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Mutations of the *A* mating type factor in *Coprinus lagopus*

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Recent studies of incompatibility in tetrapolar Hymenomycetes have revealed two features of particular interest. The first is that one or both of the mating-type factors of four species appear to be made up of two or more sub-units. The *A* and *B* factors of *Schizophyllum commune* (Raper *et al.*, 1958, 1960) and *Collybia velutipes* (Takemaru, 1961) and the *A* factor of *Coprinus lagopus* (Day, 1960*a*) are each made up of at least two linked sub-units which recombine by crossing-over. Recombination of sub-units has also been recorded for one of the mating-type factors of *Pleurotus ostreatus* (Terakawa, 1960). The second feature is that the designation of the factors is no longer arbitrary. One factor, generally called *A*, is concerned primarily with clamp connection formation; the other factor, generally called *B*, controls nuclear migration. The roles of the *A* and *B* factors were discovered by studies of the formation and properties of heterokaryons obtained by pairing different haploid monokaryotic strains of some eleven different species having either common or different *A* or *B* factors (Fulton, 1950; Swiezynski & Day, 1960; Takemaru, 1961).

During the last forty years several reports of mutation of mating-type factors have appeared. While some of these can, as Whitehouse (1949) pointed out, be the result of contamination, others, for example Kniep's (1923) report of mutation in *Schizophyllum commune* and probably also that of Zattler (1924) in *Collybia velutipes*, can most easily be explained by crossing-over between the sub-units of the parental factors to give rise to a proportion of recombinants among the progeny. There remain however some accounts of mutation, in particular those by Quintanilha (1935), Swiezynski & Day (1960) and Day (1960*b*) in *Coprinus* and Parag (1962) in *Schizophyllum* which cannot be satisfactorily explained in either way. This paper reports studies with one of the *A* factor mutants of *Coprinus* recovered by Swiezynski & Day and ten others isolated subsequently by the author. All the mutants were spontaneous in origin.

1. MATERIALS AND METHODS

The stocks of *Coprinus lagopus* used in this work were described by Day & Anderson (1961). Culture media and methods were the same as those described in Day (1959, 1960*a*) except that basidiospores were germinated on media containing 0·01% furfural and the incubation temperature was raised to 37°C.; at this temperature matings were scored for clamp connections after approximately 40 hours' incubation. Fruiting cultures were grown at 28° C. under illumination.

The *A* factor mutants were isolated by using the selection method of Swiezynski & Day (1960). Mycelia of paired monokaryons with a common *A* factor (common *A* heterokaryons) were inoculated to flasks of sterile dung which were incubated for 50 to 90 days. During this time a proportion produced fruit bodies bearing viable basidiospores, generally the result of an *A* factor mutation giving rise to a fertile dikaryon. At first wild-type stocks were used to synthesize the heterokaryons but these were subsequently replaced by stocks carrying one of the *A* linked markers *paba-1* (2519 or M2) or *ad-8* (2242), with requirements for *p*-amino benzoic acid and adenine respectively. Use of the marked stocks afforded several technical advantages. Firstly, the *A* factor mutants were already marked for further genetical analysis. Secondly, it was possible to tell, from the marker carried by the mutant, which of the two identical *A* factors had mutated and finally the presence of the markers provided a means of detecting contamination.

2. RESULTS

(i) *The mutants*

The composition of the heterokaryons employed and the mutants which were isolated from them are shown in Table 1. The mutants are denoted by the subscript of the parental factor from which they were derived to which has been added the letter *m*. The numbers which follow *m* denote mutants of independent origin.

Tetrads or random spores were taken from one fruit body in each flask. In each progeny, except one (*su-A*) which is described later, there were approximately equal numbers of the parental *A* factor, A_5 or A_6, and another mutant factor compatible with it. The two *B* factors originally present in the heterokaryon segregated in each example proving that the fruit bodies were heterokaryotic in origin. Two *A* mutants each with a different *B* factor were isolated from each progeny. All the mutants possessed hyphal septa with false clamp connections. Like the clamp connections produced by a common *B* heterokaryon the clamp outgrowth failed to fuse with the neighbouring proximal hyphal cell (Quintanilha, 1935). Some of the septa were without false clamps, the proportion which bore false clamps varied among the mutants. All the mutants produced oidia which are never formed by normal dikaryons of *Coprinus*.

