RESISTANCE OF TWENTY-ONE SWEETGUM FAMILIES TO BOTRYOSPHAERIA RIBIS

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ABSTRACT

Botryosphaeria ribis caused cankers and branch mortality in all sweetgum (Liquidambar) families inoculated in an open-pollinated progeny test. The highest infection occurred in the 21 sweetgum families inoculated in September (78%), followed by November (54%), and May (43%). There was no significant difference among sweetgum families. Some families were more susceptible than others at certain dates.

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Botryosphaeria ribis Gross & Dug. was first isolated from Liquidambar spp. in Georgia and Florida (2). In inoculation studies, Pirone (1) and Toole (6) found that the fungus causes cankers on sweetgum (Liquidambar styraciflua).

The fungus has a wide geographic distribution and reportedly attacks many hosts, including pecan, walnut, willow, elm, and oak (3, 4, 5, 7). Top dieback resulting from stem cankers caused by B. ribis infection was observed at Stoneville, Mississippi, in an 81-family sweetgum open-pollinated progeny test planting arranged in a 9 x 9 balanced lattice design.

This study evaluated the inherent resistance of 21 sweetgum families to B. ribis.

MATERIALS AND METHODS

Twenty-one families from the sweetgum open-pollinated progeny test were selected for evaluation: seven with high incidence of natural infection (48 to 74%), seven with intermediate (29 to 31%), and seven with low incidence of attack (7 to 17%).

The 8-year-old trees were inoculated bimonthly in November 1970 through September 1971 with B. ribis. On each date 5 trees for each of 4 replications per family were inoculated. Six branches in the same tree were used as inoculation points. Various colored paint was used to distinguish bimonthly inoculations. The same number of families and replications were used for control trees. Trees were inoculated by cutting a 1.27- by 2.54-cm bark flap and placing a 9-mm mycelium agar disc beneath the bark. Sterile agar discs were placed in branches of trees serving as controls. All wounds were covered with masking tape.

To determine percent of infection, the inoculation points on all trees were examined and data were tabulated 3 months after the final inoculation. Analysis of variance and Duncan's Multiple Range Test were used to reveal any significant differences among families and dates.

Table 1. Percent of cankers developed on sweetgum trees representing 21 families inoculated bimonthly with Botryosphaeria ribis.

Month of inoculation	Mean (%)
September	78 a[a]
November	54 b
May	43 c
January	21 d
March	16 d
July	6 e

[a]Different letters denote significant difference (0.05 level).

Table 2. Disease incidence in sweetgum families from natural infection and inoculation with Botryosphaeria ribis at different dates.

Family number	Natural infection	Inoculation dates: Nov.	Jan.	March	May	July	Sept.
		% Infection					
1	74	37	15	0	20	0	92
2	72	79	16	24	26	0	84
3	66	39	15	15	20	0	85
4	58	44	21	11	56	8	75
5	53	40	20	11	29	11	60
6	48	64	51	34	44	0	81
7	48	69	44	15	34	0	66
8	30	70	33	0	29	8	66
9	30	44	19	36	39	8	70
10	30	50	20	15	49	8	80
11	31	66	19	29	55	0	80
12	31	29	11	19	55	8	92
13	29	29	15	15	44	0	70
14	29	34	20	19	24	0	71
15	7	79	30	10	72	0	81
16	10	50	16	11	35	0	76
17	14	50	24	11	67	0	76
18	14	55	12	17	34	9	80
19	16	71	15	20	80	0	71
20	17	60	11	11	41	0	71
21	17	70	30	25	51	0	91

RESULTS

Typical canker symptoms developed on susceptible trees 90 days after being inoculated. Some necrotic tissues were evident at inoculation point 30 days after inoculation. Botryosphaeria ribis was reisolated from all developing cankers.

More sweetgum trees, 78%, developed cankers when inoculated in September than in any other month (Table 1). Trees inoculated in November were second with 54% and May-inoculated trees third with 43%. Some infection occurred as the result of inoculations at all dates tested. Differences between January and March were not significant. Least infection occurred in trees inoculated in July.

There was no significant difference in canker development within sweetgum families. After being inoculated, families showing low natural disease incidence developed cankers as frequently as families showing high natural disease incidence (Table 2). Inoculation beneath a bark flap appears to negate natural resistance. Apparently, resistance is related only to the bark barrier.

Literature Cited

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