EVALUATION OF THE CONCEPT OF HORIZONTAL AND VERTICAL RESISTANCE BY THE COTTON-*XANTHOMONAS MALVACEARUM* HOST-PATHOGEN SYSTEM

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Scope and Method of Study: The objectives of this study were to determine the relative levels of horizontal and vertical resistance to selected races of *Xanthomonas malvacearum* in selected cotton differentials, to quantify and compare rates of reproduction and survival in the cotton differentials of *X. malvacearum* and several other phytopathogenic bacteria which do not attack cotton, and to determine the relationship between blight severity in the field and pathogen development *in vivo*. Seven cotton differentials with various levels of bacterial blight resistance were each inoculated with eight or nine races of *X. malvacearum*. A disease severity index was obtained for each combination and rankings were made. *In vivo* studies of homologous and heterologous populations were made. Growth trends in bacterial populations were determined using the most probable number dilution technique.

Findings and Conclusions: There was a fairly constant ranking of differentials over races of the pathogen and of races over differentials, but some interactions were observed. The constancy indicates that horizontal resistance exists and is important in the cotton-*X. malvacearum* host-pathogen system, particularly in the blight immune differential Im 216. OK 1.2 and OK 2.3 contain the same genes, in different combinations, as Im 216. They each exhibit vertical resistance while Im 216 exhibits horizontal resistance. This strongly suggests a lack of difference between horizontal and vertical genes for resistance. *In vivo* studies show that homologous pathogens reach higher population levels than heterologous pathogens in fully blight susceptible and moderately resistant differentials. In Im 216 the heterologous pathogen populations were as high or higher than the homologous populations suggesting that Im 216 possesses a nonspecific resistance mechanism which inhibits the growth of all phytopathogenic bacteria. Some differences were observed between blight severity in the field and pathogen population levels in plants.

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CHAPTER I

INTRODUCTION

Bacterial blight [causal organism: Xanthomonas malvacearum (E. F. Smith) Dowson] of upland cotton (Gossypium hirsutum L.) is an economically important disease in most cotton growing areas of the world (Knight and Hutchinson, 1950; Brinkerhoff, 1970). In the United States, blight is particularly important in the more northerly portions of the Cotton Belt and at higher elevations (Brinkerhoff and Presley, 1967). Annual losses in Oklahoma and Texas averaged 0.5-1.0% in 1978, although local losses may have been much higher. Nationwide, estimated 1978 losses were 0.4% of the crop or 45,563 bales (Berry, 1979). Since 1953, the annual loss from bacterial blight in the United States has ranged from 0.38-3.42% of the crop (Watkins, 1979). Losses in many other cotton growing areas, particularly Africa, are much higher (Knight and Hutchinson, 1950).

Bacterial blight affects leaves, stems, and bolls of the cotton plant. Symptoms normally appear as round to angular, watersoaked lesions which eventually dry and become dark brown. Under severe infection, individual fruiting branches may die, but the whole plant is seldom killed. Infection by the pathogen can predispose the plant to subsequent infection by other bacteria and fungi. Economic loss is due to a decrease in cotton fiber quality and yield (Bird, 1960).

Sanitation (Brinkerhoff and Fink, 1964), quarantine (Schnathorst, 1966), and seed treatment (Hunter and Brinkerhoff, 1964) have been successfully used as cultural control measures, but use of resistant cultivars is generally considered the most effective and economical method of control (Brinkerhoff et al., 1952; Wickens, 1953; Bird, 1960; Brinkerhoff, 1963). Proper gene combinations should result in permanent, high levels of blight resistance to all races of X. malvacearum (Bird, 1960; Brinkerhoff, 1970; Brinkerhoff and Verhalen, 1976; Nelson, 1978).

The objectives of this study were: 1) to determine the relative levels of horizontal and vertical resistance to selected races of X. malvacearum in selected cotton differentials, 2) to quantify and compare rates of reproduction and survival in the cotton differentials to X. malvacearum and several other phytopathogenic bacteria which do not attack cotton, and 3) to determine the relationship between blight severity in the field and pathogen development in vivo.

CHAPTER II

LITERATURE REVIEW

Resistance to Bacterial Blight of Cotton

Knight and Hutchinson (1950) have summarized the evolution of resistance to bacterial blight in cotton. They point out that in areas where blight infection is severe, immunity or near immunity has developed in the native populations. Old and New World species of cotton differ in the amount of resistance naturally present in their respective centers of origin with Old World species generally having higher levels of resistance than New World species. Brinkerhoff (1970) listed 16 blight-resistance B genes which have been discovered in cotton and which have been described as major genes or oligogenes. Several polygene and minor gene complexes have also been described. Genes for resistance can be divided into those which have no other known effect on the host apart from providing resistance and those which incidentally confer resistance while also affecting the plant in some other way (Knight and Hutchinson, 1950; Van der Plank, 1968; Abdalla and Hermsen, 1971). Four of the five genes identified by Knight and Hutchinson (1950) belong to the first group.

Eighteen races of X. malvacearum have been identified (Brinkerhoff, 1970; Bird, 1976; Bird et al., 1977) using standard differentials developed by Hunter et al.(1968). Race 1 is the most prevalent in the

United States and is presumably the progenitor of all other races in this country (Brinkerhoff, 1961).

Breeding lines with various combinations of single and multiple genes for blight resistance have been developed which exhibit differing levels of blight resistance (Blank, 1949; Green and Brinkerhoff, 1956; Innes, 1964; El-Zik and Bird, 1970). Brinkerhoff (1961, 1963) suggested that the development of new pathogenic races of X. malvacearum would probably become a serious problem for blight-resistant cultivars of cotton. Cultivars with single genes for blight resistance have proven especially susceptible to the buildup of new races. Increased pathogen virulence has been observed in several previously resistant gene combinations (Hunter and Blank, 1954; Gunn, 1961; Cross, 1963 and 1964).

Several workers have suggested that relatively few genes can effect immunity (Bird et al., 1961; Innes, 1964; Brinkerhoff, 1967). El-Zik and Bird (1970) reported that resistance or near immunity to blight is dependent not only on the number of major genes in a strain, but also on the effectiveness of combining major and minor genes. The importance and effect of minor or modifying genes have been pointed out by Knight and Hutchinson (1950); Brinkerhoff (1961 and 1963); Bird (1964); and Innes (1964). Bird (1960 and 1966) has demonstrated that immune upland strains of cotton can be developed by transferring B genes into tolerant genotypes and then screening the segregating populations with a mixture of races of the pathogen. The immunity thus conferred appears to be stable and not subject to the development and buildup of new races (Bird, 1960; Brinkerhoff, 1963 and 1970).

Horizontal and Vertical Resistance

Van der Plank (1968) introduced the terms vertical and horizontal to describe two types of resistance present in plants. Vertical resistance has also been called field resistance, field immunity, hypersensitive resistance, major gene resistance, qualitative resistance. differential resistance, R-gene resistance, race-specific resistance, racial resistance, and specific resistance. Horizontal resistance has also been termed field resistance, general resistance, generalized resistance, minor gene resistance, nonracial resistance, nonspecific resistance, multigenic resistance, nonhypersensitive resistance, tolerance, partial resistance, polygenic resistance, quantitative resistance, quantitatively inherited resistance, race-nonspecific resistance, relative resistance, residual resistance, and uniform resistance (Robinson, 1976). Caldwell (1968) and Nelson (1978) have modified the initial definitions of Van der Plank (1968). Caldwell (1968), Posnette (1969), and Schafer (1971) pointed out that the term "tolerance" has a separate, distinct meaning and should not be confused with intermediate levels of resistance.

Vertical resistance is generally inherited oligogenically and generally provides complete, but temporary control of a disease problem (Van der Plank, 1968; Robinson, 1976). It is associated with the gene-for-gene hypothesis developed by Flor (1946, 1955). Vertical resistance delays the start of an epidemic (Van der Plank, 1968) and is easy to use in a breeding program (Abdalla and Hermsen, 1971; Simons, 1972). Robinson (1971) has listed 14 rules for assessing the value of vertical resistance in specific crop situations.

Horizontal resistance is generally inherited polygenically and provides incomplete, but permanent control of a disease problem (Van der Plank, 1968; Robinson, 1976). This type of resistance slows down the infection rate once an epidemic has started (Van der Plank, 1968). Black (1970) and Robinson (1973) have pointed out complex mechanisms which can function in horizontal resistance. Discussions of the value and importance of horizontal resistance have been published by several workers (Hooker, 1967; Caldwell, 1968; Abdalla and Hermsen, 1971; Simons, 1972; Barksdale and Stoner, 1973; Robinson, 1973; Luke, Barnett, and Pfahler, 1975; Nelson, 1978). Oligogenically controlled, horizontal resistance has been postulated for several diseases (Caldwell, 1968; Abdalla and Hermsen, 1971; Luke, Barnett, and Pfahler, 1975; Robinson, 1976).

Robinson (1976) claims:

> Vertical and horizontal resistance are entirely different characters whose inheritance is controlled by entirely different genes. It is not a question of how many genes control the inheritance, but of which genes do so. (pg. 19)

Other workers suggest a closer relationship between genes for horizontal resistance and those for vertical resistance (Abdalla and Hermsen, 1971; Parlevliet, 1976; Parlevliet and Zadoks, 1977; Nelson, 1978). Nelson (1978) summarizes similar findings by other workers. He further states:

> Vertical resistance and horizontal resistance, my argument contended, are not indications of the action of different genes, but rather are expressions of different actions of the same genes in different genetic backgrounds. There are, in fact, no major genes and minor genes. There are only genes for resistance. This concept implies that genes function vertically when they are separate and horizontally when they are together. (pp. 369-370)

Horizontal and vertical resistance genes occurring together in one

plant have been postulated for some systems (Black, 1970; Brinkerhoff, 1970; Nelson et al., 1970; Thurston, 1971; Luke, Barnett, and Chapman, 1975; Parlevliet, 1976) and perhaps for all host-pathogen systems (Van der Plank, 1968; Abdalla and Hermsen, 1971; Robinson, 1976). The value of combining different forms of resistance has been noted by a number of researchers (Schafer, 1971; Robinson, 1973; Parlevliet and Zadoks, 1977; Nelson, 1978).

Bacterial Development In Vivo

In vivo studies of bacterial populations using homologous and heterologous host-pathogen systems have exhibited definite trends (Allington and Chamberlain, 1949; Skoog, 1952; Diachun and Troutman, 1954; Maine, 1958; Scharen, 1959; Garber, 1961; Chamberlain, 1962; Klement and Lovrekovich, 1962; Chand and Walker, 1964; Klement et al., 1964; Perry, 1966; Stall and Cook, 1966 and 1968; Omer and Wood, 1969; Lozano and Sequeira, 1970; Hsu and Dickey, 1972; Young, 1974; Gross and DeVay, 1977; Essenberg et al., 1979; Johnson et al., 1979). Population trends for homologous and heterologous pathogens were similar for the first 24-48 hr after which marked differences were observed. Multiplication of the heterologous pathogens generally ceased rather abruptly around 48 hr after inoculation; populations then remained constant at about the same level or decreased for the duration of the experiment. Destruction of cellular organization often followed inhibition of multiplication (Klement et al., 1964). Peak populations and final populations were higher for homologous pathogens than they were for heterologous pathogens (Skoog, 1952; Maine, 1958; Garber, 1961; Chand

and Walker, 1964; Stall and Cook, 1966; Omer and Wood, 1969; Young, 1974; Gross and DeVay, 1977). Saprophytes tested were unable to multiply in living plant tissues (Klement and Lovrekovich, 1962; Klement et al., 1964; Young, 1974).

Logan (1960) inoculated a homologous pathogen (X. phaseoli var. fuscans) and a heterologous pathogen (X. malvacearum) into bean leaves. He did not make actual population counts, but he did observe differences in symptom expression for each phytopathogen. Smiley and Stokes (1961) observed that populations of the wildfire bacterium [Pseudomonas tabaci (Wolf and Foster) Stevens] in tobacco leaves decreased as the genes for resistance increased while holding constant chromosome number.

In some incompatible systems, inhibition observed 24-48 hr after inoculation appears to be a disease response that is localized (Essenberg et al., 1976; Essenberg et al., 1979), nonspecific (Klement and Lovrekovich, 1961), and hypersensitive in nature (Klement and Goodman, 1967). The response occurs within a specified time, regardless of the initial inoculum density (Klement and Lovrekovich, 1962; Ercolani and Crosse, 1966). Similar growth rates for bacteria in homologous and heterologous leaf extracts discredits the possibility of a preformed inhibitor in the plants (Chamberlain, 1962; Chand and Walker, 1964; Stall and Cook, 1968). Ercolani and Crosse (1966) and Ercolani (1973) discuss reasons why in vivo homologous populations are higher than in vivo heterologous populations.

CHAPTER III

MATERIALS AND METHODS

Field Study

Cotton Differentials

The seven differentials used in this study were Im 216, Ac 44, Ac B_2, Ac B_3, Ac b_7, OK 1.2, and OK 2.3. Im 216 is a blight-immune differential with at least two dominant genes and possibly one recessive genes for resistance (Brinkerhoff and Verhalen, 1976). Ac 44 is a fully blight-susceptible differential with no known genes for resistance. The near isogenic differentials on an Ac 44 background (Ac B_2, Ac B_3, and Ac b_7) possess homozygous single-gene blight resistance; these three genes have been tentatively identified as the major genes which collectively confer blight immunity to Im 216 (L. A. Brinkerhoff, (personal communication). OK 1.2 and OK 2.3 are homozygous differentials selected from segregating generations of a cross between Im 216 and Ac 44; segregating generations from this cross were repeatedly inoculated, screened, and selfed to achieve homozygosity. Present data indicate the blight resistance in OK 2.3 is due to the gene B_2 and the blight resistance in OK 1.2 is due to the genes B_3 and b_7. Plants were grown in the cotton disease nursery at Stillwater, Oklahoma. Generally, 25 plants per race per differential were selected for use in this study.

In past growing seasons, the differentials OK 1.2 and OK 2.3 have graded 1.2 and 2.3, respectively, when inoculated with X. malvacearum race 1. All OK 1.2 and OK 2.3 plants used in this study were again inoculated with race 1 to verify their uniformity for bacterial blight reaction. A tag was applied to identify those leaves which had been inoculated to prevent further testing on those specific leaves.

Bacterial Races

Virulent races of X. malvacearum were obtained from stock cultures maintained by Dr. W. M. Johnson of Langston University and Oklahoma State University and by Dr. J. S. Bird of Texas A&M University. Eighteen races of the pathogen have been identified at the present time (Brinkerhoff, 1970; Bird, 1976; Bird et al., 1977). Races 1, 2, 3, 4, 6, 7, 10, 11, and 18 were used in the field tests. All cultures were maintained on PCDA slants a potato-carrot-dextrose agar medium (Bird, 1966) at 24 C. Cultures were transferred to fresh PCDA slants at 2 wk intervals. Race identification and virulence were routinely tested by inoculations into the standard cotton differentials (Hunter et al., 1968).

Field Inoculation and Grading

Inocula for the field study were prepared by transferring a loop-ful of bacteria from a PCDA culture to 80 ml of Difco nutrient broth. This stock culture was grown for 24 hr at 24 C on a reciprocating shaker. One ml aliquots of the stock culture were transferred to culture bottles containing 80 ml of nutrient broth. The bottles of inoculum were placed on a shaker for 12-24 hr. For field inoculation,

the broth cultures were diluted with tap water on a 1:200 basis and resulted in an inoculum concentration of ca. 5.0 X 10^5 viable cells/ml.

