Ornamental Plants as Carriers of *Pseudomonas aeruginosa*

J. J. Cho, M. N. Schroth, S. D. Kominos, and S. K. Green

Former Research Assistant and Professor, Department of Plant Pathology, University of California, Berkeley 94720; Clinical Microbiologist, Mercy Hospital, Pittsburgh, Pennsylvania 15219; and Former Graduate Student, Department of Plant Pathology, University of California, Berkeley. Present address of senior author: Hawaiian Agricultural Experiment Station, Kula, Hawaii 96790.

Accepted for publication 1 November 1974.

ABSTRACT

Pseudomonas aeruginosa was isolated from foliage and soil of healthy potted ornamental plants, including African violet, azalea, chrysanthemum, hydrangea, and petunia. These plants were obtained from propagation houses, nurseries, and retail outlets. With chrysanthemums, populations of *P. aeruginosa* were as high as 50 cells/leaf and 5×10^5 cells/g of soil. Sixty-one percent of the isolates from the foliage and 5% of the isolates from the soil were identified as distinct pyocin types. The principal types, B-7, F-6, and F-2, belong to the major groups of clinical strains which cause hospital-associated infections in humans. Although potted ornamental plants present another potential source for the introduction of *P. aeruginosa* into the hospital environment, its relative importance as a disseminating agent appears inconsequential compared to other sources. Many agricultural strains of *P. aeruginosa* and corresponding clinical pyocin types were capable of rotting lettuce, celery, and potatoes, but varied in virulence.

Phytopathology 65:425-431

Hospital-contracted bacterial infections apparently have been a problem since sick people were placed together in the same rooms (14). The extensive use of antibiotics in the 1950's greatly reduced the incidence of the infections caused by gram-positive bacteria such as *Streptococcus* spp. and *Staphylococcus* spp. while allowing the gram-negative bacteria to become more prevalent (18). Among the most important of these were the facultative anaerobes, *Klebsiella pneumoniae* and *Enterobacter* spp., and the aerobe, *Pseudomonas aeruginosa* (8, 18).

Pseudomonas aeruginosa presently accounts for 5-15% of all hospital-associated infections (14). Patients most susceptible to infection by this bacterium include those with burns, immune deficiencies, premature infants, and those requiring respiratory assistance (17, 23, 25). These infections have become more serious in recent years, since debilitated patients now live longer because of advances in medical and surgical technology.

The increasing importance of *P. aeruginosa* as a human pathogen and the excellent techniques for selective isolation (4, 22, 35), identification (19, 21), and typing of strains (9, 12, 42) has encouraged epidemiological studies. Accordingly, infected patients, and moist environments including sinks, inhalation equipment, and anesthesia apparatus were implicated as reservoirs for the survival of *P. aeruginosa* (1, 13, 17, 26, 29). These reservoirs, however, appear to be transitory habitats rather than permanent or natural ones (24).

Plants and soil may be primary reservoirs for *P. aeruginosa*. For example, *P. aeruginosa* (syn. *P. polycolor*) has been cited as a pathogen on lettuce (15, 28), sugarcane (10), and tobacco (7). It has also been reported as weakly pathogenic to chrysanthemums (38). However, the methods used to identify *P. aeruginosa* in some of the reports (10, 15, 28) cast doubt as to the authenticity of the identification; the described bacterium could well have been *P. marginalis* or other species. Green et al. (20) subsequently reported that *P. aeruginosa* occurred in certain agricultural soils, as did Ringen and Drake (31), and had the capacity to multiply and persist for considerable periods in plants. Shooter et al. (34) found *P. aeruginosa* in hospital foods, and Kominos et al. (24) implicated fresh vegetables as probable sources for introduction of *P. aeruginosa* into hospitals. Kominos further showed that many isolates from vegetables were indistinguishable from clinical isolates on the basis of pyocin typing.

