N. A. Peres

University of Florida, Gulf Coast Research and Education Center, Wimauma

L. W. Timmer

University of Florida, Citrus Research and Education Center, Lake Alfred

J. E. Adaskaveg

University of California, Riverside

J. C. Correll

University of Arkansas, Fayetteville

Lifestyles of *Colletotrichum acutatum*

Colletotrichum acutatum J.H. Simmonds is a common pathogen of a wide array of crops and noncultivated plant species (2,3,8,11,22,29,56,66,83,90). The fungus is cosmopolitan in its distribution and causes extensive crop losses every year. Disease symptoms range from fruit rots to shoot, leaf, and flower blights. Common hosts include many dicotyledonous plants such as strawberry, apple, citrus, and stone fruits, but serious diseases on leatherleaf fern and pines also have been reported (22,62). There are no known reports of *C. acutatum* affecting grasses. Although *C. acutatum* has been well documented from agricultural hosts, recent reports of recovery of *C. acutatum* from a moribund sea turtle (59) and a native forest (55) suggest that this fungus may inhabit other less-studied niches.

C. acutatum was first described as a separate species by Simmonds (74,75). Many of the isolates now known to be *C. acutatum* were either included in *C. gloeosporioides* (Penz.) Penz. & Sacc. in Penz, or were considered to be species of *Gloeosporium* (von Arx). *C. acutatum* affects many hosts that are also attacked by other *Colletotrichum* species, which may have been the reason for failure to recognize it as a separate taxon. For example, strawberry is affected by *C. acutatum*, *C. gloeosporioides*, and *C. fragariae* Brooks (29,33,37,41,56,76,77). *C. gloeosporioides* and *C. fragariae* usually cause petiole and stolon lesions and crown rot on strawberry

but may also produce fruit symptoms (76). *C. acutatum* produces symptoms on flowers, leaves, petioles, and roots, but is most important as a fruit rot of strawberry. Citrus, apples, mangos, papayas, and avocados are also affected by both *C. acutatum* and *C. gloeosporioides* (3,30,83). On citrus, *C. gloeosporioides* is a common saprophyte and may cause postharvest anthracnose from preharvest quiescent infections. *C. acutatum* does not produce postharvest anthracnose on citrus fruit, but causes distinct diseases on different species of citrus, such as postbloom fruit drop and key lime anthracnose (83,84). On apple, bitter rot is caused by *C. gloeosporioides* (*Glomerella cingulata* (Stoneham.) Schrenk & Spaulding), as well as *C. acutatum* (38,73,81). A leaf spot disease caused by *Glomerella cingulata* has been reported in Brazil and the United States (38,46), but *C. acutatum* is not known to attack apple leaves.

Much of the research in recent years has been dedicated to elucidating the etiology of diseases caused by *Colletotrichum* spp. and the population dynamics of these pathogens. Research has emphasized differentiation of the species of *Colletotrichum* on each host and determining their role in producing the observed symptoms. Less effort has been dedicated to determining the life cycle of *C. acutatum* in each situation, its means of survival and dissemination, and its host specificity. In this article, we will assess the status of our knowledge on the lifestyles of *C. acutatum*, including infection and colonization strategies on different hosts, the role of various asexual structures such as conidia and appressoria, and the role of the sexual stage in selected disease cycles. To define lifestyles more effectively, we needed to clarify the taxonomy of the fungus and to differentiate those diseases caused by *C. acutatum* from those caused by other spe-

cies of *Colletotrichum*. We also explore our understanding of the ability of *C. acutatum* to move between hosts and cause disease, and the genetic relatedness of isolates of *C. acutatum* from different hosts. Information in some areas is limited, and we identify gaps in our understanding of diseases caused by *C. acutatum* and potential areas for future research.

Taxonomic Relationships

Morphology of *C. acutatum*. *C. acutatum* is an Ascomycete classified in the *Pyrenomycetes* and included in the family *Phyllachoraceae* (80). Colonies of *C. acutatum* are usually white initially and later become covered with pink to orange conidial masses (92) (Fig. 1A). Cultures from some hosts are highly chromogenic and excrete red and pink pigments into the culture media (10). The primary production of conidia is in acervuli; however, *C. acutatum* also is capable of forming secondary conidia on the surface of living leaves (49). Conidia are usually ellipsoid and fusiform (Fig. 1B), at least at one end, rather than typically having both ends rounded like *C. gloeosporioides* (1). Setae are usually sparse if produced at all. Pigmented appressoria are produced upon germination of the conidia and vary in shape and size (6).

The sexual stage of certain genetic subgroups of *C. acutatum* has been characterized under laboratory conditions and has been designated as *Glomerella acutata* (39) (Fig. 2). Guerber et al. (40) found that a large number of isolates from diverse hosts were capable of mating and forming mature perithecia in culture (Fig. 2). Crosses within isolates in the group C, and some intergroup crosses, were highly fertile, but others were weakly fertile or infertile. However, mating compatibility in *C. acutatum* and other *Colletotrichum* species is complex (40,88).

Corresponding author: N. A. Peres, Assistant Professor, University of Florida, Gulf Coast Research and Education Center, Wimauma, FL 33598; E-mail: naperes@ifas.ufl.edu

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Differentiation of *C. acutatum* from other species of *Colletotrichum*. Morphological characteristics such as colony morphology, conidial shape, the presence or absence of setae and sclerotia, and appressorium shape and size have been used for differentiation of species in the genus *Colletotrichum* (80) (Fig. 1A and B). Morphological traits, however, are highly variable among isolates and often subject to interpretation. Other characters, such as growth rates and sensitivity to benomyl, have been helpful to differentiate *C. acutatum* and *C. gloeosporioides*. *C. acutatum* is neither highly sensitive nor highly tolerant to benomyl (2,11,30,68). Levels of 0.1 to 1.0 µl/ml cause 20 to 50% growth inhibition of *C. acutatum*, but are usually completely inhibitory to other species of *Colletotrichum*. However, *C. acutatum* is more sensitive to benomyl than resistant isolates of other species of *Colletotrichum* (68).

