

# The distribution of peach scab lesions on the surface of diseased peaches

Clive H. Bock · Michael W. Hotchkiss ·  
William R. Okie · Bruce W. Wood

Accepted: 17 February 2011 / Published online: 13 March 2011  
© KNPV 2011

**Abstract** The aim of the study was to quantify peach scab (*Fusicladosporium carpophilum*) lesion distribution relative to the point of maximum lesion number on the fruit surface, the relationship between lesion count and distance from the point of maximum lesion density, and establish whether the distribution of lesions was consistent with a splash dispersed pathogen, and to assess the effect of lesion number on fruit size. Fruit of four cultivars, Jerseyqueen, Jefferson, BY07-6428r and Dixiland were collected and the fruit (assumed spherical) sliced taking three horizontal planes across the axis from the point of maximum disease, such that each horizontal zone (Z1–Z4) had the same vertical height, and thus equal surface areas. Lesion counts were analysed using general linear modeling with a Poisson distribution and a log-link function. Zones on the fruit had different numbers of lesions ( $P<0.0001$ ), with most lesions found on Z1. Cvs differed in the

number of lesions per fruit ( $P=0.0042$ – $<0.0001$ ). An analysis of covariance showed that although fruit size varied among most cvs ( $P=0.1614$ – $<0.0001$ ), the number of lesions on a fruit did not affect fruit size ( $P=0.5654$ ). Measurements of the point of maximum disease relative to the peduncle-flower scar axis of the fruit suggest that fruit are not always held upright when infection occurs, such that up to 40% of fruit showed maximum infection at an angle  $>90^\circ$  to the peduncle. This pattern of disease is consistent with observations of the splash-borne nature of conidia, with the most exposed, easily wetted, uppermost portion of fruit developing most disease.

**Keywords** Epidemiology · Disease distribution · *Fusicladosporium carpophilum* · Rain splash

## Introduction

Loss of fruit to peach scab caused by the fungal pathogen *Fusicladosporium carpophilum* (Partridge & Morgan-Jones) is a problem for peach (*Prunus persica* (L.) Batsch) producers in wet humid environments, such as those in the southeastern US (Keitt 1917; Scherm and Brannen 2005). The pathogen infects twigs and leaves of peach, and also developing fruit, where it can cause numerous lesions that can make the fruit unsuitable for fresh sale, and in extreme cases can cause cracking of the fruit and premature fruit drop (Keitt 1917; Scherm and Brannen 2005).

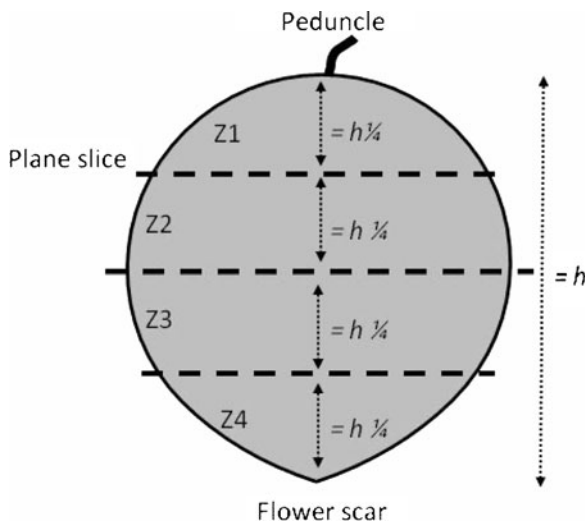
---

C. H. Bock (✉) · M. W. Hotchkiss · W. R. Okie ·  
B. W. Wood  
USDA-ARS-SEFNTRL,  
21 Dunbar Road,  
Byron, GA 31008, USA  
e-mail: clive.bock@ars.usda.gov

