

Neurospora chromosome rearrangements with mutant phenotypes provide an opportunity to sequence breakpoint junctions

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Present knowledge of junction sequences is inadequate for understanding how chromosome rearrangements originate. In *N. crassa*, cloned segments are known to cover breakpoints of *T(IR->VIR)UK-T12* (Asch et al. 1992 Genetics **130**:737-748), *T(VR;VIL)mpr15-2 am* (E.B. Cambareli and J. A. Kinsey, personal communication), *T(IR;IIR)4637 al-1* (Schmidhauser et al. 1990 Mol. Cell. Biol. **10**:5064-5070), *T(IR->VII;I;IV)AR173* (Kang and Metzenberg 1990 Mol. Cell Biol. **10**:5839-5848; S. D. Haedo, personal communication), *T(IR->VII)TM429 his-3* (Catcheside and Angel 1974 Aust. J. Biol. Sci. **27**:219-229; Legerton and Yanofsky 1985 Gene **39**:129-140, *T(VIL->IR)IBj5 cpc-1* (Paluh et al. 1990 Genetics **124**:599-606), *T(IR->VL)AR190* (Butler 1992 Genetics **131**:581-592), and *T(IIL->IIR)AR18* and *T(IIL->VI)P2869* (M. L. Smith and N. L. Glass, personal communication). However, nucleotide sequencing across junctions has been accomplished only for the first two.

Twenty-two chromosome rearrangements in *Neurospora* are associated with mutations at known gene loci (Table 1). A majority of the implicated loci have been cloned and the wild type alleles of a number of them have been sequenced. The way is thus open for determining numerous additional breakpoint junctions. Rearrangement strains are available from FGSC and are listed in the *Neurospora* Stock List, both in Part I (single mutants) and in Part VI (aberrations). Information on each rearrangement has been summarized for a forthcoming review, and I will be glad to provide copies on request. The Stock Center might well act as a clearing house to avert possible duplication of effort by anyone interested in sequencing junctions.

A caveat: For rearrangements that are placed in Table 1 solely on the basis of genetic linkage, the number of scored segregants is often not great and the possibility exists that a breakpoint is closely linked to the locus but is potentially separable by recombination. This applies to the *os-2* translocation and to most of the other rearrangements that are associated with genes having morphological or visible phenotypes. Separability is also possible though unlikely for *ad-3A*, *ad-3B*, *met-7*, *nic-2*, *ser-6*, and *thi-1*.

Table 1. Rearrangements associated with mutant phenotypes that are allelic with genes at established loci

Mutant locus	Rearrangement	References	
		Genetic	Molecular
ad-3A (IR)	T (IR<->IV) Y112M15 ad-3A	1	
	T (IR; IIR; IIR) Y155M64 ad-3A	2	
ad-3B (IR)	T (IR->IIR) Y112M4i ad-3B	1	
al-1 (IR)	T (IR; IIR) 4637 al-1	1	3
am (VR)	In (VR->VL) UK2-y am	4	5, 6
	T (VR; VIL) UK9-18 am	4	
	T (IIL; VR) mpr13-1 am	7	
	T (VR; VIL) mpr15-2 am	7	

arg-2 (IVR)	T(IL;IVR)MEP24	arg-2	9	10
arg-3 (IR)	T(IL;IVR;IVR;VR)MEP35	arg-3	9,2	11
arg-14 (IVR)	T(IVR->VIIL;IL;IIR;IVR)S1229	arg-14	1,8	12
aro-1 (IIR)	T(IIR;III)C161	aro-1	1	13
cpc-1 (VIL)	T(VIL->IR)IBj5	cpc-1	14	15
	T(IVR->VIL)MN9	cpc-1	16	
cut (IVL)	T(IL;IVL)HK53	cut	1	
eas (IIR)	T(IL;IIR)KH5-9	eas	17	18,19
his-3 (IR)	T(IR;VII)TM429	his-3	1	20
inl (VR)	T(VR;VIL)46802	inl	1	21
met-7 (VIIR)	T(I;VIIR)K79	met-7	1	22
nic-2 (IR)	T(IR<->VR)S1325	nic-2	1	
	T(IR->IIIR)4540	nic-2	1	
os-2 (IVR)	T(IVR;VI)V44o	os-2	2	
pho-4 (VII)	Ab(VII)RLM18	pho-4c	23	24
pho-5 (IVR)	T(IIIR;IVR)RLM02	pho-5c	23	25
	T(III;IVR)RLM04	pho-5c	23,2	
	T(I;IVR)RLM06	pho-5c	23,2	
	T(III;IVR)RLM08	pho-5c	23,2	
	T(IVR;VII)RLM09	pho-5c	23,2	
pk (VR)	T(VR;VII)17-088	pkD	26	
	T(IR;VR)C-1670	pk	1	
ser-6 (VIL)	T(VL;VIL)OY325	ser-6	2	
thi-1 (IR)	T(IR;VIIL)17084	thi-1	1	
wc-1 (VIIR)	T(II->VIIR)P73B159	wc-1	2	27

The normal-sequence wild type allele has been cloned for all loci except *ad-3A*, *ad-3B*, *cut*, *nic-2*, *os-2*, *pk*, *ser-6*, and *thi-1*.

References:

1. For description with primary references, see appendix in Perkins and Barry 1977 Adv. Genet. **19**:133-285.
2. Perkins, D. D., unpublished.
3. Schmidhauser et al. 1990 Mol. Cell. Biol. **10**:5064-5070.
4. Perkins et al. 1993 Genetics **134**:729-736.
5. Kinnaird and Fincham 1983 Gene **26**:253-260.
6. Kinsey and Rambosek 1984 Mol. Cell. Biol. **4**:117-122.
7. Cambareri, E. B., and J. A. Kinsey, personal communication.
8. Davis 1979 Genetics **93**:557-575.
9. Davis, R. H., personal communication.
10. Orbach et al. 1990 J. Biol. Chem. **265**:10981-10987.
11. Schmidhauser, T. J., personal communication.
12. Yu, Y. C., and R. L. Weiss, personal communication.
13. Catcheside et al. 1985 Mol. Gen. Genet. **199**:446-451.
14. Paluh et al. 1990 Genetics **124**:599-606.
15. Paluh et al. 1988 Proc. Nat. Acad. Sci. U.S.A. **85**:3728-3732.
16. Koch and Barthelmess 1988 Fungal Genet. Newsl. **35**:22-23.
17. Hasanuma 1984 Jpn. J. Genet. **59**:383-401.
18. Bell-Pederson et al. 1992 Genes Develop. **6**:2382-2394.
19. Lauter et al. 1992 Genes Develop. **6**:2373-2381.

20. Legerton and Yanofsky 1985 *Gene* **39**:129-140.
21. Akins and Lambowitz 1985 *Mol. Cell. Biol.* **5**:2272-2278.
22. Crawford et al. 1992 *Gene* **111**:265-266.
23. Versaw, W. K., and R. L. Metzenberg, personal communication.
24. Mann et al. 1988 *Mol. Cell. Biol.* **8**:1376-1379.
25. Versaw 1995 *Gene* **153**:135-139.
26. Srb, A. M., personal communication.
27. Ballario , P. and G. Macino, personal communication.