These studies prompted us to extend the epidemiological investigations of *P. aeruginosa* from the hospital to agricultural areas. We report herein the recovery of *P. aeruginosa* from potted ornamental plants from commercial sources and discuss the possibility that these plants serve as vehicles for the introduction of the bacterium into a hospital environment. An abstract of this work has been published (6).

MATERIALS AND METHODS.—*Isolation and the identification of P. aeruginosa.*—Foliage and soil samples from potted ornamental plants were examined for the presence of *P. aeruginosa*. Chrysanthemum samples included rooted cuttings from propagation houses, and potted plants from nurseries and retail outlets. Potted African violets, azaleas, and hydrangeas were sampled from both nurseries and retail outlets; petunias were sampled only from retail outlets. Soil samples were obtained from pots of the sampled plants from both nursery and retail sources. Freshly steamed sterilized soil mixtures prepared for planting were also sampled from nurseries.

Five leaves from each of 10 individual plants were assayed to estimate the average population of *P. aeruginosa* per leaf. Each leaf was triturated in 2 ml of sterile distilled water with a mortar and pestle, and a 10-fold endpoint dilution of the suspension was made in test tubes containing 4.5 ml of broth consisting of acetamide and salts (35). Estimates of the percentages of plants harboring *P. aeruginosa* were obtained by placing 10 to 20 leaves from individual plants into 250 ml Erlenmeyer flasks containing 50 ml of acetamide broth. Acetamide broth cultures from the above two experiments were subcultured on cetrimide agar (4) after 48 hours of incubation at 42 C on a rotary shaker at 150 rpm.

Soil was assayed by placing 10-g samples in 100 ml of sterile distilled water and agitating each sample for 30 minutes on a rotary shaker at 300 rpm. Soil suspensions and dilutions thereof were then plated on cetrimide agar. All cultures were incubated at 42 C.

After 24 hours of incubation, colonies that fluoresced on cetrimide agar when irradiated with a UVSL-13 ultraviolet lamp (Ultra-Violet Products, Inc., San Gabriel, California) were suspected to be *P. aeruginosa*. Additional tests for identification included growth and fluorescence in acetamide broth after 24 hours of incubation at 42 C (35), a positive oxidase test (19,21,37), pyocyanin production and production of slime in 2-ketogluconate (21), and growth on geraniol and denitrification (37).

Pyocin typing. —Strain identification of *P. aeruginosa* isolates was made on the basis of pyocin production. All isolates of *P. aeruginosa* from the foliage and 77 representative isolates from soil were pyocin typed. Typing was based on the inhibition patterns of these isolates on 11 indicator strains according to the technique developed by Darrell and Wahba (9) as modified by Zabransky and Day (42).

Plant pathogenicity tests.—The capacity of 10 clinical strains of *P. aeruginosa* to rot plant material was compared with six strains isolated from ornamental plant material. The clinical strains were common pyocin types isolated from infected hospital patients. Comparable pyocin types isolated from ornamental plants were used.

Twenty-four-hour-old cultures of *P. aeruginosa* were suspended in sterile distilled water at about 10^8 cells/ml. Five ml of each suspension was sprayed onto lettuce slices *(Lactuca sativa* L. 'Great Lakes'). Celery stalks (*Apium graveolens* L. 'Dulce') 8 cm long, and potato tuber slices *(Solanum tuberosum* L. 'White Rose'), 1.5-cm thick, were inoculated by stabbing with a dissecting needle dipped into the bacterial suspensions. Inoculated plant materials were incubated in a moist environment approximately 100% relative humidity (RH)] at 24-25 and 37 C. Rotting was observed daily up to 7 days after inoculation. Each test was run three times with three replications.

RESULTS.—*Population of P. aeruginosa on foliage.*—Ninety percent of the chrysanthemums from propagation houses harbored populations of *P.*

aeruginosa at approximately two to five cells per leaf (Table 1). The RH of these houses was approximately 90 to 100%. In nurseries with 50-60% RH, approximately 36% of the chrysanthemums harbored *P. aeruginosa*. However, the bacterium was found on only 22% of the plants in retail houses with 10 to 20% RH.

