University of Nebraska - Lincoln

DigitalCommons@University of Nebraska - Lincoln

Papers in Plant Pathology

Plant Pathology Department

1974

SURVIVAL MECHANISMS OF PHYTOPATHOGENIC BACTERIA

M. L. Schuster

D. P. Coyne

Follow this and additional works at: <https://digitalcommons.unl.edu/plantpathpapers>

Part of the [Other Plant Sciences Commons](#), [Plant Biology Commons](#), and the [Plant Pathology Commons](#)

This Article is brought to you for free and open access by the Plant Pathology Department at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Papers in Plant Pathology by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

SURVIVAL MECHANISMS OF PHYTOPATHOGENIC BACTERIA

♣3596

M. L. Schuster and D. P. Coyne

Departments of Plant Pathology and Horticulture-Forestry, respectively,
University of Nebraska, Lincoln, Nebraska 68503

INTRODUCTION

Natural habitats usually do not provide bacteria the continuity of agricultural crops. With continuous culture, perpetuation of pathogen is no problem. Although agricultural practices provide some discontinuity between crops, it is less than that in nature. Uniformity of crop germ plasm also favors inoculum buildup and perhaps perpetuation of the pathogens.

The growth of most plant pathogens is discontinuous, because of the seasonal effect upon either the pathogen or the host. A successful pathogen must be able to bridge discontinuities, such as the gaps between successive crops and seasons. To reestablish when conditions are again favorable, inoculum must survive. Facultative saprophytes or facultative parasites are not as handicapped by discontinuous growth as are obligate parasites. Discontinuous growth of the pathogen mainly decreases the amount of inoculum (bacteria that cause disease when placed in suitable contact with the host).

The success of a bacterial plant pathogen depends in part on the amount of inoculum (bacterial cells) it produces. Because bacteria have a short generation time, a small amount of surviving primary inoculum can rapidly produce an epidemic. What is the minimum amount of inoculum necessary to initiate disease? Enough for mere survival is not necessarily the answer since transmission to a host is necessary. The source, exit, and transmission of primary inoculum are all necessary for occurrence of disease, for survival, and for continuity of the bacterial species. Establishment of a "curtain" between host and pathogen may be simpler and cheaper during the primary inoculum phase than thereafter.

The longevity of primary inoculum is important in the success of bacterial pathogens and depends upon its ability to escape or endure adverse environmental conditions (62). Survival varies with the form of primary inoculum and may depend on external factors as well as on the internal makeup of the pathogen. Because there are about 200 different species of bacterial plant pathogens, variations in modes of

survival are certain to occur. The species included in this paper are limited to those that illustrate concepts and principles. Actinomycetes are excluded (14).

There are five genera of bacterial phytopathogens: *Agrobacterium* (A.), *Corynebacterium* (C.), *Erwinia* (E.), *Pseudomonas* (P.), and *Xanthomonas* (X.). They are aerobic non-spore-forming rods. Only *Corynebacterium* is Gram-positive. Most are motile with polar or peritrichous flagella, but a few are atrichous.

Phytopathogenic bacteria do not form resting spores or structures comparable to fungi or nematodes; they remain dormant during the quiescent period in association with the following animate or inanimate agencies: (a) seeds, (b) perennial plant hosts or parts, (c) insects, (d) epiphytes, (e) plant residues, (f) soil and other nonhost materials. Longevity of pathogens in these agents under natural and artificial environmental conditions are discussed in this review, and selected references are provided.

ASSOCIATION WITH SEED

"The flowers of all the tomorrows are in the seeds of today!" (22). The term *seed* is here used in a popular sense, and includes fruits, such as caryopses and achenes, but excludes vegetative propagules (e.g. seed potatoes). Seeds are an ideal agency for survival of plant pathogens when the growing host is lacking.

Orton (74) suggested in 1931 that any bacterial phytopathogen is likely to be transmitted by seed; he listed 59 such pathogens of the 128 species then described. In an annotated list of seed-borne pathogens, Noble & Richardson (73) included 95 species and varieties of bacteria, about half of the phytopathogenic species described. Baker & Smith (6) and Baker (5) outlined the essentials of the subject, referring to only a few bacterial species.

Many seeds, especially those of temperate zone plants, have a period of dormancy. Although vegetative cells of bacteria are not subject to dormancy per se, it is important that their survival correspond to that of seed viability. The occurrence of a virulent bacterium on, in, or accompanying a prescribed amount of seed does not assure transmission; various environmental and inherent seed factors could affect transmission. Transport of a bacterial pathogen in seed is an important means of survival and transfer in time and space. Another factor concerning effectiveness of seed transfer is the time of survival of the bacterium on or in seed. Most pathogens survive as long as the seed is viable.

Some bacterial pathogens die before seed loses its viability, while many survive even beyond the time of germinability. Those that infect *Phaseolus vulgaris* and other legumes are excellent examples, and *C. flaccumfaciens* was viable after 5 to 24 years (139). Schuster & Sayre (105) isolated virulent *C. flaccumfaciens* var. *aurantiacum*, *X. phaseoli*, and *X. phaseoli* var. *fuscans* from 15-year-old bean seed, and *C. flaccumfaciens* var. *violaceum* from 8-year-old seed kept at 10°C. Basu & Wallen (7) found viable *X. phaseoli* in seeds after 3 years at 20–35°C and nonviable in other 2-year-old seed. Although aging of seed was suggested as a possible control (13), it is too variable and unreliable for use as a control method. The discrepancies

in longevity of bacteria in beans apparently are due to storage temperatures and moisture, bacterial species and strains, and length of experiments.

The development of the seed-bean industry in the semiarid West was based on relative freedom from several bacterial pathogens (15): *P. phaseolicola*, *X. phaseoli*, *X. phaseoli* var. *fuscans*, *C. flaccumfaciens*, and *P. syringae*. However, the bean industry sustained disastrous economic losses during 1963–1966 from severe outbreaks of bacterial diseases. Halo blight occurred in commercial fields in Wisconsin (132) and Nebraska (101) planted with western-grown seed, as well as at Twin Falls, Idaho (41, 77, 132) where seed production is concentrated. A pilot 600-acre snap-bean acreage, for example, in McCook, Nebraska was devastated in 1964 by *P. phaseolicola* (101).

The importance of bacterial diseases to the bean industry demands the use of pathogen-free seed. Recent outbreaks of bacterial diseases of snap and dry edible beans may be attributed in part to weather favorable to halo blight (16, 41) and to changes in races of the pathogen (41) in Idaho during the 1963–1966 growing seasons. The occurrence of three bacterial pathogens in bean plants from certified seed emphasizes the problem of control of the bacterial diseases (93, 127). The explanation offered (40, 41) is that seed contamination might occur during harvesting and processing even though the disease is rare and difficult to detect by field inspection. This explanation may seem unlikely since the extensive epidemics in the 1960s imply more than rare amounts of seed infestation. Nevertheless a few infested seeds may result in considerable losses in humid bean production areas; for example, in Wisconsin a dozen seed infected with *P. phaseolicola* per acre (0.02%), distributed at random, promoted a general epidemic (132). Epidemiological work, especially in Canada, has shown that 0.5% seed infection with *X. phaseoli* var. *fuscans* can be disastrous (133). Sabet & Ishag (91) considered survival of *X. phaseoli* in *Dolichos* seeds of little practical significance even though 2% of seed had viable bacteria after storage for 549 days. They claimed that the symptoms occurring on seedlings were atypical for secondary infections, but typical for systemic infections. Nevertheless, secondary spread should be possible from systemically infected seedlings.

Sutton & Wallen (121) thought that *X. phaseoli* var. *fuscans* assumed epidemic proportions in Ontario, Canada shortly after the introduction of a new bean cultivar, Sanilac, emphasizing that a new pathogen may be introduced into an environmentally suitable region and become a serious problem. Widespread epidemics can occur when seed for large areas is grown in one locality when favorable conditions for disease occur. *P. syringae* has been isolated from Wisconsin- and Idaho-grown bean seed; Idaho beans did not exhibit the brown-spot disease in the field. Wisconsin beans recently sustained losses from this ubiquitous pathogen (47).

Taylor (125) found that a high proportion of bean seeds infected with *P. phaseolicola* failed to produce infected plants; this may account for very low levels of primary infection reported in the field. Taylor devised a means of quantitative estimation of seed infection, but no data are available on the level of seed infection necessary for primary infection. Ten percent of infected plants arose from infected seeds (125). What value is the detection of 3 bacterial cells per seed if it requires

100,000 to initiate seedling infection? Workers (47, 120, 125, 135) have painstakingly devised procedures to detect one infected seed in 10,000 to 40,000 or more, but the basic question regarding number of cells required to initiate seedling infection remains unanswered. Removal of infected seed based on discoloration did not eliminate all infected seed (125); a point not determined by Taylor was whether so-called clean seed would give rise to infected plants. The degree of success of transfer depends on the inoculum load on the seed; a heavily infested seed may not germinate or may give rise to a weak seedling that dies before emergence. *P. phaseolicola* is more apt to fall in this category than are other bean pathogens.

