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An Evaluation of the Wilt-Causing Bacterium *Ralstonia solanacearum* as a Potential Biological Control Agent for the Alien Kahili Ginger (*Hedychium gardnerianum*) in Hawaiian Forests[1]

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Kahili ginger (*Hedychium gardnerianum*) is an invasive weed in tropical forests in Hawaii and elsewhere. Bacterial wilt caused by the ginger strain of *Ralstonia* (= *Pseudomonas*) *solanacearum* systemically infects edible ginger (*Zingiber officinale*) and ornamental gingers (*Hedychium* spp.), causing wilt in infected plants. The suitability of *R. solanacearum* as a biological control agent for kahili ginger was investigated by inoculating seedlings and rooted cuttings of native forest plants, ornamental ginger, and solanaceous species to confirm host specificity. Inoculation via stem injection or root wounding with a bacterial–water suspension was followed by observation for 8 weeks. Inoculations on *H. gardnerianum* were then carried out in ohia-lehua (*Metrosideros polymorpha*) wet forests of Hawaii Volcanoes National Park to determine the bacterium's efficacy in the field. No native forest or solanaceous species developed wilt or other symptoms during the study. The bacterium caused limited infection near the inoculation site on *H. coronarium, Z. zerumbet, Heliconia latispatha,* and *Musa sapientum.* However, infection did not become systemic in any of these species, and normal growth resumed following appearance of initial symptoms. All inoculated *H. gardnerianum* plants developed irreversible chlorosis and severe wilting 3–4 weeks following inoculation. Systemic infection also caused death and decay of rhizomes. Most plants were completely dead 16–20 weeks following inoculation. The destructiveness of the ginger strain of *R. solanacearum* to edible ginger has raised questions regarding its use for biological control. However, because locations of kahili ginger infestations are often remote, the risk of contaminating edible ginger plantings is unlikely. The ability of this bacterium to cause severe disease in *H. gardnerianum* in the field, together with its lack of virulence in other ginger species, contributes to its potential as a biological control agent.

***Key Words:* *Ralstonia* (= *Pseudomonas*) *solanacearum; Hedychium gardnerianum; Hedychium coronarium; Hedychium flavescens; Metrosideros polymorpha; Alpinia purpurata; Alpinia zerumbet; Phaeomeria magnifica; Heliconia latispatha; Musa sapientum; Ilex anomala; Coprosma ochracea; Zingiber officinale; Zingiber zerumbet; Nicotiana tabacum; Arachis hypogaea;* kahili ginger; wild ginger; biological control; bacterial wilt; systemic infection; alien weed; understory invasion.**

INTRODUCTION

Kahili or wild ginger (*Hedychium gardnerianum* Ker-Gawl) (Zingiberaceae), native to eastern India (Naik and Panigrahi, 1961), has been introduced throughout the tropics and is now invasive in many forest ecosystems (Goodland and Healey, 1996; Harris *et al.*, 1996; Cronk and Fuller, 1995; Byrne, 1992; Macdonald *et al.*, 1991). Kahili ginger is also a serious pest that threatens the long-term stability of invaded native plant communities in Hawaiian ecosystems (Anderson and Gardner, 1996; Loope and Medeiros, 1994; Smith, 1985). Brought to Hawaii by the horticultural industry, it has escaped cultivation and is now naturalized in the state. First collected in 1954 at Hawaii Volcanoes National Park (HVNP) (Wester, 1992), populations are now found on all islands between sea level and 1700 m (Smith, 1985). A very aggressive shade-tolerant plant, kahili ginger is able to form dense thickets on undisturbed sites in the understory of open- and closed-canopy ohia-lehua (*Metrosideros polymorpha* Gaud.) rain forests as well as in open areas in and around HVNP. Currently, kahili ginger has invaded approximately 500 ha in HVNP forests from 1000–1300 m elevation. Like kahili ginger, the other species of *Hedychium* introduced to Hawaii (e.g., *H. flavescens*

[1] The mention of trade names in this study is solely for demonstration purposes and does not represent endorsement of these products by the Biological Resources Division, U.S. Geological Survey, National Park Service or the Pacific Cooperative Studies Unit.

