Growth of Homologous and Heterologous Phytopathogenic Bacteria in Cotton Lines Susceptible, Resistant, or Immune to Blight

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ABSTRACT

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In vivo growth studies of homologous (*Xanthomonas campestris* pv. *malvacearum* races 1, 3, 7, and 18) and heterologous (*X. campestris* pv. *campestris*, *X. campestris* pv. *phaseoli*, and *Pseudomonas syringae* pv. *pisi*) phytopathogenic bacterial populations were made to determine growth trends in blight-susceptible (line Ac 44), blight-resistant (lines Ac B_2, Ac B_3, Ac b_7, OK 1.2, and OK 2.3), and blight-immune (line Im 216) cotton (*Gossypium hirsutum*). Relative population trends for homologous pathogen-cotton line combinations were similar, in general, to those found in other host-pathogen systems. Populations of heterologous pathogens in Ac 44 declined rapidly after reaching peak levels considerably lower than the peak levels of homologous pathogens. In contrast to results reported in previous studies, heterologous populations in the most resistant host, Im 216, were similar to homologous populations in the same line. This suggests that a nonspecific resistance mechanism operates in Im 216 which inhibits growth of all of these phytopathogenic bacteria. Population trends in cotton lines with intermediate levels of homozygous resistance resulted in final populations intermediate between those of Ac 44 and Im 216.

Additional key words: bacterial blight of cotton, hypersensitive response.

Bacterial blight [causal organism: *Xanthomonas campestris* pv. *malvacearum* (Smith) Dye (abbreviation *Xcm*) (formerly *X. malvacearum* (E. F. Sm.) Dows.)] of upland cotton (*Gossypium hirsutum* L.) is an economically important disease in most cotton-growing areas of the world (7). Since 1953, the annual crop loss from bacterial blight in the United States has ranged from 0.4 to 3.4% (39). Losses in many other cotton-growing areas, particularly Africa, are much higher (7,31). The pathogen can also predispose the plant, particularly the bolls, to subsequent infection by other bacteria and fungi. Diseased bolls often do not mature properly and can produce stained lint.

Sanitation (8), quarantine (35), and seed treatment (24) have been successfully used as control measures; however, the use of blight-resistant cultivars is generally considered the most effective and economical method of control (4,6,9,40).

In vivo studies of bacterial populations in homologous and/or heterologous host-pathogen systems have exhibited definite trends (1,14–16,19,21–23,25,27,30,32–34,36,38,41). Population trends for homologous and heterologous pathogens were generally similar for the first 24–48 hr after which differences were observed. Multiplication of the heterologous pathogens generally ceased rather abruptly about 48 hr after inoculation; populations then either remained at about the same level or decreased for the duration of the experiment. In contrast, homologous pathogens continued to multiply for several more days before leveling off. Peak populations and final populations were higher for homologous pathogens than for heterologous pathogens.

Ultrastructural and biochemical differences have been observed among cotton lines inoculated with homologous pathogens (2–4,19; A. H. Al-Mousawi, P. E. Richardson, M. Essenberg, and W. M. Johnson, *unpublished*). In addition, the previous, somewhat limited studies by Johnson et al (25) and Perry (34) suggest that differences exist among population trends of *Xcm* inoculated into blight-resistant and -susceptible cotton lines. However, none of these investigators examined heterologous population trends in inoculated cotton lines. Therefore, the objective of this study was to quantify and compare population trends of *Xcm* and several other phytopathogenic bacteria which do not attack cotton in blight-susceptible, -resistant, and -immune cotton lines.

MATERIALS AND METHODS

Cotton lines. Seven cotton lines (designated as Ac 44, Ac B_2, Ac B_3, Ac b_7, OK 1.2, OK 2.3, and Im 216) were used in this study. Acala 44 (Ac 44) is a blight-susceptible line with no known major genes for resistance. Ac B_2, Ac B_3, and Ac b_7 are near-isogenic lines which were derived from a common Ac 44 background and possess homozygous single-gene blight resistance (7). These three genes (B_2, B_3, and b_7, respectively) have been tentatively identified as the major genes that collectively confer blight immunity to Immune 216 (Im 216). Cotton lines OK 1.2 and OK 2.3 are homozygous resistant lines selected from segregating generations of a cross between Ac 44 and Im 216. Blight resistance in OK 1.2 is probably due to the genes B_3 and b_7, and the resistance in OK 2.3 is probably due to the gene B_2 (W. M. Johnson, M. B. Bayles, and L. M. Verhalen, *unpublished*). Im 216 is a bacterial blight-immune line with at least two dominant and possibly one recessive gene for resistance (11). It is homozygous for the genes that determine immunity. We define immunity to bacterial blight of cotton as a response to all 18 known races (individually or combined) of *Xcm*

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which exhibits no macroscopic symptoms under field levels of inoculum (6,7,13). A discussion of the development of immunity in cotton to bacterial blight can be found in Brinkerhoff et al (12).

