Ribosomal suppressors in *Podospora anserina*: evidence for two new loci by means of a new screening procedure

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SUMMARY

I describe here a new screening procedure to isolate ribosomal suppressors in *Podospora anserina*. I have used the sporulation defect displayed by an antisuppressor mutation *AS7-2*. The revertants able to sporulate are due to either true reversions or external mutations. The mutations which restore most efficiently the sporulation show all the properties of ribosomal suppressors and are localized in two new suppressor loci *su11* and *su12*.

INTRODUCTION

In *E. coli*, genetic and biochemical analyses have shown that alteration of ribosomal proteins can modify the translational ambiguity level (for reviews, see Gorini, 1974 and Piepersberg et al. 1980): some mutations (for example *str A* mutations) diminish the level of natural misreading (i.e. enhance the translational fidelity). They restrict the efficiency of informational (tRNA) suppressors. They are called restrictive mutations and can be considered as ribosomal antisuppressors. Antagonistic ‘*ram*’ mutations were obtained which enhance the natural misreading and correspond to ribosomal suppressors.

In order to study the genetic control of translational fidelity in a eucaryotic organism, a genetic system has been developed in the fungus *Podospora anserina*. It was demonstrated that, as in bacteria, some mutations modify the ribosomal ambiguity level. Suppressors (*su*) and antisuppressors (*AS*) mutations analogous to *ram* and restrictive mutations of *E. coli* were previously described (see Picard-Bennoun, Coppin-Raynal & Dequard-Chablat, 1983 for a review). The mutations assumed to increase translational ambiguity (i.e. ribosomal suppressors) were localized in five loci: *su1, su2, su3, su5* and *su7*. They were isolated as informational suppressors (Picard, 1973; Arnaise and Mignotte, personal communication) while in *E. coli* most *ram* mutations were obtained as antagonists of restrictive mutations (Piepersberg et al. 1980). There are now biochemical arguments demonstrating that the *su* mutations increase translational ambiguity (Coppin-Raynal, 1982‘a, b). I report here the use of a new screening procedure for suppressors (*ram* like) mutations, which parallels that used in *E. coli*. The *AS7* strains were found to display a sporulation defect probably due to their high translational fidelity (Coppin-Raynal & Le Coze, 1982; Coppin-Raynal, 1982a;
### Table 1. General characteristics of the strains used in this study

<table>
<thead>
<tr>
<th>Strains</th>
<th>Localization of mutation</th>
<th>Characteristics</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>AS7-1</td>
<td>0 V</td>
<td>AS, PmR, spo~‡</td>
<td>Coppin-Raynal (1982a)</td>
</tr>
<tr>
<td>AS7-2</td>
<td></td>
<td></td>
<td>Coppin-Raynal &amp; Le Coze (1982)</td>
</tr>
<tr>
<td>AS3-1</td>
<td>40 IV</td>
<td>AS, PmR, reduced fertility</td>
<td>Picard-Bennoun (1976)</td>
</tr>
<tr>
<td>AS4-43</td>
<td>98 I</td>
<td>AS, PmR</td>
<td>Picard-Bennoun &amp; Le Coze (1980)</td>
</tr>
<tr>
<td>AS6-1</td>
<td>70 III</td>
<td>AS, PmR, ♀ sterile</td>
<td>Picard-Bennoun (1981)</td>
</tr>
<tr>
<td>193</td>
<td>0 II</td>
<td>Umpigmented spores and mycelium, nonsense mutation</td>
<td>Picard (1971)</td>
</tr>
<tr>
<td>su1-1</td>
<td>30 IV</td>
<td>Ribosomial suppressor, PmHS</td>
<td>Picard (1973)</td>
</tr>
<tr>
<td>193 su1-1</td>
<td></td>
<td>Pigmented (green) spores</td>
<td></td>
</tr>
<tr>
<td>leu1-1</td>
<td>70 VII</td>
<td>Auxotrophy for leucine</td>
<td>Crouzet (personal communication)</td>
</tr>
<tr>
<td>su2-18</td>
<td>70 VII</td>
<td>Ribosomal suppressor</td>
<td>Arnaise and Mignotte (Personal communication)</td>
</tr>
<tr>
<td>leu1-1 su2-18</td>
<td></td>
<td>Prototrophic for leucine</td>
<td></td>
</tr>
<tr>
<td>Su4-1</td>
<td>65 VII</td>
<td>tRNA type suppressor, spo~</td>
<td>Picard (1973)</td>
</tr>
<tr>
<td>cs12-1</td>
<td>0 VII</td>
<td>Cold sensitive, spo~, PmHS</td>
<td>Picard-Bennoun &amp; Le Coze (1980)</td>
</tr>
<tr>
<td>PmA-1</td>
<td>98 I</td>
<td>PmHS, spo~</td>
<td>Randscholt et al. (1982)</td>
</tr>
<tr>
<td>PmB-1</td>
<td>80 III</td>
<td>PmHS</td>
<td>Dequard et al. (1980)</td>
</tr>
<tr>
<td>Pm1</td>
<td>98 I</td>
<td>PmR</td>
<td>Coppin-Raynal &amp; Le Coze (1982)</td>
</tr>
<tr>
<td>Pm3 (= mei3)</td>
<td>65 VII</td>
<td>PmR, spo~</td>
<td></td>
</tr>
</tbody>
</table>