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Carey ex Roscoe and *H. coronarium* König) are weedy, although these are usually confined to forest edges instead of invading the understory.

A number of herbicides have been investigated and used in the past 20 years for control of kahili ginger (Harris *et al.*, 1996; Santos *et al.*, 1986). Currently, the most effective herbicide reported for kahili ginger control is Escort (metsulfuron-methyl) (Harris *et al.*, 1996). Environmental concerns regarding the use of Escort include soil leaching capacity, potential ground water contamination, and effects (usually lethal) on nontarget native species (Harris *et al.*, 1996). Because of the widespread distribution of kahili ginger in Hawaiian national parks, chemical control is cost effective and environmentally safe only in relatively small, intensively managed areas of high conservation value, such as Special Ecological Areas (SEAs) (see Tunison and Stone, 1992). Biological control is now considered the only practical approach for the long-term management of large kahili ginger infestations in native forests.

The most important concern in biological control of kahili ginger is selection of a control agent that does not damage commercial plantings of members of the order Zingiberales (e.g., *Alpinia* spp., *Heliconia* spp., *Musa* spp., and *Zingiber* spp.). A wilt disease of edible ginger (*Zingiber officinale* Roscoe) caused by a strain (=race) of the bacterium *Ralstonia solanacearum* (E. F. Smith) Yabuuchi *et al.* (=*Pseudomonas solanacearum* E. F. Smith) was brought to our attention as a possible biological control agent for kahili ginger by E. E. Trujillo of the Hawaii State and Federal Interagency Steering Committee on Biological Control. Five strains of this soilborne plant pathogen that are widely distributed in tropical, subtropical, and some warm temperate regions of the world have been described (Hayward, 1991). Strains of *R. solanacearum* are determined by host specificity, geographic distribution, and survival under different environmental conditions. The five described strains of *R. solanacearum* are Solanaceous strain (Race 1), Musaceous strain (Race 2), Potato strain (Race 3), Ginger strain (Race 4), and Mulberry strain (Race 5) (Persley *et al.*, 1986). The ginger strain of *R. solanacearum* present in Hawaii has been isolated from both edible and ornamental gingers (Ishii and Aragaki, 1963; Aragaki and Quinon, 1965). The edible ginger isolate was reported to be less virulent than the ornamental ginger isolate on *H. gardnerianum*. However, since the two isolates have similar cultural and pathogenicity characteristics and are distinct from other *R. solanacearum* strains (i.e., Solanaceous and Musaceous), they should be considered to be one strain (Aragaki and Quinon, 1965). The ornamental isolate may no longer be present because the populations of ornamental ginger from which it was isolated have been removed due to land development. The pathogen infects through wounds and becomes systemic, causing wilt and decay of infected tissues. The bacterium is easily spread via infected cuttings, contaminated equipment, and, potentially, by irrigation water that has passed through infested soil (Kelman *et al.*, 1994; Trujillo, 1964). Disease development in susceptible hosts is favored by high moisture and warm (e.g., 30–35°C) temperatures (Quinon *et al.*, 1964). Previous studies by Aragaki and Quinon (1965) and our own greenhouse experiments (Anderson and Gardner, 1996) confirmed that *R. solanacearum* is pathogenic to kahili ginger. Therefore, further investigation of its use as a possible biological control agent is warranted.

In consideration of the above, the purpose of this research was twofold: (1) to investigate the host range of the edible ginger strain of *R. solanacearum* on ornamental gingers as well as on native forest species associated with kahili ginger infestations and (2) if found to be host-specific, to determine if *R. solanacearum* could cause disease of kahili ginger in the field at higher elevations (900–1200 m) at which temperatures are cooler than the optimum for disease development and kahili ginger infestations are greatest.