Pseudomonas aeruginosa was isolated from one of 10 African violet plants from nursery sources, but was not isolated in the 10 plants sampled from retail sources. Three of 15 petunia plants sampled from retail outlets was colonized by the bacterium. *Pseudomonas aeruginosa* was not isolated from the foliage of 30 azaleas, 40 hydrangeas, and 10 hanging fern plants sampled from nursery and retail sources.

Soil population of P. aeruginosa.—Planting-soil mixtures supporting ornamental plants contained populations of *P. aeruginosa* up to 5×10^5 cells/g (Table 2). Although *P. aeruginosa* was not found on hydrangea and azalea foliage, the soil in which these plants were growing contained approximately 2×10^2 cells/g. Soil mixtures sampled prior to planting also contained *P. aeruginosa*.

The percentage of soil samples infested with the bacterium ranged from 67 to 100%. However, the

TABLE 1. Populations of *Pseudomonas aeruginosa* found on the foliage of various potted ornamental plants

Plant	Source	Plants cultured (no.)	Plants with *P. aeruginosa*	No. of leaf samples with colony counts of	
				1 to 10	11 to 100
Chrysanthemum	Propagator	30	27	27	0
Chrysanthemum	Nursery	108	50	46	4
Chrysanthemum	Retailer	61	22	22	0
African violet	Nursery	10	1	1	0
African violet	Retailer	10	0	0	0
Azalea	Nursery	5	0	0	0
Azalea	Retailer	25	0	0	0
Fern	Nursery	10	0	0	0
Hydrangea	Nursery	20	0	0	0
Hydrangea	Retailer	20	0	0	0
Petunia	Retailer	15	3	3	0

TABLE 2. Populations of *Pseudomonas aeruginosa* from the soil in which various ornamental plants were growing

Plant	Source	Soil samples cultured (no.)	No. of samples positive	No. of samples with *P. aeruginosa* (population/g of soil)			
				10^2 to 10^3	10^3 to 10^4	10^4 to 10^5	10^5 to 10^6
Chrysanthemum	Nursery	9	9	4	1	3	1
Chrysanthemum	Retailer	12	8	6	1	1	0
African violet	Nursery	5	5	5	0	0	0
African violet	Retailer	10	10	10	0	0	0
Azalea	Nursery	2	2	2	0	0	0
Hydrangea	Nursery	2	2	2	0	0	0
Petunia	Retailer	5	4	4	0	0	0
None[a]	Nursery	4	3	2	1	0	0

[a]Samples from freshly steamed pasteurized potting soil mixtures prepared for planting.

TABLE 3. Pyocin types of *Pseudomonas aeruginosa* isolated from potted ornamental plants

Plant	Source	Isolates typed (no.)	Pyocin types											
			B-6	B-7	D-2	F-2	F-4	F-6	I-1	0	R-1	T	VT[a]	NT[b]
Chrysanthemum foliage	Propagator	32	0	16	0	4	0	1	0	0	0	0	3	8
Chrysanthemum foliage	Nursery	26	2	0	0	2	0	9	1	3	0	1	1	7
Chrysanthemum foliage	Retailer	18	0	4	3	0	4	0	1	1	1	1	0	3
Chrysanthemum soil	Nursery	37	1	0	0	0	0	0	0	0	0	0	5	31
Chrysanthemum soil	Retailer	7	0	0	0	0	0	0	0	0	0	0	1	6
African violet foliage	Nursery	6	0	0	0	0	0	0	0	0	0	0	1	5
African violet soil	Nursery	10	0	0	0	0	0	0	0	0	0	0	0	10
African violet soil	Retailer	10	0	0	0	0	0	0	2	0	0	0	0	8
Azalea soil	Nursery	3	0	0	0	0	0	0	0	0	0	0	0	3
Hydrangea soil	Nursery	9	0	0	0	0	0	0	0	0	0	0	0	9
Petunia foliage	Retailer	3	0	0	0	0	0	0	0	0	0	0	0	3
Total		161	3	20	3	6	4	10	4	4	1	2	11	93

[a]VT indicates variable type.
[b]NT indicates nontypable.