* SDS %, Second division segregation percentage (postreduction percentage).
† LG, Linkage group.
‡ Symbols are as follows: PmHS and PmR: respectively more sensitive and more resistant to paromomycin than wild-type; spo~: defective in sporulation; AS: antisuppressor.
New ribosomal suppressors in Podospora

Dequard-Chablat & Coppin-Raynal, 1984). Among the revertants able to sporulate, three new ribosomal suppressors were characterized.

MATERIAL AND METHODS

(i) Organism and strains

_Podospora anserina_ is a filamentous fungus. Its biology and culture techniques have been previously described (Rizet & Engelmann, 1949; Esser, 1974). The ascus of _Podospora_ contains four spores. Each one develops around two non-sister nuclei of the post-meiotic mitosis. This property allows dominance and complementation tests in the appropriate crosses. A few asci (2–5%) contain five spores: three binucleate and two small uninucleated ones. Crosses are generally performed between homocaryotic strains derived from the small spores. A pair of alleles (mt+ and mt−) controls the mating-type.

The _AS7-2_ strain displays the same phenotypic properties as those described for _AS7-1_ (Coppin-Raynal & Le Coze, 1982): paromomycin resistance, antisuppressor effect and sporulation defect. The characteristics of the other strains used in this study are listed in Table 1.

(ii) Selection of _AS7-2_ revertants

Strains homozygous for _AS7-2_ and heterozygous for the mating-type locus (mt+/mt−) differentiate perithecia which do not contain any spores. In order to obtain revertants able to sporulate, small pieces of _AS7-2_ mt+/AS7-2 mt− mycelium were inoculated on 3 cm cellophane disks plated on solid minimal medium (M2) and grown for 2 days at 27 °C. The thalli were treated by ultraviolet irradiation (UV) at 250, 500 and 1000 ergs/mm² (respectively 30, 145 and 145 thalli). Fifty untreated thalli were kept for spontaneous reversion control. The disks were then transferred onto new M2 dishes and put back at 27 °C in the light. After about 1 week, perithecia were formed and a systematic search for the appearance of asci was undertaken.

(iii) Genetical analysis

For each sporulating sector, a sample of 6–12 asci was picked up. The progeny in the revertant sector generally come from the cross of gametes carrying _AS7-2_ revertant nuclei (_AS7-2′_) with gametes carrying the _AS7-2_ parental ones. The analysis of the offspring allows verification of the genetic nature, to follow the segregation of the sporulation phenotype and to isolate the _AS7-2_ revertant strain. Further crosses with the wild-type were made to distinguish between reversion (_AS7-2′_ = _AS7+_) (or second site mutation closely linked to _AS7-2_) and external mutations (_AS7-2′_ = _AS7-2m_) (Table 2). In the latter case the _m_ mutation was transferred from the _AS7-2_ background and characterized.

(iv) Estimation of the paromomycin resistance level in vivo

_Podospora anserina_ forms a relatively flat circular thallus growing only at the periphery. The growth curve measured by the increase in diameter of the thallus is linear. Growth curves of the various strains were determined at 27 °C on solid
minimal medium (M2) and on M2 containing 1 mM paromomycin. The diameters of at least four thalli were measured. In this study, the paromomycin resistance level was evaluated after 4 days of growth as the ratio of the average diameters of thalli on M2 + antibiotic/M2, expressed as a percentage.