M. W. Hotchkiss  
e-mail: mike.hotchkiss@ars.usda.gov

W. R. Okie  
e-mail: dick.okie@ars.usda.gov

B. W. Wood  
e-mail: bruce.wood@ars.usda.gov

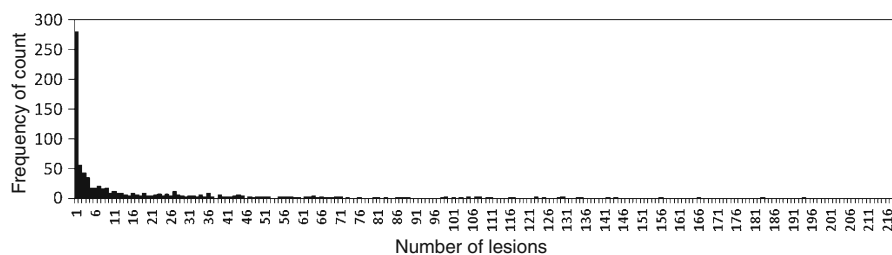


**Fig. 1** Diagram representing the slicing of the peach fruit to divide it into four segments (Z1–Z4) by taking horizontal planes across the axis ( $h$ ) from the point of maximum disease such that each segment slice had the same vertical height ( $h/4$ ). The surface area of a sphere is calculated as  $4\pi r^2$  ( $\pi$  is 3.142, and  $r$  is the radius of the sphere). Archimedes demonstrated that the surface area of a spherical cap was equal to the area of the circle whose radius is the distance from the central point of that spherical cap to its boundary, thus the four zones have equal surface areas

Lesions present on the fruit surface detract from its appearance, and in commercial operations symptomatic fruit are culled to a lower grade, or removed from the packing line entirely (Anonymous 1995), representing economic loss to the grower. Colonization of fruit by other pathogens through scab-induced damage might also occur. Peach scab is controllable with fungicides, and although costly, recently incorporated reduced fungicide spraying strategies are now superior to older strategies from both economic and environmental perspectives (Scherin and Saville 2001; Lan et al. 2003; Schnabel and Layne 2004; Scherin and Brannen 2005; Schnabel et al. 2007).

The latent period of the pathogen on fruit is  $\approx 42$  day (Keitt 1917), and in central Georgia abundant conidia are produced from mid-March to late-June (Scherin et al. 2008). In central Georgia fruit set occurs sometime in March so fruit are exposed to conidia during expansion, and early infections have time to fully develop (Keitt 1917; Scherin and Brannen 2005). Conidia are primarily splash dispersed with rain from overwintering twig infections during spring and early summer (Keitt 1917; Lan and Scherin 2003; Scherin et al. 2008); although, some are likely dispersed in air currents (Lawrence and Zehr 1982; Gottwald 1983). Once dispersed, and in the presence of prolonged surface moisture, conidia germinate and infect, with symptoms appearing as small, dark lesions. Keitt (1917) comments on the difficulty of wetting young peach fruit due to the hirsute nature of their surface, and some fruit are upturned on twigs during early development, which would affect the presentation of the fruit surface to spore infested splash or run-off from twigs. Also, on many fruit the calyx is retained and this could also result in water and spores being trapped between the fruit surface and the calyx, providing an environment for infection. As fruit progress through stages in development (Zanchin et al. 1994) and become heavier, they often hang more vertically from the peduncle, thus changing position and possibly intercepting inoculum on a different (but still uppermost) side (Keitt 1917).

The characteristic mode of dispersal (primarily rain splash), plus the difficulty of wetting young peach fruit surfaces, might result in only the most exposed parts of fruit becoming infected. This is supported in that certain authors allude to infection being greatest around the peduncle of the approximately spherical peach fruit (Keitt 1917; Scherin and Brannen 2005). Assuming that successful conidia germination and



**Fig. 2** The combined frequency of lesion counts of peach scab (caused by *Fusicladosporium carpophilum*) on the surface of four cultivars of peach fruit (Jerseyqueen, Jefferson, BY07-6428r, and Dixieland)

**Table 1** Means of surface area per zone<sup>a</sup>, lesions per fruit, and number of lesions of peach scab (caused by *Fusicladosporium carpophilum*) on four zones (Z1–Z4) of four cultivars of peach

Variable	Cultivar mean (standard deviation) <sup>b</sup>				
	Jefferson	Jerseyqueen	BY07-6428r	Dixiland	Mean data all cvs
Fruit surface area (cm <sup>2</sup> )	92.1 (13.7)	65.9 (8.9)	93.0 (14.1)	157.8 (27.2)	102.5 (38.6)
Mean lesions per fruit	11.1 (12.2)	77.5 (45.4)	125.1 (62.7)	48.8 (48.3)	63.8 (60.9)
Number lesions Z1	7.7 (8.4)	52.8 (31.1)	82.6 (47.9)	39.3 (38.5)	44.5 (43.1)
Number lesions Z2	2.7 (4.3)	21.9 (15.8)	30.6 (16.5)	7.8 (10.7)	15.3 (16.7)
Number lesions Z3	0.4 (0.9)	2.7 (3.7)	9.6 (8.4)	1.7 (3.4)	3.4 (5.8)
Number lesions Z4	0.3 (1.7)	0.04 (0.2)	2.4 (3.5)	0.06 (0.23)	0.6 (2.1)

