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Recombination and Mitotic Segregation of Mitochondrial Genes in the Crosses of Isogenic Yeast (*S. cerevisiae*) Strains

(With 1 Text-figure)

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Abstract

The investigations of the recombination and segregation of the mitochondrial drug-resistance genes were performed using pairs of the isogenic yeast (*S. cerevisiae*) strains.

1. The transmission polarity, as well as the recombination polarity, were scarcely observed in any cross between pairs of the isogenic strains.
2. The recombination frequencies in the crosses of the isogenic strains were considerably higher than those in anisogenic strains.
3. Different carbon sources in the growth media and different growth conditions prior to mating were less effective on the transmission and recombination of the mitochondrial genes of the zygotes.
4. Segregation rates of the mitochondrial genes during the growth of the zygote progeny of the isogenic strains were lower than those in the case of the anisogenic strains.

Introduction

The transmission and reco mbination frequencies of the mitochondrial genes in *S.cerevisiae* have been known to be variable with the strains used in the crosses. When two haploid strains were crossed and the zygote progeny were analysed, the ratios of the markers recovered from **a**-parent to α-parent and of one recombinat type to the other usually deviated from 1 to 1. The phenomena, called polarity of transmission and polarity of recombination, were first observed in the crosses when strains of different origins were used (Thomas and Wilkie, 1968; Coen *et al.*, 1970). "Polarity" of recombination, has been defined as the deviations induced by a mitochondrial "sex factor, ω" (Bolotin *et al.*, 1971) and the phenomenon, "bias", similar to the "polarity" as those caused by unkown nuclear gene(s) (Waxman *et al.*, 1973).

In general, it can be said that both "bias" and "polarity" have, so far, been observed in the crosses between the strains of non-isogenic mitochondrial and/or nuclear genomes. Then we attempted to perform the investigations of mitochondrial genetic analyses, where the genetic constitutions of the parental strains were as near as possible. For this purpose, we prepared a few sets of isogenic strains which were derived from a strain AU42 (**a**, *ade*1, *ura*3) (Uchida and Suda, 1978).

In this paper we describe the segregation and recombination of the mitochondrial drug-resistance markers in which crosses were conducted by using pairs of isogenic strains, and comparisons between isogenic and anisogenic crosses were made concerning the behaviors of the mitochondrial genes after zygote formation.

Materials and Methods

Strains. The strains used for the crosses are summarized in Table 1 and the lineages of isogenic strains are schematically shown in Fig. 1. The mitochondrial resistance markers to chloramphenicol, erythromycin, spiramycin, oligomycin and antimycin A are referred to as [*C*], [*E*], [*S*], [*O*] and [*A*], respectively, in this paper.

Antibiotics. The concentrations of chloramphenicol (Chloromycetin powder, Sankyo), erythromycin lactobionate (Erythrocin-I.V., Abbott), spiramycin (Kyowa), oligomycin (Sigma) and antimycin A (Boehringer Mannheim) were 3 mg, 1 mg, 4-5 mg, 30 μg and 20 ng per ml of medium, respectively.

Media, standard mating procedures and determination of drug resistances. Details were previously described (Suda and Uchida, 1974).

Isolation of isogenic strains and of drug resistant mutants, and synchronous zygote formation. Details were described elswhere (Uchida and Suda, 1978).

Temperature. All the experiments were done at 30°C.

Results

1. Cross-resistances of the mitochondrial markers

The mitochondrial drug resistant mutants isolated were examined as to resistances to other drugs. All the resistant mutants to chloramphenicol, oligomycin and antimycin A were sensitive to the other drugs, but the erythromycin resistant mutants, except the strain, ER61[*E*61], were cross-resistant to spiramycin. The strains carrying the erythromycin-resistance genes shown in Table 1, *i. e.*, [*E*106], [*E*303], [*E*102] and [*E*514], were simultaneously resistant to more than 5 mg per ml of spiramycin, whereas ER61 [*E*61] could slightly grow on the plates containing 5 mg per ml of spiramycin, and could be distinguished from the other spiramycin resistant strains. The spiramycin resistant mutants, SR6 [*S*6] and SR41 [*S*41], were sensitive to erythromycin, though the spiramycin resistances often showed cross-resistance to erythromycin.