Table 2. Expected asci in the genetical analysis of the revertants

<table>
<thead>
<tr>
<th>Crosses with</th>
<th>AS7-2*</th>
<th>AS7+ (wild-type)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type of asci . . .</td>
<td>2 (spo+)</td>
<td>2 (spo+), 4 (spo+)</td>
</tr>
<tr>
<td>Genotype</td>
<td>2 (spo-)</td>
<td>2 (spo-), 4 (spo+)</td>
</tr>
<tr>
<td>AS7+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>AS7-2</td>
<td>(No spore)</td>
<td>+</td>
</tr>
<tr>
<td>AS7-2' = AS7+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>or = AS7-2m with</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>m closely linked to AS7-2</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>AS7-2' = AS7-2m;</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>m unlinked to</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>AS7-2†</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

* AS7-2 always segregates at the first meiotic division.
† The relative amount of each category of asci depends on the post-reduction frequency of the m mutation. The selection procedure of AS7-2 revertants implies that the m mutations alleviating the sporulation defect must be at least semi-dominant.

(v) Suppression tests

The 193 mutation defective in spore pigmentation (white spores) is generally used to characterize informational suppressors (su). 193 'su' spores are more or less pigmented according to the efficiency of suppression. Other mutations altering spore pigmentation are known and their properties allow one to differentiate putative ribosomal suppressors and tRNA nonsense suppressor (Picard, 1973). We can distinguish between nonsense and missense mutations. Putative tRNA nonsense suppressors are only active on the former mutations, while ribosomal suppressors can suppress all kinds of mutations.

(vi) Antisuppression tests

They are based on the ability of the antisuppressor mutations to decrease the efficiency of a suppressor. Two strains were used: 193 sul-1 and leu1-1.su2-18 (see Table 1). The 193 sul-1 strain forms pigmented spores. The addition of an antisuppressor mutation (AS) reduces the pigmentation level (Picard-Bennoun, 1976). The leu1-1 mutation is auxotrophic for leucine. The auxotrophy is suppressed by ribosomal suppressors like su2-18 and an antisuppressor mutation abolishes the prototrophy (Coppin-Raynal, 1981).
RESULTS

(i) Selection and characterization of AS7-2 revertants

Strains homozygous for AS7-2 and heterozygous for the mating-type locus differentiate perithecia which do not contain any spores. They were used to select revertants able to sporulate as described in Materials and Methods. Of the total of 320 irradiated thalli, we identified 17 sporulating sectors (respectively 1, 13 and 3 at 250, 500 and 1000 ergs/mm²). No spontaneous revertant was obtained. The revertants were analyzed as indicated (Materials and Methods). In every case, the (spo+) phenotype was recovered in the progeny with a mendelian segregation indicating single nuclear mutations. Crosses of the revertant strains by wild-type allowed differentiation between mutational events closely linked (back mutations or second site mutations), or unlinked to AS7-2. Eleven revertants belong to the former class. They have recovered wild-type sporulation and we were unable to separate the new mutation from AS7-2 at the limit of the genetical analysis (between 25 and 50 asci analysed, according to the revertant). Two of them can be considered as true reversions because they have recovered a wild-type phenotype for all tested criteria (whole loss of antisuppressor effect and paromomycin resistance), while the nine others have kept a slight paromomycin resistance and antisuppressor effect on 193 sul-1 but not in leu-1-1 su2-18 (see Material and Methods and Table 1). For these revertants, it is impossible at the moment to differentiate between reverse mutation which do not restore the wild-type sequence exactly and closely linked suppressor mutation. The six remaining revertants contained an external mutation (called TO) unlinked to AS7-2 which more or less restores the sporulation of AS7-2 strain (Table 3). They were named m4, m6, m22, m27, m35 and m51.

(ii) Characteristics of the AS7-2 m strains

Results of the crosses AS7-2m x AS7-2 showed that the m mutations segregate mostly at the second meiotic division. As expected by the selection procedure they are at least semi-dominant. A dominance test based on sporulation showed that m22 and m27 were completely dominant. However, strains homozygote for m4 sporulated better than heterozygotic strains and we can say that m4 is partially dominant.

