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Genetics of and Breeding for Resistance to Bacterial Pathogens in Vegetable Crops[1]

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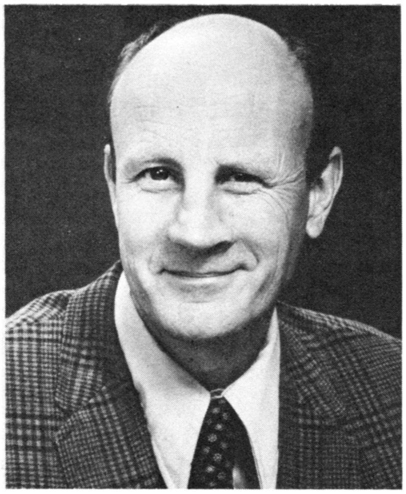

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Bacterial pathogens cause destructive diseases on many important vegetable crops throughout the world. Satisfactory chemical control measures for bacterial pathogens have not been achieved. Recommended control measures are: use of disease-free seed and transplants; hot water treatment of seed if feasible; suitable rotations; deep plowing of plant debris; and use of resistant cultivars if available (13, 50, 51, 53, 73, 75, 76, 77). Different levels of resistance to bacteria have been found in vegetable crops and various levels of resistance have been incorporated into a number of successful cultivars. The paper summarizes selected references from a vast literature and addresses current developments and future prospects on breeding vegetable crops for resistance to bacterial pathogens.

Bacterial variation, populations, and virulence.

The principal bacterial pathogens of vegetable crops are in *Xanthomonas*, *Pseudomonas*, *Corynebacterium*, and *Erwinia*. Nomenspecies of *Pseudomonas* and *Xantho-*

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monas are employed in this reveiw rather than the respective taxospecies of *P. syringae* and *X. campestris* (7, 56), because nomenspecies classifications are more meaningful to plant breeders and plant pathologists.

Variation. There is a diversity of strains which have some characters in common, but differ in others, including pathogenicity within a bacterial species. Based on recent evidence, *X. phaseoli* (E.F.S.) Dows., *X. phaseoli* var. *fuscans* (Burkh.) Starr & Burkh., and *X. vignicola* Burkh. could be considered strains of *X. phaseoli* (49, 51). Vakiki et al. (66) maintained that strains virulent for cowpeas should be in *X. vignicola*. Schroth et al. (46) contend *P. phaseolicola* (Burkh.) Dows., *P. glycinea* Coerper, and *P. mori* (Boyer and Lambert) Stevens consists of an infinite number of strains ranging from avirulent to highly virulent. They could be considered strains of *P. phaseolicola*.

Variations in virulence have been observed in *P. syringae* Van Hall (31), *P. solanacearum* E.F.S. (29), *C. flaccumfaciens* (Hedges) Dows (49), *E. tracheiphila* (E.F.Sm.) Holland (68), *E. stewartii* (E.F.Sm.) Dye (39), *C. nebraskense* Schuster, Hoff, Mandel and Lazar (48), and *C. michiganense* (E.F.Sm.) Jens (61). Strains of *P. solanacearum* (E.F.Sm.) Erw. Smith have been relegated to 3 major races (8). Race 1 (44) and race 3 (8, 54) affect potatoes and tomatoes, with the latter predominating in highland tropical regions. Three races of *X. vesicatoria* (Doidge) Dows. have been reported (16). Mutability resulted in conversion of tomato race 1 to pepper race 2 and pepper race 2 to pepper race 1, but no conversion from tomato race 1 to pepper race 1.

Variability of bacteria complicates disease control and periodic surveillance for the presence of new strains is necessary to keep abreast of the current status.

Population levels. Bacteria are confined to intercellular spaces or vascular tissue following introduction into plant tissues, and they multiply initially in these areas. Disease symptoms due to bacterial infection are associated with bacterial multiplication. Internal bacterial populations in resistant plants decline at a time when rapid multiplication continues in the congenial host, but moderately high populations of bacteria can develop and persist in resistant/germplasm (2, 13, 15, 51, 60). This could lead to a breakdown of resistance and also result in seed infection. In addition, high epiphytic populations of *X. phaseoli* exist on leaves of susceptible and moderately resistant *Phaseolus vulgaris* beans (free of leaf symptoms) (69) and on leaves of nonhost species (9). These populations provide an inoculum source which may infect the plant through wounds or during water-soaking of leaves during rainstorms.