Plants were inoculated with single nozzle guns using a power sprayer at a pressure of 200-300 psi. Plants were inoculated by spraying the abaxial side of several leaves until visible watersoaking was observed. All plants were tagged to identify the inoculated leaves because the watersoaked areas disappeared within a few hours. The differentials Ac B_2 and Ac B_3 were inadvertently not inoculated with race 10.

Individual plants were scored for their disease reactions 14 days after inoculation using the grading system described by Brinkerhoff (1963). For a more detailed explanation, see Table I.

Bacterial Development In Vivo

Cotton Differentials

The seven differentials used in the field study were also used in the laboratory study. Seed of each differential were planted in clay pots containing a commercially prepared, soilless mix of peat moss and vermiculite ("Jiffy Mix Plus", Jiffy Products of America, West Chicago, Illinois). Seedlings were initially grown in a greenhouse and were 2-3 wk old at the time of cotyledon inoculation. Inoculated plants were then transferred to a growth chamber where they were maintained under a 12 hr 26 C day and 12 hr 19 C night regime. These temperatures have been shown previously to favor bacterial blight development (Brinkerhoff and Presley, 1976).

Bacterial Races

X. malvacearum races 1, 3, 7, 8, and 18 were chosen for use in this study. In addition, three other bacterial phytopathogens which do not infect cotton were tested. *X. campestris* (Pammel) Dowson is a pathogen of cabbage, cauliflower, and related cruciferous species. *X. phaseoli* (E. F. Smith) Sowson is a pathogen of beans and *P. pisi* Sackett is a pathogen of field and garden peas. These phytopathogens were obtained from Dr. A. Novacky of the University of Missouri (*P. pisi*) and Dr. R. S. Dickey of Cornell University (*X. phaseoli* culture no. 1363 and *X. campestris* culture no. 1372). All cultures were maintained on PCDA slants as previously described.

Method of Inoculation

Inocula were prepared by transferring a small loopful of bacteria to 80 ml of nutrient broth. The broth cultures were placed on a shaker and incubated for 10-12 hr. Using a spectrophotometer, the inoculum was adjusted to an initial concentration between 1.0-2.0 X 10^9 colony forming units (CFU)/ml ($A_{600\ nm}$ = 0.2 to 0.4). Inoculum concentrations were verified using standard dilution plate count techniques. Cotyledons were inoculated using a modification of the leaf infiltration technique developed by Klement(1963). A hypodermic needle was used to prick the cuticle and epidermis of the abaxial side near the base of the center two sections of each cotyledon; this procedure allowed for more efficient watersoaking with less structural damage. The tip of a 3 cc syringe filled with inoculum was placed over the pricked area, and inoculum was injected into the tissue; complete watersoaking of each

section of cotyledon was readily achieved. Both cotyledons of a plant were inoculated and eight to 10 cotyledons were inoculated for each race or species of bacteria tested.

Inoculated plants were usually grown in a growth chamber, but two tests were conducted under greenhouse conditions due to a malfunctioning growth chamber. In each case, temperature and humidity were recorded with a hydrothermograph.

Measurement of Bacterial Populations

Isolations were made from inoculated leaf areas on the same day as inoculation (0 time), and on days 1, 2, 3, 4, 7, and 9 (unless otherwise indicated). A single cotyledon was used each day, and an effort was made to use cotyledons from the same plant on successive days to lessen the slight amount of variability that might be present among different plants. Harvested cotyledons were placed in damp paper towels and carried to the laboratory for population determinations.

Bacterial populations were determined by the "most probable number" (MPN), dilution plate technique (Cochran, 1950). Two sets of determinations were made from each cotyledon, and each dilution was plated in triplicate. For each determination, cotyledons were surface disinfected by washing in a 10% Chlorox solution for 8-10 seconds followed by a 30 second rinse in sterile distilled water. Two discs of inoculated tisssue were removed from the cotyledon with a 6 mm diameter paper punch. Whenever possible, the discs were diagonally located in the inoculated sections. Discs were macerated with 2 ml of sterile distilled water in a mortar and pestle. Dilutions were made,

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.

CHAPTER IV

RESULTS

Field Study

A bacterial blight disease index was established for differentials graded in the field. The disease indices ranged from 0.0 to 6.0; 0.0 was a totally immune population, and 6.0 was a fully susceptible one. Immunity was defined as no macroscopic symptoms of bacterial blight under field levels of inoculum. Separate determinations were made for each X. malvacearum race-cotton differential combination. The results are summarized in Fig. 1A-1G. Fig. 1H shows a mean disease severity index by race over all seven differentials , except for race 10 (which was tested on only five differentials). Ac 44 (Fig. 1A) was fully susceptible to all races, and Im 216 (Fig. 1G) was immune to every race tested. Differentials with a polygenic background (Fig. 1E and 1G) had higher levels of blight resistance and those with a monogenic background (Fig. 1B-1D and 1F) had lower levels of resistance.

Mean index values were obtained for each differential and for each race. Those values were used to rank races and differentials (Table II). Examination shows few interactions between the races and the differentials, or in other words, there was a fairly constant ranking of races when compared to the differentials and vice versa. This strongly suggests that horizontal resistance as defined by Van der Plank (1968)

and Robinson (1976) is a major component in the cotton-X. malvacearum host-pathogen system.

Ranking of the differentials can be related to their genetic condition, i.e., those with similar gene combinations showed similar disease indices. Ranking of the races according to their virulence showed that race 1 displayed the highest average virulence. However, all races tested were fully virulent on the susceptible Ac 44 (Fig. 1A). The differences in the constancy of the rankings between the races and the differentials are evidence of a vertical resistance component in some of the differentials.

Bacterial Development In Vivo

Population trends showing the development of X. malvacearum races 1, 3, 7, and 18 in the seven differentials (Ac 44, Ac B_2, Ac B_3, Ac b_7, OK 1.2, OK 2.3, and Im 216) are shown in Fig. 2 and 4-9. Development in Ac 44 and Im 216 of race 8 X. malvacearum and three bacterial phytopathogens which do not attack cotton (X. campestris, X. phaseoli, and P. pisi) are illustrated in Fig. 3 and 10, respectively.

All bacterial species studied multiplied logarithmically for the first 24-48 hr period after inoculation. In all differentials initial inoculum density was 3×10^3 to 4.5×10^4 CFU/ml. Multiplication rates for all inoculum levels appeared to be similar. Marked differences in population development between the homologous and the heterologous pathogens could generally be observed after 24-48 hr. Homologous pathogens continued to multiply reaching a final population count of 10^5-10^8 bacteria/cm^2 of tissue after 9 to 10 days in all differentials except Im 216. In Im 216, final population counts of

homologous races varied from 10^3-10^6 bacteria/cm^2 of tissue.

Heterologous pathogens in Ac 44 and both homologous and heterologous pathogens in Im 216 exhibited similar population trends. After the initial 24-48 hr period, multiplication continued until a population peak of 10^5-10^7 bacteria/cm^2 of tissue was reached 3-4 days after inoculation. Heterologous populations then declined rapidly in Ac 44 to 10^3-10^4 bacteria/cm^2 of tissue 9 days after inoculation. Heterologous populations in Im 216 declined more slowly to reach final population levels of 10^3-10^6 bacteria/cm^2 of tissue 9 days after inoculation. Race 8 in Im 216 peaked on day 4 and more or less held constant at 10^6-10^7 bacteria/cm^2 of tissue thereafter.

Two curves for races 7 and 18 in Ac 44 and Im 216 are indicated in Fig. 2 and 9, respectively. The "A" curves are the results of a preliminary study; the "B" curves are the results of the main study which also included the three single gene differentials Ac B_2, Ac B_3, and Ac b_7. The close relationship between the "A" and "B" curves provides evidence that the study is repeatable from experiment to experiment.

Multiplication of heterologous pathogens in Im 216 did not follow the population trends reported by previous workers for other heterologous relationships (Allington and Chamberlain, 1949; Ercolani and Crosse, 1966; Hsu and Dickey, 1972; Young, 1974). X. phaseoli showed considerably higher numbers of bacteria/cm^2 of tissue than any of the five races of X. malvacearum tested for 8 days after inoculation (Fig. 9 and 10). On the ninth day, X. phaseoli was surpassed only by X. malvacearum race 8. X. campestris and P. pisi had the third and fifth highest population levels peaks of the eight heterologous and homologous

pathogens tested. This differs from the general conclusion of Ercolani and Crosse (1966) that homologous pathogens grow more effectively than heterologous pathogens in the same plant, but is not necessarily a contradiction of their specific work. Rather, it is an indication of the apparently highly nonspecific resistance mechanism in Im 216 which operates effectively against both homologous and heterologous phytopathogenic bacteria. In Ac 44, a similar resistance mechanism operates against the heterologous pathogens, but not against the homologous pathogens.

Fig. 11-18 compare the growth curves of the seven differentials as a function of the X. malvacearum race with which they were inoculated. Logarithmic growth occurred in all differentials for 24-48 hr after inoculation regardless of the race used. After the initial growth period, differences in growth trends between Im 216 and the other differentials became apparent. In Im 216, with an initial concentration of 10^3-10^4 bacteria/cm^2 of tissue, the highest number of bacteria/cm^2 (10^5-10^6) occurred 3-4 days after inoculation, followed by a rapid decline to 10^3-10^5 bacteria/cm^2 of tissue after 9 days.

Differentials inoculated with races 1 and 3 continued multiplying for 6-7 days after inoculation before showing a gradual decline in numbers. During the same time period, the differentials inoculated with races 7 and 18 did not decline. Final population counts were 10^5-10^8 bacteria/cm^2 of tissue for races 1 and 3, and were 10^6-10^8 bacteria/cm^2 for races 7 and 18. Initial population levels for races 1 and 3 were ca. 10^4 bacteria/cm^2; levels for races 7 and 18 were 10^3-10^4 bacteria/cm^2 of tissue. Final populations for all four races in Im 216 were generally two to four orders of magnitude lower than

the final populations in the other differentials. Young (1974) reported a similar range in population for homologous [P. phaseolicola (Burkholder) Dowson] and heterologous [P. glycinea Coerper, P. lachrymans (Smith and Bryan) Carsner, P. syringae van Hall and P. cichorii (Swingle) Stapp wich attack soybean, cucumber, cherry, and chrysanthemum, respectively] populations inoculated at the same initial concentration into bean leaves.

Multiplication of X. malvacearum race 8 in Ac 44 and Im 216 (Fig. 19) did not follow the same pattern as the other races of X. malvacearum tested in those two differentials. Population levels were higher in Ac 44 until 9 days after inoculation when it was surpassed by Im 216. In all other instances, the race population in Im 216 was much lower than the population in Ac 44 10 days after inoculation. Further experimentation should be conducted with this particular race before any conclusions can be reached as to the apparent discrepancy.

Population trends in Ac 44 and Im 216 for X. campestris and P. pisi are illustrated in Fig. 20 and 22, respectively. X. campestris and P. pisi have similar growth curves. Numbers of bacteria/cm^2 of tissue in Im 216 generally decreased rapidly from 10^4 to 10^3 in the first 24 hr after inoculation. A gradual increase in population occurred until a peak of 10^5-10^6 bacteria/cm^2 of tissue was reached 4 days after inoculation. This was followed by another rapid decrease which resulted in a population of 10^3-10^4 bacteria/cm^2 of tissue after 9 days. When Im 216 is inoculated using concentrated inoculum (10^8-10^9 cells/ml) of P. pisi, X. campestris, and X. malvacearum race 1, the hypersensitive response to P. pisi and X. campestris becomes visable 2-6 hr earlier than X. malvacearum race 1 (W. M. Johnson,

personal communication). Such an observation would be consistent with the population trends in this study which show homologous populations peak several days later than the heterologous populations. Bacterial growth would depend on how rapidly the hypersensitive response occurs. In Ac 44, populations increased logarithmically for 24 hr and then decreased sharply. Another logarithmic growth phase occurred 2-4 days after inoculation, followed by a general decline.

Populations of X. campestris and P. pisi in Im 216 and Ac 44 all peaked on day 4 after inoculation reaching ca. 10^6 bacteria/cm^2 for X. campestris and ca. 10^5-10^6 for P. pisi. Final populations of X. campestris in Ac 44 (10^4 bacteria/cm^2 of tissue) were an order of magnitude greater than in Im 216. Populations of P. pisi on day 7 were ca. 10^4 bacteria/cm^2 of tissue and ca. 10^5 bacteria/cm^2 for Im 216 and Ac 44, respectively. Comparisons could not be made on day 9 because no value was available for Im 216.

The growth curves for X. phaseoli (Fig. 21) closely resembled that of X. malvacearum race 8 in Ac 44 3-4 days after inoculation. After 4 days the number of bacteria/cm^2 of tissue dropped off very sharply in both differentials. The final population of X. phaseoli in Im 216 is three orders of magnitude greater than in Ac 44 and corresponds to the final population of X. malvacearum race 8 in Ac 44 and Im 216. However, the final population of X. phaseoli in Ac 44 corresponds to the final population of X. campestris and P. pisi in Ac 44 and Im 216.

Relationship of Laboratory and Field Results

Results of the in vivo laboratory studies were prepared for analysis by ranking races and differentials according to the numbers of bacteria/cm^2 of tissue. Races were ranked at days 3 and 9 after inoculation (Table III). The number four was arbitrarily assigned to indicate the highest number of bacteria/cm^2 of tissue and one was assigned to indicate the lowest. The differentials were similarly ranked at days 3 and 9 after inoculation (Table IV) with the number seven assigned to indicate the highest and one to indicate the lowest number of bacteria/cm^2 of tissue within the indicated race. To facilitate comparisons between laboratory and field data, comparable races and differentials were extracted from Table II and are shown in Table V.

Examination of Tables IV and V shows a close relationship between the rankings of the differentials from the field results with the laboratory results at both days 3 and 9 after inoculation. This suggests that a high level of disease in the field should correspond with a high number of bacteria/cm^2 of tissue in the laboratory. Constant ranking of differentials and pathogens is expected in a host-pathogen system with horizontal resistance (Van der Plank, 1968).

Comparisons of race rankings from Tables III and V show that a close relationship between the rankings from field results with the day 3 laboratory results. Ranking the day 9 laboratory results places the races in almost the completely opposite order than do the field results. In other words, the highest levels of disease resistance in the field are not necessarily associated with the lowest number of bacteria/cm^2 in the laboratory.

CHAPTER V

SUMMARY AND CONCLUSIONS

Brinkerhoff *et al.* have suggested that both horizontal and vertical resistance to bacterial blight exist in cotton, particularly in the immune differential Im 216. Field tests using seven differentials and nine races of *X. malvacearum* indicate relatively few interactions between races and differentials. This strongly suggests that horizontal resistance is a major component in the cotton-*X. malvacearum* host-pathogen system.

The difference in constancy of the rankings between some of the differentials and the races suggests that vertical resistance also operates in this system. OK 1.2 and OK 2.3 contain the same genes in different combinations, as in Im 216. However, OK 1.2 and OK 2.3 exhibit vertical resistance, while Im 216 exhibits horizontal resistance. This is very strong evidence that genes for "vertical" and "horizontal" resistance are not necessarily different genes which concurs with the findings of Nelson (1978).

Population trends for homologous race-differential combinations were similar to those found in other systems. Populations of heterologous pathogens in Ac 44 declined rapidly after reaching peak levels which were considerably lower than the peak levels of the homologous pathogens. Heterologous and homologous pathogens in Im 216 had similar growth curves. Unlike previous studies, heterologous populations in

Im 216 were equal in magnitude to homologous populations. This suggests that a nonspecific resistance mechanism operates in Im 216 which inhibits growth of all phytopathogenic bacteria. Studies with other nonpathogens of cotton and saprophytes should be carried out to determine the extent of the inhibition.