TABLE 4. The capacity of clinical and plant isolates of *Pseudomonas aeruginosa* to damage various plant material[a]

Isolate	Pyocin type	Origin	Damage after 4 days		
			Celery[b]	Lettuce[b]	Potato[c]
PA-3	D-2	Human	++++	++++	++++
PA-9	D-2	Human	++++	++++	++++
P-169	D-2	Plant	−	+	+++
P-168	D-2	Plant	+	++++	++++
PA-7	J-6	Human	++	++++	++++
PA-8	B-6	Human	++++	++++	++
P-185	B-6	Plant	++++	++++	++
PA-13	B-4	Human	++	+++	++
PA-10	B-7	Human	+	+	−
P-18	B-7	Plant	++	+++	++
P-31	B-7	Plant	+	+++	++++
PA-11	F-6	Human	−	−	−
PA-12	F-6	Human	+	+	+
P-179	F-6	Plant	++	+++	+

[a]Tests were conducted under approximately 100% relative humidity at 37 C.

[b]− = no symptoms; + = one-quarter or less of the plant tissue water-soaked; ++ = one-quarter to one-half of plant tissue water-soaked; +++ = one-half to entire plant tissue water-soaked and slight rot; and ++++ = total collapse of the tissue in the case of lettuce; entire tissue water-soaked and tissue soft with celery.

[c]− = no symptoms; + = growth on the surface, but no soft rot; ++ = growth over the entire surface and slight rot only near the area of initial inoculation; +++ = growth over the entire surface and one-quarter to one-half of the slice soft; and ++++ = growth over the entire surface and the entire slice soft.

population of *P. aeruginosa* was much less in soil from retail houses than in soil from nursery sources.

Pyocin typing.—Seventy-one percent of 76 isolates from chrysanthemum foliage were identifiable as pyocin types (Table 3). The principal types were B-7, F-2, and F-6, which belong to the groups of major clinical strains isolated from infected hospital patients (23, 24). Four of these isolates gave variable typing patterns (VT) in that they exhibited different inhibition patterns on the 11 indicator strains used in replicate tests. The remaining 18 isolates did not inhibit any of the indicator strains and therefore could not be typed [nontypable (NT)]. The majority of the isolates from the foliage of the ornamental plants were nontypable. Five isolates from African violet foliage were nontypable and one produced a variable pattern. Three isolates from petunia foliage were nontypable.

Only two of 45 representative isolates from soil in which chrysanthemums were growing could be identified by pyocin typing. These strains were types B-6 and T, and were not commonly found on plant foliage. Thirty-seven isolates were nontypable and the remaining six produced a variable typing pattern. Two of 20 isolates from African violet soil mixtures could be typed. Both were of the type I group and the rest were nontypable. Three isolates from azalea soil mixtures and all nine isolates from hydrangea soil were nontypable.

Plant pathogenicity.—Several strains of *P. aeruginosa* isolated from plants and from infected human patients produced similar rotting symptoms on celery, lettuce, and potato slices (Table 4). The amount of rot varied considerably among strains with some causing none. Most nontypable isolates from ornamental plants produced neither water-soaking nor rot symptoms. Rate of symptom development on all inoculated plant material was optimal at 37 C. Similar symptoms and ranking, however, were observed on these plant materials when the incubation temperature was lowered to 30 and 24-25 C, but these symptoms were delayed for 4-7 days. Because there was little difference in the final result of these inoculations, 37 C was used routinely.

The following symptoms were observed on inoculated plant material when incubated at 37 C and approximately 100% RH: With celery, a water-soaked reaction occurred 1 day after inoculation (Fig. 1). The reaction zone turned either dark-green or brown about the inoculation site after 2 days. This water-soaking continued to spread throughout the stalk during the 4-5 day incubation period. Some of the celery stalks became soft around the inoculation site or throughout the entire stalk. Some strains did not infect the stalks.