<sup>a</sup> Fruit sliced into zones by taking the apex of the fruit as the point of maximum disease and slicing planes across this axis at three equal distances

<sup>b</sup> The sample standard deviation was calculated as  $\sqrt{(\sigma/n)}$ , where  $\sigma$  = sample variance, and  $n$  = sample size

fruit infection is on the uppermost and most exposed and easily wettable surfaces, then the resulting spatial distribution of lesions should reflect a characteristic infection pattern. The objectives of this study were to a) quantify differences in lesion distribution relative to the point of maximum lesion number on the fruit surface, b) identify the relationship between lesion number and distance from the zone of maximum lesion number on the fruit, and c) establish whether distribution of disease symptoms is consistent with

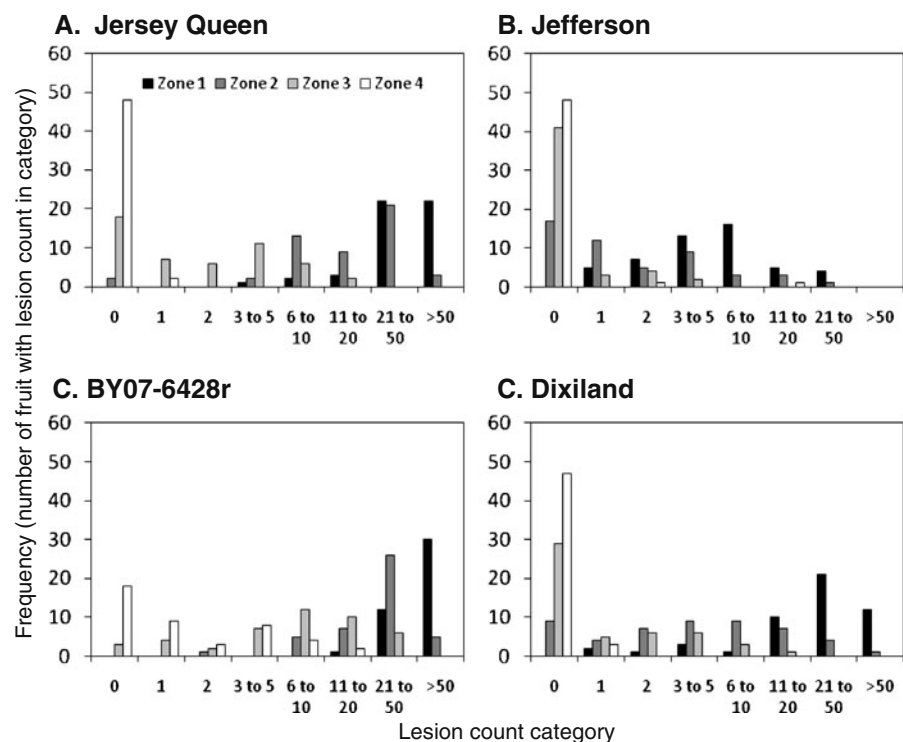
that of a splash dispersed pathogen, and iv) assess the effect of lesion number on fruit size.

## Material and methods

### Peach fruit

Two samples of fruit were taken in June–July 2010 to characterize lesion distribution. Firstly, a sample

**Fig. 3** The frequency of number of lesions of peach scab (caused by *Fusicladosporium carpophilum*) on zone (Z) 1, Z2, Z3 and Z4 on sample of four different cultivars of peach fruit. Fruit sliced into zones by taking the apex of the fruit as the point of maximum disease and slicing three planes across this axis such that it was divided into four sections of equal length