Virulence. The virulence of a bacterial population can be altered from the initial virulence level through mutation and selection during passage through various host genotypes. Successive passages of populations of *E. stewartii* through resistant maize and teosinte increased virulence, but continued passage through susceptible maize and teosinte decreased virulence (71). This phenomenon was repeated by Lincoln (32), who found that virulent strains increased through selection in resistant corn and avirulent strains increased in susceptible maize. It would be useful to determine if selection for increased virulence also occurs in epiphyte populations on leaf surfaces. We suggest, based on the above findings, that seed of resistant cultivars should be tested to determine whether or not they are free of the bacteria in order to reduce the possibility of breakdown of host resistance due to the emergence of more virulent strains. Noninfected seed should then be produced under furrow irrigation in an arid climate.

Virulence of strains of *Agrobacterium tumefaciens* (Smith & Townsend) Conn, which causes crown gall on some plant species, has been found to be associated with particular plasmids (65). Loss of virulence is associated with loss of plasmids. This may explain variations in virulence and loss of virulence in strains of several other bacterial species. Maintenance of virulence upon subculturing is a problem with bacterial pathogens and this is especially true for *Pseudomonas solanacearum*, *E. tracheiphila*, and *C. nebraskense*; their virulence can be maintained in infested host tissue. Lyophilization is another effective storage method used to maintain virulence.

Inoculation methods and evaluation of disease reactions

Inoculation methods. Diverse techniques have been successfully employed for inoculation of vegetable crops with bacterial pathogens: watersoaking; multineedle; spraying; syringe; needle(s); sandblasting; placing inoculum on cut-petiole; cut-stems; cut-root; cut-leaf, and seed dips. Some techniques are based on modes of natural entrance. Watersoaking is a more desirable method than spraying leaves for various pathogens as it duplicates natural infections during driving rainstorms. Bacterial suspensions (10^6–10^{10} cells/ml) are forced via the stomata into the intercellular spaces, the infection sites (47). The watersoaking method is effective under field as well as glasshouse conditions. The multineedle method has been found effective under glasshouse or growth chamber conditions (41, 64). Tissue wounding is required for successful inoculation with *C. nebraskense* in maize/popcorn (cutleaf, stem injections, and mutlineedle) (48), with *C. flaccumfaciens* in beans (dissecting needle) (13), with *C. michiganese* in tomato (cutstems, petioles) (18), with *E. stewartii* in maize (39), *E. tracheiphila* in cucumber (multineedle) (27) and syringe inoculation of cotyledons (R. E. Wilkinson, personal communication), and with *P. solanacearum* in potato (44) (stem puncture) and tomato (34) (clipped roots or stems). Differences in inoculation methods can cause differences in disease reactions in the host. Bean lines were found resistant to *P. phaseolicola* using a stem-stabbing-method, but were susceptible when the leaves were sprayed with a cell suspension (H. M. Munger, personal communication).

Natural infection and spread can be emulated in a disease nursery either by planting test material on a site containing diseased debris from a former crop to provide a primary inoculum source or by inoculating susceptible cultivars (''spreader rows'') spaced at intervals in the plots to provide a source of secondary inoculum for the adjacent noninoculated test plants (13, 51).

The reaction of plants to inoculations may be influenced by a variety of factors (13, 15): 1) environmental, such as temperature, moisture, light; 2) plant factors, such as cultivar and type, age, stomatal openings, openings, nutrition, subsequent treatment after inoculation, and physiological conditions; and 3) parasitic factors such as concentration and age of inoculum and strain of pathogen.