A great deal of effort has been dedicated to determining which species of *Colletotrichum* are involved in the many anthracnose-type diseases of plants, and to the development of tools to identify and differentiate them (13–15,26,27,29,44,72,78,79). As pointed out by Freeman (27), multiple hosts can be affected by a single species, and multiple species can affect a single host. Simmonds (74,75) first described *C. acutatum* as a new species, distinct from *C. gloeosporioides*, based on isolates from fruit rots in Australia. Additional detailed morphological descriptions were published later (10,23,48). Most of the fungi included in *C. acutatum* were formerly assigned to *C. gloeosporioides* with the teleomorph being *Glomerella cingulata* (10,80,89). Many species formerly considered to be *Gloeosporium* (9) are now also included in *C. acutatum*. In recent years, different tools, such as vegetative compatibility groups and isozymes, as well as molecular tools, such as random amplification of polymorphic DNA (RAPD), species-specific primers, and sequencing of the internal transcribed spacer (ITS) region of nuclear ribosomal DNA and other regions of the genome have been useful in separating *C. acutatum* from other species of *Colletotrichum* (2,12,14,29,30,33,41,44,77).

While the role and the identity of species involved in anthracnose diseases is not always clear, we now have a much better understanding of the different species and the means available to differentiate them. Almond anthracnose, for example, was thought to be caused by *C. gloeosporioides*, based on the morphology of the pathogen (33). More recently, almond isolates from California (2) and Israel (31) have been identified as *C. acutatum* primarily by use of molecular tools. Such tools have been very useful in determining, not only the identities of isolates, but also the relationships among them (1,78,79). The citrus postbloom fruit drop pathogen

was also originally identified as *C. gloeosporioides* (83), but was later found to be *C. acutatum* (14).

Intraspecific relationships. *C. acutatum sensu lato* may be considered a group species, and the relationships among populations from different host plants are not clear (28,40,48). Two subspecific forms or physiological taxa of *C. acutatum* have been described, *C. acutatum* f. sp. *pineum* (22) and *C. acutatum* f. sp. *chromogenum* (10). *C. acutatum* f. sp. *pineum* was not separated from *C. acutatum* morphologically and was designated primarily on the basis of its pathogenicity to pines. *C. acutatum* f. sp. *chromogenum* was distinguished on the basis of pink and red pigments in the agar medium in culture (10). Sutton (80) did not feel either subspecific designation was valid. However, populations of *C. acutatum* have been characterized and studied. Lardner et al. (48) found that *C. acutatum* f. sp. *pineum* from New Zealand and Australia formed two separate groups based on RAPD analysis and analysis of vegetative compatibility. According to Guerber et al. (40), isolates from pine in New Zealand were

clearly polyphyletic using restriction fragment length polymorphism (RFLP) of mtDNA and the 900-bp intron of the glutamine synthase gene, as well as DNA sequences of the latter gene and of the 200-bp intron of the glyceraldehyde-3-phosphate dehydrogenase gene. Adaskaveg and Förster (1) designated and mapped the distribution of two subgroups (e.g., pink and gray) of *C. acutatum* based on RAPD patterns and cultural morphology on almond in California over several years.

Although several studies have been conducted in recent years to characterize isolates, currently there are no formally accepted subspecific nomenclatural designations within *C. acutatum*. Often *C. acutatum sensu* Simmonds (74,75), from fruit crops is distinguished from the other groups of isolates of *C. acutatum* from diverse crop species (44,48). Lardner et al. (48) separated several groups of isolates based on morphology and found that the separation was supported by analysis of RAPDs and vegetative compatibility. Three groups (A, B, and C) included isolates from diseased fruit; two groups (D and E) were from lupine; another group

Fig. 1. A, Variation in colony color and morphology among cultures of *Colletotrichum acutatum sensu* Simmonds. Chromogenic isolates are not illustrated. Rings are due to alternating light and dark cycles; B, typical conidia.

Fig. 2. *Glomerella acutata*, the teleomorph of *Colletotrichum acutatum*: A, mating system on toothpicks in culture; B and C, perithecia formed on toothpicks in culture; D, close-up of perithecia; E and F, asci with ascospores.

included pine isolates designated *C. acutatum* f. sp. *pineum*; and a last group contained isolates of *Glomerella miyabeana* (Fukuski) Arx from willow. Guerber et al. (40) supported the recognition of *G. miyabeana* as a species distinct from *C. acutatum*, but additional work with other isolates failed to support the pine group as a unique population (40). In a study with anthracnose isolates from lupine, Nirenberg et al. (61) described a new species, *C. lupini* (Bondar) Nirenberg, Feiler & Hagedorn. Separation was based on morphological, physiological, and molecular criteria. Guerber et al. (40) found that lupine isolates were distinguished in subgroups J2 and J6 from other isolates of

C. acutatum in clades C, D, E, and J4, including one isolate from France that was placed as *C. lupini* by Nirenberg et al. (61). Talhahas et al. (82) also considered all lupine isolates as *C. acutatum* that formed a distinct subgroup separate from all isolates from other hosts. Thus, despite segregation of some isolates into new species such as *C. lupini* and others (e.g., *G. miyabeana*), *C. acutatum* remains as a genetically distinct collection of closely related isolates.

