Effects of Temperature on Isolates of *Fusarium moniliforme* Causing Fig Endosepsis and *Aspergillus niger* Causing Smut

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ABSTRACT

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Isolates of *Fusarium moniliforme* causing endosepsis, two obtained from cultivated fig and three from wild fig pollinator trees, and an isolate of *Aspergillus niger* causing smut on figs were evaluated for fruit colonization and lesion expansion at five constant temperatures. Surface-disinfested fruit halves from fig pollinator trees were inoculated individually with isolates of *F. moniliforme* and *A. niger* on the uncut surfaces and incubated at 15, 20, 25, 30, and 35°C under high humidity (>97%). Lesion sizes of smut and endosepsis were recorded after 3 and 5 days of incubation, respectively. To determine rates of lesion expansion, lesion sizes of endosepsis and smut were recorded at 48- and 24-h intervals after inoculation, respectively. Optimal temperature for *F. moniliforme* colonization was 30°C. Isolates from cultivated caprifigs failed to colonize at 35°C. Higher temperatures resulted in shorter latent periods for both *F. moniliforme* (40 to 60 h at 30°C) and *A. niger* (44 h at 35°C). *Fusarium moniliforme* isolates from the wild caprifigs had a significantly shorter latent period at 30°C (40 h) than isolates from cultivated caprifigs (60 h). Lesion sizes caused by isolates at different temperatures differed significantly ($P = 0.0001$). At each temperature, isolates from wild caprifigs caused significantly larger lesions and sporulated more than isolates from cultivated caprifigs. Optimal temperature for *A. niger* colonization was 35°C. At temperatures of <25°C, lesions areas caused by *F. moniliforme* isolates were significantly larger; at 30 and 35°C, lesion areas caused by *A. niger* were significantly larger. At 30 and 35°C, the rates of lesion expansion for *A. niger* were twice as great as rates for any isolate of *F. moniliforme*. Because temperatures of <30°C are not conducive to *A. niger* development, smut is rare on caprifigs and is common on Calimyrna figs.

Additional keyword: *Ficus carica*.

All figs (*Ficus carica* L.) are characterized by an inflorescence called a "syconium" that produces a multiple fruit. Horticulturally, fig syconia can be classified as persistent (self fertile) or caducous. Caducous figs consist of botanically distinct male (caprifig) and female (edible Calimyrna) figs, which have different types of flowers in the syconia. Caprifigs provide pollen for the pollination of the female crop and the hibernating site for the wasp pollinator *Blastophaga psenes* L., which mature in ovaries of modified pistillate flowers (3). Caprifigs produce three crops of syconia: a winter crop, a spring crop, and a summer crop. The spring crop syconia have the maximum number of staminate flowers and, consequently, the maximum amount of pollen; as a result, the syconia of the spring crop are used to pollinate the edible Calimyrna figs.

Endosepsis, named for the internal rot of figs (3,5,10), and "smut," misnamed because of a sooty black spore mass that occurs in the fig cavity (5), are two important diseases causing significant losses each year (1,5). Smut is caused by *Aspergillus niger* Tiegh. (5). Endosepsis is caused by at least three *Fusarium* species (8): *F. moniliforme* Sheld. (8,12,14) is the dominant cause, *F. solani* (Mart.) Sacc. is a significant cause, and *F. dimerum* Penz. in Sacc. is a minor cause (14). Recent evaluation of factors affecting the growth and sporulation of *F. moniliforme* and *A. niger* revealed the former to be pathogenic over a wide temperature and osmotic potential range, and the latter to be pathogenic under low temperature and high osmotic potential conditions (15). As a consequence, endosepsis is common on both inedible caprifigs and edible figs, whereas smut is common only on edible figs (15). Because both diseases develop in the fig cavity, moisture is not considered limiting for development of either disease. Temperature appears to be a major determinant of the extent of colonization of these two diseases. Evaluation of *F. moniliforme* isolates from cultivated and wild caprifigs showed that collections from the latter were generally more virulent (the degree of pathogenicity as measured by lesion area) than were collections from the former (14). Though all isolates had an optimal temperature of 25°C for mycelial growth and sporulation (15), information on the optimal temperature(s) for infection is unavailable. Determination of optimal temperature(s) for infection by isolates from cultivated and wild caprifigs would provide valuable information on the ecology of diseases caused by isolates from the two sources.

Specific temperature requirements for the development of endosepsis or smut are little understood. The objectives of this study were to determine the optimal temperature for colonization of figs by *F. moniliforme* and *A. niger*, determine the incubation and latent periods at different temperatures for endosepsis and smut, determine the relationship between lesion size and lesion expansion of endosepsis and smut at different temperatures, determine the sporulation by isolates of *F. moniliforme* at different temperatures, and determine the possible differential response of incubation and latent periods, lesion areas, rates of lesion expansion, and sporulation by *F. moniliforme* isolates from cultivated and wild caprifigs to temperature. Preliminary results have been reported previously (13).

