Infection of Peach Buds by *Botryosphaeria obtusa*

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ABSTRACT

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Three species of *Botryosphaeria* that cause peach tree fungal gummosis, *B. obtusa*, *B. dothidea*, and *B. rhodina*, were isolated from dormant peach buds in central Georgia. Only *B. obtusa* was present in buds in significant numbers, with populations peaking in January and February. Inoculations showed that both *B. obtusa* and *B. rhodina* infect and kill buds before bloom, but *B. dothidea* does not. Incidence of *B. obtusa* was greater in symptomless buds than in dead buds, indicating that infected buds often remain symptomless. Protective fungicide applied to an orchard early in January each year since planting reduced incidence of *B. obtusa* in buds and in subtending branches by 50% in the third year after planting, but there was no reduction in incidence in subtending twigs or in disease severity the fourth year after planting. Systematic isolations indicated that in mature, severely infected trees systemic growth of the fungi progressed as frequently from older infected wood into budwood as from infected floral organs into subtending twigs. A survey of twig internodes indicated that 25% of the budwood in such trees was infected in the fall of 1984. This aggressive systemic invasion may render larger but less numerous infection sites, such as wounds, more important than buds to disease development.

Additional keywords: *Prunus persica*

Fungal gummosis of peach (*Prunus persicae* L.) is a canker disease caused by three species of *Botryosphaeria* (1). *Botryosphaeria* spp. infect many deciduous hosts, usually through wounds (6,7,9). Natural openings such as growth cracks (4) and lenticels (2,10) also serve as infection sites. On apple, *B. obtusa* (Schwein.) Shoem. infects through opening buds as early as the silver-tip stage of bud phenology (8). These infections remain latent until June, when immature fruit develop a rot that progresses outward from the core, and then they abscise. Similar fruit drop has been recently observed in peaches, and *B. obtusa* has been isolated from the abscising fruit (5). Field surveys and inoculations were undertaken to determine whether buds serve as an infection site for *Botryosphaeria* on peach. Fungicide applications were tested for control potential.

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MATERIALS AND METHODS

Surveys. Buds were taken randomly from each of four mature, infected orchards near Fort Valley, GA, at biweekly intervals from August 1979 until bloom in March 1980. The same orchards were sampled on 9 March 1983, and biweekly from October 1983 through March 1984. On each sample date, 25 symptomless flower buds were surface-sterilized in 0.525% sodium hypochlorite-10% ethanol and plated on acidified potato-dextrose agar (APDA). Twenty-five nonsterilized buds also were plated. Plates were incubated under fluorescent lights (45 $\mu E \cdot cm^{-2} \cdot s^{-1}$) for 2 wk at 24 C.

Because the 1979–1980 survey indicated a decline in bud infestation before bloom, a full-bloom sample was taken to determine the impact of fungal incidence on flowering. Three orchards in central Georgia were randomly sampled on 7 March 1982; 25 live and 25 dead blooms from each orchard were surface-sterilized and plated on APDA.

Twigs bearing fertilized fruitlets were sampled in a mature, severely infected orchard on six dates, from 30 March through 30 June 1983 and from 20 March through 7 May 1984, to observe the progression of infection. Twenty-five nodes were removed on each date from each of four six-tree plots. Remnants of bud scales, fertilized fruitlets, pedicels, and 1 cm of twig subtending each node were surface-sterilized and plated. Individual nodes were numbered to determine whether all infected twigs subtended infected floral organs.

From May 1983 through April 1985, internodes of 1- and 2-yr-old twigs were sampled in each of two mature, infected orchards. Two additional orchards were surveyed the second year. Three 1-cm segments were cut from internodes of 1- and 2-yr-old growth on each of 25 symptomless twigs per orchard. The incidence of *Botryosphaeria* was determined by plating on APDA.

Inoculations. Conidial suspensions of all three species (100,000 spores/ml) were atomized on the exterior of buds of 90-cm-tall, container-grown trees (cultivar Loring) in October 1981. Ten trees were inoculated per fungal species. Ten trees atomized with sterile water served as controls. Trees remained outdoors so bud development could proceed normally. Dead and live buds were counted at bloom and the percent of dead buds on each tree was determined.