The most extensive tests were made with the mutant originally recovered by Swiezynski & Day (1960) A_{5m1}. This mutant when first isolated produced normal fruit bodies with four-spored basidia when inoculated to sterile dung. Spores from fruit bodies formed by $A_{5m1}B_5$ and $A_{5m1}B_6$ gave rise to mycelia only with parental mating types. Later attempts to fruit A_{5m1} stocks, which had then been in culture for approximately a year, failed.

When A_{5m1} stocks carrying different *B* factors were paired each stock became dikaryotized producing new growth bearing normal clamp connections and no oidia. Several attempts to fruit dikaryons homozygous for A_{5m1} were unsuccessful although dikaryons from crosses between A_{5m1} and prototrophs carrying A_{5m2}, A_{5m4} and A_{6m2} all formed normal fruit bodies. The difference between possession

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Table 1. *Mutants isolated from common* A *heterokaryons*

Heterokaryon	No. of flasks	No. of flasks fruited	Days kept	Mutants
$\frac{A_5}{A_5}\ \frac{B_5}{B_6}$	13*	1*	50–90*	A_{5m1}*
	10	1	50	*su-A*
$\frac{+ \quad A_5 \quad ad\text{-}8}{paba\text{-}1 \quad A_5 \quad +}\ \frac{B_6}{B_5}$	10	5	65	A_{5m2} *paba-1* A_{5m3} *ad-8* A_{5m4} *paba-1* A_{5m5} *ad-8* A_{5m6} *paba-1*
$\frac{A_6}{A_6}\ \frac{B_5}{B_6}$	10	0	50	—
$\frac{+ \quad A_6 \quad ad\text{-}8}{paba\text{-}1 \quad A_6 \quad +}\ \frac{B_7}{B_5}$	6	1	50	A_{6m1} *ad-8*
$\frac{+ \quad A_6 \quad ad\text{-}8}{paba\text{-}1 \quad A_6 \quad +}\ \frac{B_6}{B_5}$	10	2	65	A_{6m3} *ad-8* A_{6m4} *ad-8*
$\frac{paba\text{-}1 \quad (2519) \quad A_6}{paba\text{-}1 \quad (M2) \quad A_6}\ \frac{B_5}{B_6}$	14	2	65	A_{6m2} *paba-1*†
$\frac{paba\text{-}1 \quad (2519) \quad A_6}{paba\text{-}1 \quad (2519) \quad A_6}\ \frac{B_5}{B_6}$	29	0	65	—

* Data from Swiezynski & Day (1960).
† 2519 or M2.

of false clamps, which characterize the A mutants and the common B heterokaryon, and the true clamps found on a normal dikaryon appears to be controlled by the B factor. True clamps are formed only when the mycelium carries two different B factors.

The mutant $A_{5m1}B_5$ was crossed with A_5B_6 and both of the resulting dikaryons possessed normal clamps and produced normal fruit bodies on sterile dung. Tetrads from the fruit bodies formed by the dikaryon $A_5B_6 \times A_{5m1}B_5$ each had two A_{5m1} and two A_5 spores. Both dikaryons from the cross between $A_{5m1}B_5$ and A_6B_6 were fertile and their progenies were made up of equal numbers of A_{5m1} and A_6 spores. Further crosses of this kind were made with A_{5m1} in which the A_6 parent carried either *ad-8* or *paba-1* and in mating type tests of over 4000 spores among the progenies of these crosses no A_5 spores were found. Evidently the site of the mutation is either at or very close to the A region.