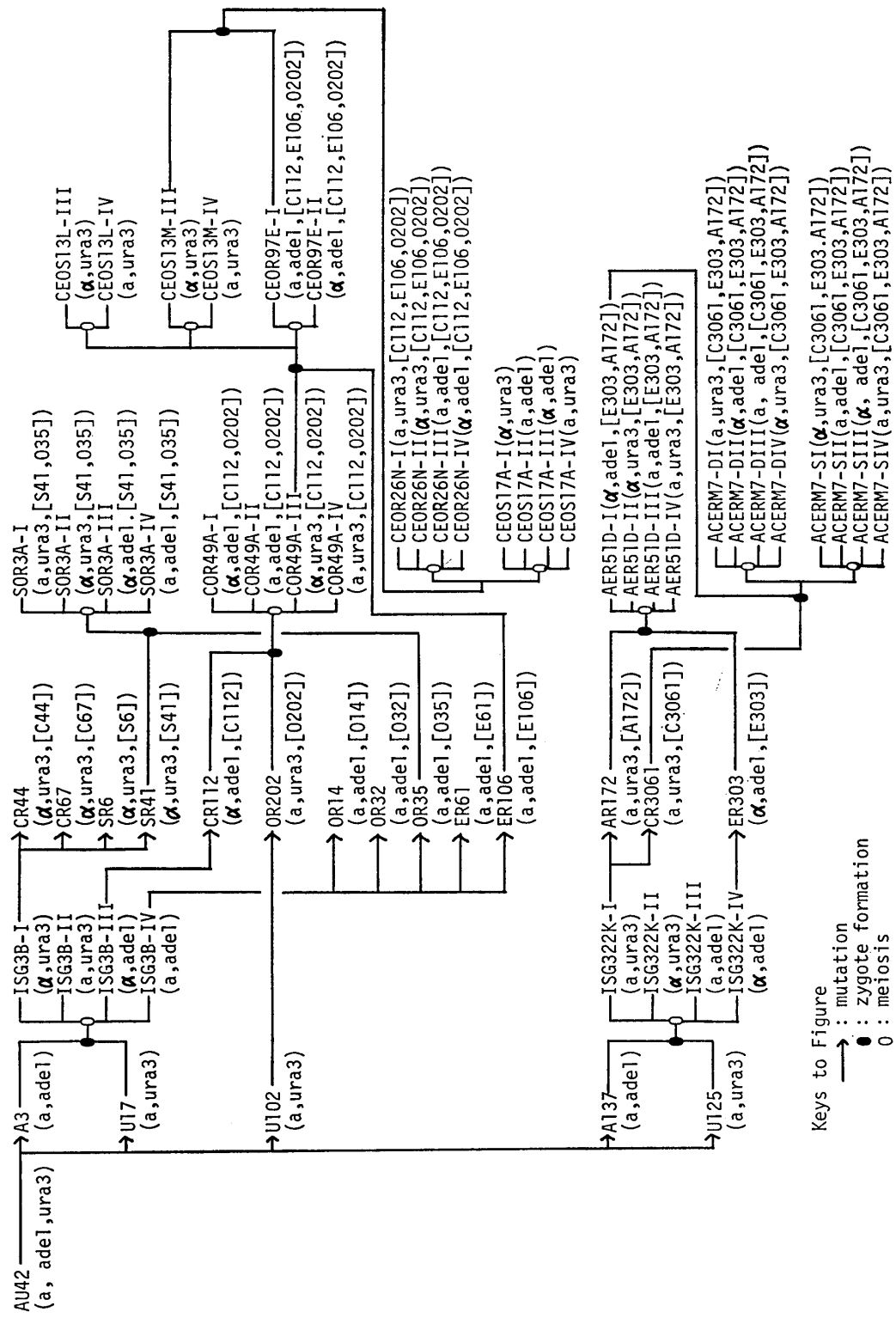

Fig. 1. Lineage of isogenic strains

Table 1. Strains used for the crosses in this paper

Strain	Marker		Source
	Nuclear	Mitochondrial	
CR44	α, *ura*3	[*C*44]	from ISG3B-I
CR67	α, *ura*3	[*C*67]	〃
SR6	α, *ura*3	[*S*6]	〃
SR41	α, *ura*3	[*S*41]	〃
ER61	**a**, *ade*1	[*E*61]	from ISG3B-IV
OR14	**a**, *ade*1	[*O*14]	〃
OR32	**a**, *ade*1	[*O*32]	〃
OR35	**a**, *ade*1	[*O*35]	〃
SOR3A-II	α, *ura*3	[*S*41, *O*35]	from the cross, SR41 and OR35
SOR3A-IV	**a**, *ade*1	[*S*41, *O*35]	〃
CEOR97E-I	**a**, *ade*1	[*C*112, *E*106, *O*202]	from the crosses, CR112, ER106 and OR202
CEOR97E-II	α, *ade*1	[*C*112, *E*106, *O*202]	〃
CEOS13L-III	α, *ura*3		〃
CEOS13L-IV	**a**, *ura*3		〃
CEOR26N-III	**a**, *ade*1	[*C*112, *E*106, *O*202]	〃
CEOR26N-IV	α, *ade*1	[*C*112, *E*106, *O*202]	〃
CEOS17A-I	α, *ura*3		〃
ACERM7-SI	α, *ura*3	[*C*3061, *E*303, *A*172]	from the crosses, CR3061, ER303 and AR172
ACERM7-SII	**a**, *ade*1	[*C*3061, *E*303, *A*172]	〃
ACERM7-SIII	α, *ade*1	[*C*3061, *E*303, *A*172]	〃
ACERM7-SIV	**a**, *ura*3	[*C*3061, *E*303, *A*172]	〃
ACERM7-DII	α, *ade*1	[*C*3061, *E*303, *A*172]	〃