MATERIALS AND METHODS

Isolation of the Pathogen

Isolates of *R. solanacearum* were collected from diseased rhizomes of *Z. officinale* obtained from a commercial operation of edible ginger production in the Hamakua region of the island of Hawaii in May 1994. The bacterium was readily isolated from rotting shoot tips by streaking the bacterial ooze onto a 0.005% tetrazolium chloride (TZC) medium (Kelman, 1954) and then incubating for 36 h at 33°C. Following incubation, creamy white virulent colonies of *R. solanacearum* were distinguished from avirulent dark red colonies and streaked again to produce numerous colonies (Kelman, 1954). Multiple virulent colonies of *R. solanacearum* were then suspended in 4-dram vials of tap water and stored at approximately 24°C for use as the stock isolate for host range testing.

Host Range Testing

To evaluate host specificity, we inoculated 20 species representing both ornamental and native plants from eight families, including the Solanaceae. Native species selected included those occurring in habitats invaded by kahili ginger or those otherwise considered ecologically significant. Young plants (10–20 cm tall) of each species were either collected in the field or grown from seed and tissue culture. All plants were potted in a growth medium of 5:2:1 (v/v/v) peat, perlite, and vermiculite, respectively. At the time of treatment, all plants grown from seed were 8 weeks old, had 6–8

expanded leaves, and were 10–30 cm tall. Before inoculation, field-collected plants were reestablished on mist benches receiving approximately 250 ml water/day in the greenhouse for 6–8 weeks or longer until new foliage developed and tissue-cultured plants were grown on nutrient agar for 8 weeks and then potted in the above growth medium for 4 weeks.

Inoculum was made by flooding petri plates covered by *R. solanacearum* and collecting the suspension in glass beakers. Inoculum concentration was adjusted spectrophotometrically to approximately 1×10^6 colony forming units (CFU)/ml before inoculation. Species with rhizomes were inoculated with a hypodermic syringe by injecting 5 ml of bacterial suspension into fleshy shoots approximately 5 cm above the soil line. Species with fibrous roots grown from seed were inoculated by cutting roots with a scalpel along three sides of the pot and pouring 10 ml of bacterial suspension over the freshly wounded roots. Controls were treated similarly, but with a sterile water suspension. Concurrently with each host species, five kahili ginger plants were also inoculated to serve as a positive control of pathogenicity. Plants received 250 ml water/day, and diurnal greenhouse temperatures ranged from 24 to 33°C during treatments. Plants were evaluated weekly for disease symptoms for 8 weeks. At the end of the experiment, one plant of each species was randomly chosen, and crown tissue was microscopically examined and cultured on TZC agar for the presence of *R. solanacearum*.

Field Evaluation

The ability to cause disease at higher elevations, at which nighttime temperatures average 10°C during the winter months, was evaluated in kahili ginger-invaded open-canopied ohia-lehua forests near the research center in HVNP. Elevation at the study sites was 1200 m, with a mean annual rainfall of 3300 mm and a mean annual temperature of 17°C (Santos *et al.*, 1986). Belt transects were established and 15 rhizome mounds falling on this transect were used to evaluate disease susceptibility in the field. Average size of each mound was 1.5 m^2 (rhizome clump diameter). Inoculum for field application was prepared as above. For inoculations, all top growth of kahili ginger mounds was removed with a machete and allowed to resprout for 6 weeks. On May 24, 1996, up to 30 young fleshy stems <50 cm tall on each mound were inoculated via stem injection as described above. Care was taken to inoculate around the perimeter and through the middle of the mound in order to facilitate maximum disease spread. Monthly records of number of infection loci and disease spread were taken for 1 year following inoculation. Disease spread was evaluated as the average distance of infection spread per month from any one

