Fungal Genetics Stock Center
Dept. of Microbiology
Univ. of Kansas Medical Center
Kansas City, KS 66103

PLEASE PROVIDE COMPLETE INFORMATION

Reprints or other data relating to this deposit will aid the Stock Center and recipients of the strain.

Accession number

SPECIES *Neurospora crassa* 4433A

GENOTYPE *cpc-1 insertional translocation VI L → IR* 4434a

MATING TYPE *A/a* LINKAGE GROUP(S) _____

DESIGNATION OF MUTANT ALLELE(S) *j-5*

STRAIN DESIGNATION IF WILD-TYPE _____

YOUR STOCK NUMBER FOR THIS CULTURE *WT548-2-22A (SiL 442)*
WT548-2-11a (SiL 441)
include stock no. from other collections

ORIGIN OF STOCK *UV induced, SL*
for example - obtained from, genetic background, from cross with; or if collected from nature, collection point, substrate and collector.

PUBLISHED REFERENCES *Barthelmess, I. B 1982, Genet. Res. Camb. 39, 169-185*

Barthelmess, I. B. 1984, MGG 194, 318-321

Janet L. Paluh,* Michael Plamann,* Dirk Krüger,† Ilse B. Barthelmess,† Charles Yanofsky*,‡ and David D. Perkins* *1990*

Determination of the Inactivating Alterations in Two Mutant Alleles of the *Neurospora crassa* Cross-Pathway Control Gene *cpc-1*

Genetics 124: 599–606 (March, 1990)

COMMENTS (special growth requirements, aberrations, heterokaryon compatibility, special uses of strain, etc.)

insertional translocation

(use additional space below or on back of page if necessary)

YOUR NAME *Dr. Ilse Babette BARTHELMESS* DATE *6-9-1990*

Additional Comments: (use back of sheet if necessary)

Northern analyses with strains carrying the *cpc-1* (j-5) allele revealed that no *cpc-1* mRNA is produced. Southern and genetic analyses established that the *cpc-1* (j-5) mutation involved a chromosomal rearrangement in which a break occurred within the *cpc-1* locus, normally resident on linkage group VI; a small fragment from the left arm of linkage group VI, containing the *cpc-1* promoter region and *ylo-1*, was translocated to the right arm of linkage group I. Other studies indicate that the *cpc-1* locus itself is not essential for viability. Lethality previously attributed to the *cpc-1* (j-5) mutation is due instead to the production of progeny that are deficient for essential genes in an adjoining segment of linkage group VI. Molecular characterization of *cpc-1* (j-5) × *ylo-1 pan-2* duplication progeny indicated that *cpc-1* is normally transcribed towards the linkage group VI centromere.