Most investigators agree that *X. malvacearum* overwinters principally in cotton seed fuzz which remains on the seed coat after delinting. The internal infection by the organism in seed rarely attains 24% in artificially inoculated bolls. On the other hand, Massey (66) reported that internal infection of seed occurs in nature, but this could not be demonstrated artificially. Brinkerhoff & Hunter (11), Hunter & Brinkerhoff (49), and others also reported internal infection. Stoughton (116) found no evidence of the bacterium within the seed; infections of the cotyledon margins occur during germination, and this process is influenced by environmental conditions. Fallen bolls containing seed furnish a source of overwintering bacteria, and volunteer seedlings from such bolls in the spring become a source of infection for the planted crop. Brinkerhoff (9) and Schnathorst (94) recovered the viable pathogen on the seed after several years, although it lost viability more rapidly than the seed. A relatively small amount of inoculum is required to initiate an epidemic (11). Conflicting results of survival in seed may be attributed to systemic vs nonsystemic infection. The preponderance of evidence indicates that angular leaf spot is nonsystemic; however, Massey (66) thinks it is systemic and that cotyledons are infected while still enclosed in the seed coat. According to him, the pathogen is never truly vascular but passes through the tissue external to the vascular strands or in the intercellular spaces of the cortical parenchyma. Wickens (136) claims the bacterium is on the seed as a surface contaminant of the micropyle. Control through acid delinting suggests that the pathogen is principally lint-borne (95, 124).

Bacterial leaf blight is recognized as one of the most important diseases of rice in Asian countries. *X. oryzae* overwinters on diseased grains stored in farmhouses as well as in rice straw. It normally is found in husk tissues (123, 131), and has not been detected in unhulled rice grains in Japan. It has been located in glumes and occasionally in the endosperm in severely infected rice in China (28). One of the most important sources of inoculum in India is infected seed, and seedlings from infected seed are usually diseased (24, 112).

Two bacterial diseases of corn deserve consideration from the standpoint of kernel infection and survival mechanisms. These are Stewart's bacterial wilt caused by *E. stewartii* and the recently discovered Nebraska leaf freckle and wilt induced by *C. nebraskense* (104). Both invade the kernels via the vascular system and contaminate the seed surface from bacterial exudate on the inner husks (51, 104, 109). The organisms are present in the old vascular tissue of the chalazal region of the endosperm (51). There is no evidence of *E. stewartii* in the embryo region. On occasion *C. nebraskense* is found around the embryo, but under these circumstances the

kernels are small and do not germinate (104). The vascular elements of the pedicel terminate in the chalazal region; further progression into the kernel is by dissolution of the chalazal areas, resulting in lysigenous cavities filled with bacterial ooze (51, 104). Both bacterial species were still viable and pathogenic in one-year-old kernels. Low seed transmission (about 2%) of *E. stewartii* was reported when kernels were planted in sterilized soil (80). Ivanoff (51), on the other hand, found no transmission to seedlings from infected seed in autoclaved or in field soil. Schuster et al (104) found that kernels infected with *C. nebraskense* gave less than 1% transmission when planted in autoclaved or in nontreated field soil. Explanations for the low amount of seedling infection from internally infected kernels are that both *E. stewartii* and *C. nebraskense* require wounding of host tissues for infection, and that there is a small percentage of infected embryos, which are the biological equivalents of infected seedlings. As in most seeds, the vascularization of corn kernels does not reach the embryo.

Shelled corn kernels usually retain the pedicels, providing a protective cover for their vascular elements and the infected chalazal regions. Because of this characteristic, 5-min treatments with tetracycline (100 and 200 ppm) or Clorox® (15%) were ineffective, whereas 10-min soak in mercuric chloride (1:1000) was effective in controlling *C. nebraskense* in the seed, based on plate culturing. Infection was decreased by 90% after 10 to 15-min soaking of *E. stewartii*-infected seed in mercuric chloride 1:1000, but chlorophol 0.25%, or several organic mercuries in up to 24 hr treatments were ineffective in decreasing the amount of infected plants from such treated seed (85). The percentage of transmission, which was at times over 50%, is surprising in light of reported average transmission of 2%. Since the experiments were field-conducted and infection data were recorded at the canning stage, insect transmission of *E. stewartii* might have been overlooked.

A few pathogens infect the seeds through the vascular elements (*X. campestris*, *X. incanae*). *C. michiganense* invades the tomato fruit via the vascular elements, but seed contamination results during extraction. Thus the tomato-canker bacterium accompanies the seed but apparently is external to it. Rates of transmission to seedlings from infested seed ranged from 1–4%. Strider (118) reviewed the variable data on rates of transmission of *C. michiganense* to seedlings from infested seed. A few diseased plants in a seed bed can result in a high percentage of diseased plants. Infested seed in a seed bed contaminate the soil as they decompose, resulting in root infection of adjacent plants. Schuster & Wagner (106) found that *C. michiganense* can infect unwounded tomato roots, resulting in 100% infected plants.

That seed anatomy is related to seed infection and transmission has already been demonstrated for two pathogens of corn. The testa of legume seed contains vascular elements that may be extensive in large seeds. In bean, cotton, cucurbits, and tomato the vascular tissue (raphe), a continuation of the funiculus, provides favorable sites for internal transmission of pathogens. The parasites are retained in the raphe and may also invade the embryo, e.g., *P. pisi* (108), *P. phaseolicola*, *X. phaseoli*, and other bean pathogens (139). The bean bacteria may gain entrance through seed openings, such as hilum, micropyle, and threshing injuries (40, 41, 139). The natural openings through tomato seed coats into the space between the testa and endosperm

cuticle is easily penetrated. In most annual plants bacterial pathogens may survive nonpathogenically on or inside the testa of dry seeds in a manner comparable to epiphytic saprophytic bacteria on plant parts.

The manner of germination may affect transmission of seed-borne bacteria. Seeds with hypogeal germination (Gramineae, pea, sweet pea) could affect infection by *C. fascians* (4), *P. pisi* (108), *X. translucens* (134), *C. nebraskense* (104), and others. This type of germination could limit transmission of bacteria that infect only aerial parts. Epigeal germination (beans, alfalfa, beet, cabbage, carrot, cotton, garden stock, lettuce, pepper, castor bean, and tomato) could favor transmission of seed-borne bacteria that infect the aerial parts (exemplified by *X. campestris* in cabbage, *P. phaseolicola* of bean, *X. incanae* of stock, *X. carotae* of carrot, and *C. michiganense* of tomato), but may limit pathogens infecting subterranean plant organs. These characteristics for five classes of seed-borne pathogens were compared by Baker (5). The success of seed-borne bacteria is dependent on their location on the seed, anatomical structure and germination type of seed, survivability, and the bacterial species itself.

C. sepedonicum, the cause of the ring rot of potatoes, can infect tomato seed, aiding its persistence and spread (58). The bacteria are restricted to the vessels of the xylem. Alfalfa seed grown in areas infested with *C. insidiosum* can provide an important means of survival and spread of this important pathogen (21). However, Cormack & Moffat (21) made no transmission studies to seedlings but thought the seed-borne organism could be introduced into the soil.

Leppik (64) made a plea to the First International Congress of Plant Pathology for post-entry surveillance of introduced seed as a safeguard against importation of seed-borne pathogens. New strains of *X. phaseoli* recently recovered from bean seeds from Colombia and Uganda were much more virulent than Nebraska strains (100). Localized production of pathogen-free seed has an inherent disadvantage in that diseases developing in these areas quickly spread through the distribution area, as shown by the bean seed infections in USA in the 1960s. Bean varieties with a narrow genetic base are vulnerable to a pathogen. This is especially true for snap beans because of the specific demands concerning the type and quality of beans (138). Through centralization of seed production, the producer may eliminate or minimize important problems for his entire clientele through distributing clean seed, if other infection sources are eliminated. Some seed-borne pathogens persist in soil in infested plant residues and serve as an infection reservoir for succeeding crops. Nevertheless, such other sources for seed-borne pathogens are not requisite for perpetuation of the diseases.

ASSOCIATION WITH PLANT RESIDUES

Although the primary inoculum source of *Xanthomonas malvacearum* is infested seed, the pathogen persists in dry plant tissues for years (9, 66). The bacterium in infested debris buried in soil was infective until debris was thoroughly decomposed (10). In the arid climate of Sudan, infested debris is a threat to the next cotton crop.

In moister Tanganyika, *X. malvacearum* barely survived between cotton crops in refuse on the soil surface.

Although seed free of *Xanthomonas phaseoli* is commonly planted, common blight occurs where crop rotation is not practiced (139). *X. phaseoli* survives at least one winter in infested bean straw (96, 102). *P. phaseolicola* can live overwinter in stems, pods, and leaves on the soil surface in New York (71). Infested bean straw is important in the overwintering of *P. phaseolicola* (Race 1, Race 2, and Nebr. 16), *X. phaseoli*, *X. phaseoli* var. *fuscans*, *C. flaccumfaciens*, *C. flaccumfaciens* var. *aurantiacum*, and *P. syringae* (98, 99, 101). Bacteria in infested straw on the soil surface survives better than in debris buried 8 inches in the soil. Recovery 22 months later was successful.

Bacteria survived best in dry bean debris (91, 98, 99). Brinkerhoff & Fink (10) found that *X. malvacearum* lost its viability sooner in nonsterilized soil than autoclaved soil. Under furrow irrigation in the semiarid Western United States, infestation with dried bean dust may be an important seed contamination, even when halo blight is rare or difficult to detect by field inspection (40, 41).

Rice stubble and straw from infested fields are sources of inoculum of *X. oryzae* in Japan (123, 131). *X. oryzae* survives until spring in dried rice straw in farmhouses, but only one or two months when straw is plowed into soil. Bacteria can survive in rice stubble in the field in the warm areas, but not in northern Japan (69).

X. vesicatoria overwinters in infested debris in tomato fields (1, 81).

Erwinia stewartii was not recovered from soil under a diseased corn crop, but was from overwintered corn stubble (51, 80, 87). *Corynebacterium nebraskense* (103, 104) and *E. stewartii* invade the vascular elements and parenchyma, producing profuse amounts of bacterial slime in the stalks and leaves. *C. nebraskense* also overwinters in the field in corn residues, particularly on the soil surface.