The three other mutations, m6, m35 and m51, are linked to the mating-type, so comparison between homocaryotic and heterocaryotic strains is impossible. In terms of restoring the spo+ phenotype of AS7-2 (Table 3), m6 was the most efficient. m22, m27 on the one hand, and m35 and m51 on the other hand, only partially relieved the sporulation defect but showed a cumulative effect (m22 plus m35 or m51, for example).

The phenotypic analysis of the revertants showed that some m mutations decreased the paromomycin resistance of AS7-2 (Table 3). Moreover there was a good correlation between the effect of the mutation on sporulation and on the decrease of the resistance: While m22 and m27 displayed no effect, m4 and m6 notably reduced the paromomycin resistance of AS7-2, the AS7-2 m6 strain even becoming more sensitive than the wild-type strain (Table 3).
Table 3. General characteristics of the m mutations recovered from the revertants

<table>
<thead>
<tr>
<th>UV doses (erg/mm²)</th>
<th>Sporulation*</th>
<th>Paromomycin† resistance in vivo</th>
<th>Paromomycin resistance of m alone</th>
<th>Suppressor‡ effect on 193</th>
<th>Localization</th>
</tr>
</thead>
<tbody>
<tr>
<td>m22 500</td>
<td>+</td>
<td>83</td>
<td>30</td>
<td>—</td>
<td>Unlinked to the mating-type locus</td>
</tr>
<tr>
<td>m27 500</td>
<td>+</td>
<td>84</td>
<td>31</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>m35 500</td>
<td>+</td>
<td>76</td>
<td>30</td>
<td>—</td>
<td>Linked to the (§) mating type locus</td>
</tr>
<tr>
<td>m51 500</td>
<td>+</td>
<td>74</td>
<td>10</td>
<td>Strong su12-1, linked to the mating type locus (§)</td>
<td></td>
</tr>
<tr>
<td>m4 1000</td>
<td>++</td>
<td>59</td>
<td>9</td>
<td>Weak su11-1, linked to the PmB</td>
<td></td>
</tr>
<tr>
<td>m6 500</td>
<td>++</td>
<td>22</td>
<td>No growth</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* The effect of the m mutation on the sporulation of AS7-2 is determined from the sporulation of a [(AS7-2 m/AS7-2 m⁺) (m⁺/m⁻)] strain. It is estimated by the number of asci projected during a day: +, about 100 asci; ++, 10000–100000 asci; ++++, more than 100000.

† The paromomycin resistance is estimated by the percentage of growth after 4 days on medium containing 1 mM paromomycin in comparison with the growth in absence of antibiotic (see Material and Methods). It is 32% for wild-type and 84% for AS7-2 in the same experiment.

‡ The suppressor effect of a mutation is determined as described in Material and Methods: ‘—’ means no visible effect. In the case of m51, the suppressor effect corresponds to the increase of the efficiency of a known ribosomal suppressor, su1-1.

§ For the moment there are no data available to determine the allelism between m35, m51 and m6 but they are very closely linked mutations and can be assumed to belong to the same locus (see in the text).
Characteristics of the m mutations

Effect on paromomycin resistance. The m mutations isolated from the AS7-2 background have been characterized. They were tested for their paromomycin resistance (Table 3). m22, m27 and m35 are as sensitive as wild-type while m4, m51 and m6 (the most efficient suppressor) displayed a hypersensitivity towards paromomycin. We found a correlation between the efficiency of the m mutation in restoring the sporulation of AS7-2 and its effect on the paromomycin resistance level. Moreover, m6 produced a much altered phenotype with a very slow growth rate and female sterility. Its growth was a little improved at 30 °C and m6 can be considered as a cold-sensitive mutation. However, AS7-2 m6 strains grow well at 27 °C and is female fertile. The other m mutants manifested no particularly altered phenotype.

The m mutations antagonize AS mutations. In Podospora, the mutants unable to sporulate constitute a very heterogeneous group involving numerous genes (Simonet & Zickler, 1972; Zickler & Simonet, 1980). We investigated whether the m mutations could suppress different types of spo~ mutations whose properties are listed in Table 1 (Pm3, cs12, su4 and PmA). No suppression was found except for AS7-1. Recently, other AS mutations were found to be sporulation deficient (AS4-43 and new AS6 mutations). m4 and m6 are able to relieve this defect. This suggests that the specificity of the m mutations is limited to AS mutations. Furthermore m4, m6 and m51 decrease strongly or weakly the paromomycin resistance associated with several antisuppressor mutations (AS4-43, AS3-1, AS6-1). They also reduce the paromomycin resistance level of Pm3 mutants which displays no antisuppressor effect.