Groupings of isolates by host or geographic region do not necessarily correspond to those based on molecular criteria. Freeman et al. (32) characterized isolates of *C. acutatum* from Israel using arbitrarily

primed PCR and AT-rich DNA analyses and found four subgroups. Subgroups were confirmed using sequences of the ITS 2 region alone or the complete ITS region. Freeman's subgroup I contained isolates from almond, apple, peach, and pecan from the United States, but Israeli isolates from almond were in subgroup IV. Guerber et al. (40) found that isolates from many different hosts and many different countries fell into the same genetic subgroups. The Guerber's subgroup C included isolates from almond, apple, avocado, blueberry, cherimoya, citrus, cranberry, dodder, grape, strawberry, and kiwi, among others, from the United States and New Zealand. Nevertheless, isolates from key lime and other citrus, as well as isolates from leatherleaf fern from Florida in the United States, fell into Guerber's subgroup J along with all of the lupine isolates from the U.K., France, and Canada. Denoyes-Rothan et al. (19) compared isolates from strawberries and other hosts using RAPDs and sequence analyses of the ITS 2 region and found that isolates from strawberries, mostly from Europe and the United States, were nearly identical, but that isolates from other hosts were variable.

Thus, the general conclusion from most of the research is that *C. acutatum* is composed of genetic subgroups that show little indication of host specificity, although there are some indications of specialization of some isolates on certain hosts. As noted above, isolates from key lime formed a single group. Studies by Peres et al. (67) using sequences of the entire ITS region and the 200-bp intron of the glyceraldehyde-3-phosphate dehydrogenase gene, as well as RAPDs, found that isolates from postbloom fruit drop-affected citrus flowers from Brazil, Costa Rica, Belize, Mexico, the Dominican Republic, and Florida were closely related and did not group based on geographic origin. Isolates from key lime from the same countries were also very closely related but differed slightly from the postbloom fruit drop isolates. Thus, isolates from postbloom fruit drop on sweet orange and those from key lime anthracnose form two separate populations regardless of their country of origin.

The situation on citrus presents an interesting opportunity to study the evolution of pathogenicity and the movement of pathogens between hosts and countries. Postbloom fruit drop is of rather recent origin. The disease and its causal agent were first described in Belize in 1979 (25). However, there are informal reports and letters from the late 1950s and the 1960s describing the symptoms on citrus in Belize and Colombia (25; W. Reuther, *personal communication*). Postbloom fruit symptoms are obvious and the effects on yield are dramatic; thus, it would be unlikely that the disease would have gone unnoticed for extended

Table 1. Plant tissues affected by *Colletotrichum acutatum* on selected hosts

	Apple/ peach	Blueberry	Sweet orange	Strawberry	Key lime	Almond
Young leaves/twigs	— ^a	(+)	—	+	+	+
Flowers	—	(+)	+	+	+	+
Fruit preharvest	—	+	—	+	+	+
Fruit postharvest	+	+	—	(+)	—	(+)
Roots/crowns	—	—	—	+	—	—

^a — = No symptoms; + = symptoms; (+) = symptoms occur but not common.

Fig. 3. Anthracnose symptoms caused by *Colletotrichum acutatum* on foliage, fruit, and branches of almond. A, Mummified almond fruit (left) with pink to orange sporulating lesions and a mature fruit (right) with recent gumming infections; B, foliage and fruit (arrow) symptoms on a detached branch; C, affected immature fruit showing external (top fruit) and internal (bottom fruit) symptoms; D, dieback of almond branches caused by anthracnose. Bars = 1 cm.

periods in commercial citrus groves. Reports of key lime anthracnose go back at least to the early 1900s (83). It was proposed originally that postbloom fruit drop may have originated when isolates from key lime anthracnose moved into other citrus species (6). The molecular data of Peres et al. (67) are not inconsistent with that hypothesis, but if the populations of *C. acutatum* on key lime and other citrus species have common ancestors, they probably diverged some time ago. Indications are that postbloom fruit drop isolates were distributed to all of the citrus-growing areas of the Americas within 30 to 40 years, probably by movement of plant material bearing appressoria.

Disease Symptoms and Tissues Affected

Symptoms caused by *C. acutatum* are primarily necroses including blights on various types of host tissues such as leaves, petioles, flowers, fruit, or even roots on a wide range of hosts. However, the pathogen is not a general necrotroph attacking

only young tissue or senescent tissues. Rather, this pathogen is highly specialized as to the tissue it attacks on each host. In some cases, one juvenile tissue may be attacked, whereas other tissues are completely immune to infection. For example, flowers of sweet orange are severely affected, but there are no symptoms produced on young leaves. With apples, the fruit is affected, but no symptoms occur on leaves. The fungus is probably best considered as a hemibiotroph in the broad sense, in that it has biotrophic and necrotrophic phases depending on the host and the tissue infected on any specific host.

C. acutatum infects susceptible cultivars of almond as a general necrotroph of young tissues, including leaves, twigs, flowers, and fruit (1,2,26) (Table 1, Fig. 3). Thus, when rain occurs during warm weather, newly developing shoots and flowers may be severely blighted. A similar situation occurs on key lime where all shoots and leaves may be killed by the pathogen (6,83). In both cases, destructive and disfiguring lesions are produced on fruit.

Interestingly, all of the young tissues of key lime are susceptible and severely attacked by *C. acutatum* (Fig. 4A, Table 1). However, on sweet orange and most other citrus crops, including Tahiti lime, only flower tissues are infected and develop postbloom fruit drop (Fig. 4B and C); vegetative tissues are immune (Table 1). Isolates of *C. acutatum* from key lime produce all of the symptoms of postbloom fruit drop on sweet orange, but isolates from postbloom fruit drop-affected sweet orange produce little anthracnose damage on key lime (6). Postbloom fruit drop of citrus appears to be unique among diseases caused by *C. acutatum* in that symptoms are induced by plant hormones (47,53). Natural citrus fruit abscission usually occurs at the base of the peduncle at the attachment to the stem, but with postbloom fruit drop, abscission is at the base of the fruit leaving persistent calyces and peduncles. Leaves around the infected inflorescences usually become small and disfigured. Genes encoding ethylene and jasmonic acid biosyntheses and indole-3-acetic acid regulation were found to be highly induced by *C. acutatum* infection (53). Indole acetic acid increased as much as 140-fold in infected petals compared with healthy controls (47).