MATERIALS AND METHODS

Isolates and inoculum production. Five single-spore isolates of *F. moniliforme* were chosen for the study. Two originated from cultivated (F23 and F32), and three from wild (F50, F59, and F62), caprifigs (14). After evaluating virulence of 10 *A. niger* isolates obtained from Calimyrna figs, and failing to detect statistically significant variation among them, we chose only one isolate (AN1) for detailed analysis. Inoculum of each of the five *F. moniliforme* isolates was produced on 25 dishes containing acidified (2.5 ml of 25% [vol/vol] lactic acid solution per liter of medium) potato-dextrose agar (APDA) by centrally seeding a 4-mm-diameter culture plug of each isolate. Because of the hydrophobicity of *A. niger* spores, suspensions were prepared in 0.1% water agar amended with four drops of Tween 80 per liter. Five microliters of spore suspension was dispensed centrally on 25 APDA dishes. All dishes of *F. moniliforme* isolates were incubated at 25 ± 1°C for 15 days and those of *A. niger* at 30 ± 1°C for 5 days. Cultures of both *F. moniliforme* and *A. niger* were then flooded with 10 ml of sterile deionized water and brushed gently with a rubber spatula to dislodge conidia. One drop of Tween 80 was added to the *A. niger* spore suspension. Density of spores was determined with a hemacytometer.

Treatments. Fruits collected from the spring crop of caprifigs near maturity were brought to the laboratory, surface disinfested with a 1% sodium hypochlorite solution for 3 min, and allowed to air dry on laboratory benches. The fruits were then sliced through the ostiole with a sterile knife, and 35 halves were placed over waxed wire screens (20 × 29.5 cm) with the cut surfaces facing down in clear plastic containers (23.5 × 32 × 10 cm). Seven halves were inoculated with each *F. moniliforme* isolate by placing 5 µl of a 10^6 conidia per ml suspension on a wound, 2 mm wide × 2 mm deep, made on the surface of each fruit half with a sterile nail. Twenty fruit halves were similarly inoculated with the *A. niger* spore suspension (10^6 conidia per ml). Two hundred milliliters of tap water was added in each container to maintain the relative humidity (>97%). Inoculated fruits were incubated in the dark at 15, 20, 25, 30, and 35°C. The incubation period (interval between inoculation and appearance of symptoms in at least 50% of the inoculated fruits) and latent period (interval between inoculation and appearance of sporulation in at least 50% of the inoculated fruits) were recorded for *A. niger* and each isolate of *F. moniliforme*. Two perpendicular diameters of each smut and endosepsis lesion were measured after 3 or 5 days incubation, respectively. The experiments were replicated eight and 10 times

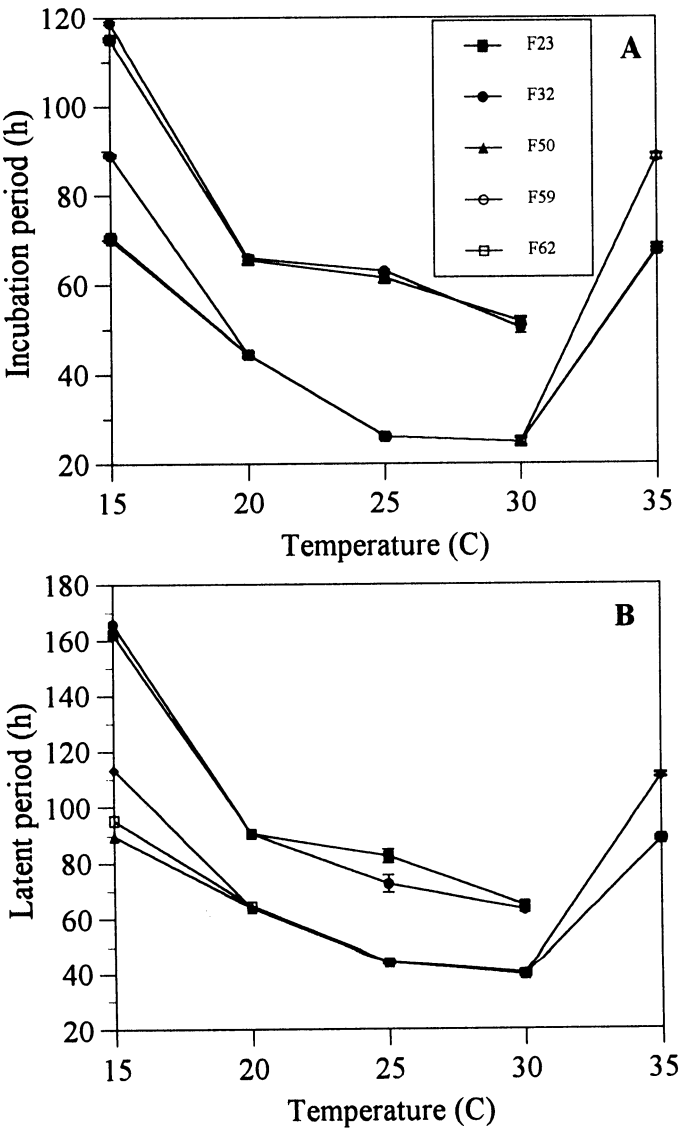

Fig. 1. Effects of temperature on incubation and latent periods (in hours) of fig endosepsis caused by five isolates of *Fusarium moniliforme* inoculated into artificial wounds on surfaces of near-mature caprifigs. **A,** incubation period (time interval between inoculation and appearance of symptoms on 50% of inoculated fruits). **B,** latent period (interval between inoculation and sporulation on 50% of inoculated fruits). Vertical bars are the standard errors of the mean.