The specificity of the m mutations at the sporulation level is limited to AS mutations while they interact with all mutations tested at the level of paromomycin resistance. In the same way, the m6 growth defect seemed to be specifically alleviated by antisuppressor mutations (AS6 mutations displayed a positive effect while Pm3-1 had no effect).

m4, m6 and m51 display a suppressor effect. The sporulation defect of AS7-2 is assumed to be the consequence of its antisuppressor property, that is its high translational fidelity (Dequard-Chablat & Coppin-Raynal, 1984). In order to determine whether the m mutations interfered with translational ambiguity, we tested their effect on the 193 nonsense mutation, an allele generally used to characterize informational suppressors and antisuppressors in Podospora. The results are reported in Table 3. m4 and m6 suppressed the 193 mutation giving light green (193 m4) or green spores (193 m6). Moreover they displayed a suppression spectrum typical of ribosomal suppressors. They were called respectively sul-1 and sul2-l. In addition, their suppression efficiency on 193 is lowered by antisuppressor mutations (AS7-2, AS3-3, AS4-43 and AS6-1) as previously observed for other ribosomal suppressors. The action of m51 on 193 is not so obvious but it increases the suppressor effect of sul-1. It can be considered as a low efficiency suppressor.

The other mutations, m22, m27 and m35 had no suppressor effect. The antisuppression tests on 193 sul-1 or leu su2-18 showed no antisuppression property.
Localization of the m mutations

The m mutations displayed post-reduction percentage between 60 and 100. They define three independent loci: a locus with m35, m51 and m6 closely linked to the mating-type locus; a locus with m4, and a third locus with m22 and m27 independent from the mating-type locus. m6, m35 and m51 are localized inside a very small region (less than 1 centimorgan) including the mating-type locus and different mutations altering the paromomycin phenotype (PmA, Pml, AS4) (Table 1). It is difficult to know the allelic relationships between these mutations. In fact, the only available complementation test is based on paromomycin hypersensitivity which is a recessive character. This property is an unreliable criterion because we have found no or very poor complementation between unlinked hypersensitive mutations. Nevertheless, m6 and PmA-1 were found to recombine at a frequency about $10^{-4}$. This indicated that they at least were localized at two different sites. The same result was found for m51. Owing to their related phenotype and genetic closeness, m6 and m51 are considered to belong to the same locus, sul2. m35 has no particular phenotype and can not be more precisely localized in this region.

Crosses with appropriate genetic markers including previously identified suppressor mutations placed m4 close to PmB (Table 1 and 3) (no recombinant for 100 ascis analysed). The previous remark on complementation tests is valid for these two mutations. However we can notice that neither PmA-1 nor PmB-1 relieved the sporulation defect of AS7-2 indicating that if PmA-1, m6 and m51 on the one hand, and m4 and PmB-1 on the other hand belong respectively to single genes, these are mutations with different effects. m22 and m27, displaying no particular phenotype, are not localized at this time.

In conclusion the m mutations define two new ribosomal suppressor loci: sul1 on chromosome III and sul2 on chromosome I. The other ribosomal suppressor loci known in Podospora are localized elsewhere (Marcou, Picard-Bennoun & Simonet, 1980).

DISCUSSION

The genetical analysis of 17 revertants of the AS7-2 mutant revealed that the mutations which restore its sporulation belongs to two classes: the first class corresponds to mutations indistinguishable from AS7-2. Two of them are assumed to be true reversions while the others did not recover a completely wild type phenotype. In these cases we were unable in the limit of the genetical analysis to differentiate between intragenic revertants and closely linked suppressor mutations antagonistic to the AS7-2 antisuppressor mutation. Indeed a ribosomal suppressor gene su3 is known to be localized very close to AS7 (Picard, 1973; Marcou et al. 1980; Coppin-Raynal & Le Coze, 1982). The second class corresponds to six external mutations (Table 3). The most efficient mutations can be considered as ribosomal suppressors localized in two new loci sul1 and sul2. In vivo they display the same properties as the previously identified ribosomal suppressors (Picard, 1973; Coppin-Raynal, 1981): Suppression of the pigmentation defect caused by 193, hypersensitivity to paromomycin, and interaction with AS7-2 and other AS mutations as revealed by the reduction of the resistance level to paromomycin and
pigmentation of 193 su AS spores. In vitro data confirm that su11 and su12 mutations increase the ribosomal ambiguity level, and electrophoretic analysis has shown that su11-1 and su12-1 affect proteins of the 40S ribosomal subunit (Dequard-Chablat & Coppin-Raynal, 1984).

