Abstract

Spread of strawberry anthracnose, resulting from the rain splash dispersal of *Colletotrichum acutatum* conidia, was determined in field plots by assessing fruit disease incidence at a range of distances from an introduced point source of infected fruit with sporulating lesions. Four within-row plant densities were established in replicated plots in each of 2 years. A generalized linear model with a logit link function and binomial distribution for incidence was used to quantify the effects of distance and side of the row relative to the inoculum source, plant density treatment, and their interactions on disease incidence. At all assessment times, there was a significant ($P <= 0.05$) decline in incidence with increasing distance from the spore source. Moreover, row side had a significant effect, with the near side having higher incidence than the far side. Plant density treatment had a significant, but nonlinear, effect on incidence, with incidence generally declining with increasing density. Side of the row relative to the inoculum source and density treatment could affect the steepness of the disease gradient (slope) as well as the overall level of disease incidence, depending on the assessment time and year. The combined effects of plant density and row side on the height and steepness of the disease gradients could be measured using the predicted distance in which incidence equals 10% ($d(10)$). Estimated $d(10)$ generally increased in a nonlinear manner with decreasing plant density. Also, plant density had a significant negative effect on the proportion of incident rain that penetrated the canopy. In a separate study, plant density did not consistently affect infection of fruit that had been placed within the canopy immediately after being inoculated in the laboratory with a controlled inoculum density, indicating that conditions favoring infection were similar for the four densities. Thus, differences in mean disease incidence and disease gradients among the treatments were mostly due to differences in dispersal and not to other components of the disease cycle. As previously reported for controlled studies using a rain simulator, however, the effects of plant density on dispersal were complex, and increasing density did not universally lead to decreasing disease incidence.