of 194 scab-infected peach fruit (cvs. Jefferson, Jerseyqueen, Dixiland and BY07-6428r, with sample sizes of 50, 50, 50 and 44 fruit, respectively) were harvested at random from plots at the USDA-ARS research farm in Peach County, GA (latitude +32° 39' 54" N, longitude +83° 44' 31" W; elevation of ≈156 m; ≈240 day freeze-free growing period with annual precipitation of ≈181 mm). Jefferson and Jerseyqueen were planted adjacent to each other and received little thinning and no scab-preventative sprays. Cvs Dixiland and BY07-6428r were in separate blocks, were thinned, and received fungicide sprays (chlorothalonil 720 at 4.7 l ha<sup>-1</sup> (Chemnut Inc., Leesburg, GA), and sulphur at 10 kg ha<sup>-1</sup> (Yellow Jacket Brand Sulfur, Georgia Gulf Sulfur Corporation, Valdosta, GA)) in April and June. Cv Dixiland received a single chlorothalonil spray and two sulphur applications, and cv BY07-6428r received two sulphur applications only. All four cvs bloomed at similar times (except BY07-6428r was slightly earlier) and ripened at similar times. Samples

were placed in a cool room at 10°C, and processed within 2 weeks of harvest. For the purpose of counting lesions, peach fruit were assumed to be a sphere (Quilot et al. 2004). Based on the diameter of the fruit (marking the point of greatest disease as the “apex”; not necessarily the peduncle end), the fruit was divided by taking three horizontal planes across the “disease” axis such that the four resulting segments each had the same vertical height (Fig. 1). If there was only a single lesion, it was taken as the apex, but if there was >1 lesion, then the mid-point of the lesion cluster was judged visually. The surface area of the peach ( $4\pi r^2$ , where  $\pi$  is 3.142, and  $r$  is the radius of a sphere) was calculated. Archimedes demonstrated that the surface area of a spherical cap was equal to the area of the circle whose radius is the distance from the central point of that spherical cap to its boundary. Thus because the vertical height between each plane through the peach was the same, it follows that the segment (the portion of a sphere cut off by two parallel planes)

**Table 2** The analysis of fixed effect parameter estimates used to explore the relationship between number of lesions of peach scab (caused by *Fusicladosporium carpophilum*) on four zones of fruit of four cultivars of peach<sup>a</sup>, and the score statistics for

the Type 3 GEE<sup>b</sup> analysis to test the significance of the fixed effects. The dependent variable data (lesion number) were analysed with a general linear model using a Poisson distribution with a log-link function

Analysis of GEE parameter estimates (empirical standard error estimates) <sup>c</sup>					Score statistics for Type 3 GEE analysis <sup>d</sup>		
Parameter		Estimate (standard error)	z <sup>e</sup>	Pr > z <sup>f</sup>	Df <sup>g</sup>	Chi-sq value <sup>h</sup>	P > Chi-Sq <sup>i</sup>
Cultivar	DL	−0.94 (0.16)	−5.98	<0.0001	3	73.61	<0.0001
	JF	−2.43 (0.17)	−14.18	<0.0001			
	JQ	−0.48 (0.11)	−4.32	<0.0001			
	BY	0.00 (0.00) <sup>j</sup>	.	.			
Zone	1	4.24 (0.23)	18.86	<0.0001	3	104.38	<0.0001
	2	3.17 (0.22)	14.31	<0.0001			
	3	1.67 (0.21)	7.86	<0.0001			
	4	0.00 (0.00)	.	.			

<sup>a</sup> Fruit sliced into zones by taking the apex of the fruit as the point of maximum disease and slicing three planes across this axis such that it was divided into four of sections equal length. Cvs JF = Jefferson, JQ = Jerseyqueen, DL = Dixiland and BY = BY07-6428r

<sup>b</sup> General Estimating Equations. The analysis was done using proc genmod (SAS V9.2), invoking the GEE option with the repeated statement (subject = individual fruit) to specify the covariance structure of the model

<sup>c</sup> Goodness-of-fit statistics for the GEE model: QIC=−5191.9, QICu=−5199.9

<sup>d</sup> Chi-square values are likelihood ratios for testing significance of the fixed effects in the model

<sup>e</sup> z-statistic for the z-distribution

<sup>f</sup> Probability that the parameter estimate z-statistic is significant

<sup>g</sup> Degrees of freedom

<sup>h</sup> The Wald *Chi-square* value for the fixed effect

<sup>i</sup> Probability that the Wald *Chi-Square* value of the fixed effects in the Type 3 GEE analysis for the full model is significant

<sup>j</sup> A parameter is given a zero value when the column in the model matrix denoting the parameter is found to be linearly dependent on columns corresponding to parameters preceding it