Population trends for race 8 did not follow the usual pattern exhibited for other races of X. malvacearum in Ac 44 and Im 216. Further study is needed to assess the validity of those observations. Results of a preliminary study using races 7 and 18 in Ac 44 and Im 216 corresponded closely to a later study with the same races and differentials. This suggests that population curves are repeatable from experiment to experiment.

Relating results between the field and laboratory presented an interesting paradox. A good relationship existed among the differential rankings based on the field study and those in the laboratory study on days 3 and 9 after inoculation. A good relationship also existed among the race rankings based on the field study and the laboratory rankings on day 3 after inoculation. However, the race rankings on day 9 after inoculation were opposite from the previous rankings. Further observations are required to verify that this difference is real.

Race 18 has the widest range of pathogenicity of all the X. malvacearum races (based on its previous reactions to a set of standard differentials). Field tests in this study did not support this observation. Race 1 gave the most severe disease readings. Race 1 also had the highest number of bacteria/cm^2 of tissue on day 3 after inoculation in the laboratory studies. It is noteworthy that the highest populations at the conclusion of the laboratory (day 9) were found for race 18

(the expected result if it were the most virulent race). Both field and laboratory studies should be repeated to furnish more insight into this apparent dicotomy.

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APPENDIXES

TABLE I

A SUMMARY OF LEAF GRADES USED TO CLASSIFY BLIGHT INFECTION IN THE FIELD[a]

Grade[b]	Host reaction	Description of infection type or types
0	Immune	No visable lesions
1	Resistant	Dry pin-point to small, round lesions
4	Susceptible	Large, watersoaked, angular lesions that turn black on drying; large, watersoaked vein lesions
Mesothetic reactions		
0.1, 0.2	Resistant	Predominantly immune; with a few dry pin-point to small, round lesions or a few dry, small, angular lesions; or more than one type may be present
1.2	Resistant	Predominantly dry, pin-point to small, round lesions; with some dry, small, angular lesions present
2.3	Intermediate	Predominantly dry, small, angular lesions; but with large, watersoaked, angular lesions also present; dry, pin-point, to small, round lesions may also be present (especially if the environment does not favor disease expression)

[a]Adapted from Brinkerhoff (1963).

[b]Blight grades are converted to disease severity indices as follows: 0.0 = 0, 0.1 = 1, 0.2 = 2, 1.0 = 3, 1.2 = 4, 2.3 = 5, 4.0 = 6.

TABLE II

NINE RACES OF *XANTHOMONAS MALVACEARUM* AND SEVEN DIFFERENTIALS OF COTTON RANKED IN ORDER OF DECREASING MEAN VIRULENCE AND MEAN DISEASE SEVERITY

	Disease Severity Index							
Race	Ac 44	Ac B_2	OK 2.3	Ac b_7	Ac B_3	OK 1.2	Im 216	Mean
1	6.0	5.0	5.0	4.0	2.0	4.0	0.0	3.7
3	6.0	4.9	4.1	2.2	2.6	2.1	0.0	3.3
2	6.0	3.6	2.2	5.2	4.5	1.3	0.0	3.3
7	6.0	4.0	3.3	2.7	2.8	2.1	0.0	3.0
10	6.0	a	2.6	4.2	a	0.7	0.0	2.7
4	6.0	4.3	3.8	1.8	1.9	0.9	0.0	2.7
11	6.0	3.4	3.2	1.8	1.5	1.4	0.0	2.4
18	6.0	2.8	3.0	2.2	2.1	0.5	0.0	2.4
6	6.0	3.2	2.8	1.4	1.4	1.6	0.0	2.4
Mean	6.0	3.9	3.5	2.8	2.4	1.6	0.0	

[a]Not tested

TABLE III

FOUR RACES OF XANTHOMONAS MALVACEARUM RANKED OVER SEVEN COTTON DIFFERENTIALS IN ORDER OF DECREASING NUMBERS OF BACTERIA PER CM^2 OF COTYLEDON TISSUE 3 AND 9 DAYS AFTER INOCULATION

Time	Numerical Ranking							
Race	Ac 44	Ac B_2	Ac B_3	Ac b_7	OK 1.2	OK 2.3	Im 216	Mean
3 Days								
1	4	4	4	3	4	3	4	3.7
3	3	2	2	2	3	4	2	2.6
18	2	3	1	4	2	2	3	2.4
7	1	1	3	1	1	1	1	1.3
9 Days								
18	3	4	4	3	4	3	4	3.6
7	4	3	3	4	3	4	3	3.4
1	2	2	1	2	2	1	2	1.7
3	1	1	2	1	1	2	1	1.3

TABLE IV

SEVEN COTTON DIFFERENTIALS RANKED OVER FOUR RACES OF *XANTHOMONAS MALVACEARUM* IN ORDER OF DECREASING NUMBERS OF BACTERIA PER CM^2 OF COTYLEDON TISSUE 3 AND 9 DAYS AFTER INOCULATION

Time	Numerical Ranking						
Race	Ac B_2	Ac 44	Ok 2.3	Ac b_7	OK 1.2	Ac B_3	Im 216
3 Days							
1	7	5	6	3	4	2	1
3	6	5	7	4	2	3	1
18	7	6	4	5	3	2	1
7	7	6	5	2	3	4	1
Mean	6.8	5.5	5.5	3.5	3.0	2.8	1.0
9 Days							
18	7	6	3	4	2	5	1
7	4	7	3	6	2	5	1
1	5	7	2	6	4	3	1
3	6	7	4	2	3	5	1
Mean	5.5	6.8	3.0	4.5	2.8	4.5	1.0

TABLE V

FOUR RACES OF XANTHOMONAS MALVACEARUM AND SEVEN DIFFERENTIALS OF COTTON RANKED IN ORDER OF DECREASING MEAN VIRULENCE AND MEAN DISEASE SEVERITY [a]

	Disease Severity Index							
Race	Ac 44	OK 2.3	Ac B_2	Ac b_7	Ac B_3	OK 1.2	Im 216	Mean
1	6.0	5.0	5.0	4.0	2.0	4.0	0.0	3.7
3	6.0	5.5	4.9	2.2	2.6	2.1	0.0	3.3
7	6.0	3.3	4.0	2.7	2.8	2.1	0.0	3.0
18	6.0	3.0	2.8	2.2	2.1	0.5	0.0	2.4
Mean	6.0	4.2	4.2	2.8	2.4	2.2	0.0	

[a] Individual data were extracted from Table II

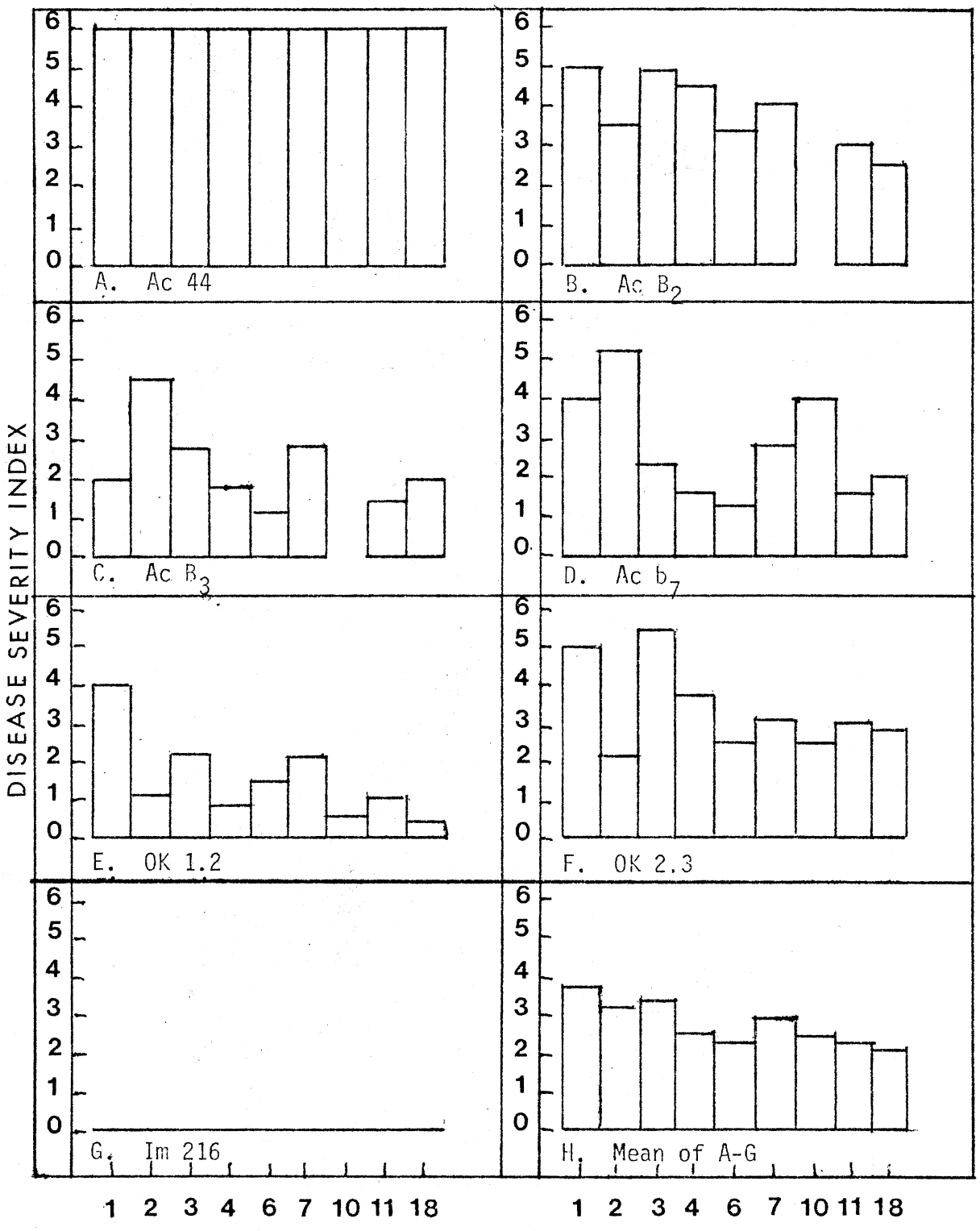

Fig. 1 Disease Severity Indices for Seven Differentials Based on Field Inoculation With Eight or Nine Races of Xanthomonas malvacearum (Race 10 was not tested on Ac B_2 or Ac B_3).

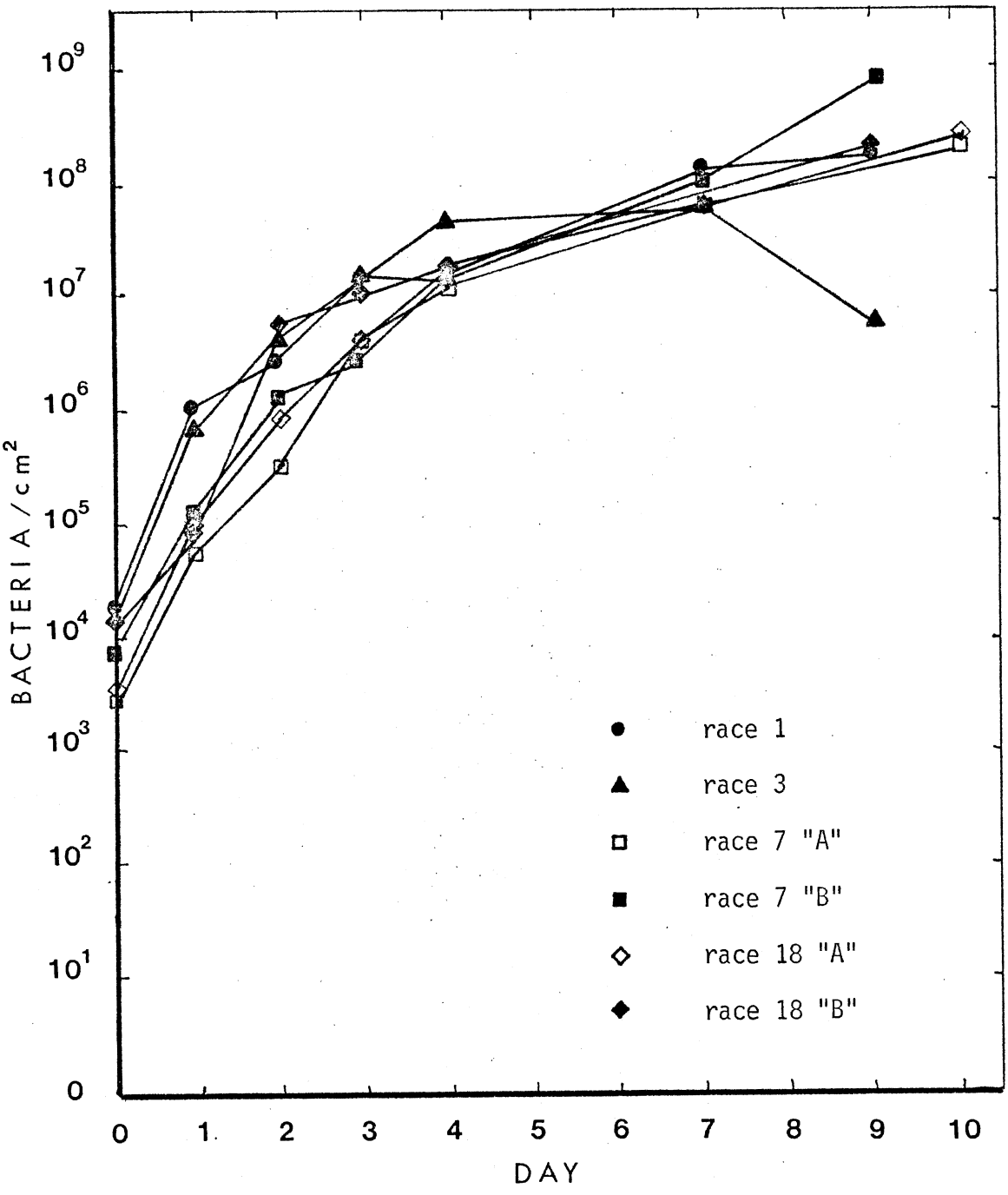

Fig. 2 Population Trends for Xanthomonas malvacearum Races 1, 3, 7, and 18 Inoculated Into Ac 44 Cotyledons.

