

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

David D. Perkins  
*Stanford University*

Follow this and additional works at: <http://newprairiepress.org/fgr>

---

### Recommended Citation

Perkins, D. D. (1995) "Neurospora chromosome rearrangements with mutant phenotypes provide an opportunity to sequence breakpoint junctions," *Fungal Genetics Reports*: Vol. 42, Article 18. <https://doi.org/10.4148/1941-4765.1348>

This Regular Paper is brought to you for free and open access by New Prairie Press. It has been accepted for inclusion in Fungal Genetics Reports by an authorized administrator of New Prairie Press. For more information, please contact [cads@k-state.edu](mailto:cads@k-state.edu).

---

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

## 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 Metzberg 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.

## Creative Commons License



This work is licensed under a [Creative Commons Attribution-Share Alike 4.0 License](https://creativecommons.org/licenses/by-sa/4.0/).

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

David D. Perkins - Department of Biological Sciences, Stanford University, Stanford CA 94305-5020

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 Metzberg 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.

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;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*.

## 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.