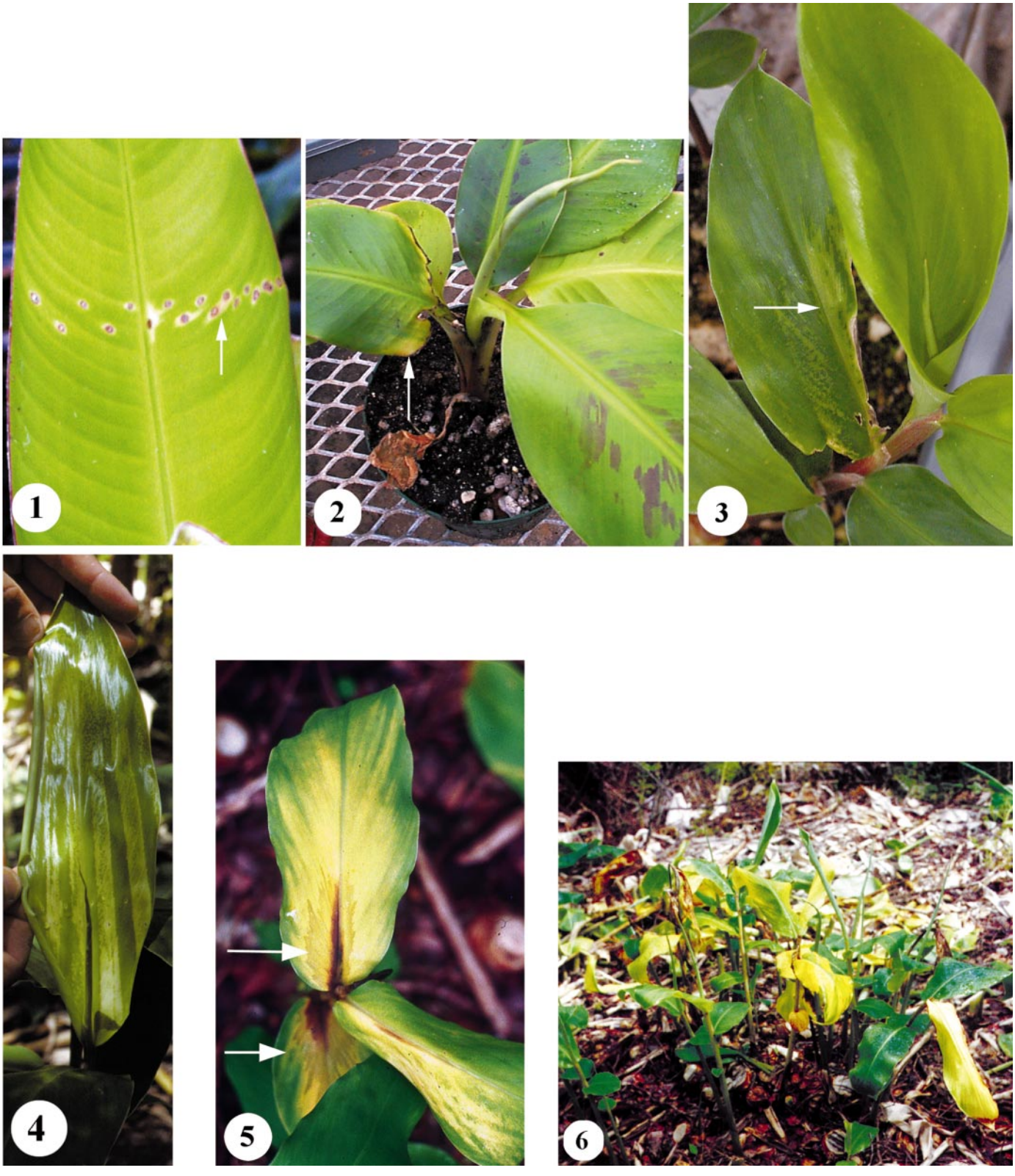
TABLE 1

Results of Greenhouse Host Range Testing for *R. solanacearum* Infection

					H. gardnerianum control[b]	
Family	Species	Common name	No. inoc.[a]	No. wilted	No. inoc.	No. wilted
Costacaceae	*Costus speciosus* (Koenig in Retz.) Sm.	Spiral ginger	8	0	5	5
Fabaceae	*Acacia koa* Gray	Koa	24	0	5	5
	Phaesolus lunatus L.	Lima bean	24	0	5	5
Heliconiaceae	*Heliconia latispatha* Benth.	Heliconia	12	0	5	5
Marantaceae	*Ctenanthe burlemarxii* H. Kenn.	Prayer plant	24	0	5	5
Musaceae	*Musa sapientum* L. var. Gros Michel	Banana	6	0	5	5
Myrtaceae	*Metrosideros polymorpha* Gaud.	Ohia-lehua	12	0	5	5
Solanaceae	*Capsicum annuum* L.	Sweet pepper	24	0	5	5
	Lycopersicon esculentum Mill. var. Healani	Tomato	24	0	5	5
	L. pimpinellifolium (Jusl.) Mill.	Currant tomato	24	0	5	5
	Nicotiana tabacum L.	Tobacco	24	0	5	5
	Physalis peruviana L.	Poha	24	0	5	5
	Solanum melongena L.	Eggplant	24	0	5	5
	S. pseudocapsicum L.	Jerusalem cherry	24	0	5	5
	S. tuberosum L.	Potato	12	0	5	5
Zingiberaceae	*Alpinia purpurata* (Viell) K. Schum	Red ginger	60	0	5	5
	A. zerumbet [=*A. nutens* Roscoe]	Shell ginger	34	0	5	5
	Hedychium coronarium J. König	White ginger	20	0	5	5
	H. flavescens N. Carey ex Roscoe	Yellow ginger	20	0	5	5
	Zingiber zerumbet (L.) Sm.	Shampoo ginger	12	0	5	5

[a] An equal number of plants was treated with sterile distilled water as controls. None of the pathogen-free control plants developed wilt symptoms or died.

[b] There was a total of 100 *H. gardnerianum* control inoculations, 5 for each set of host species inoculated.



FIGS. 1–3. Slight infections (arrows) of *H. latispatha, M. sapientum,* and *Z. zerumbet,* respectively, from greenhouse inoculation with *R. solanacearum*. Note the presence of new leaves on *M. sapientum* and *Z. zerumbet.*

FIG. 4. Interveinal chlorosis of *H. gardnerianum* in the field 2–3 weeks following inoculation with *R. solanacearum*.

FIG. 5. Advanced chlorosis of *H. gardnerianum* infected with *R. solanacearum*. Note the water-soaked appearance (arrows) and necrosis near the midrib.

FIG. 6. Typical wilt, necrosis, and flagging of infected pseudostems of *H. gardnerianum* 4–6 weeks after inoculation with *R. solanacearum*.