Fungicide applications. The protective fungicide captafol (2.14 L/ha) was applied in 1983 and 1984 to a 10-yr-old orchard with severe gummosis, in a random split-block design with six trees per plot and four replications. Four different spray dates, each 1 wk after the prior application date, were used beginning 23 January. Buds were sampled before bloom and 25 surface-sterilized and 25 nonsterilized buds were plated on APDA. Nodes bearing developing fruitlets were dissected and plated on five dates each year, as previously described, to determine the effect of this spray on bud infections, and to determine the progress of infection.

Each January from 1984 through 1987 captafol was applied in an orchard planted in January 1984 at Reynolds, GA, using a random complete block design with five replications and three rows of 10 trees for each plot. In the second, third, and fourth years, the incidence of *Botryosphaeria* in buds was assayed (22 February 1985, 15 February 1986, and 25 February 1987) by plating 50 buds from the trees in the center row of each plot. Twenty-five buds were surface-sterilized and 25 were nonsterilized. A random sample of three 1-cm internode segments from each of 25 twigs subtending buds were plated from each plot in late May 1986 and 1987.

In August 1987, the number of cankers per tree and the number of gummy spots on the trunk were counted for each tree in the center row of each plot (n = 10).

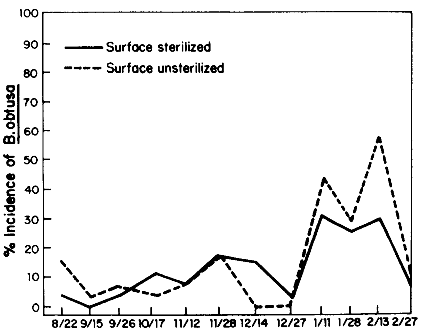

Fig. 1. Incidence of *Botryosphaeria obtusa* isolated from surface-sterilized and nonsterilized symptomless peach buds in central Georgia (1979–1980). Values are means from 25 buds from each of four orchards.

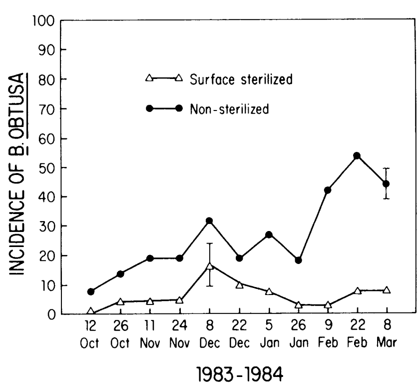

Fig. 2. Incidence of *Botryosphaeria obtusa* isolated from surface-sterilized and nonsterilized symptomless peach buds in central Georgia (1983–1984). Values are means from 25 buds from each of four orchards. Bars indicate LSD for differences due to date at P= 0.05.

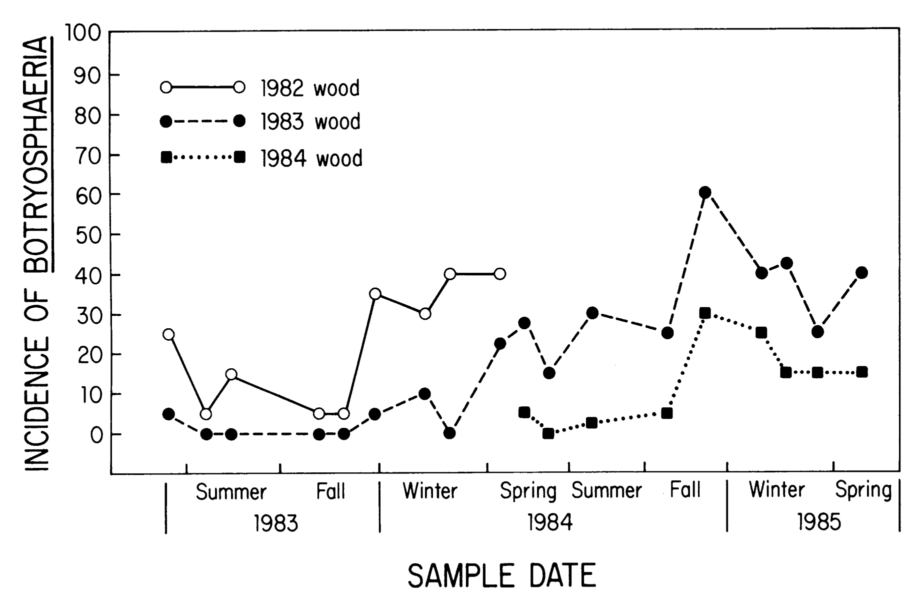

Fig. 3. Incidence of *Botryosphaeria* spp. isolated from internodes of symptomless twigs. Values are means from 25 twigs from each of two orchards in 1983 and 1984 and four orchards in 1984 and 1985.