On some hosts, such as apple and blueberry, *C. acutatum* is a preharvest problem in fruit production and also is a postharvest pathogen (45,60,73). On these plants, quiescent infections that occur prior to harvest begin to develop as the fruit ripens. Those infections then develop into ripe rot of blueberry (Fig. 5A) and bitter rot of apple (Fig. 5B) after harvest. Likewise, postharvest symptoms may occur with anthracnose diseases of peaches, almonds, and other stone fruits (11,91). There are many other examples of postharvest decays on tropical fruits such as avocado, mango, papaya, and guava that develop from qui-

Fig. 4. Symptoms caused by *Colletotrichum acutatum* on citrus: A, foliar and preharvest fruit lesions on key lime; B, infected flowers of sweet orange; C, persistent calyces remaining following fruit drop.

Fig. 5. Fruit diseases caused by *Colletotrichum acutatum*: A, ripe rot of blueberry (courtesy A. M. C. Schilder, Michigan State University); B, bitter rot of apple (courtesy J. I. Boneti, Epagri, Santa Catarina, Brazil).

escent infections occurring in the field (3,8,34,66).

C. acutatum produces a wide range of symptoms on various tissues of the strawberry plant (Table 1). The pathogen is not a general necrotroph on all tissues of strawberry, but it is most important as a necrotroph causing serious fruit rot (76). Lesions on petioles are produced from quiescent infections after the plants are established (Fig. 6A). Later, flowers may be attacked, and ripe and unripe fruit develop symptoms normally referred to as black spot (Fig. 6B and C). Strawberry is unique in that roots and crowns can be infected by *C. acutatum* (Fig. 6D, Table 1) causing severe stunting or even plant death (Fig. 6E). The source of inoculum for root infection is uncertain. Conidia or possibly appressoria on the upper parts of the plant may be washed down to the crown and roots during digging, washing, and transplanting, and mechanical damage and stress caused by these processes may facilitate root infection.

Host and Tissue Specificity

Many inoculation studies have been conducted with *Colletotrichum* spp., demonstrating that most isolates are relatively non-host-specific (7,27,30,34). However, relatively few studies have dealt with host specificity of *C. acutatum*. Bernstein et al. (11) inoculated nonwounded, detached ripe peach fruit with isolates of *Colletotrichum* spp. from a number of different hosts. They found that lesion size was not related to the source of the isolate. Freeman and Shabi (34) inoculated detached nectarine, mango, avocado, almond, and apple fruit with conidial suspensions of isolates of *C. acutatum* from apple and peach. All isolates produced lesions on fruit, but it was

necessary to wound the mango fruit to obtain infection. Peres et al. (66) found that a *C. acutatum* isolate from strawberry produced lesions on wounded, detached fruit of avocado, guava, papaya, mango, and passion fruit, but not banana. Isolates from strawberry also produced lesions on wounded and nonwounded fruit of pear, peach, nectarine, and apple (28). Thus, it would appear that *C. acutatum* has a broad host range, at least among fruit crops, and is relatively nonspecific. Many of the above studies, however, were done in the laboratory using detached and wounded fruit. Movement of *C. acutatum* from one fruit crop to another under natural conditions has not been demonstrated, and molecular groups often seem to be host specific (43).

Isolates from certain hosts do show at least some specificity. Pathogenicity of *C. acutatum* isolates from lupine, pine, tomato, and persimmon fruit was evaluated on pine and lupine seedlings (48). All isolates from pine were nonpathogenic or weakly pathogenic to lupine and vice versa. The fruit isolates were not pathogenic on either pine or lupine.

In Israel (35), inoculation of isolates of *C. acutatum* from anemone and strawberry into each host resulted in typical anthracnose symptoms on both plant species. Molecular studies indicated that the isolates from the two hosts were similar and formed a single genetic subgroup (35). Isolates of *C. acutatum* were recovered from pepper, eggplant, tomato, bean, as well as from weeds of the genera *Vicia* and *Conyza*; however, disease symptoms occurred only on strawberry (28). *C. acutatum* isolates from tulip tree in Argentina were also pathogenic to strawberry fruit (54). Thus, strawberry isolates of *C. acu-*

tatum can parasitize and cause disease on other hosts or, alternatively, survive on other crops and on weeds without producing symptoms.

Dissemination and Survival

Dispersal. Conidia of *C. acutatum* are usually produced in acervuli on host tissue and are typically rain-splash dispersed (4,57,58,63). On low-growing crops such as strawberry, conidia are spread over short distances (57,58), and use of straw as a ground cover can greatly reduce dispersal of conidia (57). On tree crops, dispersal by wind-blown rain can occur over much greater distances. With repeated cycles of infection on sweet orange flowers, post-bloom fruit drop of citrus can spread over large areas in a single season (4). On citrus, environmental factors, amounts of inoculum, and other factors affecting the epidemiology of postbloom fruit drop have been used to develop models to predict disease and forecast the need for fungicide applications (65,86).

There is no evidence that ascospores function in the dispersal of any disease caused by *C. acutatum*. Guerber and Correll (39) found nine vegetative compatibility groups (VCGs) in a single apple orchard. However, Gonzalez and Sutton (38) found only two VCGs in three North Carolina orchards and concluded that once isolates were established in an orchard, they persisted over time. Thus, additional studies are needed on the biology and population genetics of *C. acutatum* to elucidate the importance of the sexual stage in the overall life cycle.

Survival on agricultural crops. The role of epiphytic stages and quiescent infections on survival of the fungus is not clear with many diseases caused by

Fig. 6. Symptoms caused by *Colletotrichum acutatum* on strawberry: A, petiole lesions with acervuli (arrow); B, fruit lesions on unripe fruit (black spot); C, lesions on ripe fruit; D, root rot; E, plants killed or stunted by the pathogen.