The phenotypes of the remaining mutants, except *su-A*, were similar to A_{5m1} although the presence of the markers *paba-1* or *ad-8* reduced their ability to accept

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nuclei in compatible matings. Their fruiting ability in monokaryons was not studied. No phenotypic differences were detected between mutants belonging to the A_{5m} and the A_{6m} series. Both types were equally compatible with A_5 and A_6 testers as judged by the vigour of the resulting dikaryons.

(ii) *Genetic constitution of the mutants*

The A mating-type factor of *Coprinus* can be regarded as being made up of two sub-units α and β which are approximately 0·07 map units apart (Day, 1960*a*). When A_5 and A_6 recombine two non-parental factors are formed which are self-incompatible but cross-compatible with each other and with the parental factors. The two sub-units may be ordered in relation to the two markers *paba-1* and *ad-8* respectively 0·54 and 1·27 map units on either side. Thus in the cross

$$paba\text{-}1\ \alpha_5\beta_5\ ad\text{-}8^+ \times paba\text{-}1^+\ \alpha_6\beta_6\ ad\text{-}8$$

a single cross-over between α and β will produce $\alpha_6\beta_5$, a prototroph, and $\alpha_5\beta_6$ the reciprocal double mutant. Approximately 4% of the prototrophs from crosses of this kind arise from exchanges between α and β (Day, 1960*a*).

If a mutant A_5 factor is crossed with A_6 and the progeny screened for $\alpha_5\beta_6$ and $\alpha_6\beta_5$ recombinants their presence or absence should reveal whether the site of the mutation is at, or close to, α or β. For example if the mutant factor were $\alpha_5\beta_m$ recovery of the recombinant $\alpha_5\beta_6$ would show that the mutant possessed a functional α_5 sub-unit. The recombinant factor $\alpha_6\beta_m$ might be expected to be indistinguishable from $\alpha_5\beta_m$ and not to react as $\alpha_6\beta_5$. Thus the recovery of $\alpha_5\beta_6$ and the absence of $\alpha_6\beta_5$ would imply that the site of the mutation was at, or near, β. On the other hand recovery of $\alpha_6\beta_5$ and the absence of $\alpha_5\beta_6$ would imply that the site was at, or near, α.

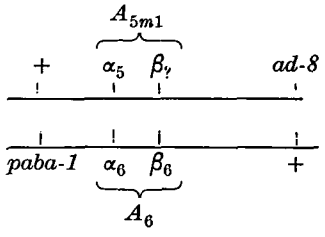
Because α and β recombine at a low-frequency test crosses were made with each of the ten A mutants using the pair of markers *paba-1* and *ad-8* in repulsion so that α–β recombinants could be detected among the prototrophs. Each A_5 mutant, carrying one of the markers, was crossed with an A_6 stock carrying the other marker and each A_6 mutant was similarly crossed with an A_5 stock. Six of the ten mutants were tested in both ways, i.e. for reciprocal recombinants. A recombinant of A_{5m1} with *paba-1* could not be recovered from tests of more than 3000 basidiospores from crosses of *paba-1* A_5 and *paba-1* A_6 with A_{5m1}. However, recombinants of A_{5m3} and A_{6m1} with *paba-1* were obtained at the expected frequencies. Prototrophs from each test cross were selected from basidiospores spread on minimal medium and mated with either $\alpha_5\beta_6$ or $\alpha_6\beta_5$ tester stocks, whichever was appropriate to detect α–β recombinants. A proportion of the prototrophs was also mated with A_5 and A_6 testers to detect regeneration of the original A factor and to measure the frequency of recombination in the two intervals on either side of the A factor. The results of the test crosses are summarized in Table 2.