ISG322K-I	**a**, *ura*3		from the cross, A137 and U125 (cf. Uchida and Suda, 1978)
ISG322K-II	α, *ura*3		〃
ISG322K-III	**a**, *ade*1		〃
ISG322K-IV	α, *ade*1		〃
AR172	**a**, *ura*3	[*A*172]	from ISG322K-I
OEC308	**a**, *ade*1	[*C*50, *E*102, *O*706]	from the crosses, CMR50, R102 and 706R1 (cf. Suda and Uchida, 1974)
SW26	α, *his*4, *leu*2	[*E*106, *O*706]	from the cross, R102 and 706R1 (cf. Suda and Uchida, 1972)
IL8-8D	**a**, *ura*1	[*C*321(*RI*), *E*514(*RIII*)]	from Dr. P. P. Slonimski
D22A15	**a**, *ade*1	[*O*114(*OII*)]	〃

2. Allelism analyses of the mitochondrial markers

The allelism tests were performed by crossing pairs of strains that were resistant to the same drug. Three hundred and twelve colonies of zygote progeny derived from each cross were examined for resistance or sensitivity to the drug. The results are shown in Table 2.

Table 2. Allelisms of mitochondrial markers

Locus	Marker
[*RI*]	[*C*50]*, [*C*44][1], [*C*67][2], [*C*112][3], [*E*61][4], [*C*3061][10]
[*R*III]	[*E*102]*, [*E*303]**, [*E*106][5], [*S*41][6], [*S*6][7]
[*O*I]	[*O*706]*, [*O*202][5], [*O*32][9]
[*O*35]	[*O*14][8], [*O*35][8]

