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

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Abstract

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. Cambareri 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->IIIR)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.

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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		References	
locus	Rearrangement	Genetic	Molecular
ad-3A (IR)	T(IR<->IV)Y112M15 ad-3A	1	
	T(IR;IIR;IIIR)Y155M64 ad-3A	2	
ad-3B (IR)	T(IR->IIIR)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*.

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