Six of the mutants possessed non-mutant α sub-units. These were recovered in recombinants which arose with frequencies of from 1·59% (A_{6m2}) to 7·85% (A_{6m4}) of prototrophs. The overall frequency of α–β recombinants for these six mutants

Table 2. *The results of test crosses of ten* A *mutants to detect* α–β *recombinants in their progenies. The percentage recombination between* paba-1 *and* ad-8 *was calculated by doubling the prototroph frequency. Stocks marked* * *are recombinants of the original mutants*

Mutant	Marker carried	No. of prototrophs scored	% Recomb. *paba-1 – ad-8*	Prototroph mating types A_{5m}	A_6	Recombinants		
A_{5m1}	* *ad-8*	223	0·69	146	68	9	α_5	β_6
A_{5m2}	* *paba-1*	392	1·12	—	—	0	α_6	β_5
	ad-8	259	0·93	81	31	0	α_5	β_6
A_{5m3}	* *ad-8*	200	1·38	—	—	4	α_5	β_6
	paba-1	267	1·30	66	81	0	α_6	β_5
A_{5m4}	* *paba-1*	203	1·05	54	149	0	α_6	β_5
	ad-8	232	0·85	169	61	0	α_5	β_5
A_{5m5}	*ad-8*	102	0·89	66	33	3	α_5	β_6
A_{5m6}	* *paba-1*	203	0·91	66	137	0	α_6	β_5
	ad-8	503	1·06	145	41	29	α_5	β_6
		2584				45		
				A_{6m}	A_5			
A_{6m1}	* *ad-8*	490	1·34	337	53	0	α_6	β_5
	paba-1	786	0·42	33	47	0	α_5	β_6
A_{6m2}	* *paba-1*	225	0·76	70	155	0	α_5	β_6
	ad-8	189	0·97	147	42	3	α_6	β_5
A_{6m3}	*ad-8*	203	1·33	174	29	0	α_6	β_5
A_{6m4}	*ad-8*	242	1·52	95	22	19	α_6	β_5
		2135				22		

The cross shown in the first line of the table may be represented as:



was 4·59% of prototrophs. Three of the mutants with parental α sub-units which were also tested for β showed no evidence of the parental β sub-unit. Neither α nor β were recovered from two A_5 mutants and one A_6 mutant which were tested for both. The negative results can of course be explained by the fact that not enough prototrophs were tested. If we assume that the proportion of α–β recombinants

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among the prototrophs was at least 1·59%, the lowest observed, the probabilities of obtaining each of the negative results by chance are all less than 4%. No A_5 spores were recovered among the prototrophs from any of the crosses involving A_5 mutants nor A_6 spores from crosses involving A_6 mutants. This confirms that the mutations either involve one or both sub-units or are very close to them.

(iii) *Tetrad analysis*

The test crosses revealed that some of the mutants had wild-type α_5 or α_6 sub-units which could be recovered in recombinants with normal functions but they provided no information about the reciprocal recombinants which were expected to have a mutant phenotype. While there is no way of distinguishing a recombinant mutant from a parental mutant among the random spores the recombinant could be identified in a tetrad from a test cross. Thus if a prototroph with a non-mutant recombinant A factor were found in a tetrad resulting from a single exchange between *paba-1* and *ad-8* the reciprocal mutant recombinant should be identifiable by its double requirement. The cross involving A_{6m4} *ad-8* was selected for tetrad analysis because it had given the highest frequencies of recombination between *paba-1* and *ad-8* (1·52%) and between α and β (7·85% of prototrophs). This cross may be represented as follows:

$$\frac{+ \quad \alpha_6\beta_{6m} \quad ad\text{-}8 \quad B_6}{paba\text{-}1 \quad \alpha_5\beta_5 \quad + \quad B_5}$$

From 313 tetrads, including seventy-four with only three spores germinated, twenty-seven tetrads each with a single prototroph were found. One of these prototrophs had the recombinant A factor $\alpha_6\beta_5$. This prototroph came from a tetrad in which all four spores had germinated and which had the following composition:

Spore 1:	+	A_m	*ad-8*	B_5
Spore 2:	+	$\alpha_6\beta_5$	+	B_6
Spore 3:	*paba-1*	A_m	*ad-8*	B_5
Spore 4:	*paba-1*	$\alpha_5\beta_5$	+	B_6

Spores 1 and 3 both had mutant A factors and, apart from the *paba-1* marker, were indistinguishable. Since spore 3 carried both *paba-1* and *ad-8* the simplest interpretation is that this spore also carried the recombinant A factor $\alpha_5\beta_{6m}$ and that spore 1 carried the parental factor $\alpha_6\beta_{6m}$. The tetrad confirms the earlier assumption that when recombination between mutant and normal factors gives rise to a functional α–β recombinant the reciprocal product does not differ phenotypically from the parent mutant.