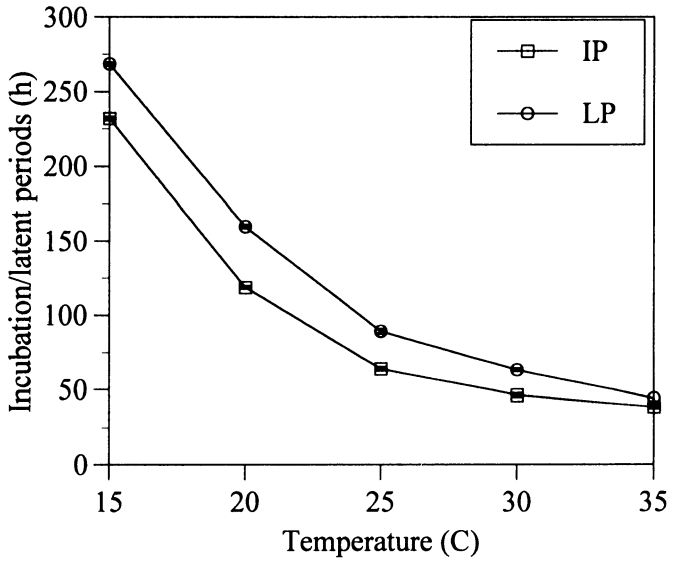

Fig. 2. Effects of temperature on incubation and latent periods (in hours) of fig smut caused by *Aspergillus niger* inoculated into artificial wounds on surfaces of near-mature caprifigs. Incubation period is the interval between inoculation and appearance of symptoms on at least 50% of inoculated fruits. Latent period is the interval between inoculation and sporulation on 50% of inoculated fruits. Vertical bars are the standard errors of the mean.

TABLE 1. Summary analysis of variance for lesion area caused by isolates of *Fusarium moniliforme* and one isolate of *Aspergillus niger* at different temperatures

Source	df[a]	Sum of squares	Mean square	$P > F^b$
Model	117	7,010.966	59.923	0.0001
Experiment	9	18.989	2.110	0.4073
Temperature	4	1,324.903	331.226	0.0001
Error a	36	70.962	1.971	. . .
Isolate	5	1,314.083	262.817	0.0001
Error b	43	51.077	1.188	. . .
Temperature × Isolate	20	3,053.460	152.673	0.0001
Error c	172	178.510	1.038	. . .

[a] Degrees of freedom. Data from all 10 experiments with *F. moniliforme* isolates were used in this analysis.

[b] Probabilities associated with individual *F* tests.

for smut and endosepsis, respectively. The assignment of an incubator to a particular temperature was random.

In a different set of experiments designed to study the rates of lesion expansion for smut and endosepsis caused by different isolates at different temperatures, 20 and seven fruit halves, respectively, were inoculated as described above with *A. niger* and with each isolate of *F. moniliforme*. The inoculated fruits were incubated at 15, 20, 25, 30, and 35°C. The lesions were measured