Evaluation. Disease rating schemes vary, depending upon the plant part inoculated (13, 15). Leaf ratings for blights were scored from 0 (no visible infection) to 4 or 5 (severe infection). Ratings in the cutleaf and multineedle methods were based on the distance the bacteria moved from points of inoculation as determined by symptom development. The degree of leaf systemic chlorosis due to a toxin, such as *P. phaseolicola* phytotoxin, was also determined. Disease reactions of plant parts, such as pods, were determined by recording the types of the reactions, i.e., watersoaking vs. necrotic lesions. Size of the diseased lesions (in mm) around needle punctures was also measured. Wilt ratings were recorded as ranging from no visible wilting to complete wilting or death. There is a dire need to standardize inoculation methods and disease rating systems in screening for resistance to different bacterial species in various vegetable crops to promote a better understanding among breeders concerning reports of resistant germplasm and cultivars (13, 45, 51).

Germplasm sources of resistance

Germplasm with various levels of resistance to the major bacterial pathogens of vegetable crops have been identified (Table 1). High levels of resistance have been found in cucumber to *E. tracheiphila* (bacterial wilt) (37) and to P. lachrymans (Smith and Bryan) Carsner (angular leaf spot) (11, 30), in sweet corn to *E. stewartii* (Stewart's wilt) (39) and *C. nebraskense* (leaf freckles and wilt) (48), in cabbage to *X. campestris* (Pammel) Dows. (black rot) (3, 13, 23), *C. flaccumfaciens* (bacterial wilt) (13, 51), in *Phaseolus acutifolius* beans to *X. phaseoli* (common blight) (13, 26, 47, 53, 76, 77), in peanuts (13) and eggplant (74) to *Pseudomonas solanacearum* (bacterial wilt), in tomato to *C. michiganense* (bacterial canker) (18, 19, 63) and to *P. tomato* (Okabe) Alstatt (bacterial speck) (40), and in pepper to *X. vesicatoria* (bacterial spot) (17, 59, 60). The level of resistance to *P. solanacearum* in tomato (1, 4, 24, 75) and potato (54) species depends upon the environment where the lines were identified, the degree of virulence of the bacterial strains, and also, in tomato, on inoculum concentration and method of inoculation (34). Sources of resistance useful in North Carolina were susceptible to tropical strains in Hawaii (1), Taiwan and the Philippines (75). Higher levels of resistance have been detected in the latter 2 countries (4, 75). Additional germplasm sources need to be identified that maintain resistance in the hot lowland tropics. Moderately high levels of resistance have been detected in *Phaseolus vulgaris* to *X. phaseoli* (13, 15, 53, 64, 76, 77), but there is a need to detect higher levels of resistance in that species to avoid problems in utilizing genes for resistance in *P. acutifolius* and *P. coccineus*, since it is difficult to combine desired horticultural traits with resistance, when using this exotic germplasm. However, Freytag (21) in Puerto Rico reported success in utilizing *P. coccineus* in breeding for resistance to *X. phaseoli* in *P. vulgaris*. Plant resistance (non-preference) in *Cucumis sativus* to the striped and spotted cucumber beetles, carriers of *E. tracheiphila*, does not provide a useful level of resistance to the bacteria, since only a few beetles feeding on susceptible plants can result in the disease. Steadman (personal communication) observed that 'County Fair', resistant to beetle feeding, was highly susceptible to the disease. Resistance to *E. tracheiphila* is best provided through the action of the *Bw* gene in cucumbers (37). No high resistance to *E. tracheiphila* has been identified in *C. melo*; only 1 cultivar, 'Burrell's Gem' with 20% plant survival, was found among 250 inoculated melon cultivars (42).