C. acutatum. With postbloom fruit drop on citrus, appressoria on mature tissues are essential for the survival of the pathogen (5,6,83,92). Since flowers are the only susceptible tissue and they occur only seasonally, the fungus survives as appressoria and quiescent infections on mature leaves (6,92). When flowers appear, leachates from flowers induce germination of appressoria and the production of conidia that infect new flowers. With strawberries, *C. acutatum* is usually introduced into production fields from nurseries. Some plants may be symptomatic, but many times the pathogen is introduced on asymptomatic leaves or petioles, presumably as appressoria and quiescent infections. Whether quiescent infections develop into lesions or whether appressoria germinate to produce conidia is not clear. However, conidia of *C. acutatum* can produce several secondary conidia from each conidium deposited on the leaf surface (49,50,51) (Fig. 7). The numbers of conidia produced suggest that the fungus is reproducing by utilizing nutrients on the leaf surface rather than reserves from the original conidium. In these cases, it would appear that the fungus is growing as an epiphyte and not necessarily producing quiescent infections. On blueberries, recent studies suggest that flower buds become infected and produce quiescent infections during the summer and that the pathogen overwinters in those structures (17,18). On almonds, it appears that mummified fruit that remain on the tree throughout tree dormancy are the main source of conidia for infections in spring (1).

Survival on alternative hosts. The role of alternative hosts in the cycle of diseases caused by *C. acutatum* also is not clear in most instances. Since *C. acutatum* from anemone also can infect strawberry, and vice versa (35), spread could occur from one crop to another. Freeman et al. (28) also found that *C. acutatum* was capable of colonizing weeds such as *Vicia* spp. and *Conyza* spp. surrounding strawberry fields and could survive 3 months on tomato, pepper, and eggplant leaves (28). Thus, the pathogen could be spreading from strawberry to weeds adjacent to strawberry fields and then reinfesting new strawberry fields the following year. Molecular and vegetative compatibility studies indicated that isolates from weeds, anemone, and strawberry form a single population in Israel (32). Further studies demonstrated that *C. acutatum* failed to penetrate initially on tissue of pepper, eggplant, and tomato but intercellular penetration occurred later, suggesting that the fungus can probably live as an endophyte on these hosts (42).

Although *C. acutatum* isolates from tropical fruits cross-infect well under laboratory conditions (7,34,66), the extent to which cross-infection occurs in nature, and the degree to which disease cycles on one

crop depend on the ability of a given group of isolates to survive on a different crop is not known. We suspect that this is not common in most situations. Despite a severe problem with anthracnose of key limes in western Mexico, postbloom fruit drop has never developed on oranges or Tahiti limes grown in the area. Both strawberries and citrus are grown in Central Florida, and there is no indication of cross-infection. Instead, molecular studies indicate that strawberry and citrus isolates differ considerably (14). Similarly, in California, genetically distinct populations were identified from adjacent almond and strawberry fields (26).

Survival in the environment. *C. acutatum* appears to compete poorly as a saprophyte (5), but can survive in soil and on inanimate surfaces for various periods depending on the conditions. Eastburn and Gubler (24) found that mycelium in strawberry tissue survived in air-dried soil for at least 7 weeks, even at temperatures up to 40°C, and it survived in moist soil for 7 weeks at 10 and 25°C. Freeman et al. (36) found that conidia of *C. acutatum* survived for at least 1 year in air-dry soil; however, in soil at field capacity, conidia survived less than 2 weeks. At 11% soil moisture, the time required for 95% loss of viability was 70 to 75 days. *C. acutatum* could not be recovered from strawberry naturally infected crowns buried in methyl-bromide fumigated or nontreated soil after 5 months. In Florida, *C. acutatum* could no longer be recovered from buried infected crowns after 56 days (87).

Conidia of *C. acutatum* from leatherleaf fern survived up to 1 year in dry soil in Florida (62). Survival was shorter in moist soil, and conidia maintained their viability for about 35 days in soils at 12 to 24% moisture. In leaf debris, the fungus was still viable after 56 days when soil moisture was 6% or less, but not at higher moisture levels. *C. acutatum* remained viable for at least 105 days when infected plants were buried in field plots, and conidia remained viable on fabric for 35 to 45 days.

Thus, conidia of *C. acutatum* can survive for extended periods in debris or soil under dry conditions. However, the fungus is unable to compete or multiply on leaf

Fig. 7. Scanning electron micrograph of secondary conidiation of *Colletotrichum acutatum* on an almond leaf. c = conidium; sc = secondary conidium.

debris or in soil under moist conditions where other organisms are active.

Host-Pathogen Interactions

Infection process and colonization.

Although *C. acutatum* usually produces necrotic symptoms of various types on most hosts, the host-pathogen interaction appears to be more biotrophic than that of some other species such as *C. gloeosporioides* (28). *C. acutatum* survives and competes poorly as a saprophyte in most cases (24,62). Nevertheless, *C. acutatum* has been isolated from many plant species and has not always been associated with disease. This species may colonize the surface of plant tissues as an epiphyte or endophyte under some circumstances, on both hosts and nonhosts, without producing symptoms (35,42,45).

O'Connell et al. (64) and Perfect et al. (69) described two types of interactions between *Colletotrichum* spp. and their hosts: intercellular hemibiotrophy and subcuticular intramural necrotrophy. With *C. acutatum*, there are few studies on the cytology of the infection process and the nature of early biotrophic relationships or quiescent infections established between pathogen and host. However, based on studies with *C. acutatum* on specific hosts, four types of interactions or infection strategies can be described (16,21,90,92).

Conidia germinate and form appressoria on the tissue surface and infection pegs that penetrate the cuticle (Fig. 8). Internal light spots at the contact point between the appressorium and the tissue surfaces have been assumed to be the points of emergence of the infection peg (16,92) (Fig. 9). However, on strawberry leaves, Leandro et al. (50) questioned whether appressorium formation was followed by quiescent infections, and whether the internal light spots represented infection pegs or were just contact points between the appressorium and the host surface. In a detailed study, Diéguez-Urbeondo et al. (20) determined that internal light spots in appressorium formation on almond leaves corresponded to penetration pores and were followed by production of an infection peg and colonization of host cells.