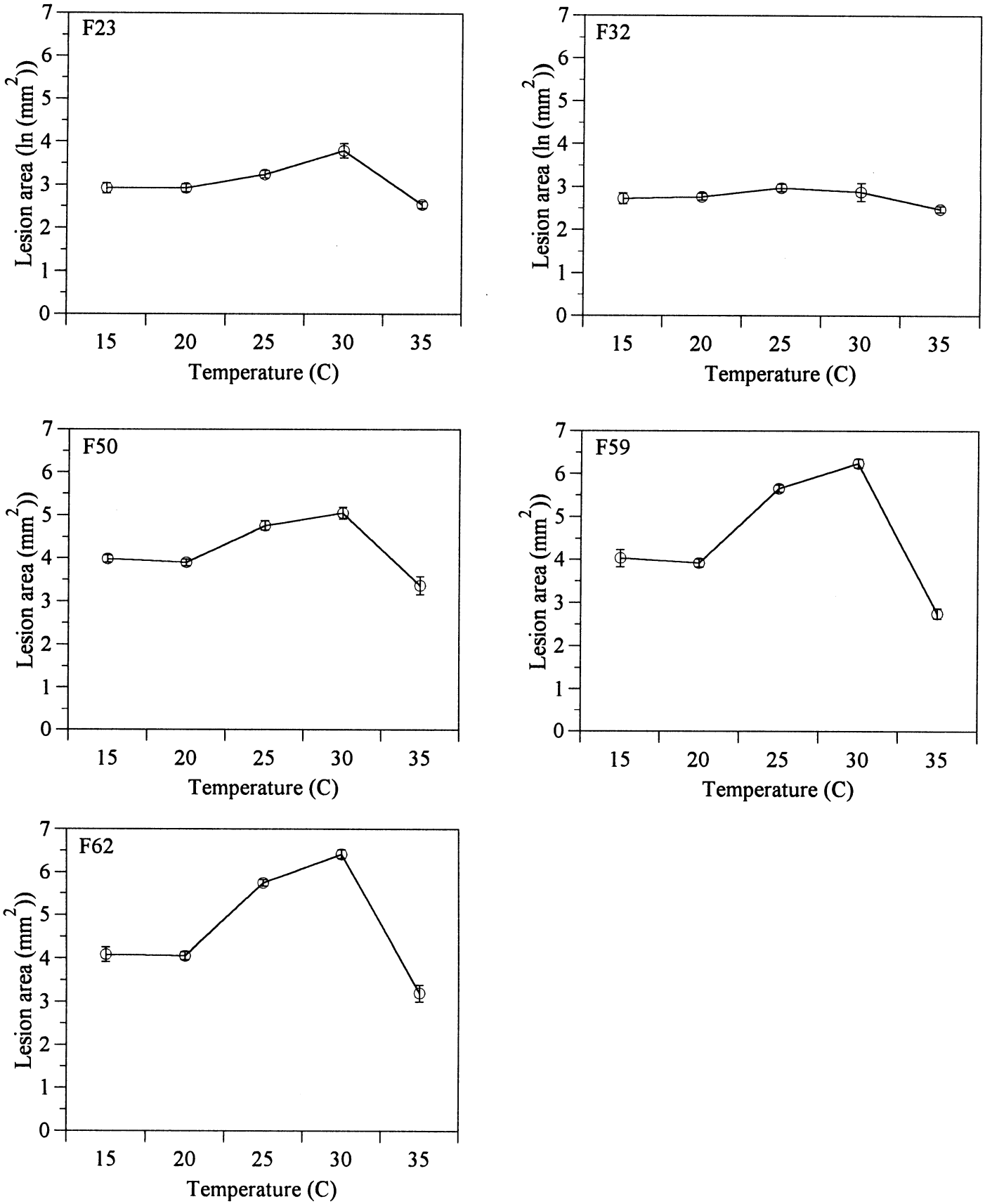

Fig. 3. Effects of temperature on lesion areas of fig endosepsis caused by isolates of *Fusarium moniliforme* inoculated into artificial wounds on surfaces of near-mature caprifigs and incubated for 6 days. Vertical bars represent the standard errors of the mean. Each point represents the mean of data from seven fig halves each in 10 experiments.

24, 48, and 72 h after inoculation for smut and 48, 96, and 144 h after inoculation for endosepsis. These experiments were conducted 10 times and the assignment of incubators to different temperatures was again random.

Sporulation. Sporulation was determined for *F. moniliforme* in the lesion size experiments only. After final lesion size determination (5 days) on fruits from different temperature treatments, lesions were excised from the infected fruits, placed in 100 ml of sterile deionized water in a large test tube, and stirred with a touch plate stirrer for 30 sec to dislodge the conidia. Densities of spores in different temperature treatments were determined with a hemacytometer. Five hemacytometer counts were made of each isolate-temperature combination, averaged and expressed as spores per mm^2 lesion area. Means and corresponding standard errors were computed for data from all experiments.

Data Analysis. Means and the corresponding standard errors were computed for incubation and latent periods for all isolates of *F. moniliforme* and *A. niger*. Polynomial models of the form $\ln(Y) = f(T)$ were evaluated in which Y was either the incubation or latent period for individual isolates of *F. moniliforme* and *A. niger*, and T was a linear combination of T, T^2, and T^3. Regression models for each pathogen isolate were evaluated by the significance of estimated parameters, the distribution of residuals, and coefficients of determination.

Because lesions were either elliptical or circular, lesion sizes were calculated using the formula lesion area (mm^2) = $(a/2 \times b/2) \times \pi$, in which a and b are the lesion diameters in two perpendicular directions. Lesion area and lesion expansion data were transformed as $\ln(x + 1)$ to stabilize variance. Experiments were considered as replications (block) for analysis of variance conducted to determine the effects of incubation temperature, pathogen isolates, isolate × temperature, and replication × isolate × temperature interactions. Both replications and isolates were considered as random effects in the analysis of variance. Because the numbers of fruits inoculated with isolates of *F. moniliforme* and *A. niger* differed, a weighted least squares analysis with frequencies of fruits as the weights was used. Because the temperature × isolate interaction was significant, there was strong evidence that the differences among isolates depended on temperature, and therefore groups of *F. moniliforme* isolates from cultivated and wild caprifig sources were compared using linear contrasts at each temperature (11). Similarly, the *A. niger* isolate was compared with individual *F. moniliforme* isolates at each temperature using linear contrasts. Because the numbers of experiments with isolates of *F. moniliforme* and *A. niger* differed, data from all 10 experiments were used to contrast isolates of *F. moniliforme*, and data from the common eight experiments were used to compare isolates of *F. moniliforme* with the isolate of *A. niger*.