Genetics of resistance

One or more major genes have been reported to control disease reactions to bacterial pathogens in the following vegetable crops: to *Pseudomonas solanacearum* in potatoes (44, 54); to *E. tracheiphila* in cucumbers (37); to *E. stewartii* in maize (70); to *X. campestris* in cabbage (72); to *X. vesicatoria* in peppers (12); to *P. phaseolicola* and *Corynebacterium flaccumfaciens* in beans (*Phaseolus vulgaris*) (13, 14, 25, 51, 53, 62, 77); and to *Pseudomonas tomato* in tomatoes (40). Various patterns of inheritance of the reaction to some pathogens were observed depending upon host genotypes and strains used in the tests. Genetic background influenced the expression of resistance to *P. phaseolicola* derived from *Phaseolus vulgaris* PI 150414 (62). Various genes were also found to control diverse symptoms of this disease and the reaction among plant parts. Coyne et al. (14) first showed that separate, linked major genes each controlled the susceptible-leafwater-soaked reaction vs. the resistant-hypersensitive reaction, and the susceptible-leaf-systemic chlorosis vs. leaf resistance to the toxin. The water-soaked reaction is due to rapid multiplication of bacteria, and systemic chlorosis is due to translocation of leaf bacterial toxin into newly formed leaves. However, the pod reaction is controlled by different genes than those that control reactions in the leaves (25). These reactions were found to be dominant in some crosses and recessive in others. This genetic analysis of disease reaction components in the same or different plant parts indicated the importance of selecting plants in a breeding program for resistant pod and leaf reactions through the use of specific environmental conditions and inoculation procedures.

Quantitative patterns of inheritance of disease reactions to bacterial pathogens have been reported in the following crops: in tomatoes to *Pseudomonas solanacearum* (1, 4, 75), *E. carotovora* (Jones) Holland (6), *C. michiganense* (19, 63), and *X. vesicatoria* (67); in *Phaseolus vulgaris* beans to *X. phaseoli* (13, 15, 26, 41, 45, 51, 53, 64, 76, 77), *C. flaccumfaciens* (13, 16, 51), and *Pseudomonas syringae* (3); in cucumber to *P. lachrymans* (11, 30), in maize/sweet corn to *C. nebraskense* (33), and in pepper to *X. vesicatoria* (12). Multiple recessive genes, acting additively, determined resistance to *P. solanacearum* in tomato (4; W. R. Henderson, personal communication). Resistance in *Lycopersicon pimpinellifolium* (PI 127805A), used in Hawaii, was incompletely dominant in young plants and recessive in mature plants (1). Many incompletely recessive genes were reported to control the resistant reaction to *P. lachrymans* in cucumber (30). Additive genetic effects were predominant in controlling the disease reactions to pathogens in the following crops: in *Phaseolus vulgaris* beans to *X. phaseoli* and *C. flaccumfaciens* (13, 15, 26, 51, 53, 64, 76, 77); in maize to *C. nebraskense* (33); and in tomato to *C. michiganense* (19). However, Thyr (63) reported that 1 to 4 incompletely dominant genes were involved in resistance to the last pathogen in tomato. Narrow sense heritability was moderately high for reaction to *X. phaseoli* in inoculated beans tested in the seedling stage in growth chambers (41) but was low under field conditions (13, 15, 51). Heritability of the reaction to *Pseudomonas syringae* (3) and to *C. flaccumfaciens* (13, 51) in beans, and to *C. michiganense* in tomatoes (19), was high. Transgressive segregation for higher levels of resistance to the pathogens was observed in the following crops: in beans to *X. phaseoli* (13, 51, 64; S. T. Mohan, personal communication), in tomato to *P. solanacearum* (4) and *C. michiganense* (19), and in cucumber to *P. solanacearum* (4) and *C. michiganense* (19), and in cucumber to *P. lachrymans* (30). Different genes controlled the reaction of leaves and pods of *Phaseolus vulgaris* to *X. phaseoli*, so that it was important to select for resistance in both organs (13, 51, 64).

Association with resistance. Linkage was detected between genes that control the tolerant reaction to *X. phaseoli* and delayed flowering under long days and high temperature in *P. vulgaris* (15, 64). The linkage was later broken and early flowering and high tolerance were recombined (13, 51). Stage of host maturity and disease reaction to *X. phaseoli* must be considered in selection and line evaluations, since tolerance decreases during pod development (15). The disease reaction of lines should be compared at the same stage of development under similar temperature regimes. Resistance in tomato to *Pseudomonas solanacearum* also changes with plant age. Resistant plants are susceptible up to 21 days of age and increase in resistance from 3 to 7 weeks, which can lead to conflicting reports in the literature on the degree of resistance to germplasm (W. R. Henderson, personal communication). Unfavorable associations were detected in tomatoes between small fruit size with bacterial wilt (1) and bacterial canker (19) resistance. However, satisfactory fruit size and bacterial wilt resistance were combined in North Carolina in utilizing different sources of resistance (24).