Fig. 8. Appressoria of *Colletotrichum acutatum* formed following germination of conidia. c = original conidium, 2-celled after germination; a = appressorium, arrow indicates the internal light spot.

The sequence and nature of the infection process, including the initial penetration phase that may be a biotrophic or quiescent relationship, and subsequent development of infection hyphae and colonization of the host varies and is not well understood in most cases. With citrus postbloom fruit drop, the infection strategy on leaves is presumably biotrophic. After the end of the bloom period, conidia germinate to

Fig. 9. Appressoria of *Colletotrichum acutatum* on the surface of almond leaves showing the internal light spot (arrow) where the penetration peg emerges from the appressorium. c = conidium; a = appressorium.

form appressoria, and quiescent infections are established that are probably intracellular (6,92) (Fig. 10A). The tissues where quiescent infections develop are never colonized and necrotrophy does not occur on leaves, but rather conidia and secondary conidia are produced following germination of the appressorium on the surface. Still, on flower petals, the pathogen is a necrotroph. With all tissues of key lime and strawberries, the biotrophic phase is very short. The pathogen produces appressoria, penetrates the host, and forms a subcuticular intramural necrotrophic association almost immediately (16) (Fig. 10B). Curry et al. (16) considered this strategy a modification of necrotrophy rather than bio- or hemibiotrophy because the biotrophic phase is so short.

With blueberry and many fruit crops (e.g., peach, apple, and many tropical fruits), other interactions or infection strategies may be occurring. Interactions can be intracellular hemibiotrophy when infecting ripe fruit of susceptible cultivars, or subcuticular intramural necrotrophy when infecting fruit of resistant cultivars

(Fig. 10C) (90). The hemibiotrophic interaction begins with appressorium formation on the fruit surface and continues with the establishment of quiescent infections with a relatively long biotrophic phase. When fruit ripen, necrotrophic inter- and intracellular colonization occurs, tissues are extensively invaded, and the fungus sporulates.

On almond leaves and presumably fruit, a combination of hemibiotrophic and subcuticular intramural necrotrophic infections occurs that results in intra- and intercellular colonization (21) (Fig. 10D). In this case, the colonization is biotrophic after fungal penetration and becomes necrotrophic during intercellular growth. This is followed by inter- and intracellular colonization and death of the tissue. The mechanism for the shift from biotrophy to necrotrophy is not certain. However, *C. acutatum*, as well as other species of *Colletotrichum*, produce ammonia causing local pH increases during the shift to necrotrophy (70). In most host-pathogen interactions, the specific interaction at the cellular level is unknown and may be a combination of strategies as shown with

Fig. 10. Schematic drawings of different host-pathogen interactions and infection strategies of *Colletotrichum acutatum*. A, Biotrophic growth of *C. acutatum* with secondary conidiation. Conidia germinate to form appressoria and quiescent infections. Secondary conidia are formed after germination of the appressorium (e.g., predominantly biotrophic disease cycle on citrus leaves) (92). B, Subcuticular, intramural necrotrophy with hyphal development within periclinal and anticlinal walls of epidermal host cells. Note that host cell walls are swollen and wider apart (e.g., predominantly necrotrophic disease cycle on strawberry) (16). C, Hemibiotrophic interaction with infection vesicle and broad primary hyphae within host cell. Inter- and intracellular secondary hyphal growth is shown illustrating the subsequent necrotrophic phase (e.g., combination of bio- and necrotrophy but mostly biotrophic disease cycle on blueberry fruit) (90). D, Hemibiotrophic and subcuticular, intra- and intercellular development of *C. acutatum* on almond (e.g., combination of bio- and necrotrophy but mostly necrotrophic disease cycle on almond leaves and fruit) (21). Cn = conidium; Gt = germ tube; Ap = appressorium; Iv = infection vesicle; Ph = primary hyphae; Sh = secondary hyphae; Sc = secondary conidium. Dead host cells are indicated with diagonal lines. Illustrations by J. E. Adaskaveg.

almond (21). Furthermore, these strategies may be dependent on host tissues. For example, in some cases on almond (21,90) and in most cases on citrus (92), petal tissues of flowers are infected and colonized without the formation of appressoria, and there is no biotrophic phase. In general, these infection strategies lead to the different lifestyles proposed below.

Disease Cycles

General disease cycles. The disease cycles of *C. acutatum* from different hosts often have attributes in common. All produce acervuli with abundant conidia on one or more host tissues, and conidia are splash-dispersed and serve as the source of inoculum for most infections. Conidia may survive in a gelatinous matrix for some time if conditions are appropriate. However, more typically, conidia germinate to produce appressoria and quiescent infections. The tissue on which sporulation and reproduction occur differs among various hosts, and the fate of appressoria and quiescent infections also varies greatly. As far as is known, the sexual stage has only a minor role in the life cycle of *C. acutatum*. Although the pathogen exhibits overlapping survival strategies, we can group different disease cycles of *C. acutatum* on different hosts according to four general lifestyles as follows:

Leaf biotrophic–flower necrotrophic lifestyle (citrus). The lifestyle of *C.*

acutatum on sweet orange is unique (83). The fungus functions as a necrotroph on flowers and as a biotroph on leaves (Table 2, Fig. 11). Thus, the fungus reproduces primarily on flower petals and does not readily colonize and form acervuli on other tissues (6,92). Acervuli only form on other tissues when the tissue is killed under artificial conditions, and the fungus does not reproduce in the absence of flowers (85,92). Survival of the fungus during

nonflowering periods occurs by means of appressoria that form on the surface of leaves, twigs, and the persistent calyces formed as a result of flower infection. The quiescent infections do not develop and result in colonization of the leaf or other vegetative tissues, and normally the pathogen cannot be isolated from leaves. Appressoria can be stimulated to germinate and produce a few conidia artificially by application of petal extracts. It is presumed