Acid-delinted seed of each line were planted in clay pots containing a commercial mixture of peat moss and vermiculite (Jiffy Mix Plus; Jiffy Products of America, West Chicago, IL). Seedlings were initially grown in a greenhouse and were 2–3 wk old at the time of inoculation. Inoculated plants then were transferred to a growth chamber and maintained under a 12-hr 26 C day and a 12-hr 19 C night regime. These temperatures have been shown to favor bacterial blight development (10).

Bacteria. *Xcm* (races 1, 3, 7, and 18) was used in this study as the homologous (natural) pathogen (17). These races elicit different levels of field resistance in the previously described cotton lines (M. B. Bayles, W. M. Johnson, and L. M. Verhalen, *unpublished*). Race identifications were made by L. S. Bird (Texas A&M University) prior to our receiving the cultures from him for these experiments. In addition, three other bacterial phytopathogens that are not pathogens of cotton served as the heterologous combinations (17). *X. campestris* pv. *campestris* (*Xcc*) is a pathogen of cabbage, cauliflower, and related cruciferous species. *X. campestris* pv. *phaseoli* (*Xcp*) is a pathogen of beans. *Pseudomonas syringae* pv. *pisi* (*Psp*) is a pathogen of field and garden peas. All cultures were maintained at 24 C on potato-carrot-dextrose agar (PCDA) tube slants (5), but without yeast extract. Before they were sent to us, these isolates were tested for virulence on their respective hosts by A. Novacky (University of Missouri) and R. S. Dickey (Cornell University).

Method of inoculation. Inocula were prepared by transferring bacteria in a 3-mm-diameter loop to 80 ml of Difco nutrient broth. Cultures were incubated on a reciprocal shaker for 10–12 hr at 24 C. Initial concentrations of inocula were adjusted by using a spectrophotometer and were verified by serial dilution plate counts on Difco nutrient agar. Cotyledons were inoculated by using a modification of the leaf infiltration technique developed by Klement (26). A 0.64-mm-diameter (22-gauge) hypodermic needle was used to lightly scratch the cuticle and epidermis of the abaxial surface near the base of the center two panels of each cotyledon; this procedure permitted efficient watersoaking with minimal structural damage. The tip of a 3-cc disposable plastic syringe containing inoculum was placed over the scratched area, and inoculum was injected into the tissue; complete watersoaking of each cotyledon panel was easily achieved.

Measurement of bacterial populations. Isolations were made from inoculated leaf tissue on the day of inoculation (0 days) and on days 1, 2, 3, 4, 7, and 9 (unless otherwise indicated). Bacterial populations were calculated from serial dilution plate counts. Two separate sets of serial dilutions were made from each cotyledon, and each dilution was plated in triplicate. Cotyledons were surface disinfested by washing them in a 0.5% sodium hypochlorite solution for 8–10 sec and rinsing them for 30 sec in sterile, deionized water. Two disks of inoculated tissue were removed from the cotyledon with a 6-mm-diameter paper punch. Whenever possible, the disks obtained were diagonally located in the two inoculated panels of a cotyledon. The two disks were macerated together in 2 ml of sterile, deionized water with a mortar and pestle. Dilutions were made in sterile, deionized water and 0.2-ml aliquots were plated on Difco nutrient agar plates. Plates were inverted and maintained at 24 C until colony counts were made.

RESULTS

Population trends showing the development of *Xcm* races 1, 3, 7, and 18 in the seven cotton lines are shown in Figs. 1 and 3–8. Development of three heterologous bacterial phytopathogens (*Xcc*, *Xcp*, and *Psp*) in Ac 44 and Im 216 are illustrated in Figs. 2 and 9, respectively. Two population curves for races 7 and 18 in Ac 44 and Im 216 are indicated in Figs. 1 and 8. The "A" curves are the results of a preliminary study; the "B" curves are from the subsequent main study which also included the three single-gene lines Ac B_2, Ac B_3, and Ac b_7 as well as OK 1.2 and OK 2.3. The similarity in curves reflects the reproducibility and uniformity of results between experiments.