ASSOCIATION WITH PERENNIAL HOST

Survival of pathogenic bacteria in perennial plants requires conditions different from those in annuals. Bacteria remain in the living, but perhaps dormant tissues of the perennial host during the off-season in temperate climates. In the tropics or where the intervals between crops is brief, the dry or dormant period may be brief or absent. Certain pathogens, such as *X. juglandis*, *P. mors-prunorum*, *X. citri*, *E. amylovora*, and *X. pruni* (23) overwinter principally in holdover cankers or blighted twigs, which may produce a bacterial exudate furnishing initial spring inoculum. Varieties most subject to fire-blight blossom infection, have, as a rule, the most overwintering sources of primary inoculum.

Although small percentages of *E. amylovora*-induced cankers contain viable bacteria, it is generally agreed that under favorable conditions a small number of holdover cankers is sufficient to initiate a spring epidemic of blight. Percentages of blight cankers harboring viable *E. amylovora* ranged from 2.0 (89) to over 50 in California (128). Viable *E. amylovora* in a blight canker is limited to the region immediately surrounding the periphery of the discolored margin. Rosen's (89)

report that cankers with well-defined, suberized, and cracked margins maintain the parasite over winter is in error.

Survival in holdover cankers does not adequately explain severe fire blight in orchards where the disease has not been noted for one to several years (53). Virulent *E. amylovora* may exist as a natural resident in healthy stems, shoots, and buds of apple and pear (54) and move into newly developed shoots, with or without producing blight symptoms. A severe outbreak of fire blight of resistant pears in northern Arkansas was associated with a severe hailstorm suggesting that the causal organism was internal (52, 129). Epidemics in orchards where the disease has not been observed may also result from wind dissemination of aerial bacterial strands. In a comparable situation, walnut catkins provide a perennial source of infection, and when mature they shed contaminated pollen; this explains the epidemic occurrence of *X. juglandis* when only a few cankers are evident (3).

Two alfalfa pathogens, *Xanthomonas alfalfae* and *Corynebacterium insidiosum*, overwinter in the host (19). Wounds are necessary for infection by the wilt bacterium (*C. insidiosum*) but not for the other pathogen. Winter frosts and mowing provide injuries for entry of the wilt pathogen. Using the invaded vessels as indicators, it was found that some active infections had occurred 17 years before. Infected plants usually succumb after the second year, but apparently spring inoculum can arise for many years from persistent infected alfalfa plants.

EPIPHYTES AS INOCULUM SOURCES

Although considered to be poor saprophytes in natural soils, phytopathogenic bacteria manifest a diversity of facultative combinations with higher plants. Evidence is accumulating that different plant organs and plant species have characteristic epiphytic bacterial floras (61, 82). These epiphytes have been found on roots (rhizoplane), buds (gemmiplane), and leaves (phylloplane). Epiphytic organisms may also survive on seed surfaces. Recent investigations have stressed the occurrence of phylloplane bacteria.

Epiphytic bacteria may be a source of primary inoculum. Hagedorn et al. (43) recovered *P. syringae* throughout the year from leaf surfaces of healthy *Vicia villosa*, and associated natural outbreaks of bean brown spot with the epiphytes on nonsusceptible hairy vetch. *P. syringae* was also isolated from bean debris overwintering in Wisconsin until April, but not May (47). Schuster (99) recovered *P. syringae* in Nebraska in May and June from overwintered bean debris. The discrepancy may be due to sensitivity of isolation methods, or differences in strains of the pathogen or in environment. That this ubiquitous pathogen resorted to resistant nonhost plant species for its survival was unexpected.

¹⁴Haas (42) found under artificial inoculations that *X. phaseoli* var. *fuscans* survived on the phylloplane of primary leaves of *Phaseolus vulgaris* cultivar Sanilac, but disappeared quickly from the unifoliate leaves. Mew & Kennedy (67) found that varieties of *Glycine max* differentially supported strains of *P. glycinea* epiphytically; on susceptible leaves the bacterium increased 1000-fold within 2 weeks, but it remained unchanged or declined on the leaf surface of resistant cultivars. Race

specificity of *P. glycinea* was correlated with the resident phase of the pathogen on the phylloplane of soybeans. Leben (61) and co-workers showed that three pathogens have a resident phase on host plants, namely *X. vesicatoria* on tomato, *P. glycinea* on soybean, and *P. syringae* on beans.

The phylloplane growth of *P. mors-prunorum* was suggested as a primary source of inoculum for infection of branches of stone fruit trees in the autumn (23). This and another important pathogen of stone fruits, *X. pruni*, have a foliar stage that alternates with an active winter stage in the branches or stems. Although the role of leaf epiphytes in the annual cycle of the disease is still obscure, fewer cells of *P. mors-prunorum* resided on leaves of resistant cherry variety than on susceptible ones. This was substantiated, using two species of bacteria on cherries and pears, with survival on the natural host in each instance (23). *X. citri* survived overwinter as a leaf epiphyte on 17 nonsusceptible weeds in citrus groves (39).

Bud epiphytes may serve as a primary inoculum source. Although they can be found on annuals, they would be especially important in perennials (61). Goodman (35) recovered overwintering *Erwinia amylovora* and *E. herbicola* from healthy apple buds; *E. herbicola* was inhibitory for *E. amylovora* (36).

Research of the aerosphere flora is limited, but that of the rhizosphere and the vicinity of roots is more extensive. Epiphytic bacteria migrate from seed and may be found on mature plants in the field. Certain phytopathogenic bacteria are able to survive in the rhizoplane of nonhost plants. *Pseudomonas tabaci* and *P. angulata* colonize root surfaces of wheat, clover, vetch, and certain weeds, which thus might be their overwintering sites; these organisms may persist in the soil indefinitely, apparently in association with roots. *X. vesicatoria* was found to overwinter on wheat roots but *P. phaseolicola* and *X. phaseoli* var. *sojense* did not (25).

Stanek & Lasik (113) discovered *X. phaseoli* var. *fuscans* colonizing bean roots, but it disappeared after two weeks; root exudates retarded the pathogen, and seed exudates stimulated its growth. Apparently plant metabolism is altered during the transition from the cotyledonary stage to the photosynthetic assimilation stage. Diachun & Valleau (25) initiated isolation studies of bacteria from wheat roots one month after seeding; that bean and soybean bacteria survived during the early development of the wheat was demonstrated in gnotobiotic tests with plants less than a week old. *X. malvacearum* isolated from roots of 14 different weed species in blighted cotton fields were thought to be unimportant in overwinter survival of the pathogen because samples collected in winter were nonpathogenic. Perhaps *X. citri* found in low numbers the year round on rhizomes and roots of *Zoysia japonica* could be important in survival, although the different strains on citrus and *Z. japonica* differ physiologically (39). Outdoors *X. citri* survived 6 months on the surface of *Calystegia japonica* rhizomes and for 5 months in the soil. Proof is still lacking that the rhizoplane bacteria on this common weed can provide primary inoculum for citrus infections. It is questionable whether the rhizoplane bacteria represent a partial and possibly transitory extension of the phylloplane phase. This might depend on the interactions of the pathogenic bacterium, host, and other microorganisms involved (33, 42, 113). Gibbins (33) thought that there was a paucity of data on relationships between pathogenic and nonpathogenic bacterial

epiphytes on leaves, and that it was necessary to investigate mixed populations of microorganisms in the different aspects of their biology.

ASSOCIATION WITH NONHOST MATERIALS

Many plant parasitic bacteria may reside in nonhost materials such as soil, or in plant parts. We should determine whether the bacteria survive in host parts or are free living in the soil. A case in point is the assumption that *X. phaseoli* (97) survived in the soil with the exact inoculum source not ascertained.

Buddenhagen (12) categorized phytopathogenic bacteria in soil into three groups: transient visitors, resident visitors, and residents. In the first group the soil phase is a rapidly declining one, commonly not contributing to the perpetuation of the pathogens. Perhaps most phytopathogenic bacteria fall in this category, although data are incomplete: *E. stewartii*, *E. amylovora*, *E. tracheiphila*, *X. citri*, *X. vesicatoria*, *X. vasculorum*, and *E. rubrifaciens* (92). Several other xanthomonads were found to overwinter in natural soil, probably in debris of diseased plants: *X. campestris*, *X. malvacearum*, *X. juglandis*, *X. vesicatoria* (1), *X. oryzae* (69), *X. translucens* (134), and *X. phaseoli*, and *X. phaseoli* var. *fuscans* (96).

X. citri underwent a rapid and continuous population decline in different nonsterile soil types tested, and vanished in about two weeks (31, 63, 79). Similar results were obtained with infested leaf debris.

Although Peltier & Frederich (79) and Goto (39) thought that *X. citri* gained entrance into the outer bark tissue through lenticels and remained dormant through the winter months, the latter worker contended that the pathogen also overwintered in soil. Peltier & Frederich (79) attempted direct soil isolations by germinating citrus seeds in infested soil. Goto (39) claimed that seedlings grown in infested soil with high *X. citri* numbers would not become infected, but that populations of 10^2 cells/ml could easily be detected by a leaf-infiltration technique. The extent to which results of the workers from USA and Japan reflected the sensitivity of the isolation method should be examined. Both Lee (63) and Fulton (31) used a very sensitive method involving inoculation of punctured leaves with soil infusion, not unlike the multineedle inoculator used by Goto. Both Lee and Fulton employed varied conditions that were much more severe than would occur under the most favorable natural conditions for spread of the bacterium from soil to plants. Isolation of *X. citri* from soil under severely infected grapefruit trees or after removal of infected trees was negative (79). Goto (39) had no direct evidence that *X. citri* at low concentrations in soil or nonhost plants served as a source of primary inoculum in the orchards under natural conditions. The work of Lee (63), Fulton (31), and Peltier & Frederich (79) is substantiated by eradication of citrus canker in citrus areas in United States through systemic destruction of diseased grove and nursery trees and by use of strict sanitation. Despite claims by Goto and co-workers, *X. citri* may not possess sufficient survivability to long maintain populations in the soil phase. Because of the contagious nature of the pathogen it seems improbable that survival in soil and nonhost plants could serve as primary inoculum sources.