Table 2. Stages of life cycles of *Colletotrichum acutatum* on different hosts: structures and tissues involved and duration of the various stages

Stage	Host			
	Blueberry	Sweet orange	Strawberry	Almond
<i>Biotrophy</i>				
Tissue	Immature fruit	Mature leaves	Leave, petioles	Leaves and fruit tissues
Structures	Appressoria	Appressoria, sec. conidia ^a	Appressoria, sec. conidia	Appressoria, sec. conidia
Duration	Weeks	Months	Days	Hours
<i>Necrotrophy</i>				
Tissue ^b	Ripe fruit	Flowers	All tissues	All tissues
<i>Overwintering</i>				
Tissue	Bud scales, dead twigs	Mature leaves	Leaves, petioles	Fruit mummies, dead twigs
Structures	Appressoria, conidia, mycelium	Appressoria	Appressoria	Conidia, mycelium

^a Secondary conidia.

^b Tissues on which acervuli and conidia are produced.

Fig. 11. Disease cycle of postbloom fruit drop on citrus caused by *Colletotrichum acutatum*. Long biotrophic phase on mature leaves and persistent calyces. Necrotrophic phase occurs only on flowers. a = appressorium; c = conidium; sc = secondary conidiation. Illustrations by Lynda E. Chandler.

that in nature, petals falling on moist leaves result in stimulation of germination of appressoria on the surface and that the few secondary conidia produced are splash dispersed to the new flowers. So *C. acutatum* behaves as a necrotroph on citrus during the flowering period and produces severe epidemics at that stage. However, for much of the year, the fungus lives biotrophically in quiescent infections on vegetative structures.

Necrotrophic lifestyle (strawberry). *C. acutatum* is primarily a necrotroph on strawberry tissues (16) (Table 2, Fig. 12). It affects flower, fruit, leaf, and petiole tissue, and also produces root necrosis, which is highly unusual for this fungus. Roots of transplants are presumably contaminated during digging, trimming, and packing operations in the nursery. Thus, roots might not normally be infected when the crop is grown as a perennial. The management of strawberry as a crop affects the cycle of the pathogen. The plant is a perennial, but as a cultivated plant, it is treated as an annual. Transplants are often produced far from commercial fields; thus, the fungus must survive as symptomatic or quiescent infections on transplants that will serve as inoculum for the commercial fields (52). The biotrophic phase is usually very short (16), but infected leaves, petioles, and runners may not show symptoms for some time. Secondary conidiation can occur on the surface of vegetative tissues,

and this can serve to augment inoculum levels to infect flowers and fruit (51). These few conidia formed on the foliage without causing symptoms are dispersed by splashing water and harvesting operations from the foliage to flowers and fruit. Primary inoculum for dispersal within the field is mostly dependent on the formation of spores in acervuli on petioles and fruit tissues.

Fruit hemibiotrophic lifestyle (blueberry-apple-stone fruit). Fruit decays represent one common lifestyle of *C. acutatum* (Table 2, Fig. 13) for diseases such as blueberry ripe rot, apple bitter rot, anthracnose of peach, and probably numerous anthracnose diseases of subtropical and tropical fruits. In the case of bitter rot of apple and ripe rot of blueberry, acervuli are produced in abundance on mature fruit (60,81). Conidia from acervuli are splash dispersed, germinate to form appressoria, and quiescent infections are established on fruit as has been demonstrated on peaches (91). These diseases occur in the field as well as after harvest. However, most of the quiescent infections develop during the course of the season on these crops. In the case of blueberry, *C. acutatum* is also known to attack young leaves and other tissues, although this is rare (60,90). Thus, conidia from acervuli on leaves and decaying fruit could serve as a source of inoculum for other fruit, and the pathogen survives as appressoria in bud

scales and dead fruiting twigs (17). With apple, the source of inoculum is not obvious since *C. acutatum* only causes disease on fruit (45,81). *C. acutatum* is apparently widespread in orchards and is readily isolated from healthy and damaged tissue. In Michigan, where bitter rot rarely occurs, *C. acutatum* is still widespread in apple orchards and commonly isolated (45). Thus, it would appear to be surviving and reproducing as an epiphyte or endophyte in those situations, similar to the epiphytic-short necrotroph lifestyle.

Biotroph-necrotroph combination lifestyle (almond-key lime). There are many diseases where *C. acutatum* functions primarily as a necrotroph but can also function as a biotroph, and thus, the pathogen has a combination of biotrophic and necrotrophic interactions. This lifestyle includes anthracnose of almond, key lime, and leatherleaf fern. In these cases, acervuli and conidia are produced in abundance on the affected tissues (Table 2). Conidia are splash-dispersed to healthy tissues that become infected in a short time (Fig. 14). The fungus can survive as dried conidia in acervuli or mycelia in dead tissues including spurs and in mummified almond fruit (1). Once new growth develops, conidia from acervuli can initiate a new epidemic. Conidia may also be dispersed to mature tissues that are not susceptible to infection. Quiescent infections could develop and result in colonization of the leaf tissue

Fig. 12. Disease cycle of anthracnose fruit rot of strawberries caused by *Colletotrichum acutatum*. Biotrophic phase primarily on runner transplants. Long necrotrophic phase on flowers, fruits, and petioles. Illustrations by Lynda E. Chandler.

Fig. 13. Disease cycle of blueberry ripe rot caused by *Colletotrichum acutatum*. Long biotrophic phase on immature berries in the spring and summer and on bud scales in the winter. Necrotrophic phase primarily on ripe fruit and to a lesser extent on shoots and other tissues. Illustrations by Lynda E. Chandler.

Fig. 14. Disease cycle of almond anthracnose caused by *Colletotrichum acutatum*. Biotrophic phase is brief; several hours after penetration, before the necrotrophic phase begins. Necrotrophic phase occurs on all tissues: flowers, leaves and fruits. Illustrations by Lynda E. Chandler.

once the leaves senesce and eventually produce acervuli.