All races of *Xcm* multiplied logarithmically during the first 24–48 hr after inoculation. Initial inoculum density in vivo, as determined by dilution plating from inoculated tissue immediately after inoculation (0 days), was 3×10^3 to 4.5×10^4 colony-forming units (cfu) per square centimeter. Multiplication rates for different initial in vivo inoculum levels were similar. Differences were detected in bacterial populations among the seven cotton lines inoculated with the same race of the homologous pathogen and also among the four races of the homologous pathogen inoculated into plants of the same cotton line. Bacterial populations generally increased for 6–7 days after inoculation before gradually declining in all cotton lines inoculated with races 1 and 3 except Im 216 and resistant Ac B_3. During the same time period, and in the same cotton lines, populations of races 7 and 18 also multiplied but did not generally show a decline after 7 days. Final population counts ranged from 10^3 to 10^8 cfu/cm^2 of tissue for races 1 and 3, and from 10^5 to 10^8 cfu/cm^2 of tissue for races 7 and 18. Populations of *Xcm* in Im 216 leveled off or declined after day 4.

Differences were observed in bacterial population trends for races of the homologous pathogen inoculated into blight-susceptible, -resistant, and -immune cotton lines. Fully susceptible Ac 44 had final populations ranging from about 10^7 to 10^9 cfu/cm^2 of tissue (Fig. 1). Im 216 displayed final populations between 10^3 and 10^6 cfu/cm^2 of tissue for the four races tested (Fig. 8). Growth trends of bacteria in cotton lines with intermediate levels of resistance had final populations intermediate between those in Ac 44 and Im 216 (Figs. 3–7). In general, cotton lines with multiple genes for bacterial blight resistance (Figs. 5 and 7) had lower final bacterial populations than did the single-gene lines (Figs. 3–5, and 7).

Population trends for heterologous pathogens in Ac 44 (Fig. 2) and for all homologous and heterologous pathogen combinations in Im 216 (Figs. 8 and 9, respectively) were similar. After the initial 24–48 hr, growth continued until populations peaked at 10^5–10^7 cfu/cm^2 of tissue 3–4 days after inoculation. Heterologous populations in Ac 44 had declined rapidly to 10^3–10^4 cfu/cm^2 of tissue 9 days after inoculation. Homologous pathogen populations in Im 216 exhibited a similar decline, reaching final populations of 10^3–10^6 cfu/cm^2 of tissue 9 days after inoculation. Final populations for races of the homologous pathogen in Im 216 were generally two to four orders of magnitude lower than were their final populations in the other cotton lines.

Throughout the experiment, after day 0, *Xcp* (a heterologous pathogen) showed considerably higher population levels in Im 216 than did any of the races of the homologous pathogen (Fig. 9). Population peaks in Im 216 for the other heterologous pathogens (*Xcc* and *Psp*) were comparable to peaks of the races of the homologous pathogen that was tested. The final population for *Xcc* was slightly lower in Im 216 than in Ac 44. For *Psp*, they were essentially the same in Im 216 and Ac 44. The final population for *Xcp* in Im 216 was three orders of magnitude greater than in Ac 44. The final population of *Xcp* in Ac 44 corresponded to the final population of *Xcc*, and *Psp* in Ac 44 and Im 216.