FIG. 7. Water-soaked appearance of outer cortex of *H. gardnerianum* rhizomes infected with *R. solanacearum*.

FIG. 8. Bacterial ooze of *R. solanacearum* exuding from an infected rhizome of *H. gardnerianum*.

FIG. 9. *H. gardnerianum* rhizome infection from *R. solanacearum* resulting in pockets of decay occurring initially around the central vascular cylinder and later throughout the rhizome.

FIG. 10. The outer cortex of *H. gardnerianum* decayed rhizomes is all that remains following infection by *R. solanacearum* and subsequent decay of infected tissues.

FIG. 11. Spread of *R. solanacearum* infection (dark rhizomes) of inoculated *H. gardnerianum*. Note the absence of new shoots.

initial infection locus. Five control mounds were treated similarly, but with sterile water.

RESULTS

Host Range Testing

The host range testing experiments indicated that of the three *Hedychium* species tested, the strain of *R. solanacearum* tested was specific in its ability to cause wilt of kahili ginger (Table 1). Slight infections and tissue discoloration around and near (within 1–2 cm) the inoculation site were observed on *H. coronarium*, *Heliconia latispatha* Benth., *Musa sapientum* L., and *Zingiber zerumbut* (L.) Sm. (Figs. 1–3). When tissue near the inoculation site of these species was plated onto TZC agar, colonies of *R. solanacearum* emerged from the tissue following incubation at 32°C for 24 h. However, these infections did not spread to cause systemic infection, and normal growth (i.e., production of new shoots and flowers) resumed following the appearance of initial symptoms. None of the inoculated species of families outside the Zingiberales developed any symptoms during the 2-month observation period. Plating of crown tissue of inoculated nonsymptomatic species yielded no bacteria resembling *R. solanacearum*. Inoculation of the positive controls of kahili ginger resulted in wilt and systemic infection in each of these tests. Initial symptoms of interveinal chlorosis developed 5–10 days after inoculation, followed by complete wilt and necrosis of pseudostems at the end of 4 weeks. Infection of rhizomes was evident by the water-soaked appearance of the outer cortex 5–6 weeks after inoculation. Rhizome tissues were in advanced decay at the end of 8–10 weeks.