The *Bw* gene in *Cucumis sativus* (PI 200815), which controls resistance to *E. tracheiphila*, was tightly linked to the *M* gene for pistillate flower (27). Bacterial wilt resistance was also associated with undesirable characteristics, such as pale foliage, late flowering, short fruit, and slow growth (35). Now, satisfactory fruit length has been recombined with wilt resistance in the 'Tablegreen' background through rigorous selection in a backcross and selfing program (H. M. Munger and R. E. Wilkinson, personal communication). The bacterial wilt-resistant lines are not satisfactory for release as cultivars because of unfavorable traits. However, these latter traits are recessive and wilt resistance is dominant, so experimental F_1 hybrids of gynoecious x bacterial wilt-resistant lines have been used to overcome the problem and show promise for possible release (H. M. Munger and R. E. Wilkinson, personal communication). Maintenance and use of the bacterial wilt-resistant parents to produce F_1 hybrids is still a problem, since they grow slowly and flower late.

Breeding methods

Pedigree selection has been highly successful in self-pollinated crops in developing cultivars and/or lines with resistance to: *X. phaseoli* (13, 15, 51, 76), *C. flaccumfaciens* (13, 51, 77), and *P. phaseolicola* (13, 51, 76) in *Phaseolus vulgaris* dry beans; to *X. vignicola* in cowpeas (13); and to *Pseudomonas solanacearum* in tomatoes (24, 75) and peanuts (13). Resistant inbred lines, for use in F_1 hybrid development, have been isolated in the cross-pollinated crops; in cabbage to *X. campestris* (73), in sweet corn to *E. stewartii* (39, 58), and in cucumbers to *P. lachrymans* (5). Increased levels of resistance to *X. phaseoli* is needed in *Phaseolus vulgaris*, in tomatoes to *Pseudomonas solanacearum*, in cucumbers to *P. lachrymans* and in maize to *C. nebraskense*. C. O. Gardner (personal communication), and G. Reed (personal communication) have made substantial gains in improving the level of resistance to *C. nebraskense* in maize and to *E. tracheiphila* in melons (*Cucumis melo*), respectively, through recurrent selection. Transgressive segregation for increased levels of resistance to *X. phaseoli* has been recorded in *Phaseolus vulgaris* (64; S. T. Mohan, personal communication) and to *Pseudomonas lachrymans* in cucumber (30). Recurrent selection would probably be useful also in the latter case since additive gene effects are important for resistance. However, this would be more difficult with the self-pollinated bean crop.

Backcross breeding has been used successfully in incorporating major genes for resistance to 1 or more pathogens into desirable recurrent parents of *Phaseolus vulgaris* (13, 51). Coyne and Schuster (13, 51) successfully combined resistance to *X. phaseoli*, which is quantitatively inherited, and *Corynebacterium flaccumfaciens*, which is simply inherited in 'Great Northern Star' *P. vulgaris*. They utilized the common blight-tolerant line GN Nebraska #1 sel. 27 as the recurrent parent in a backcross program in transferring resistance to *C. flaccumfaciens* from the donor parent PI 165078.

Dahlbeck et al. (17) showed that polygenes which control horizontal resistance and a major gene which controls vertical resistance to *X. vesicatoria* in pepper resulted in a slower rate of disease development than when either type of resistance was used alone. A polygenic system may not always provide long-term resistance to a bacterial pathogen. GN Nebraska #1 sel. 27 *P. vulgaris*, although highly resistant to strains of *X. phaseoli* in the United States, was susceptible to strains from Brazil, Colombia, and Uganda (64). According to accepted usage, horizontal resistance infers a uniformity of reaction to various races or strains of a pathogen and is polygenically inherited. The above example is an exception to this definition, since the reaction is polygenically inherited but shows a differential reaction to diverse isolates.