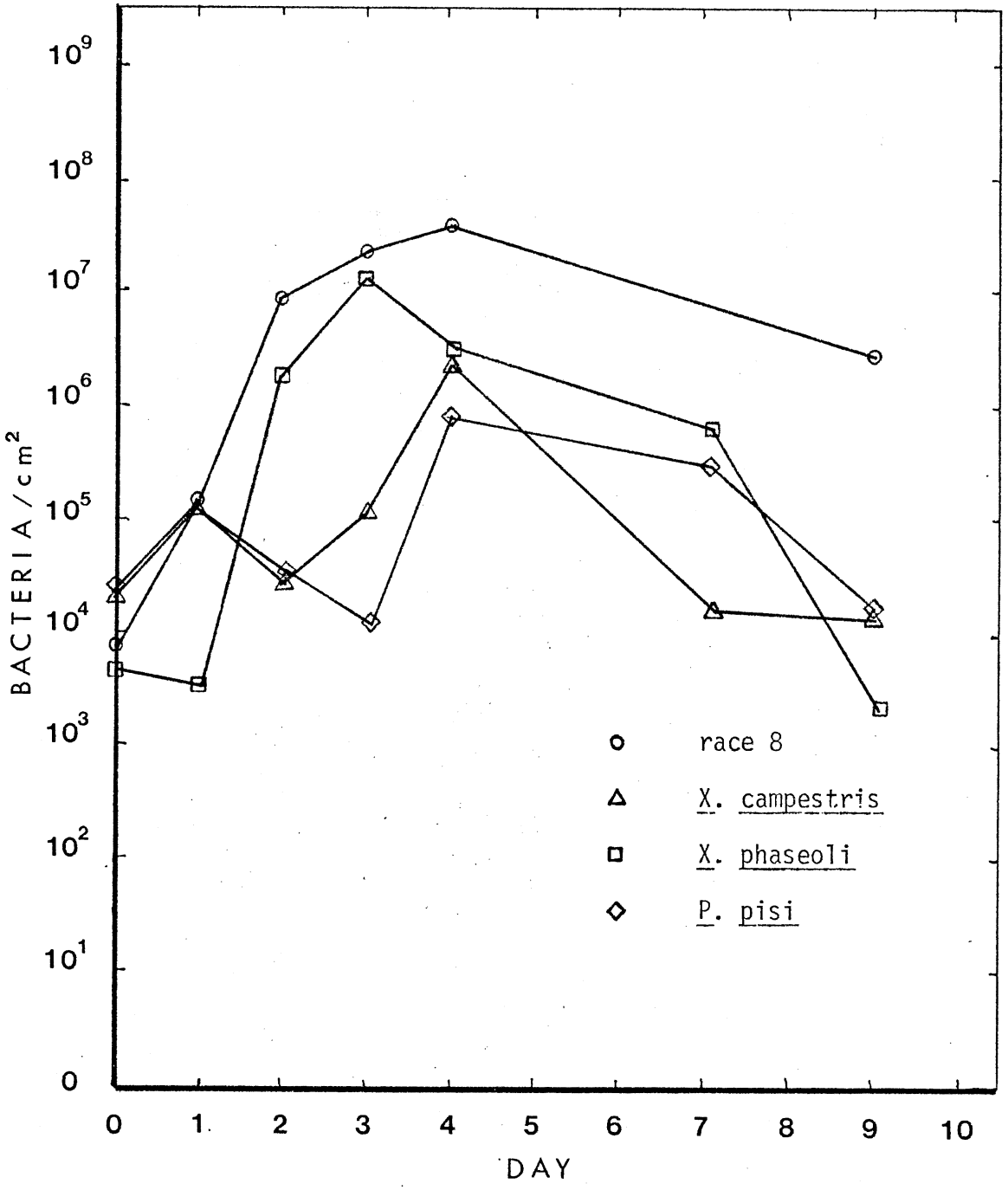

Fig. 3. Population Trends for Xanthomonas malvacearum Race 8, X. campestris, X. phaseoli, and Pseudomonas pisi Inoculated Into Ac 44 Cotyledons.

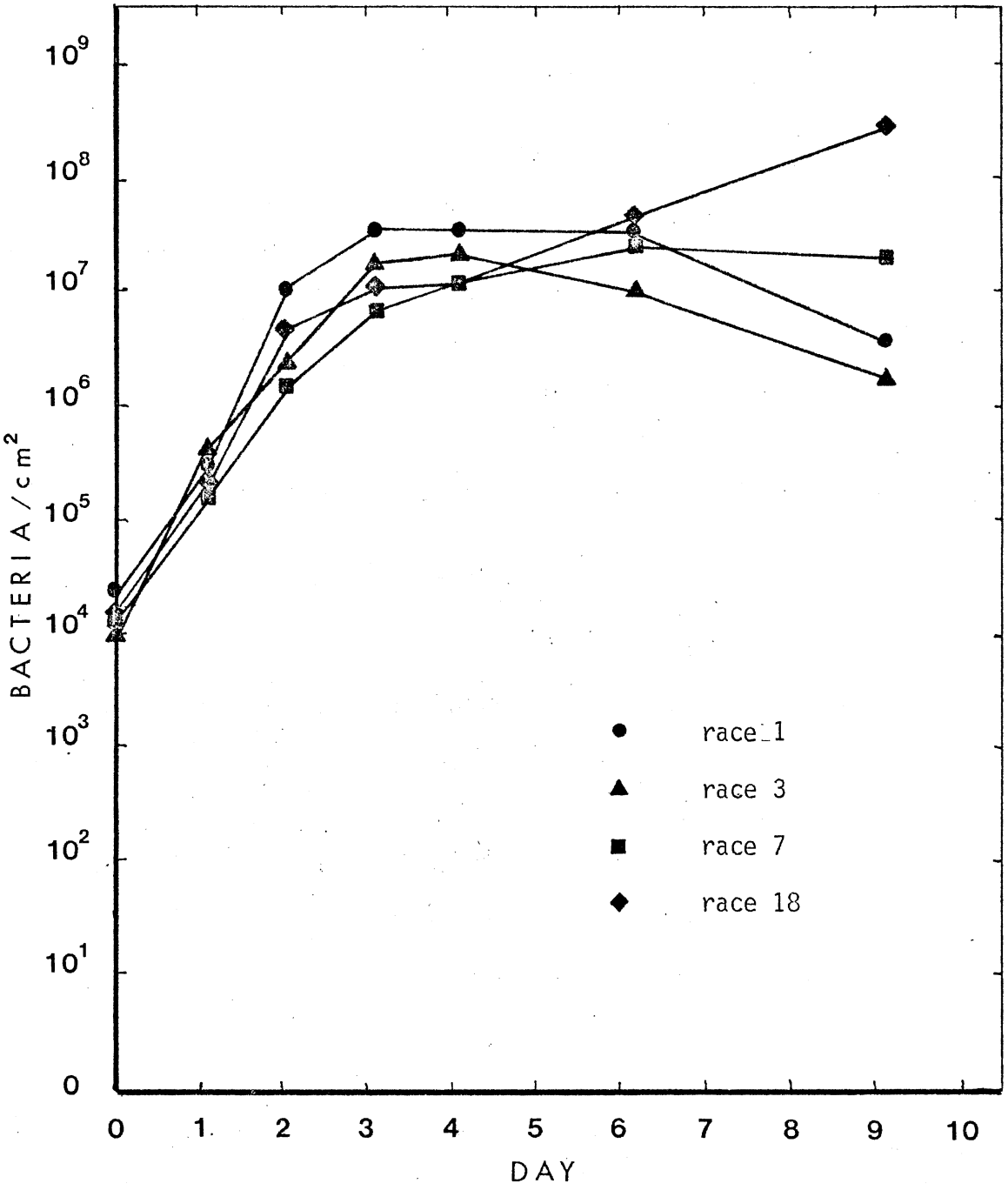

Fig. 4 Population Trends for Xanthomonas malvacearum Races 1, 3, 7, and 18 Inoculated Into Ac B_2 Cotyledons.

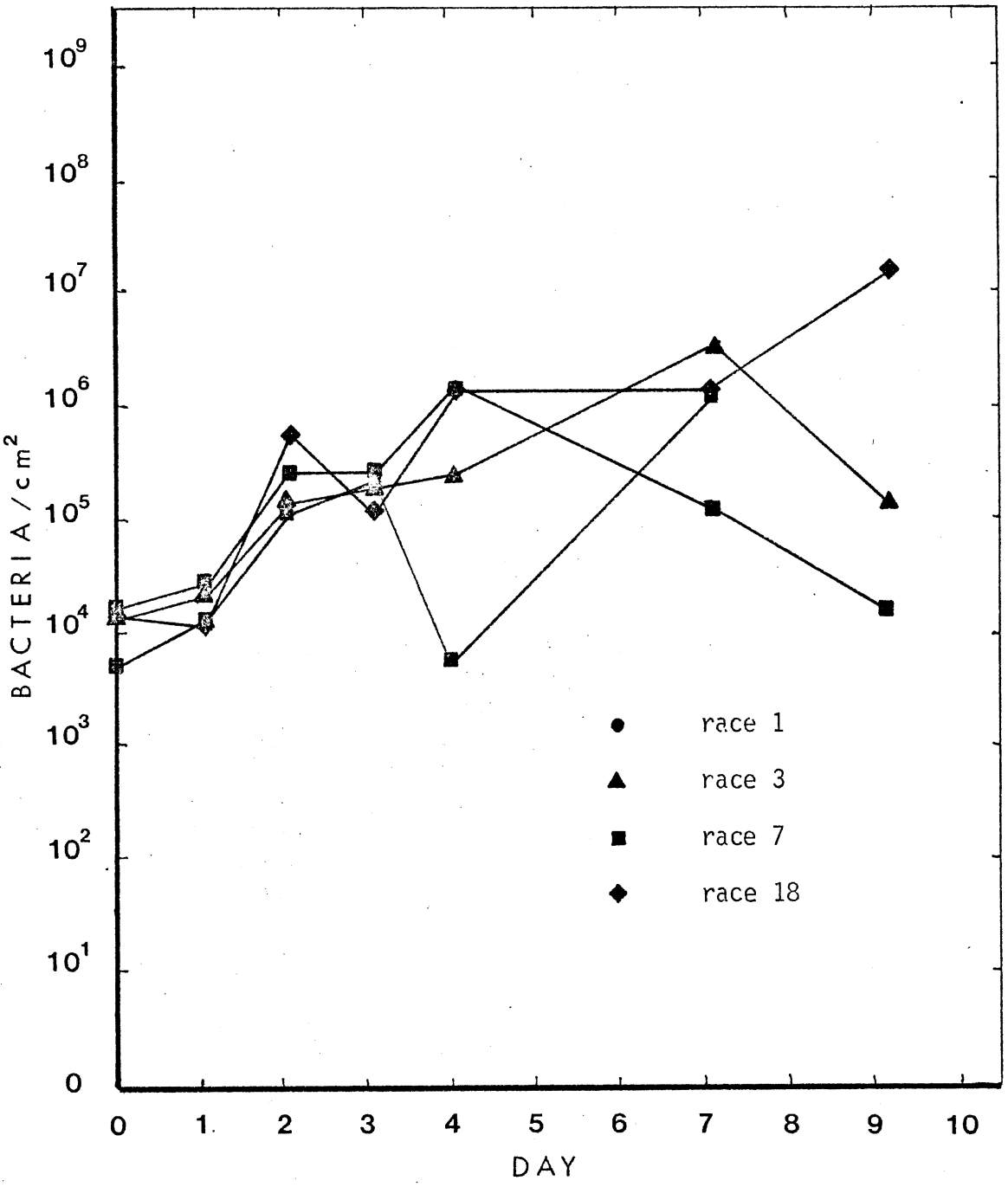

Fig. 5 Population Trends for Xanthomonas malvacearum Races 1, 3, 7, and 18 Inoculated Into Ac B_3 Cotyledons.

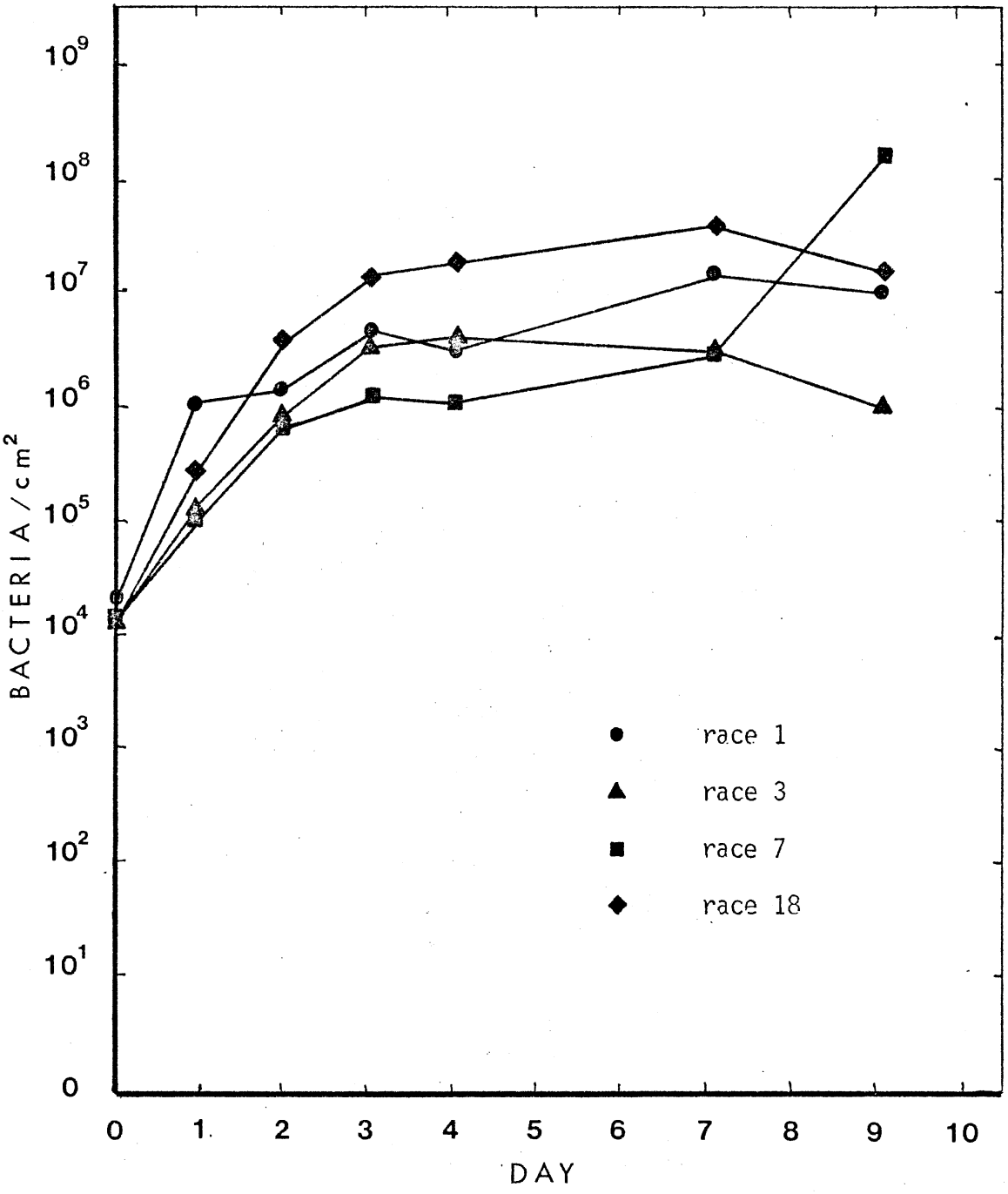

Fig. 6 Population Trends for Xanthomonas malvacearum Races 1, 3, 7, and 18 Inoculated Into Ac b_7 Cotyledons.