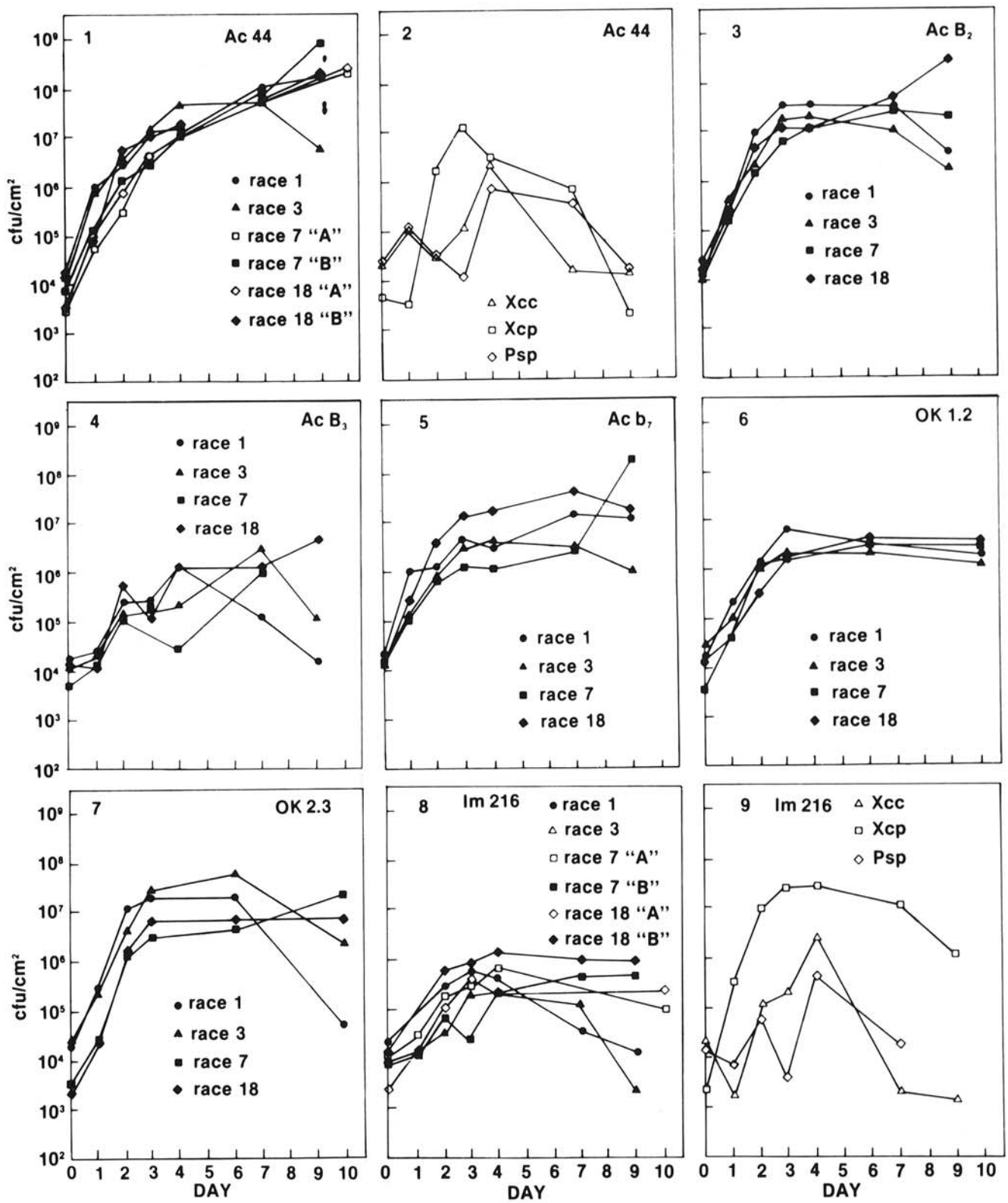
DISCUSSION

Our homologous pathogen-host combinations of four races of *Xcm* and the susceptible or resistant cotton lines exhibited population trends similar to those reported by other workers who used other homologous pathogens and plants with varying levels of resistance (21,22,32,35,36). In a smaller study, Perry (34) described similar observations of the different growth rates of *Xcm* between susceptible and resistant cotton lines. Johnson et al (25), in a preliminary study, observed that during the first 48 hr, rates of multiplication of *Xcm* in Ac 44 or Im 216 were the same regardless of initial inoculum concentration used. After 48 hr, the rate of multiplication in Im 216 was much slower than the rate in Ac 44.

The intermediate bacterial populations we observed in Ac B_2, Ac B_3, and Ac b_7, and in OK 1.2 and OK 2.3 correspond to population trends in hybrid plants with intermediate levels of resistance described by Chand and Walker (15) for cucumber and by Diachun and Troutman (16) for tobacco. Also, this system appears to react similarly to the one reported by Smiley and Stokes (37) who found that populations of the wildfire bacterium (*Pseudomonas syringae*

pv. *tabaci*) in tobacco leaves decreased as the number of genes for resistance increased and chromosome numbers were held constant.