1) Cross CR44[*C*44] x OEC308[*C*50]
2) Cross CR67[*C*67] x OEC308[*C*50]
3) Cross CR44[*C*44] x CEOR97E-I[*C*112]
4) Cross ER61[*E*61] x CR67[*C*67]
5) Cross CEOR97E-I[*E*106, *O*202] x SW26[*E*102, *O*706]
6) Cross SOR3A-II[*S*41] x CEOR97E-I[*E*106]
7) Cross SR6[*S*6] x SOR3A-IV[*S*41]
8) Cross OR14[*O*14] x SOR3A-II[*O*35]
9) Cross OR32[*O*32] x SW26[*O*706]
10) Cross ACERM7-DII[*C*3061] x IL8-8D[*C*321(*RI*)]
* (cf. Suda *et al.*, 1978)
** (cf. Uchida and Suda, 1978)

In the crosses, 1), 2), 3) , 5), 7), 8), 9) and 10), no colonies of one recombinant type that was sensitive to the drug concerned were detected, indicating that the genes used for the crosses would reside in the same loci or be very closely linked each other. In the cross, 4), the recombinant, concomitantly resistant or sensitive to both chloramphenicol and erythromycin, did not appear. Moreover, when ER61 [*E*61] was crossed to SW26 [*E*102(*R*III)], 18 erythromycin sensitive recombinants per 312 zygote progeny (5.8 %) were produced, indicating that [*E*61] locus was apparently separated from [*R*III] locus (about 12% of recombination frequency). In the cross, 6), as erythromycin-resistance marker [*E*106] was cross-resistant to spiramycin, the examination was performed for spiramycin sensitivity and the cross did not yield spiramycin sensitive recombinants in the zygote progeny.

The [*O*35] gene was considered not to be linked to the [OI] locus but somewhat closely linked to the [*O*II] locus, because the cross of SOR3A-II [*O*35] to CEOR97E-I [*O*202(*O*I)] or OR32 [*O*32(*O*I)] produced 39 or 51 oligomycin-sensitive recombinants per 312 zygote progeny each (average 14.4%; about 29% of recombination frequency)

and the cross of SOR3A-II [*O*35] to D22A15 [*O*144(*O*II)] produced 3 sensitive recombinants per 312 zygote progeny (0.9%; about 2% of recombination frequency).

3. Transmission and recombination of the mitochondrial markers

Table 3-1. Combinations of the crosses

Cross	α-parent	a-parent
1	CR44[*C*44(*R*I)]	OR35[*O*35]
2	SR41[*S*41(*R*III)]	ER61[*E*61(*R*I)]
3	SR41[*S*41(*R*III)]	OR35[*O*35]
4	SOR3A-II[*S*41(*R*III), *O*35]	ER61[*E*61(*R*I)]
5	CR44[*C*44(*R*I)]	SOR3A-IV[*S*41(*R*III), *O*35]
6	CEOS13L-III	CEOR97E-I[*C*112(*R*I), E106(*R*III), *O*202(*O*I)]
7	CEOR97E-II[*C*112(*R*I), *E*106(*R*III), *O*202(*O*I)]	CEOS13L-IV
8	CEOS17A-I	CEOR26N-III[*C*112(*R*I), *E*106(*R*III), *O*202(*O*I)]
9	CEOR26N-IV[*C*112(*R*I), *E*106(*R*III), *O*202(*O*I)]	AR172[*A*172]
10	ACERM7-SI[*C*3061(*R*I), E303(*R*III), *A*172]	ISG322K-III
11	ISG322K-II	ACERM7-SII[*C*3061(*R*I), *E*303(*R*III), *A*172]
12	ACERM7-SIII[*C*3061(*R*I), *E*303(*R*III), *A*172]	ISG322K-I
13	ISG322K-IV	ACERM7-SIV[*C*3061(*R*I), *E*303(*R*III), *A*172]

In cross 1, 2, 3, 4, 5, 6, 7, 8 and 9, 312 colonies were analysed, respectively; and in cross 10, 11, 12 and 13, 214, 202, 204 and 202 colonies were analysed, respectively

Table 3-2. Transmission frequencies (%) of the mitochondrial markers from α-parent

Cross	[*R*I]	[*R*III]	[*O*I]	[*O*35]	[*A*172]
1	51			55	
2	54	53			
3		40		48	
4	56	55		53	
5	49	50		48	
6	52	47	47		
7	51	52	52		
8	55	51	57		
9	67	71	64		60
10	51	51			50
11	60	62			60
12	45	40			46
13	62	62			61
Average	54	53	55	51	55