The first symptom observed with lettuce was water-soaking on the fringes of the slice after 2 to 3 days of incubation. This symptom progressed inward, principally from the cut edges of the slice and the noninjured tips of young leaves. Many strains of the bacterium invaded the entire slice, causing water-soaking throughout the tissue (Fig. 2). A few strains rotted the entire slice and the tissue collapsed 4 days after inoculation. However, the isolates varied in virulence and some caused little or no rotting of the lettuce tissue.

DISCUSSION.—Isolation of *P. aeruginosa* from the foliage of potted ornamental plants and soils used as planting mixtures indicated that both may serve as sources for the bacterium, thus supporting the report of Green et al. (20) who cited soil as its natural habitat. Although the bacterium readily colonizes certain plants (20, 24) under favorable environmental conditions, plants most likely are only transitory sources for the bacterium. The population of *P. aeruginosa* declines when plants are grown under relatively dry conditions (20). The present study confirms this since the bacterial population on chrysanthemums and number of infested plants decreased markedly when transferred from humid greenhouses to retail outlets with low humidities.

It appears that certain plants are colonized more readily by *P. aeruginosa*. For example, highest populations of the bacterium were isolated from tomatoes, radishes, celery, and carrots in descending order (24). Among ornamental plants, chrysanthemums supported the highest population of *P. aeruginosa*, and a greater percentage was infested than were other ornamental plants. Plants also appear to select certain strains of the bacterium from the soil reservoir; 78% of the isolates from chrysanthemums produced pyocins, of which 93% were identified as distinct pyocin types. Kominos et al. (24) further reported that a high percentage of isolates from vegetable material entering hospitals also produced pyocins of which 84% were identified as distinct pyocin types indistinguishable from clinical isolates. It was particularly noteworthy that some of the isolates from plants used in our studies also were indistinguishable from clinical strains on the basis of pyocin typing. The majority of the isolates from soil, however, were nontypable. It is possible that some non-

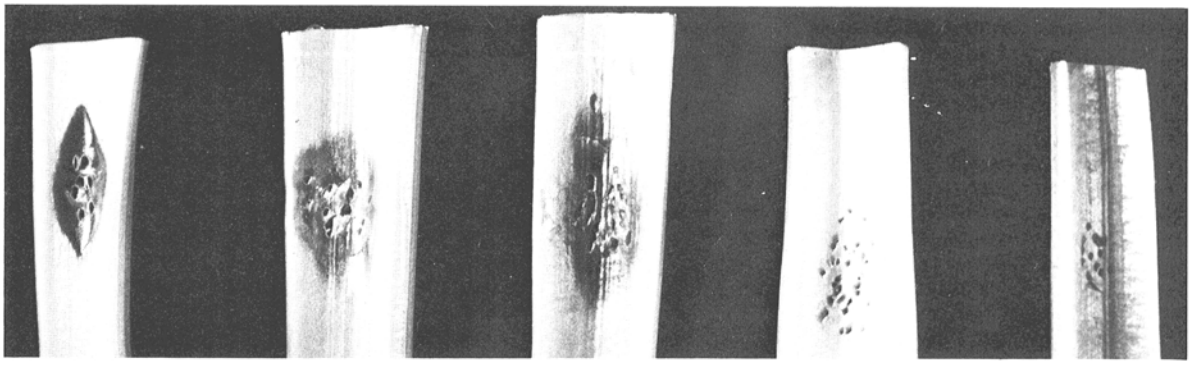

Fig. 1. Leaves of lettuce (cultivar, Great Lakes) exhibiting rot 72 hours after inoculation with a clinical strain of *Pseudomonas aeruginosa,* isolate PA-3.

pyocinogenic strains in soil are potential human pathogens and that the modes for introduction of these strains into the hospital environment are limited. The use of pyocin typing method for the identification of *P. aeruginosa* strains therefore may have to be supplemented with serological or bacteriophage typing methods (12, 16).

