at 37 °C showing autolyzing conidia. (k) brlA9, medA30 at 25 °C showing medusoid expression.

Abbreviations: m, metulae; cp, conidiophore; c, conidia; p, phialide; a, abacus-like cell; b, bristle. Bar markers represent 50 μm.
brown pigment in the conidial apparatus. This synthesis depends on the presence of wild-type alleles in the ivoA and ivoB genes (Clutterbuck, 1977), the former being concerned with phenol production and the latter with phenol oxidase synthesis. The enzyme is called cresolase from its action on cresols in vitro. Those mutants tested, e.g. leaky brlA and abaA, have normal cresolase levels (A. J. Clutterbuck, personal communication). The lack of pigment in medusa strains has not been explained but it is not due to repression of the ivoB gene, hence an absence of cresolase. Mycelial extracts of medA15 strains contain at least wild-type amounts of cresolase and possibly excess enzyme (S. Martinelli, unpublished results; confirmed by A. J. Clutterbuck, personal communication). It is curious that this inhibition occurs in mutants which are presumably blocked at an earlier structural stage than medusa, i.e. aps, brl, stu as well as mutants blocked later, i.e. aba, wet. This may extend backwards in the time period over which the medusa gene acts.

The simplified diagram of this developmental pathway published previously (Martinelli, 1974) is no longer tenable in view of the overlapping gene action seen in this work. A diagrammatic representation of conidiation in the wild-type is presented in Fig. 3, with an indication of the order in which gene action commences and finishes.

The phenomenon of ‘mixed conidial heads’ in which one vesicle gives rise to several different structures, i.e. conidiophores, bristles, metulae, is an interesting one and shown not only by brlA and medA mutants but also by aps (A. J. Clutterbuck, personal communication). In these cases, incorrectly programmed nuclei are leaving the vesicle and instead of developing further they either revert to a previous stage (conidiophore) or produce a more extreme phenotype (bristle) or form metulae. It is tempting to consider a quantitative
Conidiation mutants

explanation of gene action. The nuclei in the conidial apparatus probably never become
committed to the whole conidiation process, but sequentially to small developmental steps.
Under normal circumstances this programming is synchronous and cancels previous
programme instructions.

Some results point to the conclusion that various parts of the conidial apparatus are
dispensable and interconvertible, at least in mutant strains. stuA1 strains can apparently
produce conidia directly from a vesicle. Either metulae and phialides are dispensable or the
conidiophore is physiologically a phialide. A similar phenomenon occurs when wild-type
strains are grown in submerged liquid culture (Martinelli, 1976). The conidial apparatus is
reduced to a few phialides produced apically on a conidiophore, or, in conditions of carbon
or nitrogen starvation, to single apical or lateral phialides. In leaky brlA, apsA, apsB and
medA mutants, cells which resemble metulae can be converted to bristles or conidiophores
(Figs 2g, h) and bristles can revert to metulae.

Both interconvertibility and dispensability could be expected from a review of conidial
apparatus in the genus Aspergillus, as described by Raper & Fennell (1965). Within the
genus both uniseriate (phialides only) and biseriate (metulae and phialides) species occur,
indicating that metulae are dispensable, e.g. A. japonicus and A. carbonarius, respectively.
Vesicles must also be dispensable since A. asperescens can produce two rows of sterigmata
from a conidiophore lacking a vesicle, as can A. biplanus from mycelium at the agar surface.
In A. cremeus, a complex situation exists since the conidial head changes from being uni-
seriate to biseriate after conidial production has started, bearing septate sterigmata at the
intermediate stage. Phialides are thus converted to metulae. Other features of mutants
anticipated within the genus are the long, club-shaped, occasionally septate sterigmata at
the intermediate stage. Phialides are thus converted to metulae. Other features of mutants
anticipated within the genus are the long, club-shaped, occasionally septate metulae of the
A. niger and A. candidus groups, similar to the situation in bristle and medusa mutants.
Other genera such as Penicillium have similar forms.

It was hoped initially that a study of double mutant phenotypes would add to the
knowledge of gene function in this pathway. Unfortunately, these results only confirm
Clutterbuck's interpretation of the genetic lesions (1969, 1976, 1977), although they perhaps
emphasize some of the peculiarities of the conidiation apparatus, and the flexibility of the
process.

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