Table 3-3. Recombination frepuencies (%) between pairs of the mitochondrial markers

Cross	[*RI*] | [*R*III]	[*RI*] | [*OI*]	[*RI*] | [*O*35]	[*RI*] | [*A*172]	[*R*III] | [*OI*]	[*R*III] | [*O*35]	[*R*III] | [*A*172]	[*OI*] | [*A*172]
1			35					
2	19							
3								
4	19		34			35		
5	20		33			35		
6	19	29			28	35		
7	24	30			31			
8	21	29			31			
9	18	22		27	29		28	25
10	20			34			33	
11	25			28			26	
12	17			23			24	
13	19			30			29	
Average	20	28	34	28	30	35	28	25

Many crosses were performed between pairs of the strains carrying resistance genes in different loci. The crosses carried out are shown in Table 3-1. The transmission frequencies of the markers from α-parent to zygote progeny and the recombination frequencies between the markers are shown in Table 3-2 and 3-3, respectively.

Table3-2 indicates that the transmission frequencies were, in general, about 50%, except the crosses 9, 11 and 13; in other words, the ratio of the markers from **a**- to α-parent in the zygote progeny was about 1 to 1.

As shown in Table 3-3, the recombination frequencies are generally higher than those of the crosses where anisogenic strains were used. For example, in the anisogenic crosses involving the strain OEC1222, the recombination frequencies between [*RI*] and [*R*III] loci was about 10% and the frequencies of [*RI*]-[*O*I] and [*R*III]-[*OI*] were about 19% (Suda and Uchida, 1974). In contrast, the average recombination frequencies of [*RI*]-[*R*III], [*RI*]-[*OI*] and [*R*III]-[*OI*] were 20, 28 and 30%, respectively. In addition, the frequency of [*O*35]-[*OI*] was 13% in the anisogenic cross involving OEC1222, but it was about 29% in the isogenic cross. As a whole, the recombination frequencies in the isogenic crosses were 1.5-2.0 times as much of those in the anisogenic crosses.

The additivity of the recombination frequencies was not observed, probably due to the facts that the minimum frequency was about 20% ([*RI*]-[*R*III]) and the maximum frequeucy was about 35% ([*RI*]-[*O*35] or [*R*III]-[*O*35]).

4. Effects of carbon sources on the transmission and recombination of the mitochondrial markers

We previously described that the carbon sources in the culture media prior to mating of the parental strains affected the transmission of the mitochondrial markers to the zygote progeny in an anisogenic cross (Uchida and Suda, 1976). Here we examined if the effect would be observed in the case of the isogenic cross.

Table 4. Effects of carbon sources on transmission and recombination of the mitochondrial markers

Carbon source		Transmission frequency (%)			Recombination frequency (%)		
CEOS17A-I	CEOR26N-III	[C]	[E]	[O]	[C]-[E]	[C]-[O]	[E]-[O]
Glucose	Glucose	55	51	57	21	29	31
Glucose	Glycerol	56	53	55	21	28	26
Glycerol	Glucose	42	42	45	21	25	30
Glycerol	Glycerol	49	48	48	18	23	25

The parental strains, CEOS17A-I and CEOR26N-III were cultured independently in YPD (yeast extract 1%, peptone 1% and glucose 1%) without shaking and/or YPG (yeast extract 1%, peptone 1% and glycerol 2%) with shaking. Both strains were crossed each other by the method of the synchronous zygote formation (Uchida and Suda, 1978) and the resultant zygote progeny were examined for resistance or sensitivity to chloramphenicol, erythromycin and oligomycin. The results are summarized in Table 4, showing that the carbon sources and cultural conditions had almost no effects on both transmission and recombination frequencies, in contrast of the results in the anisogenic cross previously described (Uchida and Suda, 1976).