**Table 3** Estimates<sup>a</sup> of the mean number of lesions of peach scab (caused by *Fusicladosporium carpophilum*) on four zones<sup>b</sup> and on four cultivars<sup>c</sup> of peach

Contrast		L'Beta (standard error of L'Beta) <sup>d</sup>	Mean estimate <sup>e</sup>	Chi-sq <sup>f</sup>	P > Chi-sq <sup>g</sup>
Zone	1	3.51 (0.06)	33.34	3133.3	<0.0001
	2	2.44 (0.07)	11.44	1265.8	<0.0001
	3	0.94 (0.11)	2.56	79.03	<0.0001
	4	−0.74 (0.22)	0.48	10.90	<0.0010
Cultivar	DL	1.56 (0.14)	4.75	116.61	<0.0001
	JF	0.07 (0.17)	1.07	0.18	<0.6703
	JQ	2.02 (0.10)	7.53	418.14	<0.0001
	BY	2.50 (0.22)	12.16	563.57	<0.0001

<sup>a</sup> The mean estimates were generated using the 'lsmeans' statement in SAS applying a general linear model with a Poisson distribution and a log link function for lesion numbers

<sup>b</sup> Fruit sliced into zones by taking the apex of the fruit as the point of maximum disease and slicing three planes across this axis such that it was divided into four sections of equal length

<sup>c</sup> Cvs JF = Jefferson, JQ = Jerseyqueen, DL = Dixiland and BY = BY07-6428r

<sup>d</sup> The L'Beta is the log of the estimate

<sup>e</sup> The mean estimate is the exponentiation of the L'Beta, i.e., the "raw" mean

<sup>f</sup> The *Chi-square* value of the L'Beta estimate

<sup>g</sup> Probability that the *Chi-square* value is significantly greater than the table value

**Table 4** Estimates of the pairwise difference<sup>a</sup> in mean number of lesions of peach scab (caused by *Fusicladosporium carpophilum*) on four zones<sup>b</sup> of four cultivars<sup>c</sup> of peach The mean estimates between fixed effects of zone and cultivar are based on a general linear modeling using a Poisson distribution and a log link function for lesion numbers

Contrast		L'Beta (standard error of L'Beta) <sup>d</sup>	Mean estimate <sup>e</sup>	Chi-sq <sup>f</sup>	P > Chi-sq <sup>g</sup>
Zone	1–2	1.07 (0.06)	2.91	355.91	<0.0001
	1–3	2.57 (0.10)	13.05	652.8	<0.0001
	1–4	4.24 (0.23)	69.57	355.61	<0.0001
	2–3	1.50 (0.09)	4.48	302.51	<0.0001
	2–4	3.17 (0.22)	23.88	204.77	<0.0001
	3–4	1.67 (0.21)	5.33	61.85	<0.0001
Cultivars	DL-JF	1.49 (0.21)	4.43	51.65	<0.0001
	DL-JQ	−0.46 (0.16)	0.63	8.20	0.0042
	DL-BY	−0.94 (0.16)	0.39	35.73	<0.0001
	JF-JQ	−1.95 (0.18)	0.14	124.58	<0.0001
	JF-UK	−2.43 (0.17)	0.09	200.98	<0.0001
	JQ-BY	−0.48 (0.11)	0.62	18.64	<0.0001

<sup>a</sup> Hypothesis tests of the differences between pairs of zones and cvs were performed using the 'estimate' statement in SAS

<sup>b</sup> Fruit sliced into zones by taking the apex of the fruit as the point of maximum disease and slicing three planes across this axis such that it was divided into four sections of equal length

<sup>c</sup> Cvs JF = Jefferson, JQ = Jerseyqueen, DL = Dixiland and BY = BY07-6428r

<sup>d</sup> The L'Beta is the log of the estimate

<sup>e</sup> The mean estimate is the exponentiation of the L'Beta, i.e., the "raw" mean

<sup>f</sup> The *Chi-square* value of the L'Beta estimate

<sup>g</sup> Probability that the *Chi-square* value is significantly greater than the table value

exhibited zones (the curved surface of a spherical segment) with equal surface areas. The number of lesions on each of the four zones (designated Z1, Z2, Z3 and Z4, going down the disease axis from the point of greatest disease density) was counted. Fruit diameter was measured (before slicing planes) using digital calipers (Sylvac Ultra-Cal Mark III Electronic Caliper, Sylvac, Crissier, Switzerland), measuring along the peduncle-flower scar axis.