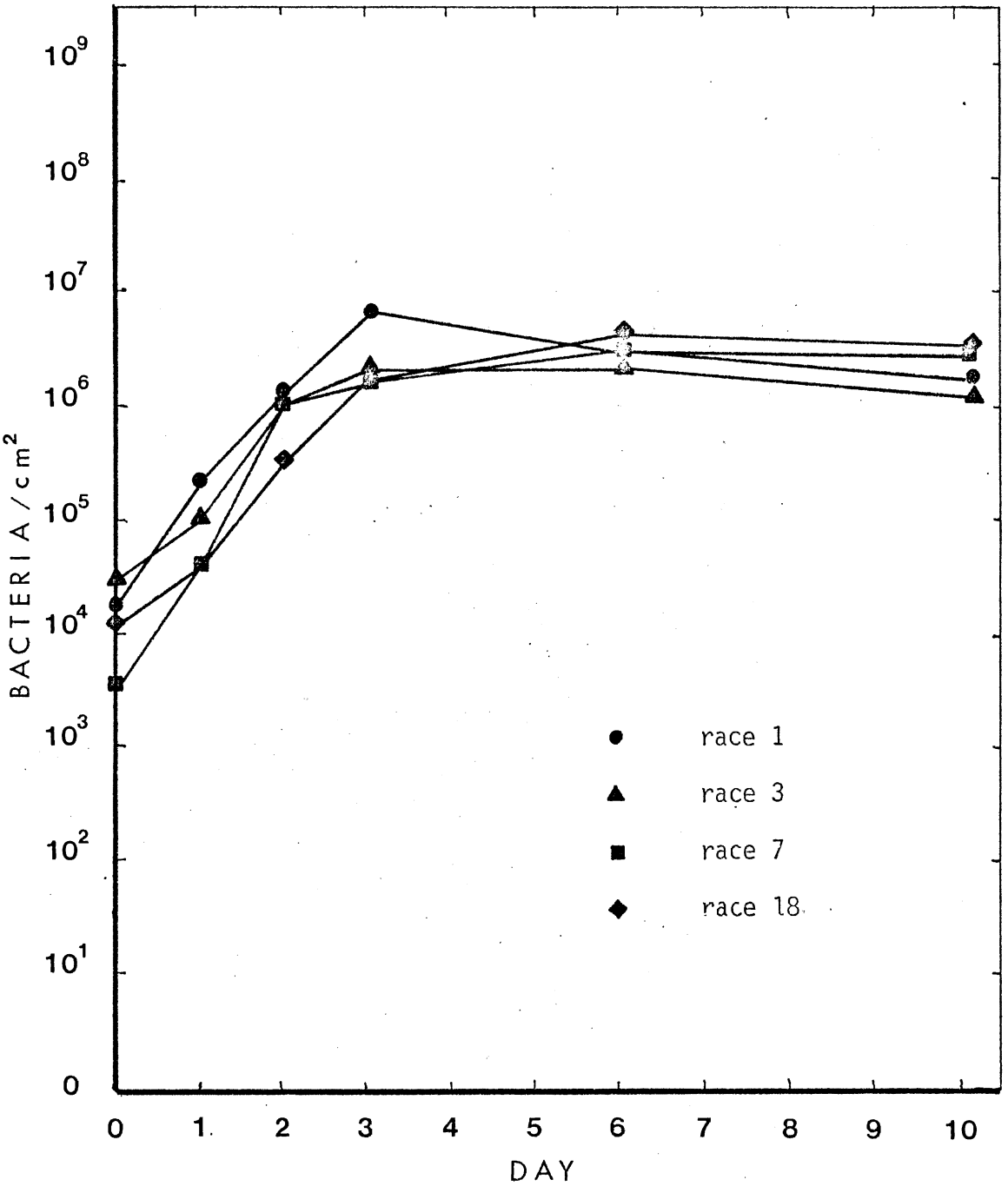

Fig. 7 Population Trends for Xanthomonas malvacearum Races 1, 3, 7, and 18 Inoculated Into OK 1.2 Cotyledons.

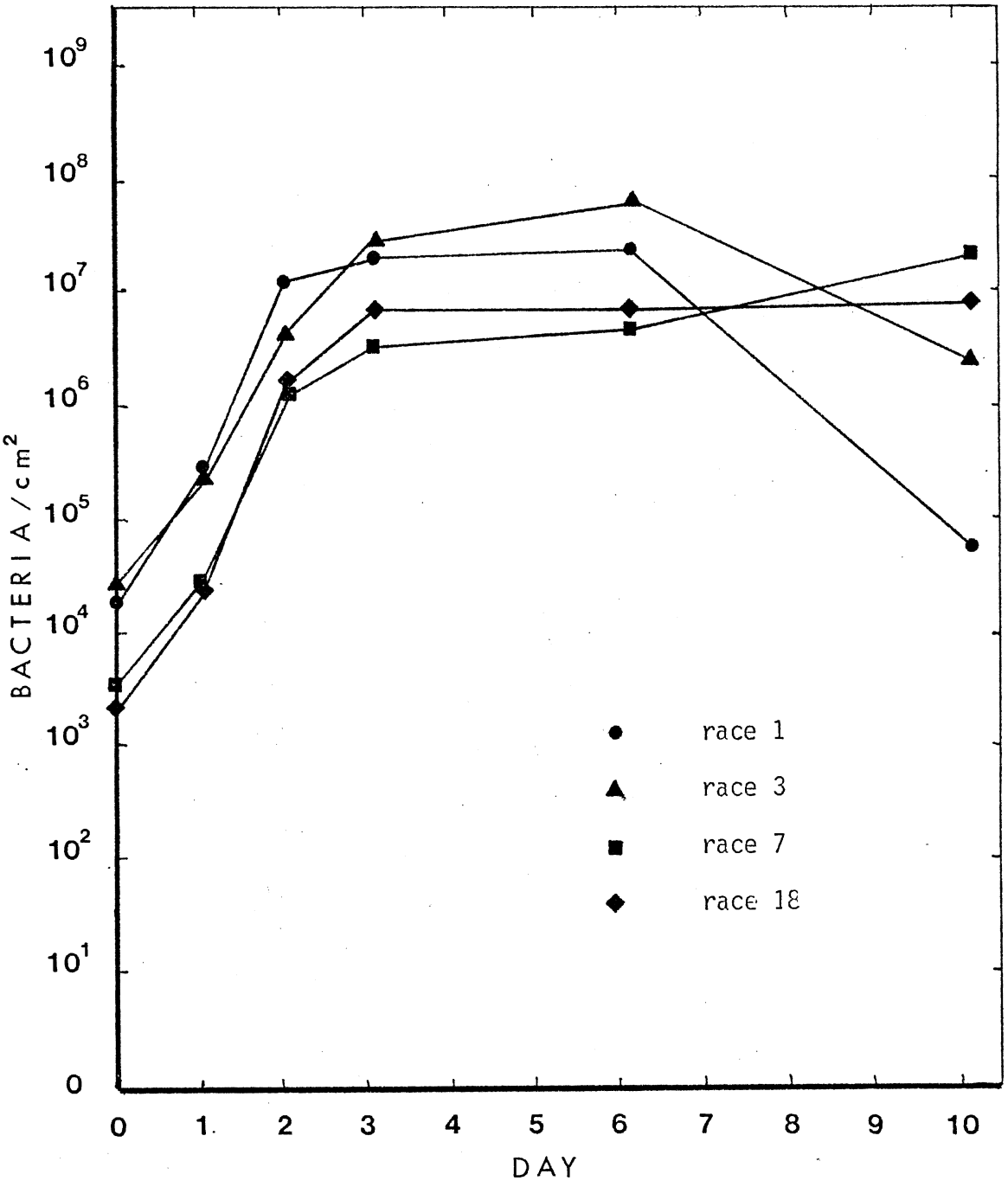

Fig. 8 Population Trends for Xanthomonas malvacearum Races 1, 3, 7, and 18 Inoculated Into Ok 2.3 Cotyledons.

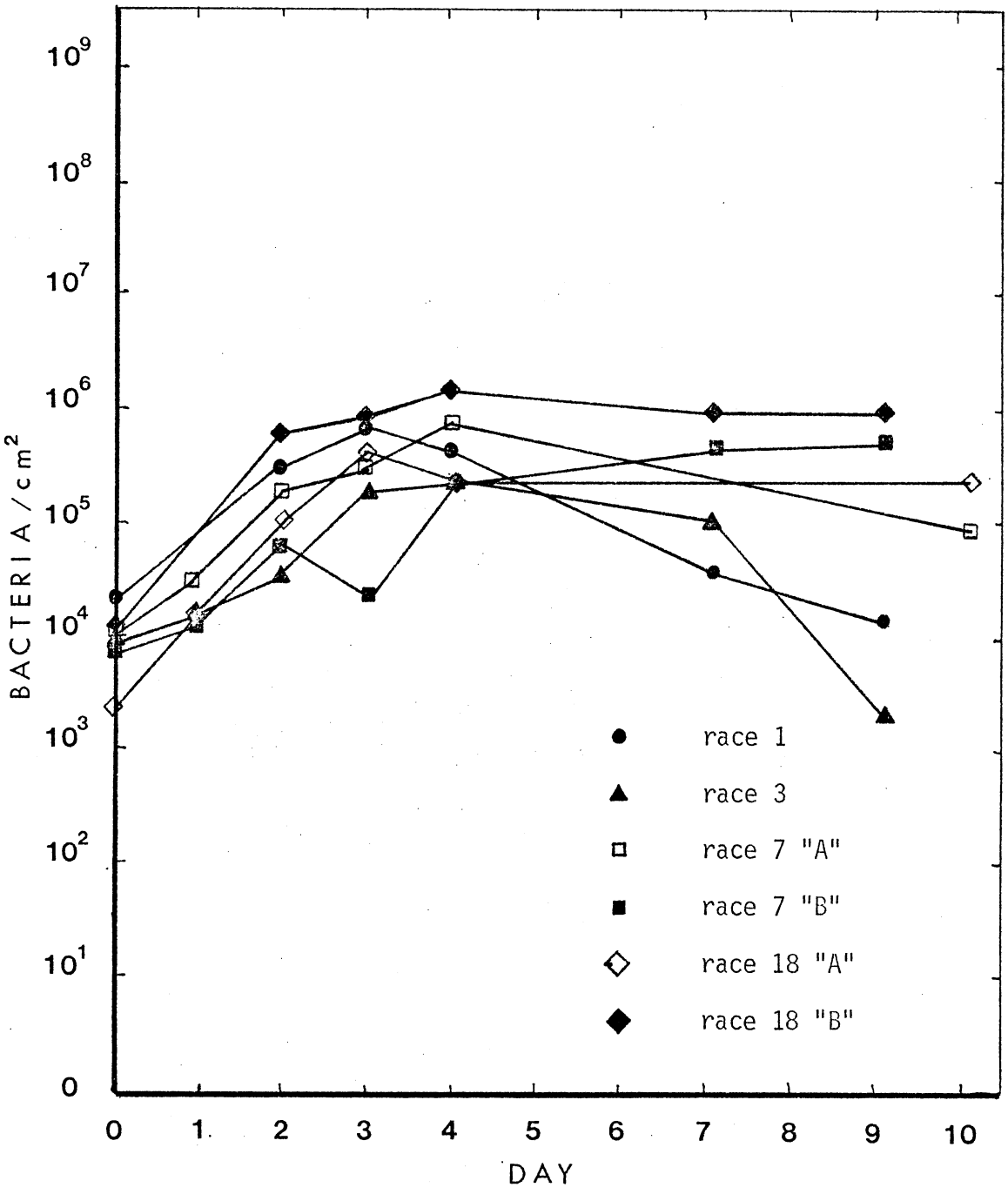

Fig. 9 Population Trends for Xanthomonas malvacearum Races 1, 3, 7, and 18 Inoculated Into Im 216 Cotyledons.

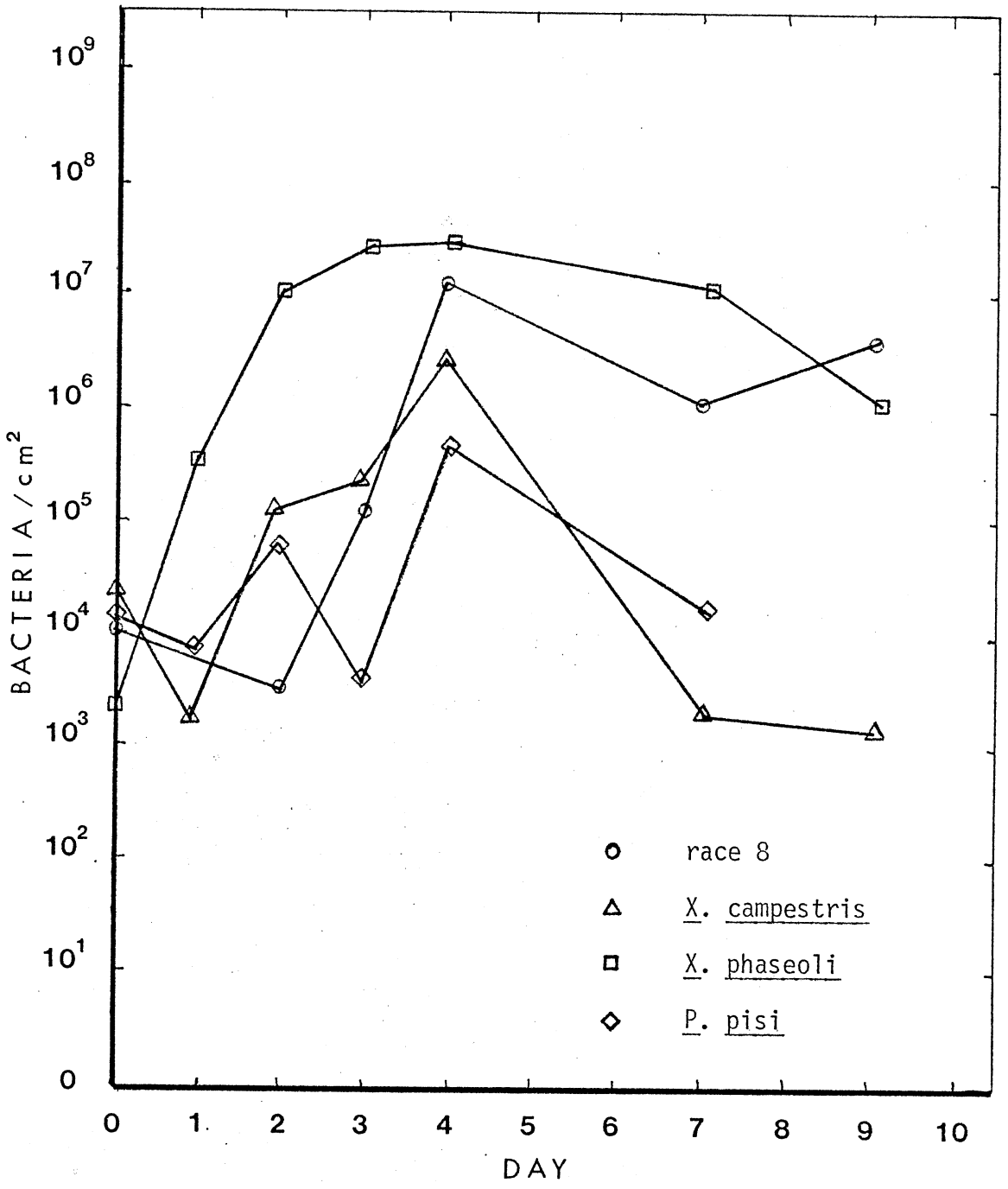

Fig. 10 Population Trends for Xanthomonas malvacearum Race 8, X. campestris, X. phaseoli, and Pseudomonas pisi Inoculated Into Im 216 Cotyledons.