5. Segregation of the mitochondrial markers from individual zygotes and their segregation rates

To examine the segregation rates of the mitochondrial markers during the zygotic growth in the isogenic cross, CEOS17A-I with CEOR26N-III, subclone analyses were conducted for the individual young zygotes synchronously formed and their progeny isolated after zygotic growth. The genetic constitutions of the subclones were shown in Table 5-1, from the individual young zygotes (A) and the individual zygote progeny after zygotic growth (B and C) and in Table 5-2 the numbers of the heteroplasmons and per cent of the total for the mitochondrial markers in the young zygotes and the zygote progeny were calculated.

As shown in Table 5-1, in the young zygotes (A. 0 hr), almost all the zygotic clones produced the subclones which carried the mitochondrial markers from either **a**- or

Table 5-1. Subclone analyses of 30 individual zygotes at various growth stages after zygote formation

A. 0 hr

[C]	[E]	[O]	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
r	*r*	*r*	18	2	14	21	8	1	0	7	2	2	5	0	4	4	4	3	3	4	8	1	4	3	4	0	5	1	1	3	11	12
r	*r*	*s*	5	2	1	1	3	3	0	4	0	1	3	0	0	2	2	0	2	0	0	1	2	1	7	9	1	1	5	0	0	1
r	*s*	*r*	0	1	3	4	0	0	0	3	0	0	0	0	0	2	1	0	0	3	2	0	2	3	4	0	2	2	1	0	0	2
r	*s*	*s*	0	0	0	0	2	5	0	2	0	2	2	6	1	5	1	1	0	0	0	2	1	3	0	15	1	1	1	0	1	1
s	*r*	*r*	0	0	1	0	1	0	1	0	5	2	1	0	0	0	0	0	1	2	0	0	0	0	1	0	0	3	4	0	1	1
s	*r*	*s*	0	1	0	0	2	0	1	3	2	6	3	0	2	3	3	4	2	4	1	7	1	1	1	1	2	1	5	0	0	1
s	*s*	*r*	1	3	0	0	2	0	0	0	2	0	2	0	6	3	0	1	3	0	3	3	5	0	0	0	2	11	2	0	0	4
s	*s*	*s*	2	17	7	0	8	17	24	7	15	13	10	20	13	7	15	17	15	13	12	12	11	15	9	1	13	6	7	23	13	4

B. 6 hr

[C]	[E]	[O]	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
r	*r*	*r*	0	8	0	9	2	0	15	3	24	5	5	0	14	1	2	7	0	0	0	1	0	10	0	19	5	2	3	8	0	6
r	*r*	*s*	0	1	0	4	20	0	1	2	2	1	4	4	1	11	5	2	0	0	1	2	0	6	0	1	1	3	1	2	1	0
r	*s*	*r*	0	2	0	1	0	0	1	3	0	1	1	0	1	0	2	1	0	0	0	3	0	0	0	4	3	0	0	0	0	1
r	*s*	*s*	2	1	0	3	0	0	0	8	0	0	1	4	2	7	1	0	6	0	13	3	0	0	1	0	0	0	0	0	1	1
s	*r*	*r*	0	6	0	0	0	0	1	3	0	6	1	0	2	0	2	1	0	1	0	0	0	1	5	1	1	12	9	1	0	3
s	*r*	*s*	0	1	6	0	3	0	0	1	0	1	0	12	0	7	2	0	0	2	1	2	2	0	7	0	3	1	4	0	2	0
s	*s*	*r*	3	6	3	1	0	0	1	5	0	2	0	0	1	0	1	2	0	7	0	3	12	3	6	0	2	8	9	5	0	2
s	*s*	*s*	21	1	17	8	1	26	7	1	0	10	14	6	5	0	11	13	20	16	11	12	12	6	7	1	11	0	0	10	22	13