Corynebacteria do not survive very long in the free state, but may overwinter in soil. *C. sepedonicum* (110), *C. insidiosum* (72), *C. nebraskense* (104), and *C. flaccumfaciens* (13) have poor survival in natural soil. Strider (118) thought that *C. michiganense* could persist in soil for a long period of time. The pathogen kept in tubes of air-dried soil persisted outdoors for five years. Circumstantial evidence of survival for several years in field soil is commonly reported without experimental proof. *C. fascians* is common in soil (4), but there is some uncertainty regarding its biology (12) and taxonomic position there.

Certain pseudomonads are incapable of persisting in the free state in natural soils for extended periods: *P. syringae*, *P. pisi*, *P. phaseolicola*, *P. solanacearum* race 2, *P. tabaci*, *P. glycinea*, *P. lachrymans*, *P. mors-prunorum*, *P. savastanoi*, *X. pruni*, and others.

Buddenhagen's second category is characterized by organisms whose numbers gradually decline in soil; their long-term occurrence there is host-dependent, and their populations increase or gradually decrease according to cropping practices. Pathogens with an extended soil phase include *A. tumefaciens*, *P. solanacearum* race 1, and *E. carotovora*. The pathogens *P. solanacearum* and *Agrobacterium* spp. can be considered true soil-borne pathogens, yet both are wound parasites. Host wounding may be common.

Few bacteria survive in soil in a free state. One of the most successful pathogens in this regard is the common and important *P. solanacearum* race 1. This pathogen survives four to six years under bare fallow or up to 10 years in soils cropped to nonsusceptible plants (55). The disease may occur in the first planting of a susceptible crop on virgin land; this has been attributed to the presence of susceptible weed hosts in the natural flora (107). Because of the pathogen's low tolerance of desiccation, increase in microbial antagonism, exposure to sunlight, and absence of weed hosts, it gradually decreases in some soils under cultivation. Since these reports have been based on the occurrence of brown rot in crops replanted in the field, the differences may be due to soil factors and bacterial strains (12).

The brown-rot pathogen enters the plant through wounds in the roots and invades the xylem, inducing wilt and possible death of the plant (56). Although races 2 and 3 are transmitted independently of the soil, soil is the principal source of inoculum. Loss of viability in infested plant residues is associated with desiccation rate of tissue. Persistence in potato tubers was comparable to that in cultures exposed to cold-storage temperature. This is not in agreement with Smith's (109) report that the bacterium can survive in vitro exposure to -77°C .

Some aspects of ecology of *P. solanacearum* are still obscure (23). It has a host range of 200 plant species, wider than that of any other bacterial plant pathogen (55). Its host range, occurrence as pathogen in indigenous plants, and adaptability are consistent with root inhabitants (32). Records of the distribution of *P. solanacearum* are based more on the occurrence of disease than on the occurrence of the species. It may be widely distributed as a harmless rhizoplane organism.

A. tumefaciens, the crown-gall bacterium, is universally distributed. This wound parasite commonly survives in soil long enough to infect crops the following year. The bacteria can be reisolated after 669 days in artificially infested nonsterile soil

without plant cover, but they gradually decrease in numbers in natural soils. They are favored by moderate temperature, alkaline, and moist soil (26). Field observations of *A. tumefaciens* suggest it is capable of long survival in soil, but, as with *P. solanacearum*, this interpretation is confused by the extensive host range, survival in plant residue, and independently distributed strains in plant material. There is, however, some evidence of prolonged persistence in soil in warm moist climates. The *Erwinia* soft-rot bacteria may be included in the category with *P. solanacearum* and *A. tumefaciens*. However, research might show them to be root epiphytes.

Buddenhagen's third ecological group of phytopathogens is typified by bacteria that reproduce in soil and by root epiphytes whose relation to plant disease is ephemeral. These include green-fluorescent *Pseudomonas* spp. that produce soft rot of plants in or near the soil (27), as well as rhizoplane bacteria and true soil saprophytes. Some of the *Erwinia* (*Pectobacterium*) soft-rot bacteria also might be included here. Vorokovich (130) thought that *Erwinia* soft-rot bacteria were not capable of long persistence in soil but survive in plant residue until it is decomposed. Evidence on soil relations of soft-rot bacteria of *Erwinia* is conflicting (12).

A frequent explanation for poor survivability of plant pathogenic bacteria in soil is that they are inhibited by antagonistic microflora. Brian (8) discussed antibiotic production in this respect. Patrick (78) demonstrated that many soil actinomycetes, bacteria, and fungi are antibiotic to phytopathogenic bacteria in culture. Of 1200 microorganisms, 120 produced large inhibition zones with 28 species of plant-pathogenic bacteria. Addition of organic matter to soil increases antagonistic microorganisms and decreases *A. tumefaciens* (26). In mixed cultures, antagonistic bacteria decreased infection by several pathogens (13, 126).

Bacteriophages are common in soil in association with diseased plants (23) and have been thought to reduce bacterial pathogens in soil (66), but this seems improbable. The requisite for control are high bacteriophage concentration and a low concentration of sensitive bacterial cells. Anderson (2) reported that, under favorable situations in vitro, the lowest initial concentration of a virulent bacteriophage required to eliminate a single cell of *Salmonella typhi* was about 10^7 particles/ml. He concluded that change in natural bacterial numbers due to elimination of sensitive cells was improbable. Crosse (23) found that soils enriched with concentrated suspensions of *P. syringae* and *P. mors-prunorum* have rarely exceeded 10^2 phage particles/ml of soil suspension. This yield would only lyse two or three cells, and the chance of absorption onto sensitive cells would be remote. Sutton & Wallen (120) failed to detect *X. phaseoli* in soil from infested fields or to isolate phages of this pathogen without phage enrichment. It is improbable that bacteriophage significantly affects the occurrence of *X. phaseoli* strains. Obligately parasitic bdellovibrios (114, 115) or predacious protozoans and free-living nematodes should be studied with respect to bacterial survival.

Certain bacteria can survive on nonhost materials other than soil. An example is *C. sepedonicum* which persists on planting equipment, harvesting and grading machinery, sacks, and storage bins, and is resistant to heat and desiccation (86). Planting and harvesting equipment can also be survival sites for *X. malvacearum* (94) and *P. phaseolicola* (40). The importance of nonhost agencies should not be

minimized for contagious organisms such as *C. sepedonicum*, *P. phaseolicola*, and *X. malvacearum*.

ASSOCIATION WITH INSECTS

Insects are *socius criminis* with certain bacterial pathogens in inciting plant diseases. A few bacteria transmitted primarily by insects are capable of survival within the bodies of the insects vectors. Specialized relationships between the bacterial pathogens and the transmitting insects have been summarized (17, 59).

Because seed and soil transmission have failed to explain the prevalence and dissemination of Stewart's bacterial wilt of corn, insect vectors have been extensively studied. The geographical distribution of this disease coincides with the prevalence of the 12-spotted cucumber beetle (*Diabrotica undecimpunctata howardi*), the corn flea beetle (*Chaetocnema pulicaria*), and the toothed flea beetle (*C. denticulata*). Dissemination of the wilt organism by adult 12-spotted cucumber beetles is not important, although the insect harbors the organism in its alimentary tract for considerable periods of time. Field observations and tests in Maryland demonstrated that most of the late spring and summer infection occurred from bacteria carried by corn flea beetles.

Of 40 insects studied, *E. stewartii* was isolated only from the intestinal tract of overwintered adults of the corn flea beetle *C. pulicaria*. Out of every 100 of these insects from different hosts and localities, 75 contained the wilt bacterium. Robert (87) found that 10–20% of the beetles emerging from hibernation carried *E. stewartii*; up to 75% of the beetles feeding on corn in midsummer may be carriers. Root-feeding insects (*Phyllophaga* spp., *Diabrotica longicornis*, *Hylemya platura*) expedite invasion by *E. stewartii* of seedlings from infected seed due to root injury. Since 160 of the more than 350 species of insects attacking corn cause noticeable damage, it is possible that other insects can also act in transmission and survival of the bacteria.

There is an apparent relation between winter temperatures and prevalence of Stewart's bacterial wilt. Low temperatures decrease the number of pathogen-harboring insects that overwinter. Forecasting of this disease is based on the sum of the mean temperatures for December, January, and February. In winters with mean temperatures above 37–38°C, large numbers of beetles survive. Cold winters (sum of mean temperatures below 32°C) reduce the populations and limit disease development (80).

C. nebraskense, causal agent of leaf freckles and wilt (LFW) of corn, is very similar in symptomatology to Stewart's disease. It does not overwinter in the corn flea beetle, *C. pulicaria* (104). LFW could become a problem in other corn-growing areas if a relationship similar to that in Stewart's disease were established with insects such as the corn flea beetle.

The cucurbit-wilt bacterium, *E. tracheiphila*, is completely dependent on cucumber beetles for its survival between seasons. Smith (109) thought that the striped cucumber beetle (*Acalymma vittatum*) was the only disseminator of the pathogen. Careful research showed that primary infection was not associated with soil or seed,

but that the hibernating adult striped cucumber beetle (*A. vittatum*) and the 12-spotted cucumber beetle (*D. undecimpunctata howardi*) harbor the pathogen overwinter in their intestinal tracts and transmit it. Primary infection in the spring originates from feeding punctures of such overwintered beetles. The bacterium was recovered from a relatively small percentage of the overwintered beetles tested, but this is adequate to establish infection centers for secondary spread. The dependence of the pathogen on the insects is therefore complete. Feces from infective beetles contain virulent bacteria and may serve as primary inoculum if dropped into fresh wounds. Overwintered adults of *A. vittatum* feed on plants of wild cucumber in the spring before migration to cucurbit seedlings, and may obtain fresh inoculum. Infection of cucurbits therefore may be from secondary spread, a primary inoculum source, or both. Weather conditions that affect the abundance of cucumber beetles also influence the prevalence of bacterial wilt of cucurbits.