Interspecific hybridization. This approach has been useful in a few cases in transferring genes for a high level of resistance to several bacterial pathogens from exotic species into modern cultivars/lines. Genes for resistance to *Pseudomonas solanacearum* have been transferred from *Solanum phureja* Juz and Buk. to *S. tuberosum* L. (54), and from *L. pimpinellifolium* (Jusl.) Mill. to *L. esculentum* (24); to *C. michiganense* from *L. pimpinellifolium* to *L. esculentum* (19), to *X. phaseoli* from *Phaseolus coccineus* L. (21) and *P. acutifolius* Gray (Tepary bean) (13, 26) to *P. vulgaris*. Honma (26) successfully used embryo culture in the latter cross to overcome F_1 embryo abortion and to produce several fertile F_1 hybrids. Coyne and Schuster (13) later found a common blight-resistant selection, designated #27, in the 'GN Nebraska #1' cultivar derived from this cross, which has since become a useful parent for other breeders (13).

Cell culture. Cells and regenerated plants resistant to a bacterial toxin have been isolated from susceptible germplasm. Carlson (10) used methionine sulfoximine, an analog of the wildfire toxin, to screen for resistance to this toxin in haploid cells of tobacco. Diploid resistant plants were regenerated from resistant cells and resistance was controlled by a single semidominant gene. This procedure may offer a new method to plant breeders to develop plants resistant to bac-

Table 1. Sources of resistance to bacterial pathogens used by vegetable crop breeders.

Crop	Pathogen (Disease)	Source[z]
Potato	*Pseudomonas solanacearum* E.F.Sm. (Bacterial wilt)	*S. phureja*
Eggplant		Matale, Kopek
Tomato		*L.* var. *pyriforme* Beltsville #3814 *L.e.* var. *cerasiforme* PI 129080 (Colombia) *L.p.* PI 127805A *L.e.* VC 8-1-2-1 *L.e.* Llanos de Colce
	Pseudomonas tomato (Okobe) Alls. (Bacterial speck)	*L.e.* Farthest North
	Corynebacterium michiganense (E.F.Sm.) H. L. Jens. (Bacterial canker)	*L.e.* Bulgaria 12 (PI 336729) *L.p.* PI 344102 *L.p.* Utah 737 *L. hirsutum* PI 251305
Pepper	*Xanthomonas vesicatoria* (Doidge) Dows. (Leaf spot)	PI 163192, many others
Cucumber	*Erwinia tracheiphila* (E.F.Sm.) Holland (Bacterial wilt)	PI 200818
	Pseudomonas lachrymans (E.F.Sm. & Bryan) Carsner (Angular leaf spot)	PI 169400, PI 197087 Polish and U.S. inbreds
Sweet corn	*Erwinia stewartii* (E.F.Sm.) Dye (Stewart's wilt)	Numerous inbreds
	Corynebacterium nebraskense Schuster, Hoff, Mandel & Lazar (Leaf freckles and wilt)	Several inbreds
Cabbage	*Xanthomonas campestris* (Pam.) Dows. (Black rot)	Early Fuji Wisconsin inbreds
Common beans	*Pseudomonas phaseolicola* (Burkh.) Dows. (Halo blight)	PI 150414 GN Nebr. #1 sel. 27 Red Mexican UI-3 Negro Vaine Blanca El Conge
	Pseudomonas syringae Van Hall (Brown spot)	PI 313537
	Corynebacterium flaccumfaciens (Hedges) Dows. (Bacterial wilt)	PI 165078
	Xanthomonas phaseoli (E.F.Sm.) Dows. (Common blight)	GN Nebr. #1 sel. 27 PI 207262
Tepary beans	*Xanthomonas phaseoli* (E.F.Sm.) Dows.	Tepary beans
Peanut	*Pseudomonas solanacearum* E.F.Sm. (Bacterial wilt)	Schwartz 21

[z] Abbreviations: *S* = *solanum*, *L.e.* = *Lycopersicon esculentum*
L.p. = *Lycopersicon pimpinellifolium*

terial toxins where genes for resistance have not been identified in existing germplasm collections.