The present findings, which show a wide difference between susceptible Ac 44 and Im 216 and intermediate responses in other cultivars, are in accordance with previous reports of ultrastructural and biochemical differences among these cultivars made by members of the Oklahoma Cotton Disease Resistance Program. Cason et al (13), Al-Mousawi et al (2,3), and A. H. Al-Mousawi, P. E. Richardson, M. Essenberg, and W. M. Johnson (*unpublished*) observed morphological differences over time among blight-



Figs. 1–9. Population trends for *Xanthomonas campestris* pv. *malvacearum* (*Xcm*), *X. campestris* pv. *campestris* (*Xcc*), *X. campestris* pv. *phaseoli* (*Xcp*), and *Pseudomonas syringae* pv. *pisi* (*Psp*) following inoculation into cotyledons of selected cotton cultivars. **1,** *Xcm* races 1, 3, 7, and 18 in Ac 44. "A" and "B" here and in Fig. 8 indicate results from a preliminary experiment and the main experiment, respectively. **2,** *Xcc, Xcp,* and *Psp* in Ac 44. **3,** *Xcm* races 1, 3, 7, and 18 in Ac B_2. **4,** *Xcm* races 1, 3, 7, and 18 in Ac B_3. **5,** *Xcm* races 1, 3, 7, and 18 in Ac b_7. **6,** *Xcm* races 1, 3, 7, and 18 in OK 1.2. **7,** *Xcm* races 1, 3, 7, and 18 in OK 2.3. **8,** *Xcm* races 1, 3, 7, and 18 in Im 216. **9,** *Xcc, Xcp*, and *Psp* in Im 216.

susceptible, -resistant, and -immune cotton lines in electron micrographs of inoculated leaves. Al-Mousawi et al (2) reported that *Xcm* was not attached to host cell walls by enveloping fibrillar material in leaves and cotyledons of susceptible Ac 44 cotton; however, *Xcm* enveloped and attached to host cell walls within a few hours after inoculation in OK 1.2 and OK 2.3 that possess lower levels of genetically determined resistance than Im 216. These envelopes later ruptured as the bacteria multiplied (A. H. Al-Mousawi, P. E. Richardson, M. Essenberg, and W. M. Johnson, *unpublished*). In Im 216, *Xcm* is enveloped by fibrillar material with an outer border of more dense cuticle-like material during the first 4 hr following inoculation (3,13). Essenberg et al (20) reported that inoculated blight-susceptible and blight-resistant leaves and uninoculated blight-resistant leaves contained compounds inhibitory to *Xcm*. However, levels of the compounds found in inoculated blight-susceptible or uninoculated blight-resistant leaves were much lower than in the inoculated blight-resistant leaves. It is possible that these mechanisms may account for the differences in population trends observed; however, further research in this area is being conducted.

Population trends for the heterologous pathogens (*Xcc*, *Xcp*, and *Psp*) in Ac 44 were also similar to trends reported by other researchers for other heterologous relationships (1,17,23,27,41). However, multiplication of heterologous pathogens in Im 216 did *not* follow the population trends previously mentioned. This observation differs from the general conclusion of Ercolani and Crosse (17) that homologous pathogens grow more effectively than heterologous pathogens in the same plant, but it is not necessarily a contradiction of their work on specific host-pathogen systems. Rather, it is an indication of an apparently nonspecific resistance mechanism in Im 216 which operates effectively against both homologous and heterologous phytopathogenic bacteria. In Ac 44, a similar resistance mechanism may operate against the heterologous pathogens, but not against the homologous pathogen.

When Im 216 cotyledons are inoculated with concentrated inocula (10^8–10^9 cfu/ml) of *Psp*, *Xcc*, or *Xcm* race 1, a hypersensitive response in cotyledons to *Psp* or *Xcc* becomes visible 2–6 hr earlier than the response to *Xcm* race 1 (W. M. Johnson, *unpublished*). In the present study, homologous populations peaked shortly after the heterologous populations. Bacterial growth would depend on how rapidly the hypersensitive response occurs. Stall and Cook (38) reported a hypersensitive reaction in pepper leaves within 8–12 hr after inoculation, whereas, inhibition in susceptible leaves was not observed until 48 hr after inoculation.

In Im 216, inhibition observed 24–48 hr after inoculation appears to be a disease response that is localized (18,19) and nonspecific in that it effectively inhibits both homologous and heterologous pathogens (29), and is hypersensitive in nature (28). The response occurs within a specified time, regardless of the initial inoculum density (17,30). Similar initial growth rates for bacteria in homologous and heterologous leaf extracts discredit the possibility of a preformed inhibitor in the plants (14,15,38). The resistance mechanism observed in Im 216 differs from those reported for other incompatibility systems which are effective against heterologous pathogens in that the resistance in Im 216 is equally effective against *both* heterologous and homologous pathogens.

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