C. 12 hr

[C]	[E]	[O]	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
r	*r*	*r*	17	0	24	17	0	0	0	17	4	25	24	0	7	26	0	26	4	13	0	0	0	0	0	0	7	11	0	26	0	0
r	*r*	*s*	1	0	2	0	0	6	0	9	0	0	0	0	18	0	0	0	20	3	0	0	2	0	1	0	13	15	0	0	0	0
r	*s*	*r*	2	13	0	9	0	3	0	0	10	1	0	0	0	0	0	0	0	0	0	0	0	1	0	1	3	0	2	0	0	0
r	*s*	*s*	0	12	0	0	0	8	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	2	0	0	0	0	0
s	*r*	*r*	4	0	0	0	5	0	0	0	3	0	2	5	1	0	1	0	1	8	0	0	0	1	0	0	1	0	12	0	0	0
s	*r*	*s*	0	0	0	0	1	4	0	0	0	0	0	2	0	0	11	0	1	2	0	0	5	23	1	0	0	0	0	0	11	0
s	*s*	*r*	2	1	0	0	19	0	0	0	9	0	0	0	0	0	0	0	0	0	0	0	0	0	4	0	0	0	12	0	0	0
s	*s*	*s*	0	0	0	0	1	5	26	0	0	0	0	19	0	0	14	0	0	0	26	26	19	1	20	24	0	0	0	0	15	26

Table 5-2. Numbers of the heteroplasmons for [*C*], [*E*] and [*O*] alleles during the growth of zygotes calculated from the data in Table 5-1

Marker	0 hr	6 hr	12 hr
[*C*]	28 (93%)	25 (83%)	14 (47%)
[*E*]	29 (97%)	26 (87%)	14 (47%)
[*O*]	28 (93%)	25 (83%)	16 (53%)

α-parent, that is, most of the young zygotes retained both of the mitochondrial alleles. The degrees of heterogeneity of the mitochondrial markers were decreased with the zygotic growth (B. 6 hr and C. 12 hr). Furthermore, it can be said that the segregation rates of chloramphenicol-, erythromycin- and oligomycin-resistance markers, that is, the rates of the decreasing of heteroplasmons, were nearly equal, but rather low when compared with those of the anisogenic crosses previously described (Suda, 1976; Uchida and Suda, 1976).

Discussion

1. Transmission and recombination of the mitochondrial markers in the isogenic crosses

Many investigations have been done concerning the distribution of the mitochondrial markers among zygote progeny and subsequently polarities of transmission and of recombination have been described (*e. g.* Coen *et al.*, 1970). These phenomena, however, resulted from the crosses between parental strains of different origin. In most cases of the isogenic crosses in this paper, it was evident that no polarities of both transmission and recombination were observed. Therefore, it is considered that when parental strain has more genetical traits in common with the other, the cross is less affected by covered genes, either chlomosomal or mitochondrial, thus the transmission of the markers from **a**- and α- parents will occur with the same frequency.

The recombination frequencies between any pairs of the markers in the isogenic crosses were considerably higher than those in the anisogenic crosses. This may be elucidated by 1) higher frequency of pairing event between the mitochondrial genomes from **a**- and α-parents or 2) lower rate of degradation or elimination, if any, of one of the mitochondrial genomes either from **a**- or α-parent after zygote formation. Firstly, the cell lineage analysis has suggested that the mitochondrial genomes of yeast are not always panmictic after zygote formation, mainly due to bud position of the primary zygote (Strausberg and Perlman, 1978). If the parental strains used were isogenic, it can be assumed that the poor panmix of the mitochondrial genomes would be improved, and, therefore, the probability of the pairing event of the genomes would increase.

Secondarily, the fact that the distribution of the mitochondrial genes was asymmetrical has brought forth many hypotheses for the mechanisms (cf. Birky, 1975). One of the proposed mechanisms was preferential degradation of the mitochpndrial genomes from **a**- or α-parent. If such degradation would not occur or would occur with a lower frequency in the young zygote of the isogenic parents, the chance of recombination would increase.

As the recombination frequencies were comparatively high, the additivities of the frequencies were not consistent. The most probable, however, gene arrangement derived from the results is the order, [*R*III]•[*R*I]-[*O*I]•[*A*172]-[*O*35], and this arrangement is in agreement with the results of deletion mapping (unpublished data).

