

Effect of ret-3 on hirt-5

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Effect of *ret-3* on *hirt-5*

Abstract

Effect of *ret-3* on *hirt-5*

Smith, B. R The effect of the
recombination-3 gene on histidine-5.

The recombination-3 gene described by D. G. Catcheside (1966 Austral. J. Biol. Sci. 19: 1039) controls the frequency of recombination between pairs of auxotrophic amination alleles in such a way that crosses bearing the dominant rec-3⁺ allele in one or both parents give frequencies of prototrophic recombinants that are around 15 times lower than those in crosses homozygous for the recessive rec-3 allele. The recombination-3 gene does not control recombination at the histidine-1 locus which is linked to amination on chromosome V or at the histidine-3 locus on chromosome I (Jha 1967 Genetics 57:365), indicating that its effect is locus specific. Since rec-3 is linked to mating type in linkage group I, its effect on the histidine-5 gene in linkage group IV could be easily tested.

The tests measured recombination between the his-5 alleles K553 and K512. The K553 a; rec-3 stock isolated from the wild type Em a; rec-3 was crossed to each of five K512 A stocks isolated from a cross of K512 a of unknown ret-3 constitution with the wild type Em A; rec-3⁺. Frequencies of prototrophic recombinants arising in the progeny of these five crosses ranged from 7.4 to 11.7 per 10⁵ ascospores. Since ret-3 is only 12 map units from mating type, the probability that at least one of the crosses bears the dominant rec-3⁺ allele is 0.999 or unity if the K512 a stock is rec-3⁺. Five isolates of K512 of mating type A were isolated from a cross of a with a rec-3 stock cot-1 (C102); am (47305), isolate no. 3675 supplied by D. G. Catcheside. Each of these five isolates was crossed to the K553 a; ret-3 stock and the frequency of histidine prototrophs in the progeny was determined. Frequencies ranged from 8.4 to 12.6 per 10⁵ ascospores. The probability that at least one of the five crosses was homozygous for rec-3 is 0.999. It may be confidently assumed therefore that recombination-3 differences do not control recombination between K553 and K512, or if they do then the effect is only very slight.

Since ret-3 controls recombination frequency between all pairs of amination alleles tested, the absence of any detectable control of recombination between the his-5 auxotrophs K553 and K512 adds considerable weight to the supposition that control by rec-3 is locus specific. Further tests will be needed to determine whether rec-3 controls recombination at loci other than amination. ■ ■ ■ Department of Genetics, University of Leeds, Leeds 2, England.