Pathogenicity tests demonstrated the similarity between clinical and plant isolates in their capacity to rot plant material. No correlations, however, could be made between pyocin types and rotting capacity, or between rotting capacity and sources of isolation, clinical or agricultural, because of the variation in virulence observed among the isolates. Perhaps a relationship may be detected using a greater number of isolates.

The question of whether or not *P. aeruginosa* is a true plant pathogen appears somewhat controversial. Although it has been reported as a pathogen on several plants (7, 10, 15, 28), it has not been readily accepted as a "typical" plant pathogen, presumably because it does not cause macroscopic damage unless the plants are subjected to conditions of high moisture. Our pathogenicity tests, however, suggest that certain strains are capable of rotting plant material under environmental conditions that can occur in the field or during the marketing process. We therefore are inclined to view *P. aeruginosa* as a "quasi-pathogen" that is part of the continuum extending from saprophytism to parasitism. It probably is representative of a number of bacteria, such as *P. viridiflava* (2, 41), that have pathogenic potential, which is expressed during certain environmental conditions, especially those unfavorable to plant growth.

Thus far, clinical microbiologists have not traced a single nosocomial infection to an ornamental plant source (33), and there is no evidence that these plants constitute a primary source of bacteria for hospital infections. Although Taplin and Mertz (40) found large populations of *P. aeruginosa* and other gram-negative bacteria in vase water with cut flowers, they do not feel that flowers constitute a major threat to most patients in general hospitals. They suggested, however, that plants should not be introduced into wards with highly susceptible patients. Further, the population of *P. aeruginosa* carried by ornamental plants may be insignificant when considering that salads and vegetables

Fig. 2. Celery stalks 72 hours after inoculation with clinical and agricultural strains of *Pseudomonas aeruginosa* showing differences in strain virulence; left to right, PA-3 (clinical), PA-8 (clinical), P-185, PA-11 (clinical), and P-169 (agricultural).

in hospitals harbor relatively high populations of *P. aeruginosa* (24). Patients consuming these salads could ingest as many as 5×10^4 cells of the bacterium.

Although pyocin typing indicates that human and certain plant strains of *P. aeruginosa* are similar, this offers only circumstantial evidence to the supposition that patients acquire the organism from vegetables. Final proof depends upon further studies such as the one currently underway at Mercy Hospital, Pittsburgh, Pennsylvania. Patients who are admitted free of *P. aeruginosa* are examined to determine whether or not they can acquire the bacterium via consumption of fresh

vegetable salads and become colonized in the gastrointestinal tract. In addition, these studies are designed to determine whether or not *P. aeruginosa* acquired from vegetables can lead to infection of debilitated patients.

These studies with the objective of determining the role of a plant bacterium in human disease shows the beginning of cooperative efforts between plant pathologists and medical microbiologists. Other species of bacteria involved in human disease and reportedly isolated from plants are *Klebsiella pneumoniae*, *Enterobacter cloacae*, *Enterobacter agglomerans* (syn. *Erwinia herbicola - lathyri* group), *Serratia-marcescens* (3, 24, 27, 30, 32, 34, 39) and *Pseudomonas cepacia* (5, 11, 36). We believe that these two fields should intensify cooperation, since previous studies suggest that plants may serve as natural reservoirs for microorganisms that have pathogenic potential to man.