Lesion expansion rates for both endosepsis and smut were calculated and expressed as mm^2 per 24 h. Three rates each were calculated for smut and endosepsis using the equation $R_k = (Y_{tj} - Y_{ti})/(t_j - t_i)$, in which R_k is the rate of lesion expansion at interval k, and Y_{ti} and Y_{tj} are lesion areas at times i and j, respectively. Repeated measures analysis of variance was used to evaluate the effects of experiment (block), temperature, isolate, assessment in-

TABLE 2. Linear contrasts between *Fusarium moniliforme* isolates from cultivated (C) and wild (W) caprifigs at different temperatures

Contrast	df[a]	Contrast sum of squares	$P > F$[b]
F. moniliforme C vs W at 15°C	1	122.710	0.0001
F. moniliforme C vs W at 20°C	1	113.515	0.0001
F. moniliforme C vs W at 25°C	1	460.836	0.0001
F. moniliforme C vs W at 30°C	1	539.077	0.0001
F. moniliforme C vs W at 35°C	1	29.497	0.0001

[a] Degrees of freedom. Data from all 10 experiments with *F. moniliforme* isolates were used in this analysis.
[b] Probabilities associated with individual F tests.

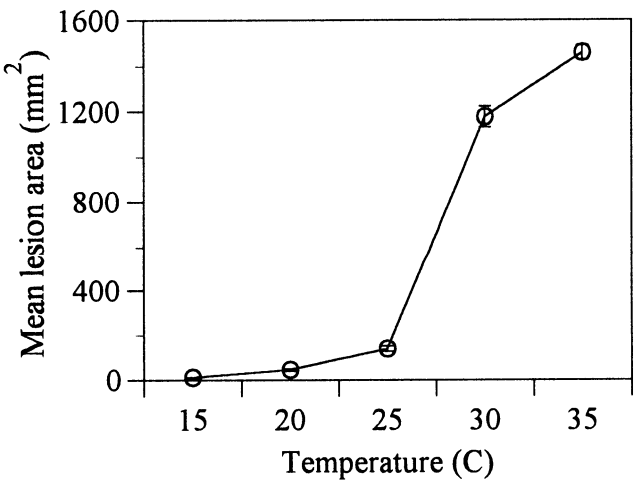

Fig. 4. Effects of temperature on lesion areas of fig smut caused by *Aspergillus niger* inoculated into artificial wounds on surfaces of near-mature caprifigs and incubated for 3 days. Vertical bars represent the standard errors of the mean. Each point represents the average of data from 20 fig halves each in eight experiments.

TABLE 3. Summary analysis of variance for lesion area caused by isolates of *Fusarium moniliforme* and one isolate of *Aspergillus niger*

Source	df[a]	Sum of squares	Mean square	$P > F$[b]
Model	99	6,498.136	65.638	0.0001
Experiment	7	15.337	2.191	0.4202
Temperature	4	1,082.874	270.719	0.0001
Error a	28	58.428	2.087	...
Isolate	5	1,126.895	225.379	0.0001
Error b	35	45.343	1.296	...
Temperature × Isolate	20	2,972.697	148.635	0.0001
Error c	140	145.575	1.040	...

[a] Degrees of freedom. Data from eight experiments common to both *F. moniliforme* and *A. niger* were used in this analysis.
[b] Probabilities associated with individual F tests.

TABLE 4. Linear contrasts between *Aspergillus niger* (AN1) and *Fusarium moniliforme* (F) isolates at different temperatures

Contrast	df[a]	Sum of squares	$P > F$[b]
AN1 vs F23 at 15°C	1	131.567	0.0001
AN1 vs F23 at 20°C	1	0.574	0.4588
AN1 vs F23 at 25°C	1	54.216	0.0001
AN1 vs F23 at 30°C	1	392.691	0.0001
AN1 vs F23 at 35°C	1	890.549	0.0001
AN1 vs F32 at 15°C	1	86.308	0.0001
AN1 vs F32 at 20°C	1	2.703	0.1092
AN1 vs F32 at 25°C	1	88.349	0.0001
AN1 vs F32 at 30°C	1	679.714	0.0001
AN1 vs F32 at 35°C	1	914.748	0.0001
AN1 vs F50 at 15°C	1	330.454	0.0001
AN1 vs F50 at 20°C	1	28.221	0.0001
AN1 vs F50 at 25°C	1	17.602	0.0001
AN1 vs F50 at 30°C	1	139.682	0.0001
AN1 vs F50 at 35°C	1	618.588	0.0001
AN1 vs F59 at 15°C	1	368.423	0.0001
AN1 vs F59 at 20°C	1	35.130	0.0001
AN1 vs F59 at 25°C	1	59.447	0.0001
AN1 vs F59 at 30°C	1	16.892	0.0001
AN1 vs F59 at 35°C	1	805.664	0.0001
AN1 vs F62 at 15°C	1	389.463	0.0001
AN1 vs F62 at 20°C	1	58.402	0.0001
AN1 vs F62 at 25°C	1	73.102	0.0001
AN1 vs F62 at 30°C	1	18.260	0.0001
AN1 vs F62 at 35°C	1	660.343	0.0001

[a] Degrees of freedom. Data from eight experiments common to both *F. moniliforme* and *A. niger* were used in this analysis.
[b] Probabilities associated with individual F tests.