The potato blackleg organism, *E. atroseptica*, can live in all stages of the seed-corn maggot (*Hylemya platura*), and may persist in this insect, even though it can also persist in tubers and soil (59). The insect increases disease incidence by transporting the pathogen and placing it in ideal infection courts. Because *E. atroseptica* is a facultative anaerobe, and cork formation by the host is diminished in wet soils, infection is increased by wet, poorly drained soils. Leach (59) demonstrated that the bacterium is present in the intestinal tracts of both adult flies and larvae. Since the pathogen survives pupation, the emerged adult may contaminate eggs as they are laid. *E. carotovora* survives in the castout linings of the fore and hind intestines and in the lumen of the pupal midintestine. Survival in the fore and hind intestines must be entirely saprophytic. Leach (59) believed that soft rot of crucifers associated with cabbage maggot (*H. brassicae*) was similar to potato blackleg.

Survival of bacterial pathogens in the alimentary canals of insects is not, however, an assurance of transmission. Beetles in the subfamily Chrysomilidae commonly regurgitate their food and have been shown to transmit plant viruses (30). It is logical that, in representatives (*A. vittatum*, *D. undecimpunctata howardi*) of this subfamily, the mechanism of transmission could be by regurgitation. Other related beetles (*C. pulicaria*, *C. denticulata*) in the subfamily Galerucinae also regurgitate and presumably thus transmit internally borne bacteria. Feces, regurgitated fluids, and crushed bodies of the insects were found to carry the bacteria.

It is unlikely that symbiosis is involved in the insect-pathogen relationship of bacterial wilt of cucurbits and Stewart's bacterial wilt of corn since the vectors of both are chewing insects, and the bacteria in plant tissues on which they feed enter and pass through the intestinal tract. The *E. atroseptica* seed-corn maggot association suggests symbiosis; the association cannot be parasitic because the bacteria develop on castoff tissues. No specificity is associated with the pupation-surviving bacteria.

The relationship between *Daucus oleae*, the olive fly, and *P. savastanoi*, cause of olive-knot disease, is in a different category from the above cases. The pathogen enters the egg through the micropyle. The diverticulum is a reservoir for the pathogen, from which the alimentary tract of the fly becomes contaminated. These spe-

cialized organs indicate a highly developed association. The olive fly is not, however, essential to survival and transmission of *P. savastanoi*.

A number of aspects of insect-bacterial associations need investigation. Strains of *E. stewartii* of differing virulence should be investigated to ascertain the effect of virulence on survival in the intestinal tract and in passage through wild host plants. A similar situation may occur in cucurbit wilt. Indiscriminate feeders, such as the cucumber beetles, should also be studied. Other areas for study include determining the minimal bacterial dosage, and the effect of pathogen retention and mode of inoculation on efficiency of transmission. Does the pathogen multiply in the vector, and is it there symbiotic or pathogenic? Aposymbiosis is an antibiotic method to demonstrate symbiosis in insect vectors. For example, antibiotics eliminated pseudomonads in maggots, especially *P. savastanoi*, the microsymbiote of *D. oleae*. Streptomycin applied to adult flies prevented larval development in olives. Thus it is possible to treat plants simultaneously against hibernating pathogens and symbiote-dependent insects (57). What is the relationship of plant-pathogenic Rickettsia-like bacteria and insects in the epidemiology of a disease such as Pierce's disease of grapes? Could the leafhopper be a winter carrier of the Rickettsia-like bacterium (34)?

DISCUSSION

Without means of distribution and the ability to live through the winter in the temperate zone or the dry periods in the tropics, a plant pathogenic bacterium would not long survive. The common pathogens are perhaps universal because they have succeeded in both dissemination and survival.

The mechanisms pathogens adopt in overwintering are few and uncomplicated. Survival through unfavorable conditions can be in association with animate or inanimate agencies. Any survival site is not mutually exclusive for individual species. In survival in host plants some pathogens have become so dependent that they become quite vulnerable when this association fails.

Although plant-pathogenic bacteria are non-sporeforming, many are tolerant of desiccation and survive for relatively long periods under dry conditions. Are these bacteria protected by some agency or material? Bacterial exudate, ooze, or slime has been considered as offering protection. This substance is commonly found in infested seed, cankers, or in living or dead plant parts. *C. flaccumfaciens*, for example, was shown to survive about five years in "dried bacterial ooze." In fact the viability of this and other bean bacterial pathogens exceeds that of the bean seed.

Workers have found, for the following bacteria, that the production of bacterial exudate, ooze, or polysaccharides in culture is comparable with that in infested host plants: *X. campestris* (122); *E. amylovora* (29, 45, 70); *X. phaseoli* (20, 59, 137); *E. stewartii* (29, 37); *E. carotovora* (20); *A. tumefaciens* (20, 46); *C. michiganense* (83); *C. insidiosum* (111); *C. sepedonicum* (111); *E. rubrifaciens* (92); and *P. solanacearum* (50). Hedrick (44) reported that 16 species of pathogens in five genera

(*Bacterium*, *Corynebacterium*, *Erwinia*, *Pseudomonas*, and *Xanthomonas*) produced exudate on media containing sucrose or glucose in basal casein hydrolysate.

The ooze produced by *E. amylovora* has been given much attention with respect to bacterial survival. It was believed that *E. amylovora* was very susceptible to desiccation, although the fire blight bacterium was known to survive a long time in dried exudate. Rosen (89, 90) found that the pathogen thus survived for more than 300 days at different temperatures and relative humidities. Hildebrand (45) recovered virulent cultures of *E. amylovora* from dried exudate after 15 and 25 months, but the organism survived only 13 days in moist exudate. When the exudate becomes moist in the spring the opportunity for infection is short indeed. Bacteria survived only 13 days in nutrient media in which capsules or slimy layers around the bacterial cells were absent. *E. amylovora* remains viable up to 12 months in aerial strands, another form of ooze. Both aerial strands and ooze are composed of about four-fifths matrix and one-fifth bacterial cells. Because of their ease of spread by wind the aerial strands may explain severe outbreaks of fire blight not explainable by survival in holdover cankers.

Hedrick (44), Leach et al (60), Feder & Ark (29), Corey & Starr (20), and others thought that bacterial exudates behave as a hydrophilic colloid. Its high water-holding capacity may aid bacterial survival during unfavorable conditions and seasons. This has been reported for *E. amylovora*. Leach et al (60) found that appreciable numbers of *X. phaseoli* cells survived in exudate for as long as 1325 days under a variety of conditions.

Maintenance under dry conditions commonly favors bacterial survival in plant residues. Residues on or near the soil surface are more favorable to bacterial survival than those incorporated in soil. By inference, the pathogens in dry undecayed residues are protected from antagonistic microflora (78) and from voracious protozoa (117) and free-living nematodes (75). Moisture is necessary for movement of these microfauna. Protozoa and nematodes were estimated to consume 9×10^{14} bacterial cells/m² and about one ton of bacteria per hectare/year, respectively. These microfauna therefore may be involved in bacterial survival. Decreasing moisture is one factor that causes bacteria to become dormant. Control may thus be effected by maintaining the survival site in a moist condition. This is possible under irrigation systems. *P. solanacearum* and *A. tumefaciens* both survive better under moist conditions, and for this reason are true soil pathogens.

Living plant tissue and the debris of dead plants provide more favorable loci for bacteria than does the soil matrix. Lucas (65) showed that the morphology of an individual piece of straw may influence the species colonizing it. After some decomposition of colonized materials in soil some species disappear, while other fungi and bacteria (119) continue in sites in humus specially favorable for survival. This may be due in part to protection afforded by organic matter (84). The rate of decomposition of infested plant parts may have a bearing on longevity. For example, virulent *C. nebraskense* was recovered for a longer time from parts of corn plants resistant to decay. Organic matter in tropical soils is decomposed very rapidly, whereas in temperate zones it reaches a fairly stable equilibrium level; repeated addition of organic manures causes no permanent increase (23). Bacterial pathogens in dead

residues obviously will be differentially reduced in the two climatic zones in a given period of time.

Presumably some bacterial phytopathogens may once have been soil inhabitants. Changes due to mutations may have caused free-living types to assume a parasitic habit and in due time become dependent on the plant for their nutritional requirements (68). Under natural conditions these bacteria are essentially obligate parasites, since they presumably lost their saprophytic capability.

"Bacterial plant pathogens are not soil-inhabiting organisms and are apparently unable to stand the competition in nature" (13). Many pathogens have a soil phase, even if only one of a rapidly declining population. Soil matrix may be protective in function during survival because of its colloidal properties. Chen & Alexander (18) and Robinson et al (88) found that certain asporogenous soil bacteria unprotected by colloids survived in extremely dry conditions for long periods. They found that a higher percentage of drought-tolerant than drought-sensitive bacteria were able to grow under dry conditions. They also found that the bacteria remained viable under dry conditions if collected in the stationary phase, but became nonviable if harvested in the exponential phase. It was thought that the effect of age resulted from differences in internal osmotic pressure.