LITERATURE CITED

1. BASSETT, D. J. C. 1971. Causes and prevention of sepsis due to gram-negative bacteria: common-source outbreaks. Proc. Roy. Soc. Med. 64:980-986.
2. BILLING, E. 1970. Pseudomonas viridiflava (Burkholder, 1930; Clara, 1934). J. Appl. Bacteriol. 33:492-500.
3. BOTTONE, E., and S. S. SCHNEIERSON. 1971. Erwinia species: an emerging human pathogen. Am. J. Clin. Pathol. 57:400-405.
4. BROWN, V. I., and E. J. L. LOWBURY. 1965. Use of an improved cetrimide agar medium and other culture methods for Pseudomonas aeruginosa. J. Clin. Pathol. (London) 18:752-756.
5. BURKHOLDER, W. H. 1950. Sour skin, a bacterial rot of onion bulbs. Phytopathology 40:115-117.
6. CHO, J. J., S. K. GREEN, M. N. SCHROTH, and S. D. KOMINOS. 1973 Potted ornamental plants: another source for introduction of Pseudomonas aeruginosa into hospitals. Phytopathology 63:1215 (Abstr.).
7. CLARA, F. M. 1930. A new bacterial leaf disease of tobacco in the Philippines. Phytopathology 20:691-706.
8. CURTIN, J. A., R. G. PETERSDORF, and I. L. BENNETT, JR. 1961. Pseudomonas bacteremia:review of ninety-one cases. Ann. Intern. Med. 54:1077-1107.
9. DARRELL, J. H., and A. H. WAHBA. 1964. Pyocin-typing of hospital strains of Pseudomonas pyocyanea. J. Clin. Pathol. (London) 17:236-242.
10. DESAI, S. V. 1935. Stinking rot of sugarcane. Indian J. Agric. Sci. 5:387-392.
11. EDERER, G. M., and J. M. MATSEN. 1972. Colonization and infection with Pseudomonas cepacia. J. Infect. Dis. 125:613-618.
12. EDMONDS, P., R. R. SUSKIND, B. G. MACMILLAN, and I. A. HOLDER. 1972. Epidemiology of Pseudomonas aeruginosa in a burns hospital: evaluation of serological, bacteriophage, and pyocin typing methods. Appl. Microbiol. 24:213-218.
13. EDMONDS, P., R. R. SUSKIND, B. G. MACMILLAN, and I. A. HOLDER. 1972. Epidemiology of Pseudomonas aeruginosa in a burns hospital: surveillance by a combined typing system. Appl. Microbiol. 24:219-225.
15. ELROD, R. P., and A. C. BRAUN. 1942. Pseudomonas aeruginosa, its role as a plant apthogen. J. Bacteriol. 44:633-644.
16. FARMER, J. J. III., and L. G. HERMAN. 1969. Epidemiological fingerprinting of Pseudomonas aeruginosa by the production of and sensitivity to pyocin and bacteriophage. Appl. Microbiol. 18:760-765.
17. FIERER, J., P. M. TAYLOR, and H. M. GEZON. 1967. Pseudomonas aeruginosa epidemic traced to delivery-room resuscitators. N. Engl. J. Med. 276:991-996.
18. FINLAND, M. 1970. Changing ecology of bacterial infections as related to antibacterial therapy. J. Infect. Dis. 122:419-431.
19. GILARDI, G. L. 1971. Characterization of Pseudomonas species isolated from clinical specimens. Appl. Microbiol. 21:414-419.
20. GREEN, S. K., M. N. SCHROTH, J. J. CHO, and S. D. KOMINOS. 1974. Agricultural plants and soil as a possible reservoir for Pseudomonas aeruginosa. Appl. Microbiol. (In press).
21. HAYNES, W. C. 1951. Pseudomonas aeruginosa–its characterization and identification. J. Gen. Microbiol. 5:939-950.
22. HEDBERG, M. 1969. Acetamide agar medium selective for Pseudomonas aeruginosa. Appl. Microbiol. 17:481.
23. KOMINOS, S. D., C. E. COPELAND, and B. GROSIAK. 1972. Mode of transmission of Pseudomonas aeruginosa in a burn unit and an intensive care unit in a general hospital. Appl. Microbiol. 23:309-312.
24. KOMINOS, S. D., C. E. COPELAND, B. GROSIAK, and B. POSTIC. 1972. Introduction of Pseudomonas aeruginosa into a hospital via vegetables. Appl. Microbiol. 24:567-570.
25. LEVINE, A. S., R. G. GRAW, JR., and R. C. YOUNG. 1972. Management of infections in patients with leukemia and lymphoma: current concepts and experimental approaches. Seminars Hematol. 9:141-179.
26. LOWBURY, E. J. L., B. T. THOM, H. A. LILLY, J. R. BABB, and K. WHITTALL. 1970. Sources of infection with Pseudomonas aeruginosa in patients with tracheostomy. J. Med. Microbiol. 3:39-56.
27. MEYERS, B. R., E. BOTTONE, S. Z. HIRSCHMAN, and S. S. SCHNEIERSON. 1972. Infections caused by microorganisms of the genus Erwinia. Ann. Intern. Med. 76:9-14.
28. PAINE, S. G., and J. M. BRANFOOT. 1924. Studies in bacteriosis. XI. A bacterial disease of lettuce. Ann. Appl. Biol. 11:312-317.
29. PHILLIPS, I., and G. SPENCER. 1965. Pseudomonas aeruginosa cross-infection due to contaminated respiratory apparatus. Lancet 2:1325-1327.
30. PIEN, F. D., W. J. MARTIN, P. E. HERMANS, and J. A. WASHINGTON, II. 1972. Clinical and bacteriologic observations on the proposed species, Enterobacter agglomerans (the Herbicola-Lathyri bacteria). Mayo Clin. Proc. 47:739-745.
31. RINGEN, L. M., and C. H. DRAKE. 1952. A study of the incidence of Pseudomonas aeruginosa from various natural sources. J. Bacteriol. 64:841-845.
32. SAMISH, Z., and R. ETINGER-TULCZYNSKA. 1963. Distribution of bacteria within the tissue of healthy tomatoes. Appl. Microbiol. 11:7-10.
33. SCHROTH, M. N., J. J. CHO, and S. D. KOMINOS. 1973. No evidence that Pseudomonas on chrysanthemums harms patients. Lancet 2:906-907.
34. SHOOTER, R. A., E. M. COOKE, M. C. FAIERS, A. L. BREADEN, and S. M. O'FARRELL. 1971. Isolation of Escherichia coli, Pseudomonas aeruginosa, and Klebsiella from foods in hospitals, canteens, and schools. Lancet 2:390-392.
35. SMITH, R. F., and S. L. DAYTON. 1972. Use of acetamide broth in the isolation of Pseudomonas aeruginosa from rectal swabs. Appl. Microbiol. 24:143-145.
36. SNELL, J. J. S., L. R. HILL, S. P. LAPAGE, and M. A.