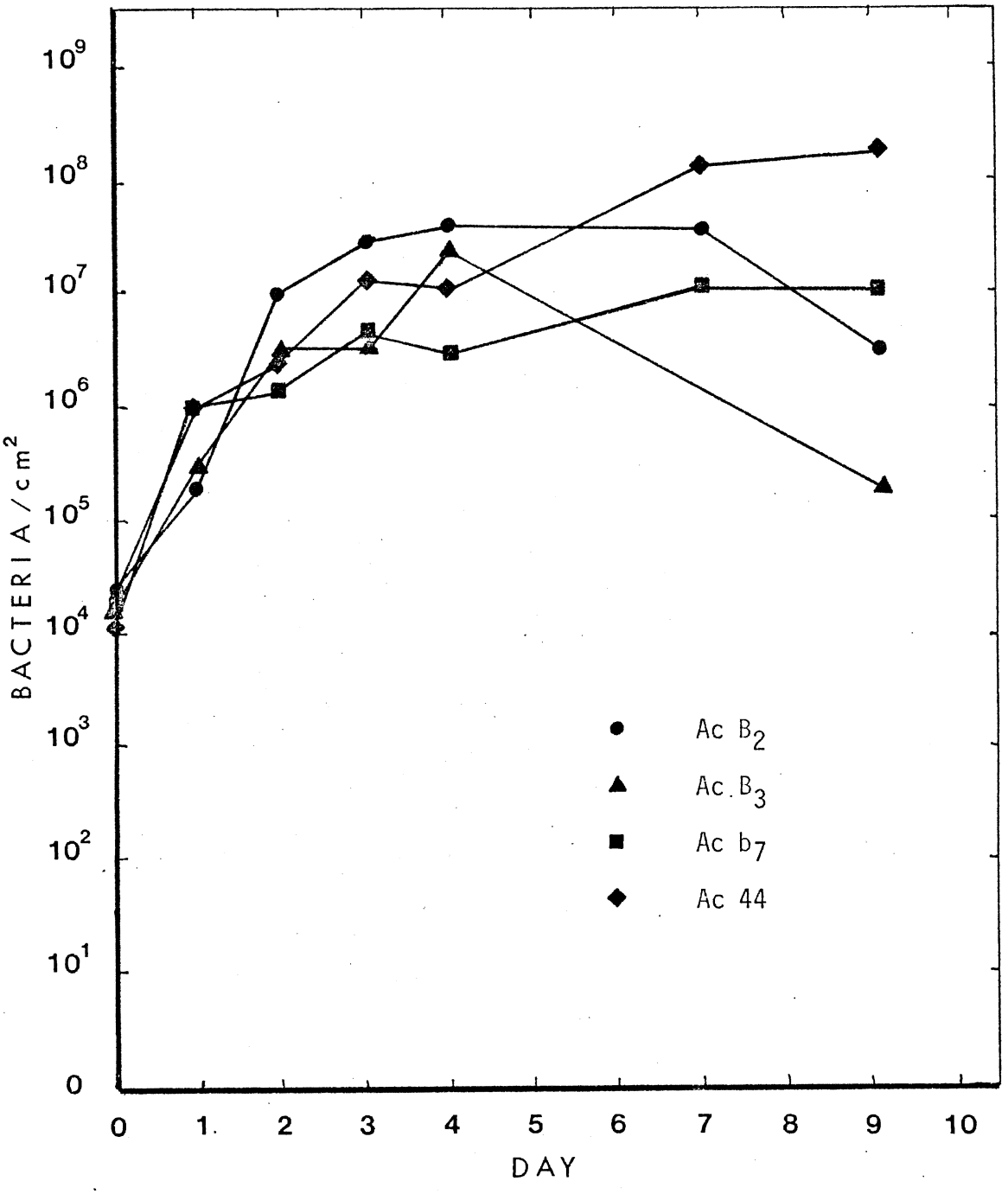

Fig. 11 Population Trends for Xanthomonas malvacearum Race 1 Inoculated Into Ac B_2, Ac B_3, Ac b_7, and Ac44

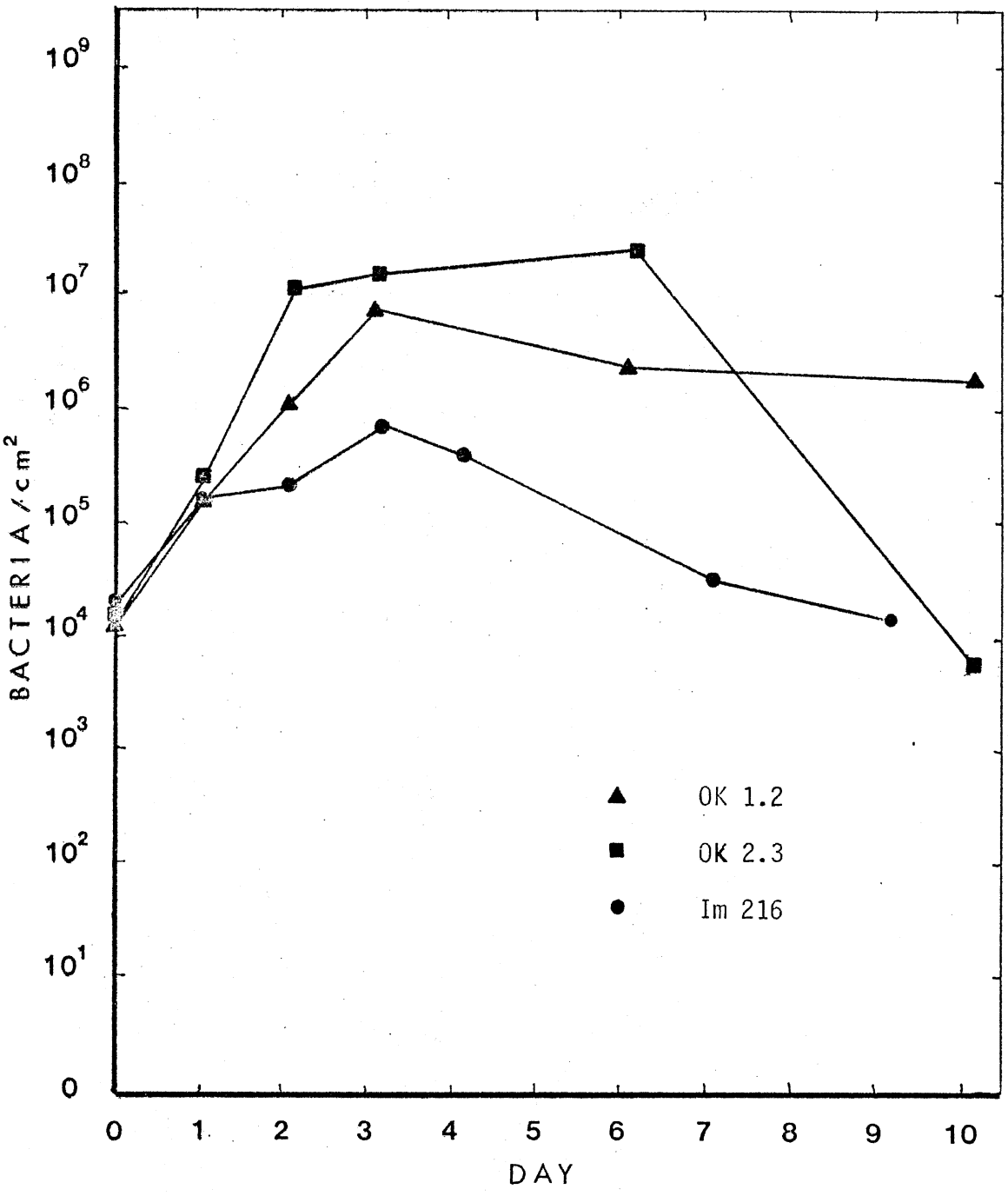

Fig. 12 Population Trends for Xanthomonas malvacearum Race 1 Inoculated Into Ok 1.2, Ok2.3, and Im 216.

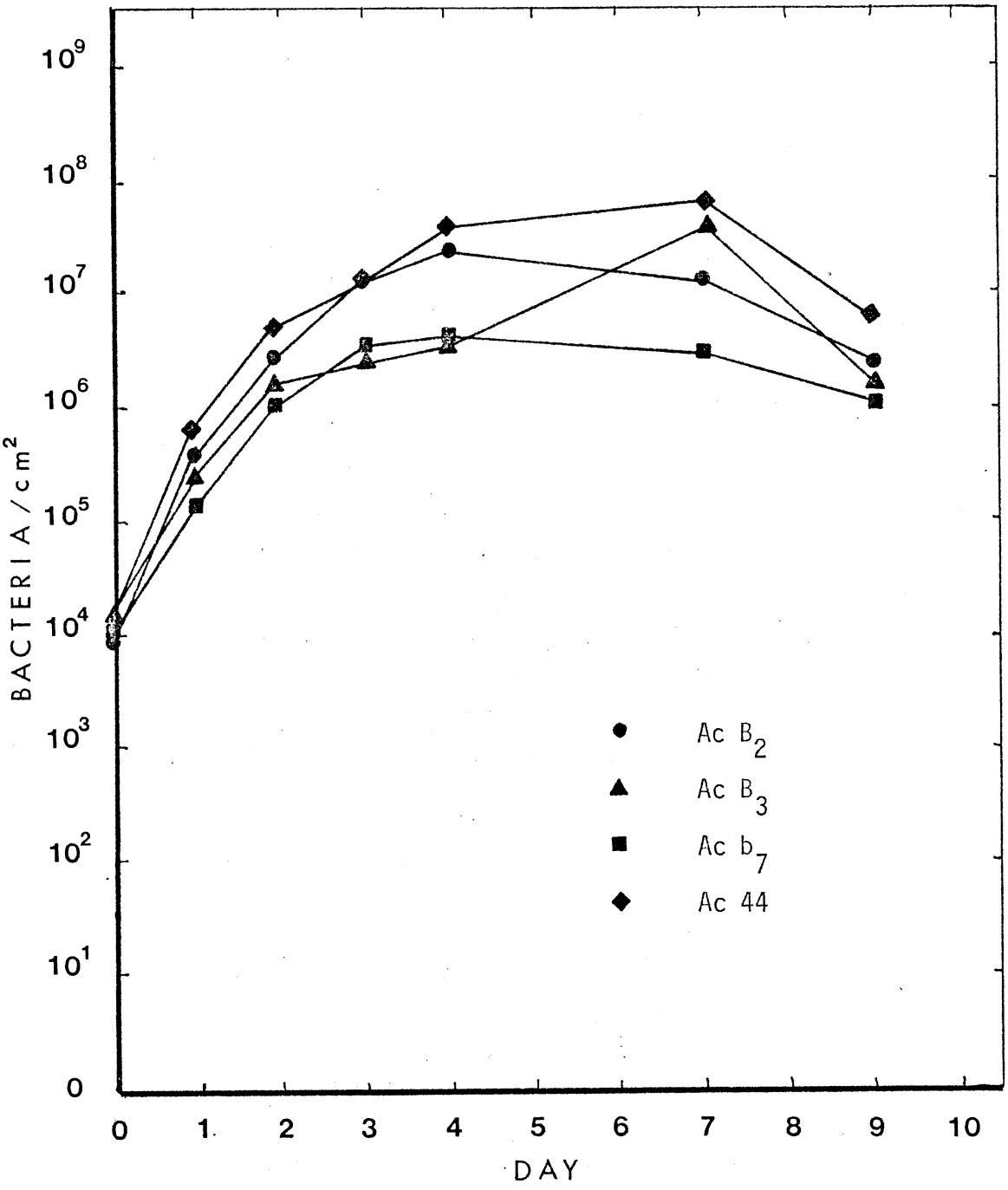

Fig. 13 Population Trends for Xanthomonas malvacearum Race 3 Inoculated Into Ac B_2, Ac B_3, Ac b_7, and Ac 44.

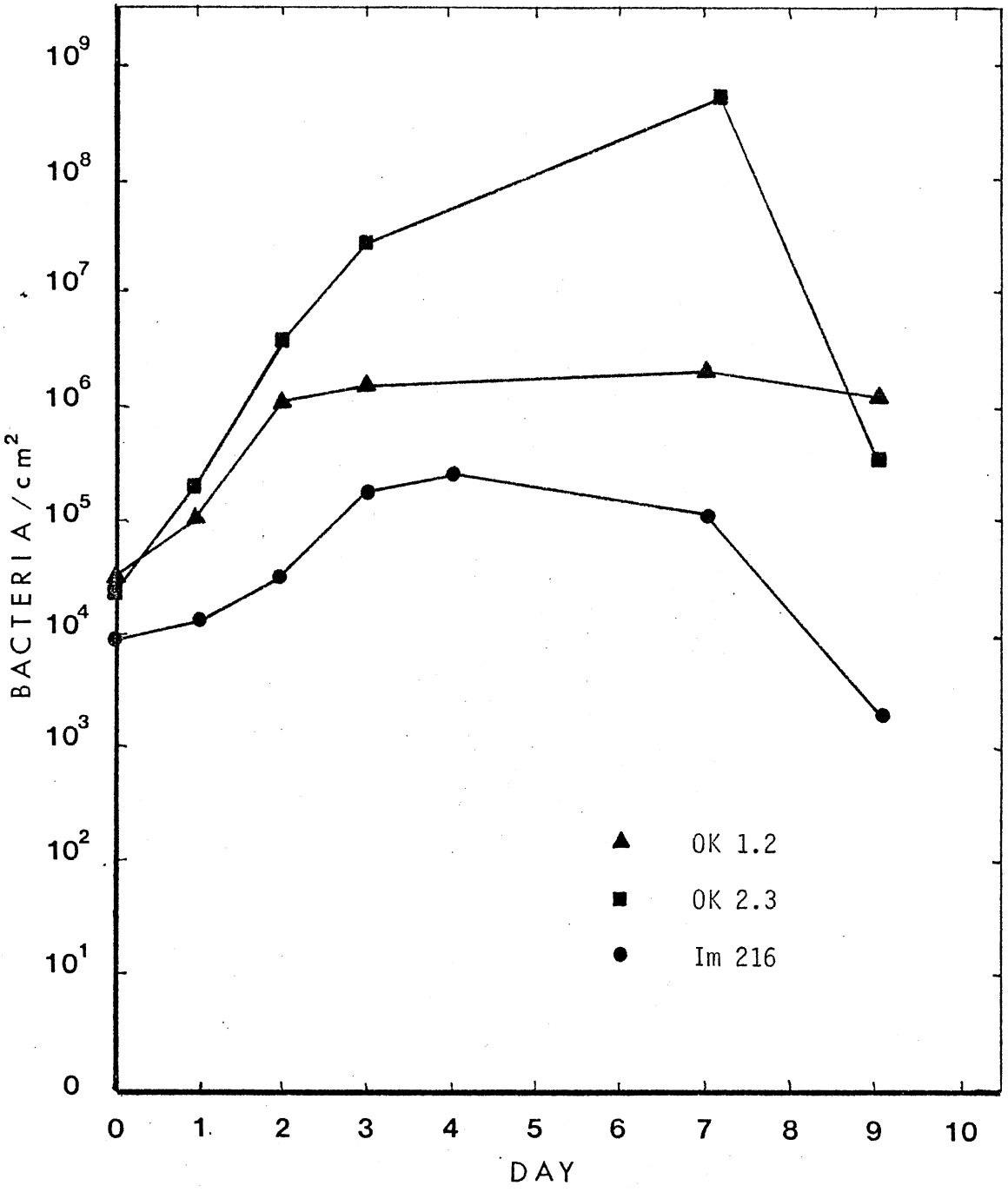

Fig. 14 Population Trends for Xanthomonas malvacearum Race 3 Inoculated Into OK 1.2, OK 2.3, and Im 216.

