

Endothia parasitica (Murr.) And. (Sphaeriales, Diaporthaceae) is the pathogen which causes chestnut tree blight. The American chestnut (*Castanea dentata*) was almost completely wiped out when this fungus was introduced into this country at the turn of the century, presumably from the Orient. Besides being a classic among plant pathogens, the organism has recently provided an example of a new kind of biological control system strains of the fungus which contain double-stranded RNA, the genetic material of most fungal viruses, do not have normal pathogenicity and can control disease-causing strains in the host (Day et al., 1977 *Phytopath.* 67:1393). E. parasitica? is grown commercially as a source of renin for cheese making (Sardinas 1968 *App. Microbiol.* 16:248).

Growth conditions have been studied and mutants obtained (Puhalls and Anagnostakis 1971 *Phytopath.* 61:169), a vegetative compatibility system has been reported (Anagnostakis 1977 *Exp. Mycol.* 1:306), and a technique detailed for making controlled crosses in the laboratory (Anagnostakis 1979 *Mycol.* 71:213).

Sexual incompatibility is homogenic. So far, 108 strains have been crossed with two mating type testers and only one mating type locus with two alleles has been found. Some strains function only as males, notably the auxotrophs. Occasionally evidence for homothallism is seen, such as: 1) perithecia produced by a strain cultured alone (two Italian isolates), and 2) ascospore progeny from a single perithecium that fail to segregate a given marker while progeny from other perithecia in the same cross are segregating.

Vegetative incompatibility is heterogenic. The mating type gene does not function as a vegetative compatibility (v-c) gene in vegetative interactions as it does in *Neurospora crassa*. So far 77 different v-c groups have been found. Strains in a given group are vegetatively compatible with each other but not with strains in other groups. If all loci have only two alleles and interactions are allelic (not occurring between alleles at two different loci), then at least 7 loci would be needed to yield 77 different v-c groups (128 v-c groups maximum). Some crosses have been made to define the genes responsible, and results are shown in Table 1.

TABLE 1.

Evidence for genetic control of vegetative incompatibility

V-C types crossed	random ascospore progeny	v-c types resulting	probable number of genes segregating
5x 8	213	5,8,39,71	2
5 x 39	76	5,39	1
10 x 17	197	10,17	1
5 x 10	90	5,10,9,24,36,40,42,49,68, and nine others	at least 5
5 x 17	37	5,17,3,6,27,30,36,43,55,56,60, and four others	at least 4
8 x 17	262	8,17, and twenty others	at least 5

Even though these data represent rather small samples, the following tentative genotypes might be used as a working model:

Y-C 5	B C D E F G H	V-c 71	B c D E F G H	v-c 17	B C d e f g H
Y-C 39	b C D E F G H	v-c 8	b c D E F G H	v-c 10	B C d e f g h

TABLE 2.

Available mutants characterized

designation	ATCC#	description	origin	V-c type	mating type	pathogenicity
col-1	22509	highly branched, thick restricted growth, recessive	UV	8	a	
col-2	22510	growth more restricted than <u>col-1</u> , recessive	UV	8	a	
cre-1	38981	light cream colored conidia and mycelium recessive	UV	5	A	(+)
arg-1	22506	arginine requiring, responds to citrulline, recessive	UV	8	a	
met-1	22508	methionine requiring, does not respond to homocysteine, recessive	UV	8	a	+
br-1		brown mycelium and conidia, W MacDonald field isolate, (W.Va.)	S			+
ts-1	38982	grows normally at 20°C, and not at all at 35°C, nutritionally irreparable but osmotically repairable, dominant?	UV	5	A	(+)

The double mutant we-1, ts-1 (ATCC #22507) is pathogenic.

So far no linkage has been found between any of these mutants and any of the v-c genes examined

The importance of this fungus in plant pathology and industry makes it a good choice for general genetic studies. I hope that the information reported here will encourage more people to work with the organism