Secondly, 100 fruit of each of three cvs (cvs. Jefferson, Jerseyqueen, and Dixiland) were chosen at random from the same trees as the previous samples, and the angle at which the maximum disease density occurred in relation to the peduncle-flower scar axis was measured. An Angle Finder (Swanson Tool Co., Inc., Frankfort, IL) was used to measure the angle of the axis of greatest disease density against the peduncle-flower scar axis. Reports suggest disease most often develops on the most exposed, upper surface (Keitt 1917), and since much of the visible disease is from infection caused earlier in the sizing process (Keitt 1917), by taking this angle it is possible to gain some insight into the angle fruit might have been when subject to infection.

## Data analysis

Analyses were performed in SAS V9.2 (SAS Systems, Cary, NC). The lesion count data were not normally distributed (Fig. 2) and initially the analysis of the distribution of lesion counts was compared using a Poisson and a negative binomial distribution for the null model (using proc genmod with a log-link function). Goodness-of-fit for the general linear models was based on the Quasi-likelihood Information Criterion (QIC), and on QICu, an approximation of QIC based on the number of regression parameters. These goodness-of-fit statistics provide a measure of the ability of different models derived using General Estimating Equations (GEEs) to describe the data—the smaller the number, the more appropriate the model. The GEEs provide a semi-parametric technique for estimating effects of parameters where unknown correlations exist. GEEs also generate robust standard errors. The covariance structure based on individual fruit (minimum cluster size=4) was identified using the ‘repeated’ statement in SAS, which also invokes the GEE process. The QIC and QICu for the null model indicated distribution of scab lesions on individual fruit was

**Table 5** Analysis of covariance<sup>a</sup> of the fixed effects of cultivar and number of lesions of peach scab (caused by *Fusicladosporium carpophilum*) on the size of peach fruit, with Type 3 score statistics to test the significance of the fixed effects

Analysis of fixed effects					Type 3 tests of fixed effects		
Parameter		Estimate (standard error)	<i>t</i> -value <sup>b</sup>	<i>P</i> > <i>t</i> <sup>c</sup>	Df <sup>d</sup>	<i>F</i> -value <sup>e</sup>	<i>P</i> > <i>F</i> <sup>f</sup>
Intercept		24.82 (1.49)	16.72	<0.0001			
Cultivar <sup>g</sup>	DL	14.94 (1.73)	8.64	<0.0001	3	101.54	<0.0001
	JF	−2.40 (1.71)	−1.41	<0.1614			
	JQ	−7.83 (1.93)	−4.06	<0.0001			
	BY	0 (0)	.	.			
Lesions (Cultivar)	DL	−0.01 (0.01)	−0.48	0.6316	4	0.74	0.5654
	JF	−0.05 (0.05)	1.06	0.2923			
	JQ	−0.01 (0.01)	−0.48	0.6329			
	BY	−0.01 (0.01)	−1.18	0.2403			

<sup>a</sup> Analysis was done using proc mixed in SAS V9.2, with lesion number as a covariate with cultivar. Goodness of fit statistics for the model: AIC=1654.0, AICc=1654.0, BIC=1657.2

<sup>b</sup> *t*-distribution value to gauge significance of the model parameter

<sup>c</sup> *P* = Probability the *t*-value for the model parameter is significant

<sup>d</sup> Df = degrees of freedom

<sup>e</sup> *F*-distribution value of fixed effect in model

<sup>f</sup> Probability the *F*-value for the model fixed effect significant

<sup>g</sup> Cvs JF = Jefferson, JQ = Jerseyqueen, DL = Dixiland and BY = BY07-6428r