Resistant cultivars. High levels of resistance to bacterial pathogens have been incorporated into cultivars and/or lines in the following vegetable crops: in sweet corn to *E. stewartii* (39, 58); in cucumber to *E. tracheiphila* (35) and *Pseudomonas lachrymans* (5); in cabbage to *X. campestris* (73); in *Phaseolus vulgaris* beans to *Pseudomonas phaseolicola* and *C. flaccumfaciens* (13, 51, 77); and in peanuts (13) and eggplant (74) to *P. solanacearum* (Table 2). The development of sweet corn resistant to Stewart's wilt and peanuts resistant to bacterial wilt has removed the threat of these diseases in several regions. Moderately high resistance has been incorporated in tomato to *C. michiganense* (Table 2) (20). Several 'Great Northern' dry bean cultivars with moderately high leaf and pod resistance to *X. phaseoli* and *X.p.* var. *fuscans* have been developed in Nebraska (Table 2) (13, 51). These cultivars also show a reduction in seed transmission of the bacteria in comparison with susceptible cultivars (52). Katherman et al. (28) also found a reduction of seed transmission of *P. phaseolicola* in cultivars of dry beans with intermediate resistance in comparison with a susceptible cultivar. 'Venus' and 'Saturn' were the first tomato cultivars released with resistance to *P. solanacearum* (24). However, these cultivars, adapted to North Carolina, were susceptible under high temperatures in the tropics and in the presence of more virulent strains of the pathogen. 'VC-4' and '1169' tomatoes developed in the Philippines, lines developed by AVRDC, Taiwan, and 'Kewalo' from Hawaii, express useful levels of resistance under tropical conditions. 'Caxamarca' and 'Molinera' potatoes developed in Peru, have high resistance to *P. solanacearum*, but become susceptible under high temperatures (54). Cabbage cultivars have recently been released with resistance to *X. campestris*, but there is still a need to improve the marketable characteristics of resistant cabbage. There are no resistant cultivars to bacterial pathogens in the following crops: in sweet corn to *C. nebraskense*; in pepper and tomato to *X. vesicatoria*; in *Phaseolus vulgaris* beans to *Pseudomonas syringae*; and in melons (*Cucumis melo*) to *E. tracheiphila*. However, lines of pepper and beans (*Phaseolus vulgaris*) with major gene and/or polygenic resistance to *X. vesicatoria* (R. E. Stall, personal communication) and to *Pseudomonas syringae* (23) respectively, have been developed. No breeding lines of tomato with useful levels of resistance to *X. vesicatoria* have been developed (66).

Nature of resistance

Bacterial toxins include phaseotoxin isolated from *P. phaseolicola*, syringomycin from *P. syringae*, tabtoxins from *P. tabaci*, and rhizobitoxine from *Rhizobium japonicum* (38). Certain of these toxins can reproduce symptoms that their parent bacteria incite. However, the bacterium and the toxin exhibit dissimilar host specificity. The exact mechanisms of action of certain bacterial toxins in the host have not been determined. Phaseotoxin may act through inhibition of L-ornithine carbamyl transferase, with accumulation of ornithine in toxin-treated bean leaf tissue as the result (38).

Bacteria may be inhibited by inherent or induced types of resistance (13, 51). Induced types constitute either hypersensitive or protective reactions. Inherent resistance may involve inhibition by innate substances that are toxic per se or are converted into a toxic form upon cell injury. Overall information is inconclusive, despite attempts to associate phytoalexin production with induced resistance, such as the accumulation of anti-bacterial isoflavonoids in hypersensitively reacting bean leaf tissues inoculated with *P. phaseolicola* (22).

Recent research indicates that lectins are involved in the attachment of avirulent cells of some bacterial pathogen species and so may play a role in induced resistance. Avirulent cells of *P. solanacearum* in a tobacco host and those of *P. phaseolicola* in a bean host, respectively, became attached to the plant cell walls while virulent cells remained free in the intercellular spaces (43, 55). Slusarenko and Wood (57) reported a fraction from the cotyledons of *Phaseolus vulgaris* cv. Red Mexican caused greater agglutination of avirulent race 1 isolates to *Pseudomonas phaseolicola* than virulent race 2 isolates. They speculated that the dominant gene for resistance was involved in the alteration of polysaccharide cell structure in the susceptible host to produce an agglutination of race 1 isolates.

Needs, prospects, and directions.