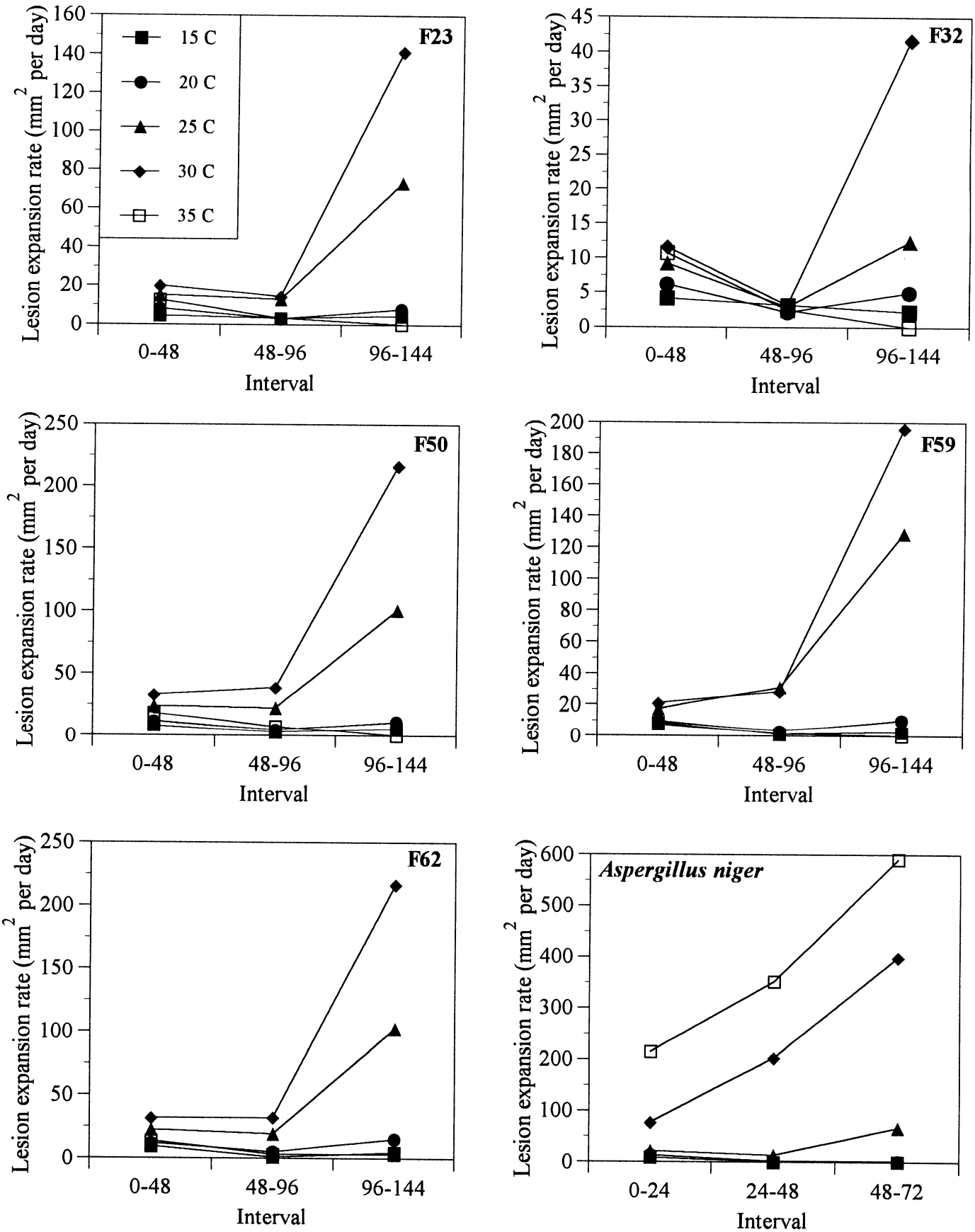

Fig. 5. Effects of temperature on rates of lesion expansion caused by different isolates of *Fusarium moniliforme* (mm^2 per day) after 48, 96, and 144 h of incubation, and by *Aspergillus niger* (mm^2 per day) after 24, 48, and 72 h of incubation at different temperatures. The points are averages of data from 10 experiments.

terval, and interactions on lesion expansion rates of *F. moniliforme* and *A. niger*. Mean lesion rates among assessment intervals and isolates were compared using the least significant difference test ($P < 0.05$) (11). All data analyses were performed using SAS (release 6.03 ed., SAS Institute, Inc., Cary, N.C.).