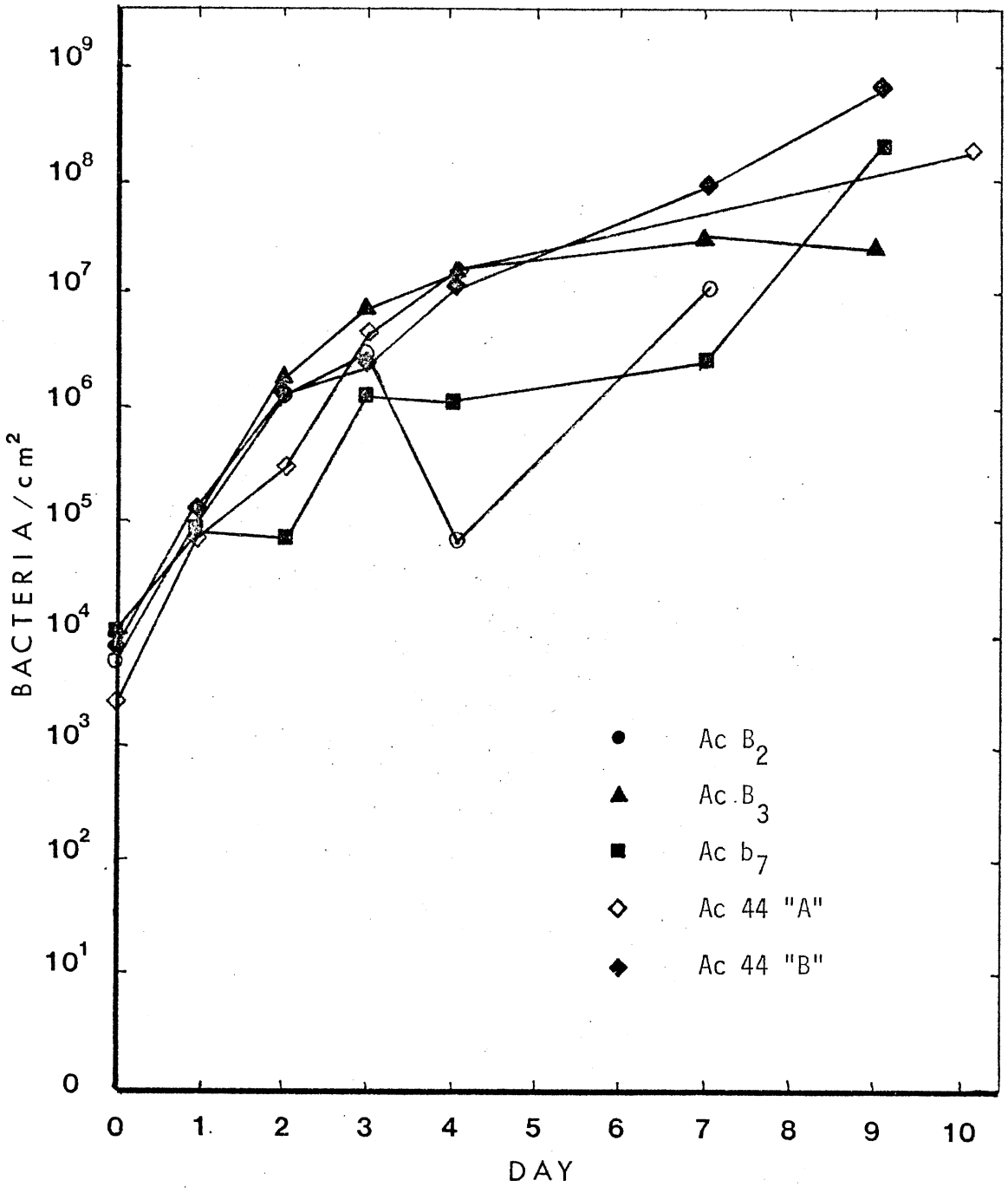

Fig. 15 Population Trends for Xanthomonas malvacearum Race 7 Inoculated Into Ac B_2, Ac B_3, Ac b_7, and Ac 44.

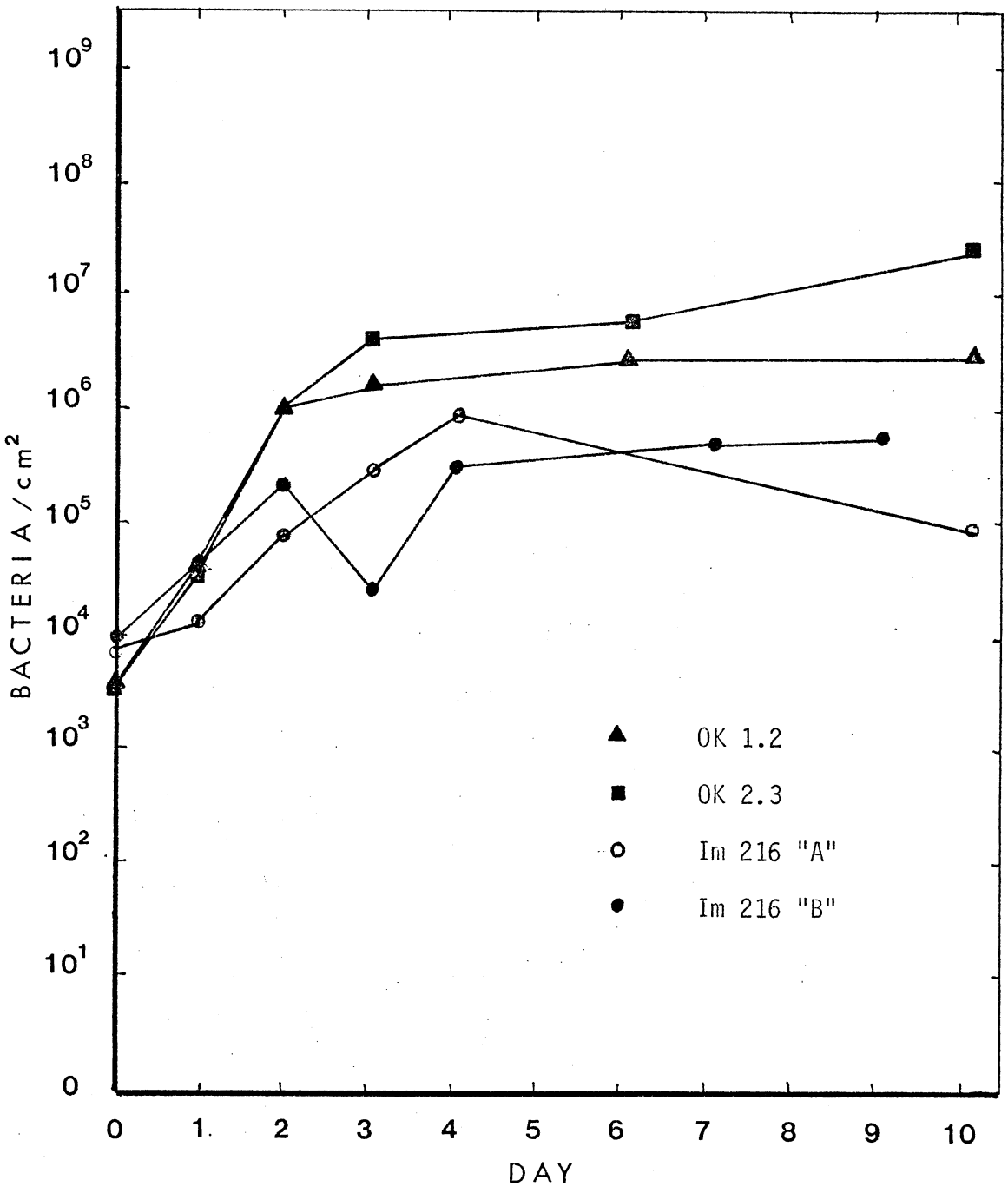

Fig. 16 Population Trends for Xanthomonas malvacearum Race 7 Inoculated Into OK 1.2, OK 2.3, and Im 216.

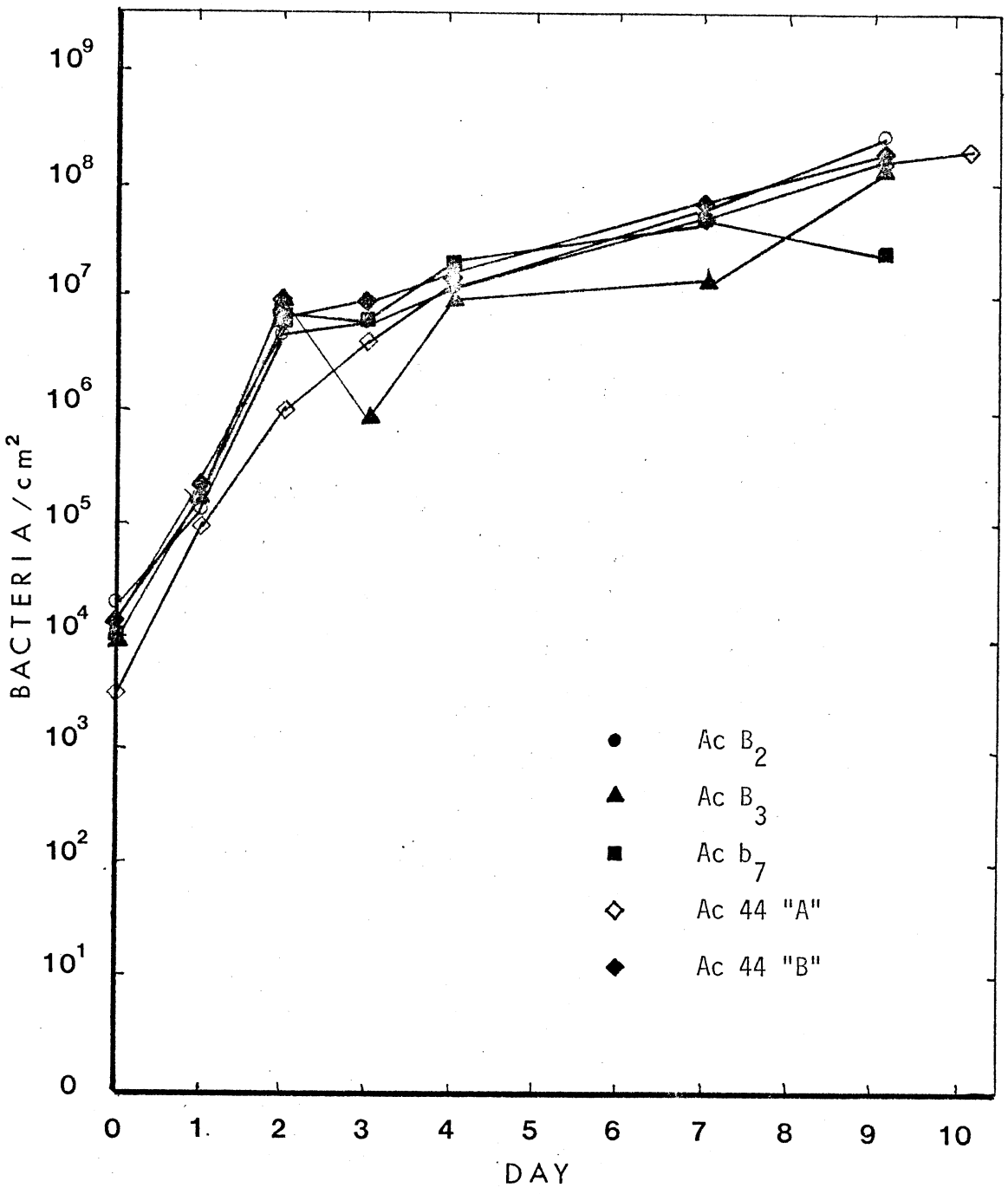

Fig. 17 Population Trends for Xanthomonas malvacearum Race 18 Inoculated Into Ac B_2, Ac B_3, Ac b_7, and Ac 44.

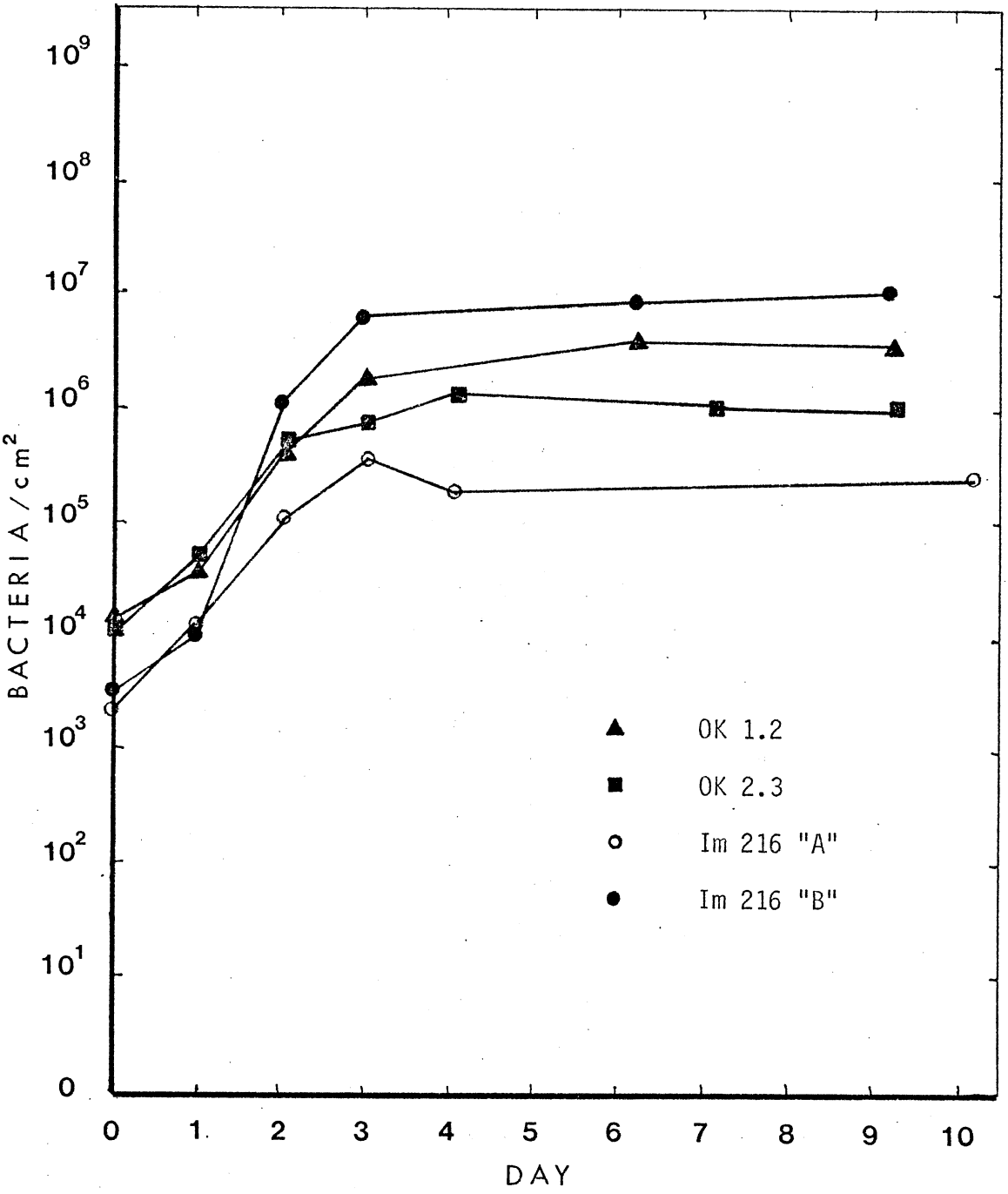

Fig. 18 Population Trends for Xanthomonas malvacearum Race 18 Inoculated Into OK 1.2, OK 2.3, and Im 216.