Four of the six mutants with a functional α sub-unit which were tested for β thus appear to have mutant β sub-units. These are A_{5m3}, A_{5m6}, A_{6m2} (Table 2) and A_{6m4} (tetrad data). A_{5m1} and A_{5m5}, which also have functional α sub-units but were not tested for β, probably also have mutant β sub-units. The three remaining mutants, A_{5m2}, A_{5m4} and A_{6m1} from which neither α nor β functional sub-units were recovered, and possibly also A_{6m3}, only tested for α, may either be mutant in both sub-units or altered in such a way as to prevent or restrict crossing-over in the A region. A small deletion or inversion might have this effect.

(iv) *The suppressor mutant—su-A*

The remaining mutant, *su-A*, was recovered from a fruit body which appeared to be produced by a common A_5 heterokaryon (Table 1). A sample of sixteen basidiospores from this fruit body were tested and proved to be all of the same mating type, namely A_5B_5. Evidently the fruit body had originated from a monokaryotic mycelium. The mycelia produced by the spores bore false clamps but were unable to dikaryotize A_5 or B_5 tester stocks or be dikaryotized by them. Four of the mycelia were inoculated to sterile dung and produced fruit bodies with pale instead of the normal black gills. The gills bore varying proportions of abnormal tetrads. The frequencies of one-, two-, three- and four-spored basidia are shown in Table 3 with, for comparison, the frequencies found in an earlier example of tetrad abnormality which was shown to be cytoplasmically controlled (Day, 1959).

Table 3. *Frequencies of different tetrad classes found in homokaryotic fruit bodies formed by four random sister spores of the* su-A *mutant compared with those found in a fruit body formed by a cytoplasmic 'pale gill' mutant dikaryon* (* *data from Day, 1959*)

Spore No.	No. of tetrads scored	% tetrads with 1 spore	2 spores	3 spores	4 spores	5 spores	6 spores	Tetrad density per sq. mm.
1	342	5·0	43·3	10·8	40·9	0	0	617
2	318	10·7	43·7	3·8	41·8	0	0	—
3	626	5·1	43·4	3·7	47·8	0	0	558
4	1150	3·2	10·1	25·4	61·3	0	0	289
Cytoplasmic example*	2000	0	0·3	7·9	87·1	4·5	0·2	0–600

While the densities of tetrads in these fruit bodies are similar to those found in the cytoplasmic example the relative frequencies of the different classes are dissimilar. The fruit bodies from spores 1, 2 and 3 show a bimodal distribution of tetrad frequencies with approximately equal numbers of two- and four-spored basidia and much lower numbers of one- and three-spored basidia. The fruit body from spore 4 shows a distribution more like that of the cytoplasmic example but

compared with it has a greater total of abnormal tetrads which includes one-spored forms but none with five or six spores.

Tetrads with two and four spores isolated from second-generation fruit bodies were uniformly A_5B_5, although possessing false clamps.

The mutant A_5B_5 stock was crossed with A_6B_6 and a normal dikaryon was formed on the A_6B_6 mycelium. Fruit bodies formed by this dikaryon were normal and bore only four-spored tetrads. Some twenty-one complete tetrads were isolated and scored for A and B factors, possession of false clamps and ability to fruit. Two cultures from each tetrad bore false clamps but only six pairs and six other single cultures had fruited after 30 days out of the total of forty-two cultures with false clamps. No cultures without false clamps fruited. The A and B factors segregated independently of false clamps and fruiting ability. Evidently the formation of false clamps was due to mutation at an unlinked locus *su-A* which has the effect of suppressing the A factor. The mutation is recessive since A_5 and A_6 stocks carrying it are incompatible with their respective wild-type testers carrying *su-A*$^+$. Test crosses with four other wild stocks (A_3B_1, A_7B_7, A_9B_9 and $A_{13}B_{13}$) also gave independent segregations for A, B and the suppressor. The mutant can thus suppress at least six different A factors apparently indiscriminately. The fact that only eighteen of the forty-two cultures with false clamps produced fruit bodies suggests that the presence of *su-A* alone is not sufficient for fruit-body formation by a monokaryon but that it must be accompanied by another factor, or factors, not present in the A_6B_6 parent.