least for the model assuming the Poisson distribution (QIC=−825.4, QICu=−825.5, with intercept 2.77 (se 0.07),  $z=40.48$  ( $P<0.0001$ )), rather than a negative binomial distribution (QIC=−83623.1, QICu=−83623.3, with intercept=0.35 (se 0.27),  $z=1.27$  ( $P=0.2032$ )). Subsequently, fixed effects of cultivar and zone were analysed for effect on the dependent variable lesion number using a Poisson distribution with a log-link function. Unbiased estimates of the means (and associated standard errors) for zone and cultivar were generated using the 'lsmeans' statement in SAS. 'Estimate' statements were used to test hypotheses that lesion counts were different between zones and between cvs (the null hypothesis,  $H_0$  = no difference between means). The effect of lesion number on fruit size (surface area, mm<sup>2</sup>) was explored with analysis of covariance (proc mixed) using individual fruit and cultivar as class effects, and number of lesions per fruit as the covariate. Goodness of model fit was assessed using Akaike's Information Criterion (AIC), the Corrected Akaike's Information Criterion (AICc), and the Bayesian Information Criterion (BIC, as with the QIC, the smaller the number, the more appropriate the model). 'Estimate' and 'contrast' statements were used to compare lesion numbers among cultivars, and to test hypotheses of the effect of minimum, mean and maximum lesion count on fruit size for each cv. Depending on the particular analysis, the significance of a parameter, fixed effects or differences between means was tested using a Chi-square test, a  $t$ -tests, the  $z$ -statistic, or the  $F$ -test. In addition, standard errors were used to gauge the quality of the parameter estimates and mean values.

## Results

### Lesion distribution

The fruit surface area ranged from 65.9 cm<sup>2</sup> for Jerseyqueen to 157.8 cm<sup>2</sup> for Dixiland (Table 1). The mean number of lesion per fruit was greatest for BY07-6428r (125.1 lesions per fruit), and least for Jefferson (11.1 lesions per fruit). Most lesions were found on the upper-most zone of the fruit, and for all cvs there was a greater frequency of higher lesion counts in Z1 and Z2 of the fruit for each cultivar (Fig. 3).

The GLM analysis using a Poisson distribution with a log-link function (Table 2) demonstrated

parameters of the fixed effects of both cultivar (Jefferson, Jerseyqueen, Dixiland and BY07-6428r) and fruit zone (Z1-4) contributed significantly to the model. Furthermore, the goodness-of-fit parameters were superior when based on the Poisson distribution (QIC=−5191.9, QICu=−5199.9) compared to the negative binomial distribution (QIC=−22808.7, QICu=−22804.5). Estimates of the mean number of lesions on each zone demonstrated each was highly significant (Table 3) with fewest lesions on the lower zones. The estimates of cultivar means were significant, except for cv. Jefferson which also had consistently fewest lesions. Estimates of the pairwise differences among zones and among cvs showed that each comparison was significantly different (Table 4).

There was no effect of lesion number on fruit size, but cultivar had a significant effect (Table 5). Model-based estimates of the fruit mean size at the minimum, mean and maximum lesion count for each

**Table 6** Estimates<sup>a</sup> of the size (surface area, cm<sup>2</sup>) of peach fruit of four cultivars infected with the minimum, mean and maximum number of lesions of peach scab (caused by *Fusicladosporium carpophilum*) for each cultivar

Cultivar <sup>b</sup>	Lesion number <sup>c</sup>	Estimate (standard error)	$t$ -value <sup>d</sup>	$P > t^e$
DL	Min	159.0 (3.5)	45.4	<0.0001
	Mean	157.8 (2.5)	63.8	<0.0001
	Max	152.4 (11.6)	13.2	<0.0001
JF	Min	89.9 (3.2)	27.9	<0.0001
	Mean	92.1 (2.5)	37.2	<0.0015
	Max	102.1 (9.8)	10.5	<0.0001
JQ	Min	67.8 (4.6)	14.7	<0.0001
	Mean	65.9 (2.5)	26.7	<0.0001
	Max	62.0 (8.5)	7.3	<0.0001
BY	Min	98.6 (5.4)	18.2	<0.0001
	Mean	93.0 (2.6)	35.3	<0.0001
	Max	86.1 (6.4)	13.5	<0.0001

<sup>a</sup> The mean estimates of fruit size were generated using the 'estimate' statement in SAS

<sup>b</sup> Cvs JF = Jefferson, JQ = Jerseyqueen, DL = Dixiland and BY = BY07-6428r

<sup>c</sup> Cultivar minimum, mean and maximum: DL = 1, 48 and 267; JF = 1, 11, and 57; JQ = 7, 77 and 226; and BY = 1, 125 and 262 lesions per fruit, respectively

<sup>d</sup>  $t$  =  $t$ -distribution value of the estimate

<sup>e</sup>  $P$  = Probability the  $t$ -value is significant



**Table 7** Estimates of the pairwise difference<sup>a</sup> in size of peach fruit with symptoms of scab (caused by *Fusicladosporium carpophilum*). The mean estimates between fixed effects of zone and cultivar are based on a general linear model with fixed effects of cultivar and number of lesions