Many refined techniques have been developed to detect surviving bacteria, but we question their relation to natural conditions. For example, the vacuum method of seed inoculation (38) or the use of additives are artificial. The water-soaking method or its modifications tend to duplicate nature in detecting viable pathogenic cells (25, 96). Leaf clipping or root injury duplicate natural conditions for certain diseases. Infested residues, nonhost materials, vegetative propagules, and seed lend themselves to field tests of survival. In greenhouse tests it is nearly impossible to simulate population levels and environmental conditions that occur in the field. Some pathogenic bacteria have the ability to persist on the root surface or in leaves of nonhost crops and weeds. Perhaps true soil-borne pathogens tend to survive in the rhizoplane rather than saprophytically. Pathogens that survive poorly in soil may have lost their saprophytic ability during the change from the saprophytic to parasitic habit or they could be sensitive to antagonistic microorganisms. We commonly assume that soil bacteria usually change in the direction of parasitism. Virulent isolates become avirulent in culture; why not in nature? Plant pathologists cannot readily recognize avirulent isolates. Differential selection pressures in nature affect persistence of bacterial strains and species. The greater vulnerability to antibiotics of root pathogens than soil saprophytes is in part responsible for their low competitive saprophytic ability (8). Differential persistence has been noted for races of *X. malvacearum* on different host varieties (9). Buddenhagen (12) suggested that races of pathogens best adapted to saprophytic survival are not necessarily the most virulent. However, because of their population buildup during their pathogenic phase these strains might effect saprophytic survival. We have found that the more virulent strains of *P. phaseolicola* and *C. flaccumfaciens* are better adapted for survival, and that two equally virulent strains of *C. nebraskense* differed in overwinter survival.

Phytopathogenic bacteria must have evolved successful survival mechanisms or they would have become extinct. The best example of man's attempts to eliminate

primary inoculum sources is the eradication of citrus canker between 1914–1927. Recently this method also proved successful in California for *X. malvacearum* (95). Other general controls of primary inoculum consist of the use of pathogen-free seed, tubers, and cuttings, cultural practices, and resistant cultivars.

Hollis (48) discussed the origin of different disease types, and Buddenhagen (12) suggested ways in which competitive saprophytic ability of bacterial pathogens may have been altered. Crosse (23) postulated a localized origin of a bacterial pathogen. A recent example, the new bacterial pathogen *C. nebraskense*, was first noted in two locations in south central Nebraska in 1969, apparently of local origin. The primary source of inoculum is corn stubble on or near the soil surface; seed transmission is negligible. Minimum tillage and corn monoculture had been widely practiced in south central Nebraska for a decade before the sudden and widespread appearance of this new disease in Nebraska. Were the corn roots exposed to the bacteria long enough for buildup of inoculum, or for soil bacteria or another vascular parasite to adapt to corn? It is improbable that the new bacterium appeared *de novo* and assumed serious proportions in less than a decade. Because of the narrow corn germ-plasm base, the pathogen was favored. Perhaps the organism was present as an epiphyte or as a parasite of green foxtail (*Setaria viridis*), shatter cane (*Sorghum* spp.), or common weeds.

That nonpathogenic bacteria predominate over pathogenic in association with higher plant relations is well established. During the evolution of plants, roots have been subjected to bacteria so long that the survivors are tolerant of most soil-borne bacteria. The effect of host and nonsusceptible crops on survival needs clarification. For example, *X. phaseoli* and *C. flaccumfaciens* var. *aurantiacum* overwintered inside weeds (99). Do different bacterial strains possess the ability to colonize different plant parts? What are the active and inactive stages of survival in soil? We need to determine the environmental conditions in which activity and survival occur. What effect does cultivation have on survival in contrast to noncultivation? Debris may differ from mineral soil matrix as a site for survival. Factors inimical to survival might differentially affect bacteria in their active and inactive phases; the periods of active and inactive phases are perhaps not the same for all pathogens. Nutrient deficiencies and competition with other organisms could operate only during the active phases since the inactive stage has no demands on nutrients. Antibiosis could operate during the active stage and possibly to some extent during the dormant stage. Senescence of inactive stages may be expected to occur in soil as it does in culture and may account for decline in populations. Survival in the free state in soil may be misinterpreted; bacteria may occur not as single cells but as microcolonies in a mucilaginous matrix. The bacteria also may assume L-forms or resting cells induced by antibiosis or unfavorable conditions (76).

ACKNOWLEDGMENTS

This work was done under Nebraska Agricultural Experiment Station Project No. 20-016. The authors gratefully acknowledge assistance of Kathryn Radant and Kathie Bragg for their patience, kindness, and diligence in translating longhand to legible type.

Literature Cited

- Allington, W. B. 1961. Plant Pathology. Report of progress. *Nebr. Agr. Exp. Sta. Quart.*, 7(4):7-10
- Anderson, E. S. 1957. The relations of bacteriophages to bacterial ecology. *Symp. Soc. Gen. Microbiol.* 7:189-217
- Ark, P. A. 1944. Pollen as a source of walnut bacterial blight infection. *Phytopathology* 34:330-34
- Baker, K. F. 1950. Bacterial fasciation disease of ornamental plants in California. *Plant Dis. Repr.* 34:121-26
- Baker, K. F. 1972. Seed Pathology. In *Seed Biology*, ed. T. T. Kozlowski, 1:317-416. New York: Academic
- Baker, K. F., Smith, S. H. 1966. Dynamics of seed transmission of plant pathogens. *Ann. Rev. Phytopathol.* 4: 311-34
- Basu, P. K., Wallen, V. R. 1966. Influence of temperature on the viability, virulence, and physiologic characteristics of *Xanthomonas phaseoli*. *Can. J. Bot.* 44:1239-45
- Brian, P. W. 1957. The ecological significance of antibiotic production. *Symp. Soc. Gen. Microbiol.* 7:168-88
- Brinkerhoff, L. A. 1970. Variation in *Xanthomonas malvacearum* and its relation to control. *Ann. Rev. Phytopathol.* 8:85-110
- Brinkerhoff, L. A., Fink, G. B. 1964. Survival and infectivity of *Xanthomonas malvacearum* in cotton plant debris and soil. *Phytopathology* 54: 1198-1201
- Brinkerhoff, L. A., Hunter, R. E. 1963. Internally infected seed as a source of inoculum for the primary cycle of bacterial blight of cotton. *Phytopathology* 53:1397-1401
- Buddenhagen, I. W. 1965. The relation of plant-pathogenic bacteria to the soil. In *Ecology of Soil-borne Plant Pathogens*, ed. K. F. Baker, W. C. Snyder. pp. 269-284. Berkeley: Univ. Calif. Press 571 pp.
- Burkholder, W. H. 1948. Bacteria as plant pathogens. *Ann. Rev. Microbiol.* 2:389-412
- Burkholder, W. H. 1959. Present-day problems pertaining to the nomenclature and taxonomy of the phytopathogenic bacteria. In *Omaggio lui Traian Săvulescu*, 119-27. Bucharest: Ed. Acad. Repub. Pop. Romine
- Butcher, C. L., Dean, L. L., Guthrie, J. W. 1969. Effectiveness of halo blight control in Idaho bean seed crops. *Plant Dis. Repr.* 53:894-96
- Butcher, C. L., Dean, L. L., Laferriere, L. 1967. Incidence of halo blight in Idaho. *Plant Dis. Repr.* 51:310-11
- Carter, W. 1962. *Insects in Relation to Plant Disease*. New York: Interscience 705 pp.
- Chen, M., Alexander, M. 1973. Survival of soil bacteria during prolonged desiccation. *Soil Biol. Biochem.* 5:213-21
- Clafin, L. E., Stuteville, D. L. 1973. Survival of *Xanthomonas alfalfae* in alfalfa debris and soil. *Plant Dis. Repr.* 57:52-53
- Corey, R. R., Starr, M. P. 1957. Colony types of *Xanthomonas phaseoli*. *J. Bacteriol.* 74:137-40
- Cormack, M. W., Moffat, J. E. 1956. Occurrence of the bacterial wilt organism in alfalfa seed. *Phytopathology* 46: 407-9
- Cowan, J. R. 1973. The seed. *Agron. J.* 65:1-5
- Crosse, J. E. 1968. Plant pathogenic bacteria in soil. In *Ecology of Soil Bacteria*, ed. T. R. Gray, D. Parkinson. pp. 552-72. Toronto: Univ. Toronto Press. 681 pp.
- Devadath, S. 1969. *Studies on Xanthomonas oryzae (causal organism of bacterial blight) occurring on rice*. PhD thesis. Utkal Univ., Cuttack, India
- Diachun, S., Valteau, W. D. 1946. Growth and overwintering of *Xanthomonas vesicatoria* in association with wheat roots. *Phytopathology* 36:277-80
- Dickey, R. S. 1961. Relation of some edaphic factors to *Agrobacterium tumefaciens*. *Phytopathology* 51:607-14
- Dowson, W. J. 1958. The present position of bacterial plant diseases, and subjects for future research. *Commonw. Phytopathol. News* 4:33-35
- Fang, C. T., Lin, C. F., Chu, C. L. 1956. A preliminary study on the disease cycle of the bacterial leaf blight of rice. *Acta Phytopathol. Sinica* 2:173-85 (In Chinese with English summary)
- Feder, W. A., Ark, P. A. 1951. Wilt-inducing polysaccharides derived from crown-gall, bean blight, and soft-rot bacteria. *Phytopathology* 41:804-8
- Freitag, J. H. 1956. Beetle transmission, host range, and properties of squash mosaic virus. *Phytopathology* 46:73-81
- Fulton, H. R. 1920. Decline of *Pseudomonas citri* in the soil. *J. Agr. Res.* 19:207-23
- Garrett, S. D. 1950. *Ecology of Root Inhabiting Fungi*. London: Cambridge Univ. Press 292 pp.