Stocks carrying *su-A* are strongly unilateral in matings and will only donate nuclei. For this reason it was not possible to determine whether a heterokaryon homozygous for A and *su-A* but heretozygous for B has true or false clamps.

3. DISCUSSION

Two of the spontaneous mutants reported by Quintanilha (1935) in *Coprinus fimetarius* are very similar to the mutants described above. The first, denoted A'', arose in a common A heterokaryon. It possessed false clamps and behaved like the A_5 and A_6 mutants. The second mutant, denoted K, arose in a common B heterokaryon. K also produced false clamps and formed monokaryotic fruit bodies but still retained its A and B mating-type specificities. When K was crossed with a compatible stock a vigorous dikaryon was formed but a fruit body produced by the dikaryon gave only monotypic tetrads, each spore having the mating type and characteristics of the K parent. The other parent had made no apparent contribution to the fruit body. We cannot tell whether K was an A factor mutant or, as seems more likely, a recessive suppressor like *su-A*.

In *Schizophyllum commune* Parag (1962) isolated one spontaneous and two nitrogen mustard induced mutants of the mating-type factor B_{41} using a similar method to that employed here. These mutants lacked any B mating-type specificity and resembled the common A heterokaryon of *Schizophyllum* which has a distinctive 'flat' morphology. Parag's analysis revealed that all three B mutants

possessed functional α sub-units indicating that the mutations had occurred in or near the β sub-unit. The parallel with the six A mutants of *Coprinus*, four from A_5 and two from A_6, which also have functional α sub-units is interesting. It could mean that in the factors of both organisms either one of the sub-units is more mutable than the other or that one of the sub-units controls an indispensable function and only mutates to an inviable condition.

Raper & Raper (1962, and personal communication) have also found suppressor, or modifier, mutants which disrupt the normal function of the A factor in *Schizophyllum*. Nine mutants tested so far differ in several ways from the suppressor mutant of *Coprinus*. For example they are all dominant to their wild-type alleles. Most of the mutants are only expressed in the presence of two compatible B factors in a common A heterokaryon or a disomic for B, or, in a haploid monokaryon, when the B factor is represented by a mutant allele. However, as in *Coprinus*, the mutants suppressed all the A factors with which they were tested.

The A or B factor mutants found so far in *Coprinus* and *Schizophyllum* can all be interpreted as losses of factor function. Although the selection method employed should reveal mutations to new specificities none have been found. Of equal interest would be the possibility of mutations to dual or multiple specificities since factors of this kind have never been recorded. However, they would require special methods for their selection and none are available at the present time.

It seems likely that mutants of the kind found in the laboratory may also occur in nature and might account for some of the reports of self-fertile forms of normally heterothallic species (Fincham & Day, in press).

4. SUMMARY

Ten A factor mutants and an unlinked A suppressor mutant were isolated from fruit bodies formed by common A heterokaryons of *Coprinus lagopus*. All A factor mutants formed false clamps and lacked A mating-type specificity. Four A_5 and two A_6 mutants had functional α sub-units which were recovered in tests for intra-factor recombination in matings between mutant and wild-type factors. No functional β sub-units were recovered in tests of two of the A_5 mutants and the two A_6 mutants which possessed functional α sub-units. From three other mutants (two A_5 and one A_6) neither α nor β sub-units were recovered.

The suppressor mutant also formed false clamps but was recessive since stocks carrying it retained their A and B specificities when mated with wild-type testers. Fruit bodies formed by monokaryons carrying the suppressor mutant had pale gills bearing varying proportions of abnormal tetrads with less than four spores.

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