RESULTS

Surveys. The 1979–1980 survey of peach buds near Fort Valley indicated *B. obtusa* infestation occurred throughout dormancy, with low incidence from August through December, followed by a rapid buildup in early January in both nonsterilized (44%) and surface-sterilized buds (32%) (Fig. 1). This same trend was observed in each of the four orchards surveyed. As buds began to swell in late February, incidence of *B. obtusa* declined. A maximum of 1.2% of these buds contained *B. dothidea* (Moug. ex Fr.) Ces. & de Not., and no *B. rhodina* (Berk. & Curt.) Arx was isolated. *Cytospora* spp. were isolated from less than 1% of the buds. On 9 March 1983, the mean incidence of *B. obtusa* in the four orchards was 24% in surface-sterilized buds and 54% in nonsterilized buds. In 1984, isolations from dormant buds indicated very little infection because only 8% of surface-sterilized buds contained *B. obtusa*, despite 50% incidence of *B. obtusa* in nonsterilized buds (Fig. 2).

When buds in three central Georgia locations were surveyed before bloom on 7 March 1982, 6% bud mortality had occurred. Ten percent of the surface-sterilized dead buds contained *B. obtusa*, and approximately 2% contained the other two species. Of symptomless buds sampled on the same date, 17% of the surface-sterilized, and 45% of the nonsterilized buds contained *B. obtusa*.

Isolations in 1983 from nodes bearing developing fruitlets in the 10-yr-old orchard showed 23% of the twigs were infected on 30 March. A significant increase in incidence occurred in June (45% infected). Remnants of bud scales were 18% infected on 30 March, and increased to 57% infected by 27 April. After this date, all bud scales were dropped from the developing fruits. Fruits and the subtending pedicels averaged 3.3 and 14.6% infected, respectively, with no significant changes from March through June.

Isolation data for individual nodes were analyzed by creating discrete variables to indicate various degrees of progressive infection (i.e., if one floral part was infected and the subtending twig was not, then $I = A$. If one floral part and the pedicel were both infected and the subtending twig was not infected, then $I = D$. If no floral parts were infected, but the subtending twig was infected, then $I = U$.) The frequency of each value for I was then compared for each sample date. There were no differences in the frequency of each value for I due to date, nor were there significant differences between the frequency of A or D (indicating downward progress of infection from floral organs to twigs) and U (indicating upward progress of infection from twigs to floral parts).

Isolations of dissected nodes and floral organs in 1984 showed significantly higher incidence of *B. obtusa* in developing fruitlets in samples taken 2 April (12%) and 9 April (7%) than in fruitlets sampled 20 March, 16 April, or 7 May (average 3%). No differences in incidence due to date were significant in samples of twigs (average 9.5%), bud scales (11.6%), calyx cup (2.7%), or anthers (1.2%). There were again no significant differences in the frequency of the variables created to identify downward or upward progress of infection in individual nodes.

Internodes sampled from mature, severely infected trees from May 1983 through April 1985 indicated that 25% of symptomless twigs produced in 1982 were infected by May 1983. There was a large increase in *Botryosphaeria* incidence in these second-year twigs in the fall of 1983, and 40% became infected by April 1984. The twigs produced in 1983 were also 25% infected by the following spring and 40% infected 1 yr later. In the fall of 1984, incidence of *Botryosphaeria* increased in both first- and second-year twigs (Fig. 3).

Inoculations. Bud inoculations demonstrated that both *B. obtusa* and *B. rhodina* significantly increased bud mortality (mean 20.5 and 32.6%, respectively). These treatments did not differ significantly. In the control trees, the incidence of dead buds was 6.0%. Of the buds inoculated with *B. dothidea*, 12.5% died during the winter. This did not differ significantly from the control at $P = 0.05$.