RESULTS

Incubation and latent periods. The incubation period for all isolates of *F. moniliforme* decreased as temperatures increased up to 30°C (Fig. 1A). Incubation periods at different temperatures were nearly identical for isolates from wild caprifigs. Similarly, differences in incubation periods of isolates from cultivated caprifigs were not significant (Fig. 1A). Incubation periods at 35°C were as long as those observed at 15°C for isolates from wild caprifigs. Only at the end of 6 days did fruit inoculated with isolates from cultivated caprifigs show measurable lesions at 35°C. The length of latent period of isolates from wild caprifigs as a function of temperature was similar to the response obtained for incubation period. The latent period was shortest at 30°C (Fig. 1B). A second-order polynomial model best described the incubation and latent period responses to temperature for isolates from cultivated caprifigs ($R^2 = 0.93 - 0.98$) while a third-order polynomial model best described the response for isolates from wild caprifigs ($R^2 = 0.98 - 0.99$).

Both incubation and latent periods for *A. niger* were longest at 15°C and progressively decreased as the temperature increased. At 35°C, both incubation and latent periods were nearly identical (Fig. 2). A second-order polynomial model best described the relationships between both incubation and latent periods with temperature ($R^2 = 0.99$)

Lesion areas at different temperatures. Endosepsis lesion areas caused by the different isolates were consistent across all experiments and block effects were not significant ($P < 0.4073$), but effects of temperature, isolate, and isolate × temperature interactions were significant (Table 1). Lesion areas caused by *F. moniliforme* increased with temperature up to 30°C for all isolates tested and then declined. At 15 and 20°C, lesion areas were similar. Incremental increases in temperature from 20 up to 30°C resulted in significant increases in lesion areas (Fig. 3). Increases in lesion area in response to temperature were consistent across all isolates of *F. moniliforme*, but the magnitude of response was significantly different among isolates tested (Fig. 3). At each temperature, lesion areas caused by isolates from cultivated caprifigs were significantly smaller than lesion areas caused by isolates from wild caprifigs (Table 2). Isolate F62 was the most virulent while isolate F32 was the least (Fig. 3).

Lesion areas caused by *A. niger*, however, resulted in a sigmoidal curve with no significant increases up to 25°C. Lesion areas were largest at 35°C (Fig. 4). The lesion areas caused by the *A. niger* isolate were significantly larger than those caused by isolates of *F. moniliforme* in the temperature range 25 to 35°C (Tables 3 and 4; Figs. 3 and 4). At temperatures <25°C, however, lesion areas caused by all isolates of *F. moniliforme*, except for isolates from cultivated caprifigs at 20°C, were significantly larger than those caused by the *A. niger* isolate (Tables 3 and 4).

Lesion expansion rates and temperature. Regardless of the isolate, the rates were optimal at 30°C for endosepsis and at 35°C for smut (Fig. 5). At each temperature, both endosepsis and smut lesions expanded during the final assessment interval at a significantly ($P < 0.05$) higher rate than during the first two intervals. While the rates of lesion expansion were comparable among the isolates from wild caprifigs, they were significantly ($P < 0.05$) higher than those for the isolates obtained from cultivated caprifigs. Lesions caused by isolate F32 had the slowest rate of expansion. The rates of lesion expansion caused by *F. moniliforme* isolates were significantly lower than those caused by *A. niger*. Consistently, *A. niger* lesions occupied nearly the entire surface of the fruit within 3 days at optimal temperatures, whereas *F. moniliforme* isolates took 6 days to colonize the same surface area.

Sporulation. Sporulation by different isolates of *F. moniliforme* followed the patterns of lesion sizes at different temperatures. Sporulation of all isolates was maximum at 30°C (Fig. 6). Sporulation by isolates from wild caprifigs was significantly higher at 30°C than that by isolates from cultivated caprifigs.

DISCUSSION

This study documents the effects of temperature on several components in the disease cycles of fig endosepsis and smut. Temperature influenced incubation and latent periods, lesion sizes, and rate of lesion expansion of both pathogens. Temperatures tested also affected sporulation by isolates of *F. moniliforme*. These two diseases differ from other foliar and fruit diseases that are usually affected by both temperature and wetness duration (2,4,6,7) in that infection by these two pathogens begins in the fig cavity where moisture is limiting only at the very last stage of fruit ripening. Temperature plays the most important role in determining the incidence and severity of these two diseases on the different fig crops. The optimal temperature for all of the disease components measured in this study was 30°C for *F. moniliforme* isolates and 35°C for *A. niger*. We had previously determined that the optimal temperature for mycelial growth of *F. moniliforme* and *A. niger* was 25 and 30°C, respectively (15). Differences in optimal temperatures for mycelial growth and plant infection have been previously documented for other diseases (7).