These results suggest several remarks:

1) The obtaining of ‘true’ reversions of the AS7-2 mutant demonstrates that its various properties (antisuppression, paromomycin resistance, sporulation deficiency) are the result of a single mutation with pleiotropic effects. The fact that a high translational fidelity blocks sporulation in Podospora is discussed elsewhere (Dequard-Chablat & Coppin-Raynal, 1984) within the context of a more general hypothesis proposed by Picard-Bennoun (1982).

2) Besides the three well defined external mutations localized in su11 and su12, three mutations were found with a lower efficiency: m35, m22 and m27. m35 lies in the same region as su12 but it does not display a suppressor effect or paromomycin hypersensitivity. On the contrary, unpublished in vitro experiments suggest that m35 might correspond to a weak ribosomal antisuppressor mutation displaying an antagonistic action on AS7-2. In Podospora, antagonistic effects have already been described for suppressors (Picard-Bennoun, 1976; Coppin-Raynal & Le Coze, 1982). Further experiments are needed to know whether m35 lies in the AS4 gene which is closely linked to su12.

m22 and m27 belong to the same locus and have no suppressor or antisuppressor effect in vivo (Table 3). Unreported data showed that, in vitro, they did not seem to modify the misreading level of the ribosomes. However they might have a slight effect on misreading level which could occur specifically during the sporulation process. This effect would not therefore be seen at the spore or vegetative level. An alternative explanation is that m22 and m27 alter some genes involved in sporulation.

3) Until this work, all mutations increasing or assumed to increase the level of translational errors had been selected as suppressors of the 193 (Picard, 1973) or the leu1-1 (Arnais and Mignotte, personal communication) nonsense mutations. These ribosomal suppressors were localized in five loci su1, su2, su3, su5 and su7. Of these suppressors 90%, particularly the most efficient ones, map in the su1 and su2 loci. In the present case, the suppressors have been selected against an antisuppressor, a procedure used in E. coli to isolate ram mutation (for a review, see Piepersberg et al. 1980). This screening procedure has allowed the identification of two new ribosomal suppressors in Podospora. Their expression required that they were epistatic to AS7-2 (decrease of paromomycin resistance and translational fidelity, restoration of sporulation) and thus somewhat different from the previous suppressor mutations described. In effect, the AS7-1 and AS7-2 mutations are epistatic to su1 and su2 mutations both in vivo and in vitro. Even strong suppressor mutations like su1-60 and su2-5 cannot relieve the sporulation defect of AS7-1 or notably decrease its paromomycin resistance. Moreover the ribosomes from the double mutants su1-60.AS7-1 or su2-5.AS7-1 behave like AS7-1 ribosomes in in vitro experiments (Coppin-Raynal & Le Coze 1982; Coppin-Raynal 1982a, b). These data might explain why no su1 and su2 mutations were found in the AS7-2 revertants.

In E. coli, mutations in two ribosomal genes can increase the misreading level.
They fall in the structural genes for ribosomal proteins S4 and S5. In yeast, three omnipotent suppressor loci are now well characterized (Inge-Vechtomov & Andrianova, 1970; Hawthorne & Leupold, 1974; Gerlach 1975; Ono, Stewart & Sherman, 1981). So far, the alteration of one ribosomal protein (S11) has been identified, for sup46 (Ishiguro et al. 1981). In Podospora, we have identified seven putative ribosomal suppressors loci. For three of them, we have evidence for the alteration of a ribosomal protein (Dequard-Chablat & Coppin-Raynal, 1984; Dequard-Chablat et al. in preparation). This result does not imply that the genetic control of translational fidelity is more complex in Podospora: Perhaps the variety of screening procedures used in this organism has allowed identification of a greater number of genes than in other organisms. The present results support this conclusion because a new screening procedure leads to two new ribosomal suppressor loci.

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