33. Gibbins, L. N. 1971. Relationships between pathogenic and non-pathogenic bacterial inhabitants of aerial plant surfaces. In *Plant Pathogenic Bacteria*. Geesteranus, H. P. M., Ed. pp. 15-24. *Proc. 3rd Int. Conf. Plant Pathog. Bact. Wageningen*. 365 pp.
34. Goheen, A. C., Nyland, G., Lowe, S. K. 1973. Association of a rickettsialike organism with Pierce's disease of grapevines and alfalfa dwarf and heat therapy of the disease in grapevines. *Phytopathology* 63:341-45
35. Goodman, R. N. 1965. In vitro and in vivo interaction between components of mixed bacterial cultures isolated from apple buds. *Phytopathology* 55:217-21
36. Goodman, R. N. 1967. Protection of apple stem tissue against *Erwinia amylovora* infection by avirulent strains of three other bacterial species. *Phytopathology* 57:22-24
37. Gorin, P. A. J., Spencer, J. F. T. 1961. Structural relationship of extracellular polysaccharides from phytopathogenic *Xanthomonas* spp. Part I. Structure of the extracellular polysaccharides from *Xanthomonas stewartii*. *Can. J. Chem.* 39:2282-89
38. Goth, R. W. 1966. The use of partial vacuum to inoculate bean seeds with phytopathogenic bacteria. *Plant Dis. Repr.* 50:110-11
39. Goto, M. 1971. The significance of the vegetation for the survival of pathogenic bacteria. See Ref. 33, pp. 39-53
40. Grogan, R. G., Kimble, K. A. 1967. The role of seed contamination in the transmission of *Pseudomonas phaseolicola* in *Phaseolus vulgaris*. *Phytopathology* 57:28-31
41. Guthrie, J. W., Fenwick, H. S. 1967. Bacterial pathogens of beans. *Idaho Agr. Res. Progr. Rept.* 121:1-36
42. Haas, J. H. 1972. Epiphytic populations of *Xanthomonas phaseoli* var. *fuscans* on *Phaseolus vulgaris* "Sanilac." *Proc. 38th Can. Phytopathol. Soc.* 39:31
43. Hagedorn, D. J., Rand, R. E., Ercolani, G. L. 1972. Survival of *Pseudomonas syringae* on hairy vetch in relation to epidemiology of bacterial brown spot of bean. *Phytopathology* 62:672
44. Hedrick, H. G. 1956. Exudates produced by phytopathogenic bacteria. *Phytopathology* 46:14-15
45. Hildebrand, E. M. 1939. Studies on fire-blight ooze. *Phytopathology* 29: 142-55
46. Hodgson, R., Riker, A. J., Peterson, W. H. 1945. Polysaccharide production by virulent and attenuated crown gall bacteria. *J. Biol. Chem.* 158:89-100
47. Hoitink, H. A. J., Hagedorn, D. J., McCoy, E. 1968. Survival, transmission, and taxonomy of *Pseudomonas syringae* van Hall, the causal organism of bacterial brown spot of bean (*Phaseolus vulgaris* L.) *Can. J. Microbiol.* 14: 437-41
48. Hollis, J. P. 1952. On the origin of diseases in plants. *Plant Dis. Repr.* 36: 319-27
49. Hunter, R. E., Brinkerhoff, L. A. 1964. Longevity of *Xanthomonas malvacearum* on and in cotton seed. *Phytopathology* 54:617
50. Husain, A., Kelman, A. 1958. Relation of slime production to mechanism of wilting and pathogenicity of *Pseudomonas solanacearum*. *Phytopathology* 48: 155-65
51. Ivanoff, S. S. 1933. Stewart's wilt disease of corn, with emphasis on the life history of *Phytoplasma stewartii* in relation to pathogenesis. *J. Agr. Res.* 47:749-70
52. Keil, H. L., Imale, B. C., Wilson, R. A. 1964. Longevity of fire blight organism, *Erwinia amylovora* on pear trees in the greenhouse as demonstrated by infection after sandblast injury. *Phytopathology* 54:747
53. Keil, H. L., van der Zwet, T. 1972. Aerial strands of *Erwinia amylovora*: structure and enhanced production by pesticide oil. *Phytopathology* 62:355-61
54. Keil, H. L., van der Zwet, T. 1972. Recovery of *Erwinia amylovora* from symptomless stems and shoots of Jonathan apple and Bartlett pear trees. *Phytopathology* 62:39-42
55. Kelman, A. 1953. The bacterial wilt caused by *Pseudomonas solanacearum*. *N.C. Agr. Exp. Sta. Tech. Bull.* 99: 1-194
56. Kelman, A., Sequeira, L. 1965. Root-to-root spread of *Pseudomonas solanacearum*. *Phytopathology* 55:304-9
57. Krieg, A. 1971. Aposymbiosis, a possible method of antimicrobial control of arthropods. In *Microbial Control of Insects and Mites*, ed. H. D. Burges, N. W. Hussey, pp. 673-77. London New York: Academic 861 pp.
58. Larson, R. H. 1944. The ring rot bacterium in relation to tomato and eggplant. *J. Agr. Res.* 69:309-25
59. Leach, J. G. 1940. *Insect Transmission of Plant Diseases*. New York London: McGraw-Hill. 615 pp.
60. Leach, J. G., Lilly, V. G., Wilson, H. A., Purvis, M. R. Jr. 1957. Bacterial

- polysaccharides: The nature and function of the exudate produced by *Xanthomonas phaseoli*. *Phytopathology* 47: 113-20
61. Leben, C. 1965. Epiphytic microorganisms in relation to plant disease. *Ann. Rev. Phytopathol.* 3:209-30
 62. Leben, C. 1973. Survival of plant pathogenic bacteria. Abstr. 0326, 2nd Int. Congr. Plant Pathol. Minneapolis
 63. Lee, H. A. 1920. Behavior of the citrus-canker organism in the soil. *J. Agr. Res.* 19:189-206
 64. Leppik, E. E. 1968. Quarantine and seed pathology. 1st Int. Congr. Plant Pathol. London Abstr. p. 114
 65. Lucas, R. L. 1955. A comparative study of *Ophiobolus graminis* and *Fusarium culmorum* in saprophytic colonization of wheat straw. *Ann. Appl. Biol.* 43: 134-43
 66. Massey, R. E. 1931. Studies on black-arm disease of cotton. II. *Emp. Cotton Grow. Rev.* 8:187-213
 67. Mew, T. W., Kennedy, B. W. 1971. Growth of *Pseudomonas glycinea* on the surface of soybean leaves. *Phytopathology* 61:715-16
 68. Misaghi, I., Grogan, R. G. 1969. Nutritional and biochemical comparisons of plant-pathogenic and saprophytic fluorescent pseudomonads. *Phytopathology* 59:1436-50
 69. Mizukami, T., Wakimoto, S. 1969. Epidemiology and control of bacterial leaf blight of rice. *Ann. Rev. Phytopathol.* 7:51-72
 70. Moore, L. W., Hildebrand, D. C. 1966. Electron microscopy of *Erwinia amylovora* and *Pseudomonas phaseolicola* in bacterial ooze. *Phytopathology* 56:891
 71. Natti, J. J. 1967. Overwinter survival of *Pseudomonas phaseolicola* in New York. *Phytopathology* 57:343
 72. Nelson, G. A., Semeniuk, G. 1963. Persistence of *Corynebacterium insidiosum* in the soil. *Phytopathology* 53:1167-69
 73. Noble, M., Richardson, M. J. 1968. An annotated list of seed-borne diseases. *Commonw. Mycol. Inst. Phytopathol. Papers* 8:1-191
 74. Orton, C. R. 1931. Seed-borne parasites. A bibliography. *W. V. Agr. Exp. Sta. Bull.* 245:1-47
 75. Overgaard-Nielsen, C. 1949. Studies on the soil microfauna. 2. The soil inhabiting nematodes. *Natura Jutlandica* 2: 1-131
 76. Park, D. 1965. Survival of microorganisms in soil. See Ref. 12, pp. 82-98
 77. Patel, P. N., Walker, J. C. 1965. Resistance in *Phaseolus* to halo blight. *Phytopathology* 55:889-94
 78. Patrick, Z. A. 1954. The antibiotic activity of soil microorganisms as related to bacterial plant pathogens. *Can. J. Bot.* 32:705-35
 79. Peltier, G. L., Frederich, W. J. 1926. Further studies on the overwintering of *Pseudomonas citri*. *J. Agr. Res.* 32: 335-45
 80. Pepper, E. H. 1967. Stewart's bacterial wilt of corn. *Am. Phytopathol. Soc. Monogr.* 4. St. Paul, Minn: Am. Phytopathol. Soc. 36 pp.
 81. Peterson, G. H. 1963. Survival of *Xanthomonas vesicatoria* in soil and diseased tomato plants. *Phytopathology* 53:765-67
 82. Preece, T. F., Dickinson, C. H. Ed. 1971. *Ecology of Leaf Surface Microorganisms*. London New York: Academic 640 pp.
 83. Rai, P. V., Strobel G. A. 1969. Phytotoxic glycopeptides produced by *Corynebacterium michiganense* II. Biological properties. *Phytopathology* 59: 53-57
 84. Record, B. R., Taylor, R. 1953. Some factors influencing the survival of *Bacterium coli* on freeze-drying. *J. Gen. Microbiol.* 9:475-84
 85. Reddy, C. S., Holbert, J. R., Erwin, A. T. 1926. Seed treatments for sweet-corn diseases. *J. Agr. Res.* 33:769-79
 86. Richardson, L. T. 1957. Quantitative determination of viability of potato ring rot bacteria following storage, heat, and gas treatments. *Can. J. Bot.* 35:647-56
 87. Robert, A. L. 1955. Bacterial wilt and Stewart's leaf blight of corn. *US Dep. Agr. Farmers Bull.* 2092:1-13
 88. Robinson, J. B., Salonijs, P. O., Chase, F. E. 1965. A note on the differential response of *Arthrobacter* spp. and *Pseudomonas* spp. to drying in soil. *Can. J. Microbiol.* 11:746-48
 89. Rosen, H. R. 1929. The life history of the fire blight pathogen, *Bacillus amylovorus*, as related to the means of overwintering and dissemination. *Ark. Agr. Exp. Sta. Bull.* 244:1-96
 90. Rosen, H. R. 1938. Life span and morphology of the fire blight bacteria as influenced by relative humidity, temperature and nutrition. *J. Agr. Res.* 56:239-58
 91. Sabet, K. A., Ishag, F. 1969. Studies on the bacterial diseases of Sudan crops. VIII. Survival and dissemination of *Xanthomonas phaseoli* (E. F. Smith) Dowson. *Ann. Appl. Biol.* 64:65-74

