DEA Catcheside 1974 Aust. J. Biol. Sci. 27:561-73.

(10575) : VI *a lys-5* (DS6-85) *ylo-1* (Y30539y) and *a tryp-2* (A60) : VII *a nt* (65001). The group VII marker stock: *A nic-3* (Y31881) was obtained from the Fungal Genetics Stock Centre (FGSC stock number 121).

The genotype and origin of all other cultures is detailed in Table 1.

Stock number 1728 was obtained by crossing 1693 to D. G. Catcheside's stock number 3814, an adenine-requiring isolate (*A his*$^+$ *crisp*$^+$) derived from a cross between Emerson *A* wild type and FGSC stock number 246, which is *a ad-5* (Y152 M40) *his-2* (Y152 M14) *crisp* (B123). Analysis showed DGC 3814 to contain a new *ad-7* allele rather than *ad-5*. The spontaneous acquisition of additional mutations blocking the adenine pathway in adenine auxotrophs is a well-known phenomenon (Mitchell and Mitchell 1950) and presumably this event occurred in FGSC 246. Unlike the *ad-5* allele, the new mutant (MN227) is not linked to *his-2* and *crisp* (see Table 2) and hence would be expected to be the predominant *ad* allele amongst *his*$^+$ *crisp*$^+$ progeny obtained when FGSC 246 is crossed to wild type. The location of *ad* (MN227) 14·7 map units from *am-1* on linkage group V suggested that it is an *ad-7* allele. This hypothesis was tested by constructing a forced heterokaryon with the following genotype: [*a al* (15300); *ad* (MN227) *asp* (MN137)] + [*nit-2* (MN72) *a*; *cot-1* (C102); *am-1*^{6} *his-1* (K83) *ad-7* (K77)]. Since the heterokaryon fails to grow unless adenine is present in the culture medium, the *ad-7* (K77) and *ad* (MN227) mutations are allelic; they affect the same function in adenine biosynthesis.

Table 2. Linkage data for *ad* (MN227) obtained from a cross between DGC 3814 and 1693

The data are abstracted from determinations of the full genotype of 190 viable ascospores

Gene pair[A]	Total number of: Parental types	Recombinant types	P[B]	Map distance (centimorgans)
ad nit-2	96	94	0·9	—
ad mating type	102	88	0·3	—
ad al-2	104	86	0·2	—
ad am-1	162	28	$\ll$0·001	14·7

[A] The markers *nit-2*, *al-2* and mating type are located on linkage group I and *am-1* on linkage group V. Since *ad* (MN227) is linked to *am-1* it will not show linkage to the group I markers *his-2* and *crisp* present in FGSC 246.

[B] Probability of obtaining as great or greater deviation from equality of parental and recombinant types if the genes are truly independently inherited.

Stock 4212 was isolated from a cross between 3882 [an isolate from a cross between 3871 and 2794 having the genotype *A al-2; cot-1; am-1 his-1 inos asp* (C123) *rec-1*$^+$] and 1880 [a sibling of 1885 having the genotype *nit-2* (MN72) *a rec-3 al-2; cot-1; am-1 rec-z*].

Stock number 4277 was constructed as follows: from a cross between 1885 and 3872 a strain having the genotype *nit-2* (MN72) *A al-2; cot-1; am-1*^{6} *his-1 ad-7 rec-1*$^+$ was isolated. The *rec* constitution was determined in crosses to the testers 4322 and 2207. 4277 proved to contain both *rec-1*$^+$ and *rec-z*$^+$ (indicating that *rec-z*$^+$ was present in 3872). The *am-1* (47305) allele present in 4277 was identified by examining the properties of the NADP-specific glutamate dehydrogenase in cell-free extracts, using the methods detailed by D. E. A. Catcheside (1968). The mutant enzyme from *am-1* (47305) cells retains the ability to deaminate glutamate whilst the enzyme from *am-1*^{6} cells is unable either to deaminate glutamate or to aminate α-ketoglutarate.

Stock 4801 was constructed in two steps. First, a new mutation causing a requirement for asparagine, *asp* (MN137), was inserted into stock 2794 by mutation. This was achieved by treating conidia with ultraviolet light followed by filtration enrichment using the method of D. G. Catcheside (1954). Next, the new mutant was crossed to 1883 (a sibling of 1886 having the same genotype). Determination of the genotypes of a random sample of ascospores revealed that 21 were *asp his* and 25