<u>Käfer, E.</u>
ssbA, a suppressor of <u>sbA</u>
in stock strains of
<u>Aspergillus nidulans</u>
<u>ssbAl</u> (suppressor of <u>sbA</u>).
Recent analysis of haploid and aneuploid segregants from a diploid with markers on all 16 chromosomes (diploid no. 26, Käfer 1984 Mutat. Res. <u>135</u>:53-75) produced inconsistent segregation for <u>sbA3</u> (inability to use sorbitol as a carbon source). Detailed testing of haploid mitotic segregants from this diploid revealed as the probable cause a mutation on chromosome IV of the <u>sb^+</u> haploid component (M2468; Table I). This mutation apparently inhibits the expression of <u>sbA3</u> and was given the symbol

been found to affect any other carbon source mutants.

To discover the origin of ssbAl which would identify strains unlikely to contain it, and to construct ssbA free diploids for tests of environmentally induced chromosome malsegregation, parental and ancestor strains of M2468 were tested for the presence of ssbAl. Two replacement strains, free of ssbA, were constructed which are available from FGSC as A591 and A593. These strains, in combination with A592 and A594, respectively, produce the test diploids No. 29 and 30 (Käfer et al. 1986 Mutation Res. 167:9-34), which have all chromosomes marked.

To check for the <u>sbA</u> suppressor in strains with a direct ancestor or progeny which showed this mutation, such strains were combined into diploids with suitable tester strains that carried <u>sbA3</u> on chromosome VI and usually <u>methG1</u> on chromosome IV (none of the suspect strains contained <u>sbA3</u> and very few were <u>methG</u> but most carried <u>pyroA4</u> on IV). From these heterozygous diploids, haploids which result from malsegregation of chromosomes were induced with benlate (on complete medium supplemented with methionine to improve the imperfect recovery of <u>methG</u> haploids; Table I). Haploid segregants were tested for markers on IV and VI. Observed ratios for <u>sbA3</u> : + among <u>methG</u> haploids which cannot have <u>ssbA1</u> if it is on chromosome IV, should be 1:1. On the other hand, <u>met^+</u> haploids will show such 1 : 1 ratios only if <u>ssbA</u> is not present, but segregate 0 : 2 for sb : + phenotypes if <u>ssbA1</u> is present since all <u>met^+</u> haploids must have <u>ssbA1</u>. As expected, it was found that for each ssbA1 strain at least one parent carried <u>ssbA1</u> it was assumed, without testing, that all progeny carried <u>ssbA</u>. Fortunately, most of the suspect FGSC strains were without the suppressor (right half and footnote of Table I.)

Table I.

Segregation of the "sb" phenotype in haploid segregants from diploids which combine strains that may carry <u>ssbA1</u> with <u>sbA3;methG1</u> tester strains.

Tested strains with ssbA1*

Strains normal (ssb⁺)**

	Haploids	: me	tł	ı G	vs.	me	et.	h^+			Haplo	ids:	m	eth	G	vs.	me	th^+
FGSC	no.	sb	:	+		sb	:	+	FGSC	r	no.	sb	:	+		sb	:	+
A75	(M 391)	40	:	30		0	:	68	A271	(M2008)	8	:	: 5		9	:	13
A452	(M2162)	20	:	10		0	:	31	A283	(M1655)	16	:	: 9		21	:	16
A473	(M2233)	13	:	14		0	:	17	A363	(M1274)	19	:	: 17		23	:	28
A513	(M2308)	53	:	8		0	:	62	A426	ĺ	M2023)	7	:	: 8		26	:	54
-	(M2468)	21	:	46		0	:	148	A468	(M2216)	11	:	: 23		11	:	21
Not									A479	(M2243),	18	_ :	: _21		16	:	31
FGSC	8 other								A514	(M2356)	11	:	14		20	:	23
	M strains	144	:	169		0	:	447	A550	Ì	M2358)	13	:	14		16	:	21
14 strains,					A608	Ć	M1996)	15	:	9		21	:	19				
comb	oined total	315	:	291		0	:	819	_	(M2257)	12		11		26	:	30
									10 st	tr	rains							
									tota	ıl		130	:	131		189	:	256

* To replace with strains, which are unlikely to carry ssbA1; proposed for A473: A614 (M2843), A615 (M2844) or A616 (M2485) for A513: A617 (M2848), A618 (M2851)

** Other tested FGSC strains which are not ssbA1: A55 (M892), A164 (M645), A213 (M1634), A348 (M921), A453 (2165), A474 (M2234), A477 (M2240), A487 (M2269), A502 (M2285), A511 (M2325) To check for meiotic segregation and linkage, various ssbA strains were crossed to methG and pyroA. No meiotic linkage could be demonstrated (recombination over 40%). As expected, such crosses produced 25% sb- progeny when they were heterozygous for sbA3 (68/274 for ssbA x sbA, and 21/77 for ssbA;sbA x +;+) and 50% sb- in crosses homozygous for sbA3 (51/100); 50% sb- was found also in control crosses heterozygous for sbA3 (206/438).

The ssbAl mutation was traced back through 15 generations to strain FGSC A75 (progeny of cross 362, top of Fig. 2 in Barratt et al. 1965 Genetics 52:233-246). Further tracing to the strain of origin of ssbAl is not possible because few of the ancestral stocks are available by now. Exceptions are the original mutagenized strains in which the various mutants were induced by UV or X-rays that are in the pedigree of A75. Only two of these are fairly direct ancestors of A75 and probably worth testing (Al, choAl and #5, riboB2) Please note that galD5 was prematurely assumed to be induced in the same strain as ssbAl, and contrary to the listing in NN 1985 32:41, strain A213 does not carry ssbAl.

Table II

Untested FGSC strains with an ancestor or progeny that carry ssbA1

a) Strains to be replaced	Replacement strains
FGSC A342 (M771)	FGSC A607 (M2878)
A494 (M2277)	A608 (M1966)
A507 (M2295)	(M2275)
b) Strains to be discontinued	c) Strains to be tested A1 (M7) A155 (M1056)
A455 (M2175)	A5 (M149) A466 (M2213)
A465 (M2212)	A44 (M857) A483 (M2255)
A476 (M2239)	A108 (M455) A499 (M2282)

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