92. Schaad, N. W., Wilson, E. E. 1971. The ecology of *Erwinia rubrifaciens* in the development of phloem canker of Persian walnut. *Ann. Appl. Biol.* 69:125-36
93. Schnathorst, W. C. 1954. Bacteria and fungi in seeds and plants of certified bean varieties. *Phytopathology* 44: 588-92
94. Schnathorst, W. C. 1964. Longevity of *Xanthomonas malvacearum* in dried cotton plants and its significance in dissemination of the pathogen on seed. *Phytopathology* 54:1009-11
95. Schnathorst, W. C. 1966. Eradication of *Xanthomonas malvacearum* from California through sanitation. *Plant Dis. Repr.* 50:168-71
96. Schuster, M. L. 1955. A method for testing resistance of beans to bacterial blights. *Phytopathology* 45:519-20
97. Schuster, M. L. 1955. Bean blight survives winter in soil. *Nebr. Agr. Exp. Sta. Quart.* 3(4):3
98. Schuster, M. L. 1967. Survival of bean bacterial pathogens in the field and greenhouse under different environmental conditions. *Phytopathology* 57:830
99. Schuster, M. L. 1970. Survival of bacterial pathogens of beans. *Bean Impr. Coop.* 13:68-70
100. Schuster, M. L., Coyne, D. P., Hoff, B. 1973. Comparative virulence of *Xanthomonas phaseoli* strains from Uganda, Colombia, and Nebraska. *Plant Dis. Repr.* 57:74-75
101. Schuster, M. L., Coyne, D. P., Kerr, E. D. 1965. New virulent strains of halo blight bacterium overwinters in the field. *Phytopathology* 55:1075
102. Schuster, M. L., Harris, L. 1957. Find new ground for your 1958 bean crop. *Nebr. Agr. Exp. Sta. Quart.* 5(1):3-4
103. Schuster, M. L., Hoff, B. 1973. Epidemiology of leaf freckles and wilt of corn. *2nd Int. Congr. Plant Pathol. Minneapolis Abstr.* 0817
104. Schuster, M. L., Hoff, B., Mandel, M., Lazar, I. 1973. Leaf freckles and wilt, a new corn disease. *Proc. Ann. Corn Sorghum Res. Conf. Chicago* 27:176-91
105. Schuster, M. L., Sayre, R. M. 1967. A coryneform bacterium induces purple-colored seed and leaf hypertrophy of *Phaseolus vulgaris* and other Leguminosae. *Phytopathology* 57:1064-66
106. Schuster, M. L., Wagner, L. J. 1972. Control of bacterial canker and root knot of hydroponic tomatoes. *Plant Dis. Repr.* 56:139-40
107. Sequeira, L. 1962. Control of bacterial wilt of bananas by crop rotation and following. *Trop. Agr. London* 39: 211-17
108. Skoric, V. 1927. Bacterial blight of peas; overwintering, dissemination, and pathological histology. *Phytopathology* 17: 611-27
109. Smith, E. F. 1911. *Bacteria in Relation to Plant Diseases*. Carnegie Inst. Wash. Publ. 27, Vol. 2. 368 pp.
110. Snieszko, S. F., Bonde, R. 1943. Studies on the morphology, physiology, serology, longevity and pathogenicity of *Corynebacterium sepedonicum*. *Phytopathology* 33:1032-44
111. Spencer, J. F. T., Gorin, P. A. J. 1961. The occurrence in the host of physiologically active gums produced by *Corynebacterium insidiosum* and *Corynebacterium sepedonicum*. *Can. J. Microbiol.* 7:185-88
112. Srivastava, D. N., Rao, Y. P. 1964. Seed transmission and epidemiology of bacterial blight disease of rice in North India. *Indian Phytopathol.* 17:77-78
113. Stanek, M., Lasik, J. 1965. The occurrence of microorganisms parasitizing on the over-ground parts of plants in the rhizosphere. pp. 300-307. In *Plant Microbes Relationships*, ed. J. Macura, V. Vančura. Prague: Publ. House Czech. Acad. Sci. 333 pp.
114. Starr, M. P., Seidler, R. J. 1971. The bdellovibrios. *Ann. Rev. Microbiol.* 25:649-78
115. Stolp, H., Petzold, H. 1962. Untersuchungen über einen obligat parasitischen Mikroorganismus mit lytischer Aktivität für *Pseudomonas-Bakterien*. *Phytopathol. Z.* 45:364-90
116. Stoughton, R. H. 1930. Angular leaf spot disease of cotton. *Nature* 125: 350-51
117. Stout, J. D., Heal, O. W. 1967. Protozoa. In *Soil Biology*, ed. A. Burges, F. Raw. pp. 149-95. New York: Academic 532 pp.
118. Strider, D. L. 1969. Bacterial canker of tomato caused by *Corynebacterium michiganense*. *N.C. Agr. Exp. Sta. Tech. Bull.* 193:1-110
119. Strigger, S. 1948. Fluorescence microscope examination of bacteria in soil. *Can. J. Res., Sec. C.* 26:188-93
120. Sutton, M. D., Wallen, V. R. 1967. Phage types of *Xanthomonas phaseoli* isolated from beans. *Can. J. Bot.* 45: 267-80
121. Sutton, M. D., Wallen, V. R. 1970. Epidemiological and ecological relations of *Xanthomonas phaseoli* and *X. phaseoli* var. *fuscans* on beans in south-

- from culture and from infested cabbage leaves. *Can. J. Bot.* 48:645-52
123. Tagami, Y. et al 1964. Epidemiological studies on the bacterial leaf blight of rice, *Xanthomonas oryzae* (Uyeda et Ishiyama) Dowson. I. The overwintering of the pathogen. *Bull. Kyushu Agr. Exp. Sta.* 9:89-122
 124. Tarr, S. A. J. 1953. Seed treatment against blackarm disease of cotton in the Anglo-Egyptian Sudan. I. Dry dressings, laboratory evaluation of bactericides and the toxicity of mercuric chlorida-iodide powders to cotton seed. *Emp. Cotton Grow. Rev.* 30:19-33
 125. Taylor, J. D. 1970. The quantitative estimation of the infection of bean seed with *Pseudomonas phaseolicola* (Burkh.) Dowson. *Ann. Appl. Biol.* 66:29-36
 126. Teliz-Ortiz, M., Burkholder, W. H. 1960. A strain of *Pseudomonas fluorescens* antagonistic to *Pseudomonas phaseolicola* and other bacterial plant pathogens. *Phytopathology* 50:119-23
 127. Thomas, W. D. Jr., Graham, R. W. 1952. Bacteria in apparently healthy pinto beans. *Phytopathology* 42:214
 128. Van der Zwet, T. 1969. Study of fire blight cankers and associated bacteria in pear. *Phytopathology* 59:607-13
 129. Van der Zwet, T., Keil, H. L. Smale, B. C. 1969. Fire blight in the Magness pear cultivar in north central Arkansas. *Plant Dis. Repr.* 53:686-89
 130. Vorokovich, I. W. 1960. On the survival in the soil of bacteria of the genus *Erwinia*, causal agents of soft rots in plants. *Bull. Soc. Nat. Moscow. Ser. Biol.* 65:95-105
 131. Wakimoto, S. 1955. Overwintering of *Xanthomonas oryzae* on unhulled grains of rice. *Agr. Hort.* 30:1501
 132. Walker, J. C., Patel, P. N. 1964. Splash dispersal and wind as factors in epidemiology of halo blight of bean. *Phytopathology* 54:140-41
 133. Wallen, V. R., Sutton, M. D. 1965. *Xanthomonas phaseoli* var. *fuscans* (Burkh.) Starr & Burkh. on field bean in Ontario. *Can. J. Bot.* 43:437-46
 134. Wallin, J. R. 1946. Seed and seedling infection of barley, bromegrass, and wheat by *Xanthomonas translucens* var. *cerealis*. *Phytopathology* 36:446-59
 135. Wharton, A. L. 1967. Detection of infection by *Pseudomonas phaseolicola* (Burkh.) Dows. in white-seeded dwarf bean seed stocks. *Ann. Appl. Biol.* 60:305-12
 136. Wickens, G. M. 1953. Bacterial blight of cotton. *Emp. Cotton Grow. Rev.* 30:81-103
 137. Wilson, H. A., Lilly, V. G., Leach, J. G. 1965. Bacterial polysaccharides. IV. Longevity of *Xanthomonas phaseoli* and *Serratia marcescens* in bacterial exudates. *Phytopathology* 55:1135-38
 138. Zaumeyer, W. J. 1972. Genetic vulnerability in snap beans. *Bean Impr. Coop.* 15:37-40
 139. Zaumeyer, W. J., Thomas, H. R. 1957. A monographic study of bean diseases and methods for their control. *US Dep. Agr. Tech. Bull.* 868:1-255