At the corresponding optimal temperatures, no significant differences occurred in the incubation and latent periods between isolates of *F. moniliforme* and the isolate of *A. niger*. However, lesions caused by the isolate of *A. niger* were significantly larger and rates of lesion expansion significantly higher than those occurring in any of the *F. moniliforme* isolates. Even at 30°C, which is optimal for fruit infection and sporulation by *F. moniliforme*, lesions caused by the *A. niger* isolate were significantly larger. Thus, at temperatures above 30°C, this isolate of *A. niger* may have a competitive advantage over *F. moniliforme*. If both pathogens are simultaneously present in the fig cavity and the prevailing temperatures are 30°C or above, our results suggest that fruit

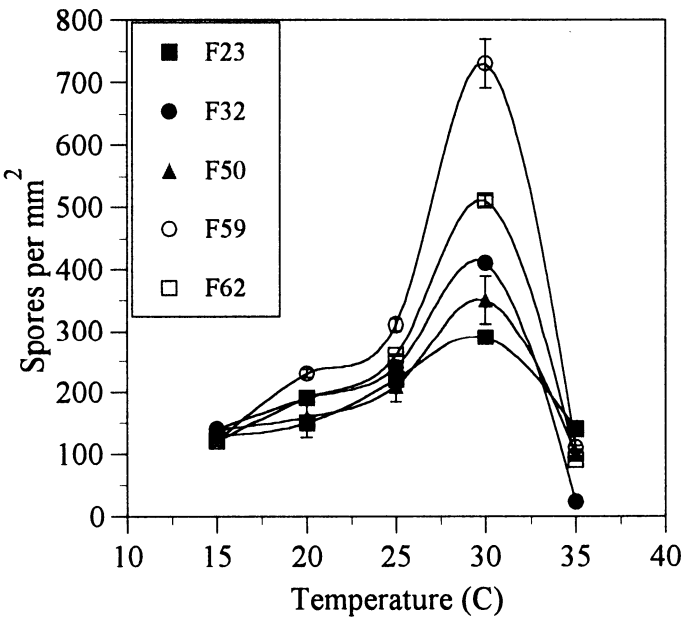

Fig. 6. Effects of temperature on sporulation (spores per mm² lesion area) on fig fruits by five *Fusarium moniliforme* isolates inoculated into artificial wounds on surfaces of near-mature caprifigs and incubated for 6 days.

symptoms typical of smut instead of endosepsis will result. Nevertheless, such conditions seldom occur during caprifig cultivation in California and, thus, smut is very rare on caprifigs. The conditions described above occur routinely during the Calimyrna fig season and therefore may explain why smut is more common in this crop season. *Fusarium moniliforme* isolates evaluated in this study were able to cause endosepsis at temperatures between 15 and 30°C. Because *F. moniliforme* is pathogenic over a wider temperature range, it has the potential to occur more frequently than *A. niger* on figs. Evaluation of mycelial growth of these two pathogens under combinations of temperature and osmotic potentials provided similar results (15).

The disease cycle of *F. moniliforme* is inextricably linked to the fig pollination cycle (3,8). The pathogen is introduced into the cavities of caprifigs and Calimyrna figs during the obligatory entry of the pollinator insect, *B. psenes*. In contrast, *A. niger* inoculum is introduced into the fig cavity by a variety of means such as nitidulid beetles that visit Calimyrna fruits and via the direct deposition of spore around the ostiole (9). Even though the two diseases are introduced into the fig cavities by different mechanisms and the infection begins in the cavities (3,8,9), the effects of temperature on different aspects of endosepsis and smut disease cycles were evaluated in this study by inoculating fig halves on the surface. Inoculation of both pathogens on the surface yields distinct, regular, measurable lesions that are circular. Because inoculations in the cavity result in irregular lesions that are difficult to quantify (14), and the results can be confounded by background inoculum already present in the fig cavities, evaluation of isolates or pathogens can be obtained more objectively with surface inoculations.

In a detailed analysis of the virulence of *F. moniliforme* isolates from cultivated and wild caprifigs, we determined that the latter were much more virulent than the former (14). In this study, we evaluated two isolates collected from cultivated and three isolates collected from wild caprifigs for different disease components at different temperatures. Isolates from wild caprifigs had significantly shorter incubation and latent periods, produced significantly larger lesions and greater numbers of spores per lesion, and had significantly faster lesion expansion rates than isolates from cultivated caprifigs. Isolates from within the wild caprifigs differed significantly in virulence. It is unclear why the isolates from the wild caprifigs were much more virulent than those from cultivated caprifigs. A plausible explanation could be that the continuous management of endosepsis in cultivated caprifig orchards by the use of fungicides since the early 1930s (10) has resulted in an apparent loss of virulence in the isolates from cultivated caprifig orchards. In contrast, the wild caprifig trees are located in the Sierra Nevada mountains isolated from the commercial fig production areas, resulting in both physical and genetic isolation of the pathogen. Because of their greater virulence over a wider temperature range compared with isolates from cultivated caprifigs, and the progressive unavailability of commonly used fungicides, isolates from wild caprifigs have the potential to cause widespread losses if incorporated into the agroecosystem. In some years, caprifig growers augment the supply of cultivated caprifigs with caprifigs from trees growing wild in the Sierra Nevada mountains (14). In so doing, virulent isolates of *F. moniliforme* are brought into the cultivated agroecosystem that may cause significant long-term problems for fig production in California. Growers often face the dilemma of choosing between producing a Calimyrna fig crop by augmenting their supply of caprifigs for pollination with wild caprifigs, and having insufficient caprifigs to produce an edible crop. The effect of using wild caprifigs on the severity of endosepsis in subsequent fig crops was unknown until recently. This work provides further evidence to discourage such a practice.

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