CURTIS. 1972. Identification of Pseudomonas cepacia Burkholder and its synonomy with Pseudomonas kingii Jonsson. Int. J. Syst. Bacteriol. 22:127-138.
37. STANIER, R. Y., N. J. PALLERONI, and M. DUODOROFF. 1966. The aerobic pseudomonads: a taxonomic study. J. Gen. Microbiol. 43:159-271.
38. STAPP, C. 1934. Eine bakteriose an Chrysanthemen. Zentralbl. für Bakteriol., Parasitenkd. Infektionsk. Abt. II, 90:320-329.
39. STARR, M. P., and A. K. CHATTERJEE. 1972. The genus Erwinia: Enterobacteria pathogenic to plants and animals. Annu. Rev. Microbiol. 26:389-426.
40. TAPLIN, D., and P. M. MERTZ. 1973. Flower vases in hospitals as reservoirs of pathogens. Lancet 2:1279-1281.
41. WILKE, J. P., D. W. DYE, and D. R. W. WATSON. 1973. Further hosts of Pseudomonas viridiflava. N.Z.J. Agric. Res. 16:315-323.
42. ZABRANSKY, R. J., and F. E. DAY. 1969. Pyocine typing of clinical strains of Pseudomonas aeruginosa. Appl. Microbiol. 17:293-296.