Field Testing

The field inoculation experiments confirmed that *R. solanacearum* is able to infect kahili ginger in upper elevation forests and survive cooler winter temperatures than those at elevations closer to sea level at which this pathogen is a problem in commercial plantings. All inoculated pseudostems developed interveinal chlorosis and necrosis, followed by typical wilt symptoms 3–5 weeks following inoculation (Figs. 4 and 5). Initial chlorosis in the youngest leaves was followed by complete wilt occurring within 3–4 weeks after the onset of the initial symptoms (Fig. 6). As the infection spread acropetally, pseudostems became detached as the bacteria moved into the rhizome. Infected rhizomes first developed a water-soaked appearance on the periphery (Fig. 7) and often exuded a creamy bacterial ooze when cut (Fig. 8). Rhizome decay began initially around the central vascular cylinder (Fig. 9) and eventually encompassed the entire rhizome (Fig. 10). The infections spread throughout the rhizome mound at a rate of 25–75 cm^3/month, causing many secondary infection loci. Complete death of smaller mounds occurred within 4–6 months following observation of initial symptoms. Larger mounds were still alive at the end of the study, but had many infection loci, causing disruption of normal growth (Fig. 11). Underground infections extending to a depth of 15 cm were also observed on larger mounds. In addition to rhizome disintegration, infection of new pseudostems, epinasty, and seedling wilt were observed. Apparently healthy seedlings of the native species ohia-lehua, kawau (*Ilex anomala* Hook and Arnott), and pilo (*Coprosma ochracea* W. Oliver) were observed growing in and near bacterial-decayed rhizome mounds.

DISCUSSION

Because of its destructive potential to crops, the control of *R. solanacearum* has attracted much research attention (Hayward and Hartman, 1994). However, studies involving other species of bacteria as biological control agents have demonstrated the potential usefulness of these organisms in controlling invasive weeds (Johnson *et al.*, 1996; Zidack and Backman, 1996). Results of the present study have shown that the ginger strain of *R. solanacearum* can be considered a useful biological control agent for kahili ginger. Although we did not attempt direct isolation of *R. solanacearum* from the soil surrounding inoculated rhizome mounds, infection of adjacent uninoculated mounds suggested the ability of this bacterium to move through the soil. As described by Kelman and Sequeira (1965), this may occur as a result of large numbers of bacteria being released into the soil following the decay of diseased roots and rhizomes. In addition to its ease of infection, the bacterium spreads substantially within inoculated mounds. Although infection progresses slowly, the bacteria eventually cause severe wilt and death of infected plants.

Bacterial wilt of edible ginger causes significant losses each year to Hawaii's commercial ginger industry. Because of this apparent conflict of interest, the use of *R. solanacearum* as a biological control agent has been questioned. The specificity of the Hawaiian ginger strains of *R. solanacearum* among commercial species of the Zingiberales has been well established (Aragaki and Quinon, 1965; Quinon *et al.*, 1964; Ishii and Aragaki, 1963). These investigators concluded that both the ornamental and the edible ginger strains are not virulent to red ginger (*Alpinia purpurata* (Veill) K. Schum), shell ginger (*A. zerumbet* Roscoe), and torch ginger (*Phaeomeria magnifica* (Roscoe) K. Schum). Although we did not test torch ginger, our experiments confirmed that red and shell gingers were not susceptible to the strain that we tested. Our experiments also indicated that the edible ginger strain of *R. sola-*

