

Bacterial Biocontrol of White Mold Disease (Sclerotinia sclerotiorum)**G. Y. Yuen, E. D. Kerr, J. R. Steadman, and M. L. Craig****Department of Plant Pathology
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Some strains of blossom-resident bacteria were reported to be antagonistic to Sclerotinia sclerotiorum in vitro, and when applied to bean blossoms under greenhouse conditions, produced antibiotics that suppressed infection by ascospores of the pathogen and development of white mold (Report of the Bean Improvement Cooperative 33:47, 1990.). In an initial field experiment, effective control of white mold by Erwinia herbicola strain B1 was associated with the ability of the bacterium to survive on leaves and to multiply on developing blossoms. However, the biocontrol proved to be ineffective in later field experiments, despite the bacterium colonizing bean blossoms to mean populations of log 4 to log 6 colony forming units (cfu) per blossom in all experiments.

In laboratory investigations on the process of antagonism by strain B1, it was found that the strain B1 multiplied rapidly to approx. 10^6 cfu/blossom in 12 to 16 hr. when applied at an initial population of 100 cfu/blossom. When ascospores of S. sclerotiorum were added to blossoms at various time periods after the application of B1, germination of the ascospores was not inhibited unless B1 had been incubated on the blossoms for 24 hr. prior to inoculation with the fungus. At 24 hr., the bacterium was in stationary growth phase. Similarly, when blossoms treated with B1 and ascospores were placed on bean leaves, the bacterium inhibited disease development only after a 24-hr. preincubation period (Fig. 1). These results suggest explanations for the lack of efficacy of B1 in the field. First, the high population level of B1 (10^6 cfu/blossom) required for effective inhibition of the pathogen is much higher than normally found in the field after application of B1. Second, antagonism is related to production of second metabolites during stationary phase. In the field, numbers of blossoms with B1 reaching this growth stage may be low. Stationary phase also may be achieved too late relative to the deposition and germination of ascospores.

An in planta screening of more than 300 strains of bacteria isolated from bean blossoms was conducted to identify those that could inhibit S. sclerotiorum at low populations without the necessity of multiplying to stationary phase. In this procedure, bacterial isolates were incubated on blossoms for only 6 hr. and then ascospores were applied to the bacteria-treated blossoms. Population levels of the bacteria, germination of ascospores, and development of white mold lesions from blossoms placed on bean leaves were examined. The experiments resulted in Erwinia herbicola strains that arrested white mold development at approximately 10^5 cfu/blossom (Fig. 2), when the bacteria were in early exponential growth phase. Inhibition of white mold at low bacterial populations was related to inhibition of ascospore germination by some strains (eg. strain B346 in Fig. 2).

These strains were tested on dry bean in the field in 1991 and found to survive on leaves and to colonize blossoms equally well in comparison to strain B1. However, the experiments were inconclusive due to a high degree of variability in disease levels in the plots, and thus, efficacy against white mold in the field remains to be confirmed.

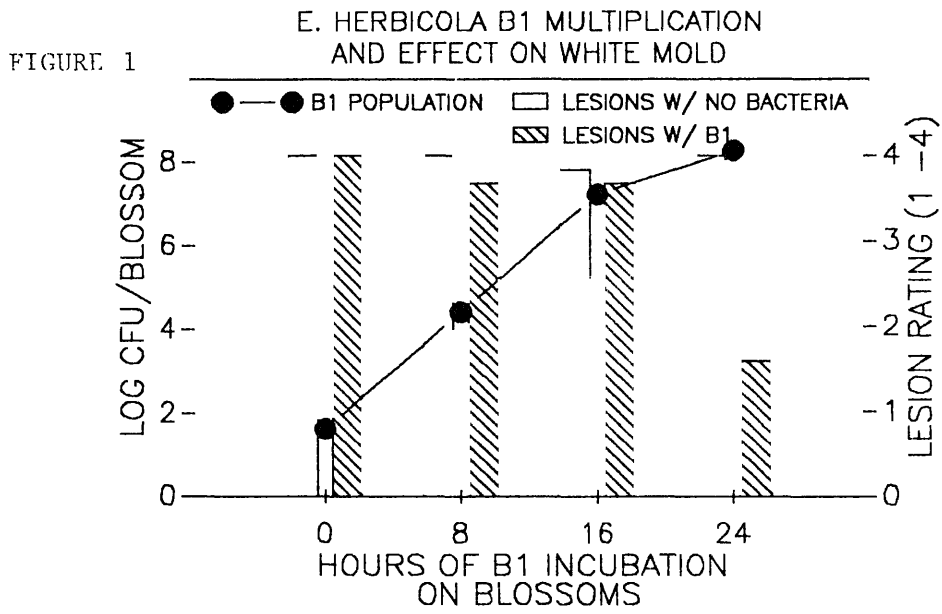


FIGURE 2