2. Segregation of the mitochondrial markers from the individual zygotic clones

The asymmetry of the transmission was not observed, as above mentioned, when the progeny from whole zygotes of an isogenic cross were analysed. But when individual zygotes were isolated and their progeny were inspected, the distribution of the mitochondrial genes issued from each zygote was apparently asymmetrical. As shown in Table 5-1. A, the distributions of the mitochondrial markers were markedly variable. Most of the distributions of the markers among the subclones was asymmetrical and the extremes were the clones, *4*, *7*, *12* and *28*, whereas the symmetrical distribution was only observed in 3 clones (clones, *5*, *8* and *29*), indicating that an apparent uniparental transmission of the markers from **a**- or α-parent was observed. In the results of the anisogenic cross (Suda, 1976), the asymmetrical distribution was more serious, though the experimental conditions were different. Those seem likely to support the interpretation that the mitochondrial genomes were distributed uniparentally to the zygote progeny. In fact, Uchida and Suda (1978) observed the uniparental distribution of the mitochondrial genes from young zygotes to tetrads. As shown in Table 5-2, the segregation rates of the three markers were equal and were lower than those in the anisogenic crosses (Suda, 1976; Uchida and Suda, 1976). Those suggest that less degradation of the mitochondrial genomes from **a**- or α-parent would occur in the young zygotes derived from the isogenic cross and the rate of the degradation in the zygote progeny would be lower than that of anisogenic cross.

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References

Birky, C. W., Jr. : Zygote heterogeneity and uniparental inheritance of mitochondrial genes in yeast. Molec. Gen. Genet. **141**, 41-58 (1975)

Bolotin, M., D. Coen, J. Deutsch, B. Dujon and P. P. Slonimski: La recombinaison des mitochondries chez *Saccharomyces cerevisiae*. Bull. Inst. Pasteur **69**, 215-239 (1971)

Coen, D., J. Deutsch, P. Netter, E. Petrochilo and P. P. Slonimski: Mitochondrial genetics I. Methodology and Phenomenology. In: Control of organelle development. Soc. Exp. Biol. Symp. Vol 24 (Miller, P. L., ed.), pp. 449-496. Cambridge: University Press 1970

Strausberg, R. L. and P. S. Perlman: The effect of zygotic bud position on the transmission of mitochondrial genes in *Saccharomyces cerevisiae*. Molec. Gen. Genet. **163**, 131-144 (1978)

Suda, K. and A. Uchida: Segregation and recombination of cytoplasmic drug-resistance factors in *Saccharonyces cerevisiae*. Japan. J. Genet. **47**, 441-444 (1972)

Suda, K. and A. Uchida: The linkage relationship of the cytoplasmic drng-resistance factors in *Saccharomyces cerevisiae*. Molec. Gen. Genet. **128**, 331-339 (1974)

Suda, K.: Somatic segregation, recombination and elimination of mitochondrial genes in *Saccharomyces cerevisiae*. Mem. Facult. Sci. Univ. Kyoto, Series Biol. **7**, 57-66 (1976)

Suda, K., K. Suda and A. Uchida: Degradation of mitochondrial genome of an unstable strain in *Saccharomyces cerevisiae* as revealed by loss and retention of the drug-resistance factors. Bull. Nara Univ. Educat. **27** (2), 71-83 (1978)

Thomas, D. Y. and D. Wilkie: Recombination of mitochondrial drug-resistance factors in *Saccharomyces cerevisiae*. Biochem. Biophys. Res. Commun. **30**, 368-372 (1968)

Uchida, A. and K. Suda: Pattern of somatic segregation of the cytoplasmic drug-resistance factors in yeast. Molec. Gen. Genet. **145**, 159-163 (1976)

Uchida, A. and K. Suda: Distribution of mitochondrially inherited drug-resistance genes to tetrads from young zygotes in yeast. Molec Gen. Genet. **165**, 191-197 (1978)

Waxman, M. F., N. Eaton and D. Wilkie: Effect of antibiotics on the transmission of the mitochondrial factors in *Saccharomyces cerevisiae*. Molec. Gen. Genet. **127**, 277-284 (1973)