Nearly all of the predominant dry bean, green bean, pea, and peanut cultivars used in the United States are genetically uniform and have a narrow genetic base (36). About 80% of the seed production of green beans and most of the certified seed of 'Pinto', 'Red Mexican', and 'Great Northern' dry beans are produced in southern Idaho. This entire bean crop could be destroyed if conditions were favorable for an epiphytotic. Serious bean seed losses, due to an epidemic of the bacterial disease halo blight, occurred in Idaho during 1963–1967. This clearly demonstrated a need for decentralization of seed production, for more intensive surveillance of changes in virulence of bacterial pathogens, and for broadening the genetic base of beans.

Many of the existing cultivars resistant to bacterial disease possess the same major genes for resistance. There is a good probability that new virulent strains of the bacterial pathogens will evolve to overcome host resistance. Efforts need to be made to recombine diverse resistance genes in the same genetic background.

Increased research is still needed to improve the level of resistance to the following pathogens: to *E. tracheiphila* in melons; to *P. solanacearum* in tomatoes and potatoes; to *X. vesicatoria* in tomatoes and peppers; to *C. michiganense* in tomatoes; to *C. nebraskense* in sweet corn; and to *X. phaseoli* in beans (*Phaseolus vulgaris*). Germplasm with increased levels of resistance to these pathogens needs to be identified. Furthermore, breeding strategies have to be utilized to recombine diverse genes present in germplasm already available to obtain increased levels of resistance. Emphasis should only be placed on selection of resistant plants with no or

Table 2. Vegetable cultivars developed for resistance to bacterial pathogens.

Crop	Pathogen	Cultivars (country of origin)
Tomato	*Pseudomonas solanacearum* E.F.Sm.	Venus, Saturn (USA) VC-4, 1169 (Philippines)
Potato	*Pseudomonas solanacearum* E.F.Sm.	Caxamarca, Molinera (Peru)
Peanut	*Pseudomonas solanacearum* E.F.Sm.	Schwartz 21 (Indonesia)
Eggplant	*Pseudomonas solanacearum* E.F.Sm.	Matale (Sri Lanka), Kopek (Indonesia)
Tomato	*Corynebacterium michiganense* (E.F.Sm.) H. L. Jens.	Bulgaria 12 (Bulgaria) H2990 (USA)
Cucumber	*Erwinia tracheiphila* (E.F.Sm.) Holland	Saladin Pickler (USA)
	Pseudomonas lachrymans (E.F.Sm.) Carsner	Poinsett (OP), Carolina Hybrid, Calypso Hybrid (USA)
Corn	*Erwinia stewartii* (E.F.Sm.) Dye	Golden Cross Bantam (USA)
Cabbage	*Xanthomonas campestris* (Pam.) Dows	Guardian, Defender, Hancock (USA)
Common beans	*Pseudomonas phaseolicola* (Burkh.) Dows.	Redkote, Redkloud, Montcalm, Mecosta (USA)
	Corynebacterium flaccumfaciens (Hedges) Dows.	GN Star, GN Emerson (USA)
	Xanthomonas phaseoli (E.F.Sm.) Dows.	GN Tara, GN Valley, GN Harris, GN Star (USA)

reduced seed transmission of bacteria, since seed is often a source of inoculum for the emerging crop.

Near-term prospects are promising for the introduction of horticulturally improved cultivars with relatively high resistance levels to the following pathogens: to *E. tracheiphila* in cucumbers; to *X. campestris* in cabbage; to *X. phaseoli* in beans; to *X. vesicatoria* in peppers; and to *P. solanacearum* in tomatoes and potatoes. It will be necessary to increase even further the present levels of resistance in these crops if tomatoes and potatoes are to be grown successfully in the hot lowland tropics.

Novel methods—using cell and tissue culture for the induction and isolation of genetic variants, and transfer of genes that control resistance to bacteria from distant genera using plasmids or other genetic carriers—will continue to receive increased attention and may provide some important breakthroughs. However, traditional plant breeding approaches will be used to develop resistant cultivars in the near future. These will be combined with an integrated management approach of clean seed and transplants, suitable rotations, and suitable disposition of infected debris.

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