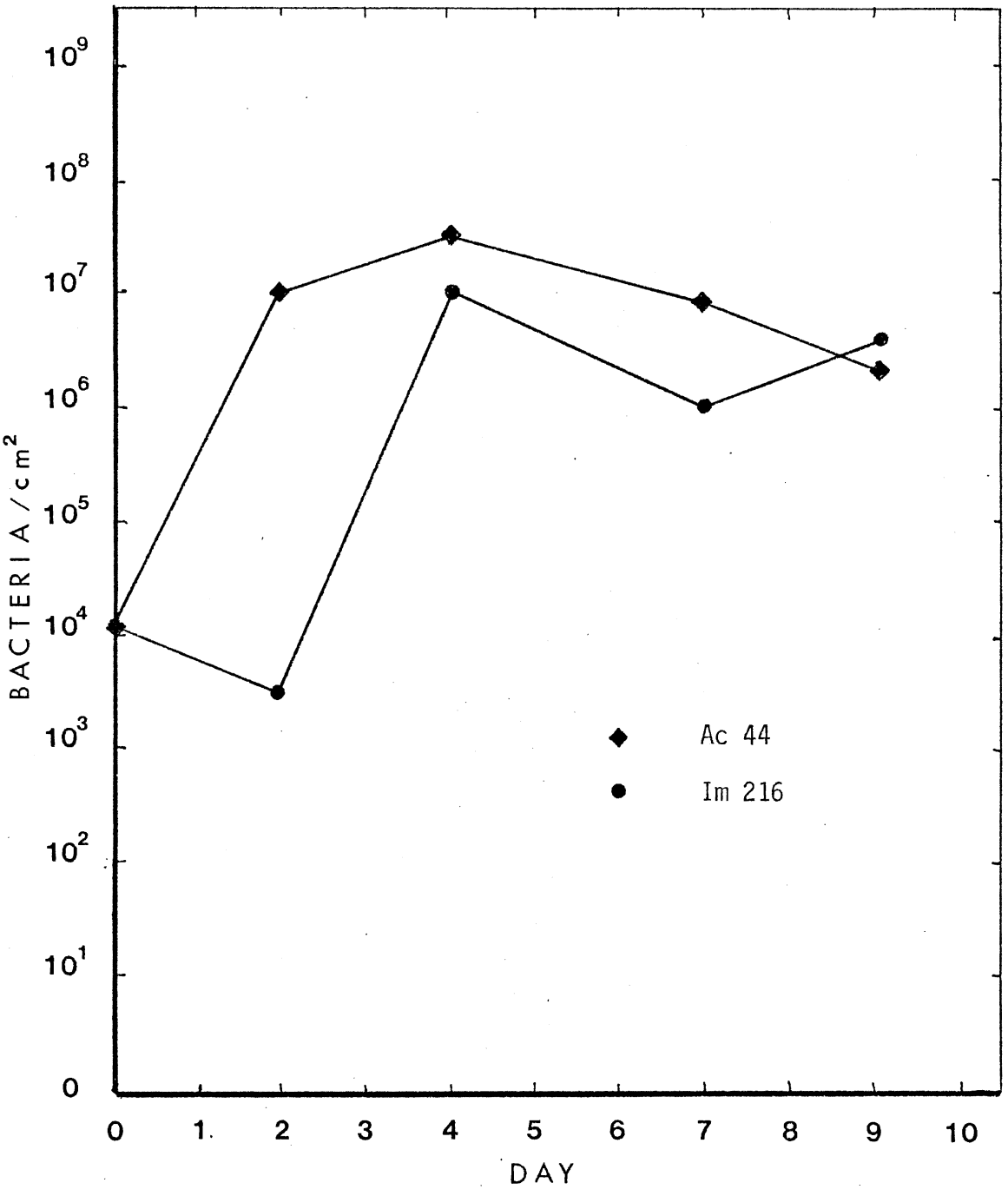

Fig. 19 Population Trends for Xanthomonas malvacearum Race 8 Inoculated Into Ac 44 and Im 216.

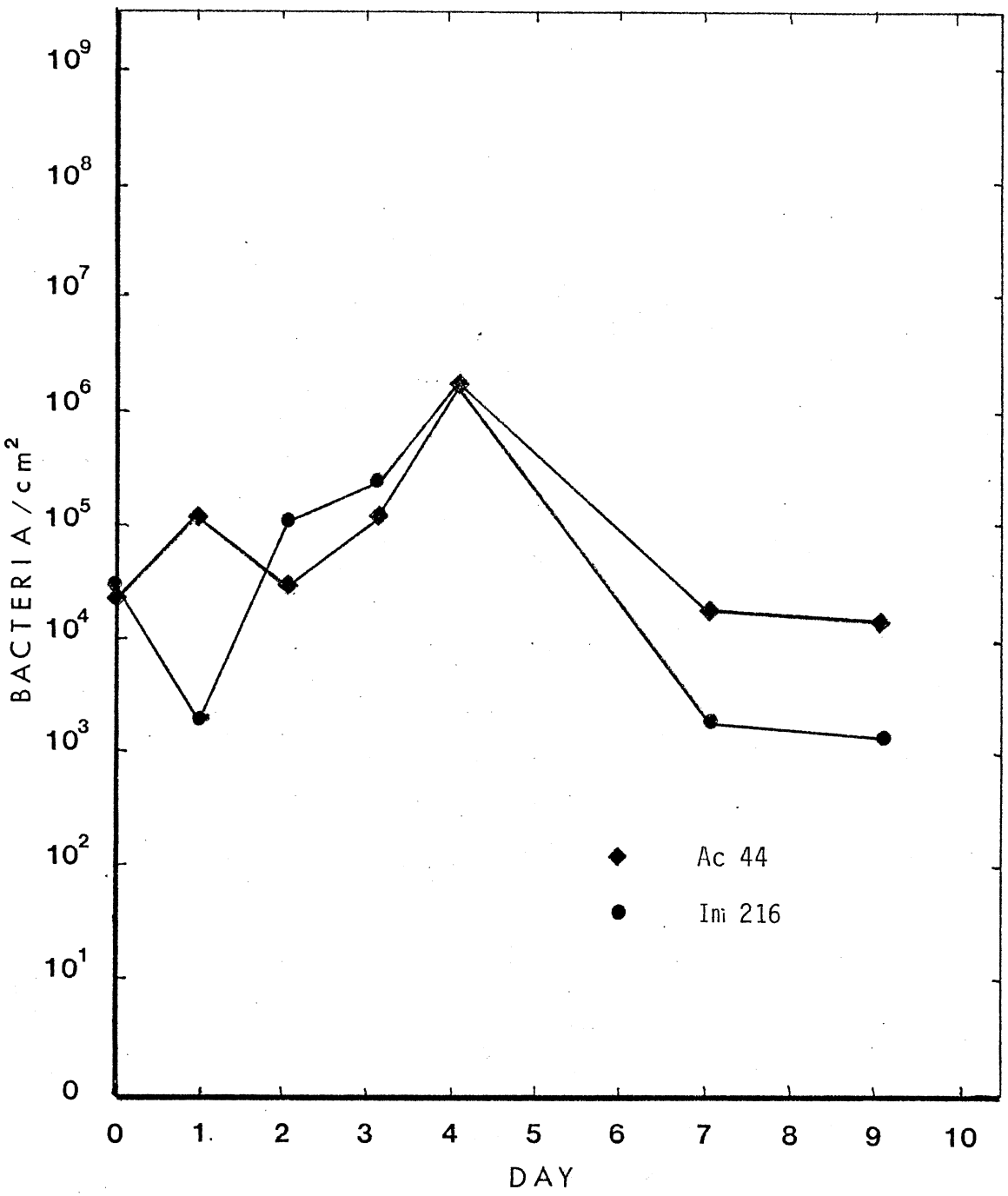

Fig 20 Population Trends for Xanthomonas campestris Inoculated Into Ac 44 and Im 216.

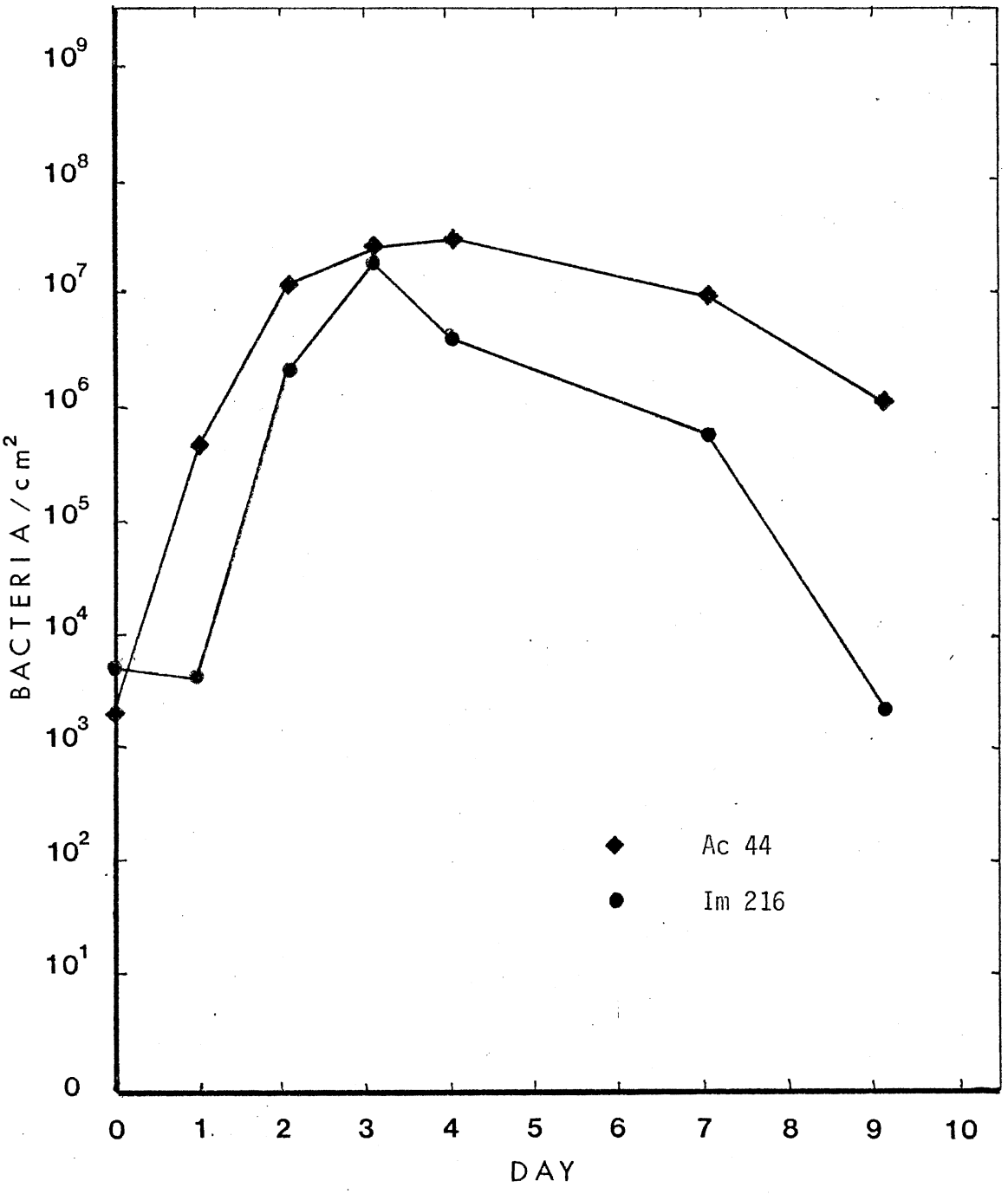

Fig. 21 Population Trends for Xanthomonas phaseoli Inoculated Into Ac 44 and Im 216.

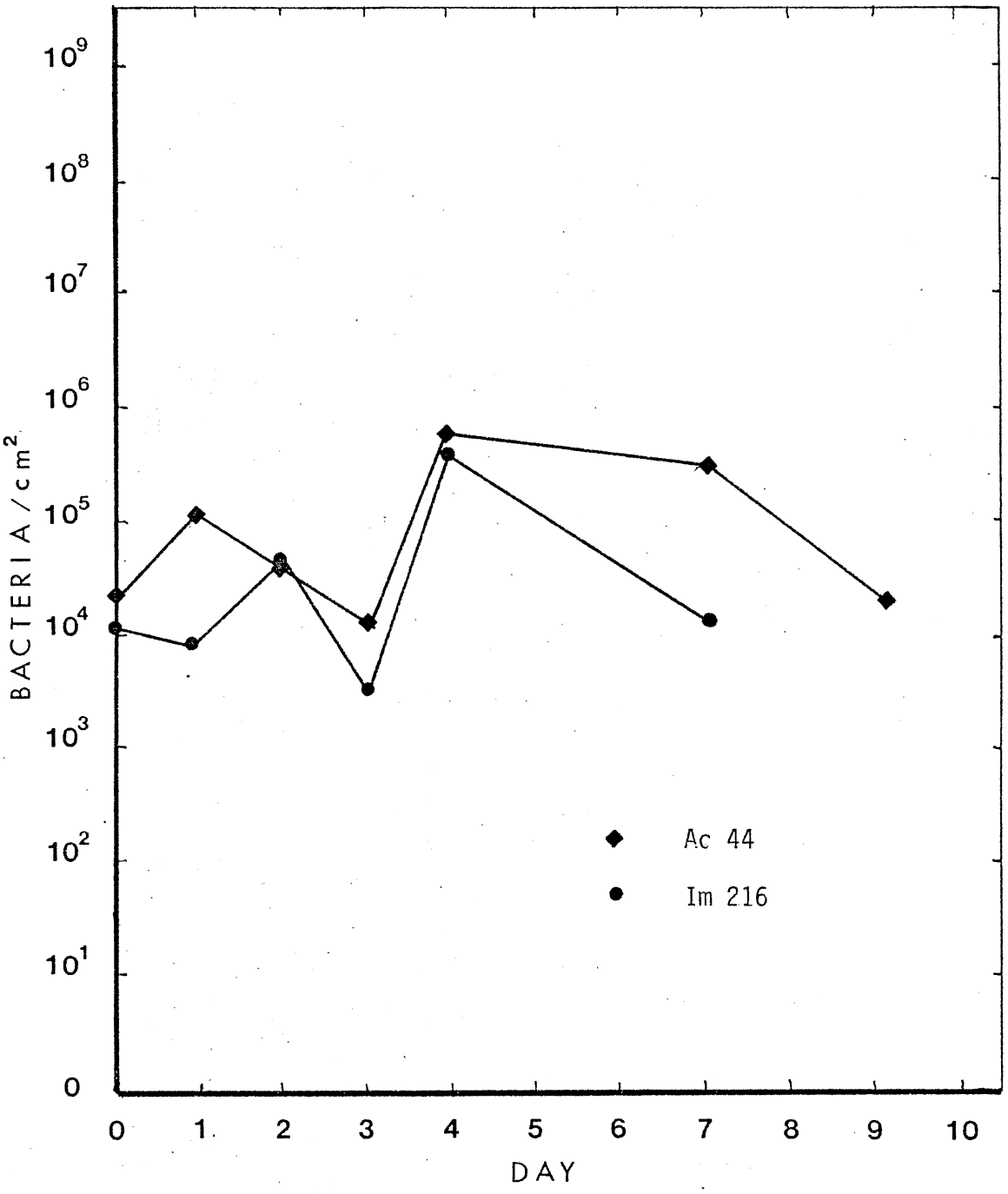

Fig. 22 Population Trends for Pseudomonas pisi Inoculated Into Ac 44 and Im 216.

VITA

Melanie Barnes

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Master of Science

Thesis: EVALUATION OF THE CONCEPT OF HORIZONTAL AND VERTICAL RESISTANCE BY THE COTTON-*XANTHOMONAS MALVACEARUM* HOST-PATHOGEN SYSTEM

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