nacearum is only weakly pathogenic to yellow, white, and shampoo (*Zingiber zerumbet* L.) gingers and is unlikely to cause wilt on these hosts. This is advantageous because these species are valued for ornamental, cultural, and ethnobotanical uses. Regarding the virulence of *R. solanacearum* on plants other than the Zingiberales, previous researchers have described the edible ginger strain of this pathogen as lacking in virulence. Ishii and Aragaki (1963) reported that inoculation with this ginger strain on tomato (*Lycopersicon esculetum* Mill.), eggplant (*Solanum melongena* L.), and sweet pepper (*Capsicum annuum* L.) caused limited infection (2–5 cm) near the inoculation site, but failed to cause wilt after 4 weeks. Quinon *et al.* (1964) also reported the lack of virulence of the edible ginger strain to solanaceous hosts. The ginger strain was weakly pathogenic or avirulent to most of the hosts tested. In later studies by Lum (1973), the Malayan edible ginger strain of *R. solanacearum* did not wilt tomato, tobacco (*Nicotiana tabacum* L.), or peanut (*Arachis hypogaea* L.). Our results confirmed these earlier reports of the lack of virulence of the edible ginger strain to species outside of the Zingiberales, as no infection or wilt was observed on any of the species of Fabaceae or Solanaceae tested. In contrast, studies conducted in the Philippines and Australia reported wilt and death of solanaceous species inoculated with the ginger strain of *R. solanacearum* (Zehr, 1969; Pegg and Moffet, 1971). However, the virulence of the ginger strain reported in these studies was variable, and with few exceptions most isolates were weakly pathogenic to solanaceous hosts.

Aside from the host specificity of *R. solanacearum* shown by our host range studies, the remote location of kahili ginger infestations is favorable to consideration of *R. solanacearum* as a biological control agent. Infestations of kahili ginger often occur in upper-elevation forested areas well removed from cultivated lands. Because there is little evidence documenting long-distance field to field spread (Kelman *et al.*, 1994), contamination of agricultural lands from application of *R. solanacearum* in Hawaiian conservation areas is considered unlikely. Although dispersal of bacterial plant pathogens may be enhanced by irrigation water in agricultural settings, we do not consider this a significant safety concern in our study site. The highly porous young volcanic soils do not support water runoff; therefore, this type of dispersal is unlikely. However, the proposed use of *R. solanacearum* as a biological control agent in other management situations should be evaluated for the possibility of contamination of nearby agricultural lands through runoff water originating from treatment areas. The use of this bacterium represents an alternative management strategy that can be applied over large areas without the undesirable effects of excessive herbicide. While considering the above constraints, the current investigation may be expanded for application of this strain of *R. solanacearum* to control kahili ginger in other tropical areas where infestations are a problem (e.g., Azores, Jamaica, Mauritius, and La Réunion) and in particular where the Zingiberaceae are not components of the native flora (e.g., New Zealand and Hawaii).

The method of inoculation used in this study is not practical for application of the control agent over large areas. Observations of disease progression suggest that a uniform inoculation of the entire rhizome mound could produce higher levels of disease and therefore increased control of treated mounds. The use of surfactants (see Zidack and Backman, 1996) in applying *R. solanacearum* to cut rhizomes is suggested as a practical method of application. In preliminary experiments using the organosilicone surfactant Silwet L-77 (polyalkyleneoxide-modified polydimethylsiloxane; OSi Specialties, Tarrytown, NY) mixed with bacterial inoculum (prepared as above), we were able to infect kahili ginger mounds in the field. Nine months after inoculation, some mounds had no regrowth, while others produced predominantly deformed shoots that did not mature. *R. solanacearum* was reisolated from stem and rhizome tissue, thus demonstrating Koch's postulates for the spray application (Anderson and Gardner, unpubl. data). We are continuing with this investigation to determine the optimum methods of field application and will publish later. Specifically, these investigations are focused on the concentration of surfactant needed, the timing of treatment (e.g., summer vs winter inoculation), and the number of treatments needed for acceptable levels of control.

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