Fungicide applications. In the mature orchard, bud and node isolations indicated captafol did not prevent bud infections. However, if data for each positive isolation from all floral organs for a given node were added together, significant differences between sprayed and unsprayed trees occurred, because these data magnified treatment effects when more than one floral organ per node was infected. This information, coupled with data indicating that twigs subtending buds were already 25% infected in mature orchards, caused us to move the bud spray test to a new orchard, to avoid the confounding influence of systemic infections.

Table 1. Incidence of *Botryosphaeria obtusa* in buds and twig internodes and disease severity on sprayed and unsprayed trees at Reynolds, GA

Tissue sampled	Incidence of *B. obtusa*: No fungicide	Captafol	P^a
1986			
Surface-sterilized buds	41	17	0.034
Nonsterilized buds	54	32	0.005
Branch internodes	61	34	0.006
1987			
Surface-sterilized buds	24	10	0.004
Nonsterilized buds	70	30	0.0001
Branch internodes	7	15	0.101
Cankers per tree	10	11	0.772
Gummy spots per trunk	8	8	0.719

[a] Probability that difference between sprayed and unsprayed trees is not significant ($P > F$ under H_0).

In the Reynolds, GA, orchard that was planted in 1984 and sprayed each January from 1984 through 1987, there was very low incidence of *B. obtusa* in 1985. In 1986, incidence increased to 54% in nonsterilized buds (Table 1). Captafol applications reduced *B. obtusa* incidence in both buds and in subtending twig nodes by approximately 50% in 1986. In 1987, the incidence of *B. obtusa* in buds was reduced 50% by fungicide application, but there was no reduction in the incidence of *Botryosphaeria* in subtending branches. There was no significant difference in the number of cankers per tree or the number of seeping spots per trunk of sprayed versus unsprayed trees in August 1987.

DISCUSSION

Field surveys indicated that *B. obtusa* commonly infests peach buds in Georgia in January and February. Bud infection occurs before bloom and, although buds may be killed, they often remain symptomless. These infections may remain latent or may progress into the twig or into the fruit giving rise to latent infections that cause immature fruit to abscise in June (5).

Although *B. rhodina* also increased bud mortality significantly in the inoculation test, trees in the field apparently escape bud infection by this species because *B. rhodina* conidia were not isolated from buds.

Internode surveys of severely infected orchards in the fall of 1983 indicated a greater increase in *Botryosphaeria* incidence in 2-yr-old twigs, which bore few buds, than in 1-yr-old twigs on which most of the buds are borne. Infection of bud-bearing twigs lagged approximately 3 mo behind increased infection in older twigs. In the fall of 1984, the rate of increase in incidence in 2-yr-old twigs equalled that of 1-yr-old twigs. Therefore, in severely infected orchards, systemic growth of *B. obtusa* from established infections caused at least as many twig infections as were due to bud infections. Furthermore, comparison of variables created from dissection data showed that there was as much upward progress of infection (from twigs) as downward progress (from floral organs). This systemic infection may explain the lack of significant differences in the mature, severely infected orchard on the incidence of *B. obtusa* in twigs of sprayed and unsprayed trees.

We relocated bud-swell spray tests to a newly planted orchard, because if healthy trees could be protected from bud infections each year the impact of these infections on the incidence of *B. obtusa* in the branches and on symptom severity could be determined. In the first 2 yr there was very little *Botryosphaeria* present in the new orchard. In the third year (1986), incidence increased and captafol application reduced bud infections and subsequent twig infections 50%. In the fourth year (1987), bud infections were again reduced 50%, but the population of *Botryosphaeria* in the twigs was not affected by captafol application. The trees, which are unusually vigorous, did not express symptoms until 1987. Canker counts indicated that annual captafol application in January did not reduce gummosis severity. It is possible that latent systemic growth of the fungus from pruning wounds and other infection sites had already invaded the bud-bearing twigs, rendering bud infection control ineffectual in reducing the incidence of *Botryosphaeria* in twigs.

The economic importance of bud infections lies in the formation of cankers, rather than in the loss of buds. Cankers slowly expand and may girdle branches, eventually killing fruiting wood. Unless they are pruned out, they will also result in further increases in inoculum, because the pathogens sporulate as long as the bark remains on the wood (3). Whether these factors justify the use of protective fungicides at bud swell depends upon the severity of gummosis in the orchard and whether infection through other infection sites, such as pruning wounds, can be prevented.

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