Alternative lifestyles? Almost all reports of the natural recovery of *C. acutatum* have been from agricultural hosts. However, recent reports indicate that *C. acutatum* may inhabit a number of unexplored niches. For example, *C. acutatum* has been recovered from an immune-compromised sea turtle (59). Also, a single isolate of *C. acutatum* was recovered apparently colonizing the leaves of a tree species in a native forest (55). Whether such isolates originated from agricultural ecosystems or could represent a source of such pathogens remains largely unexplored.

Concluding Remarks

Considerable progress has been made in recent years in delineating anthracnose diseases and their causal agents. Molecular tools have been effectively utilized to distinguish *Colletotrichum* species, clearly separate *C. acutatum* from *C. gloeosporioides*, and identify genetically distinct subgroups with *C. acutatum*. However, the disease cycles of *C. acutatum* in many host-pathogen systems still remain poorly understood, even on economically important crops. This pathogen has the ability to reproduce on many different types of tissues on various hosts and can exist as a necrotroph, biotroph, endophyte, or epiphyte depending on the plant species and tissue involved. The tissues affected also are very specific for each host combination, and the pathogen develops a highly specialized relationship at the cellular level that is reflected in the disease cycle in each host case. The biotrophic and epiphytic phases of the pathogen and the means of reproduction of *C. acutatum* in the absence of pathogenesis are in need of further investigation. Tissue specificity and life cycles of *C. acutatum* perhaps are clearly under genetic control, and it may be possible to induce mutants that are no longer pathogenic to certain hosts or tissues and determine the genetic basis for tissue or host specificity as has been done with other species of *Colletotrichum* (71).

Few investigations of cross pathogenicity and the host range of *C. acutatum* isolates in nature have been conducted. If this pathogen truly has a broad host range and can move readily between hosts, it is interesting that it has developed unique relationships with each plant species. Many investigators have limited crop responsibilities, do not have facilities to handle temperate or tropical crops, or are limited in the isolates they can study due to biosecurity regulations. Thus, these factors have limited studies of host ranges with large numbers of isolates. Perhaps, with ingenuity and cooperative efforts, at least some of these studies can be conducted, and eventually we should be able to develop a better understanding of movement and host

specificity of *C. acutatum* and determine where restrictions on movement of plant materials are warranted.

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Natalia A. Peres

L. W. "Pete" Timmer

J. E. Adaskaveg

J. C. Correll

Dr. Peres is assistant professor of plant pathology at the University of Florida, Gulf Coast Research and Education Center in Wimauma. She received her B.S. in agronomy in 1995, an M.S. in horticulture in 1998, and a Ph.D. in plant pathology in 2002 at São Paulo State University in Botucatu, São Paulo, Brazil. Her dissertation work involved the biology and management of postbloom fruit drop of citrus caused by *Colletotrichum acutatum*. After she received her Ph.D., she worked as program manager for a project on foliar fungal diseases of citrus at the Instituto Biológico in São Paulo until 2003. She joined the faculty at the University of Florida in November 2003. Her research program focuses on etiology, epidemiology, and control of anthracnose and other diseases of strawberry and management of ornamental diseases.

Dr. Timmer is professor of plant pathology at the University of Florida. He received his B.S. in botany and plant pathology from Michigan State University and his Ph.D. at the University of California at Riverside. He spent two years doing research on citrus diseases in Argentina during his doctoral program. From 1970 to 1978, he did research and extension on citrus diseases at the then Texas A&I University Citrus Center (now the Texas A&M Kingsville Citrus Center) in Weslaco, TX. Subsequently, he accepted a position with the University of Florida, Citrus Research and Education Center in Lake Alfred, where he is currently employed. His research program focuses on fungal diseases of fruit and foliage of citrus trees including etiology, epidemiology, and disease control. For many years, his assignment was 100% research, but it was changed to 70% extension three years ago. Dr. Timmer has been active in APS and has served terms as associate and senior editor of *Phytopathology* and associate editor and, now, senior editor of *Plant Disease*. He was senior editor of the *Compendium of Citrus Diseases* and the *Citrus Health Management* book. Pete received the Lee Hutchins Award for excellence in research on tropical fruit crops and is a Fellow of APS.

Dr. Adaskaveg is associate professor of plant pathology at the University of California, Riverside. He received a B.S. degree in agronomy in 1982 at the University of Connecticut, Storrs, and M.S. and Ph.D. degrees in plant pathology at the University of Arizona, Tucson, in 1984 and 1986, respectively. He was a postdoctoral research associate and a research plant pathologist at the University of California, Davis, until 1995. Dr. Adaskaveg currently leads a diverse research program on the biology, ecology, epidemiology, and management of pre- and postharvest fungal diseases of tree fruit and nut crops in California. Part of his research focuses on anthracnose diseases of citrus, almond, and peach. Recent studies have provided new information on the ultra-structure of host-pathogen relationships, pathogen biology, epidemiology of anthracnose, and molecular techniques for the detection and identification of *Colletotrichum* species and other fungi.

Dr. Correll is professor of plant pathology at the University of Arkansas in Fayetteville. He received his B.S. from Pennsylvania State University and his M.S. and Ph.D. degrees at the University of California at Berkeley. From 1986 to 1989, he was a postdoctoral fellow at Kansas State University and University of California, Berkeley, and he joined the faculty at the University of Arkansas in 1989. His current research program focuses on basic research on the population genetics of fungi such as *Pyricularia*, *Colletotrichum*, and *Fusarium*. He also has a large program dealing with management of diseases of vegetables and rice using cultivar resistance, cultural practices, and chemical and nonchemical methods. He has many international research projects, especially in Asia and Africa, and travels extensively. Dr. Correll has been active in APS and has served terms as associate and senior editors of *Plant Disease*, associate editor of *Phytopathology*, and currently is senior editor of *Phytopathology*. He has been an invited speaker at many APS symposia and has also been active in the Mycological Society of America.