Cultivar contrast <sup>b</sup>	<i>F</i> -value <sup>c</sup>	<i>P</i> > <i>F</i> <sup>d</sup>
DL-JF	202.09	<0.0001
DL-JQ	225.30	<0.0001
DL-BY	74.71	<0.0001
JF-JQ	13.27	<0.0003
JF-BY	1.98	<0.1614
JQ-BY	16.47	<0.0001

<sup>a</sup> Hypothesis tests of the differences in size between pairs of cvs were performed using the ‘contrast’ statement in SAS

<sup>b</sup> Cvs JF = Jefferson, JQ = Jerseyqueen, DL = Dixiland and BY = BY07-6428r

<sup>c</sup> *F*-distribution value of the pairwise difference

<sup>d</sup> Probability the *F*-value for the model parameter is significant

cultivar confirmed no effect of these lesion number ranges on fruit size (Table 6), although the pairwise comparisons of the mean estimates of fruit size between cvs showed all were significantly different, except for cvs Jefferson and BY07-6428r (Table 7).

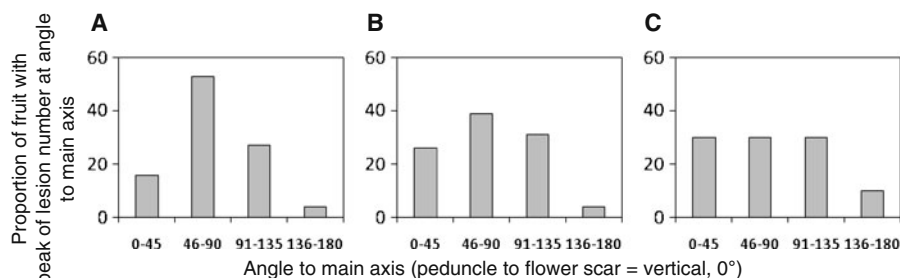
#### Fruit angle in relation to scab

Among the three cvs where the angle was measured, the majority of fruit had maximum scab density at 0° to 90° from the vertical axis (Fig. 4, assuming the peduncle the apex, the vertical axis, 0°, was from peduncle to flower scar). A proportion of the fruit in all cvs had greatest disease density on the flower scar end (at angles >90° to the fruit axis), but this proportion was less for all four cvs, although it accounted for up to 40% of fruit with cv. Dixiland.

## Discussion

As previously suggested (Keitt 1917; Scherm and Brannen 2005), these data demonstrate that peach scab lesions are not uniformly distributed over the surface of peach fruit. Lesion distribution is therefore definable and predictable. There is a rapid decline in lesions from the point of greatest lesion numbers. The zones of the fruit opposite the point with the greatest lesion numbers rarely have any infection at all. The relationship was described by a general linear model using a Poisson distribution with a log-link function. Indeed, on this “disease axis”, very few lesions were observed below the “equator”. The distribution of lesions on fruit has rarely been studied, but the characteristics of the distribution of the peach scab lesions on peach are similar to that reported for citrus canker on grapefruit in Florida (Bock et al. 2010), which is also a splash-dispersed pathogen.

The different peach cvs had different lesion counts, although on all cvs the greatest counts were on Z1. Thus the point at which a peach fruit is most exposed to inbound inoculum and infection appears to result in the same characteristic distribution of infection over the fruit surface. The most exposed point is probably the uppermost face of the fruit (Keitt 1917), although this remains to be confirmed. Cvs do vary in size and shape (Okie 1998), although many approximate to spherical (Quilot et al. 2004), which might explain certain differences in disease distribution on fruit surfaces, although the particular cvs in this test had similar, generally spherical shapes. There was difference among cvs in fruit size, probably in part due to particularly heavy crops on Jerseyqueen, Jefferson and the BY07-6428r trees, compared to the Dixiland, which was thinned. At the time of infection, most developing fruit are somewhat elongated, but the



**Fig. 4** The numbers of peach fruit with the peak in peach scab (caused by *Fusicladosporium carpophilum*) lesion density relative to the peduncle—flower scar axis (= 0°) on three cultivars of peach, **a** Jerseyqueen, **b** Jefferson, and **c** Dixiland



major difference in lesion counts between cvs is most likely due to differences in inoculum pressure, spray treatments, fruit development patterns or cv. resistance to infection.

The peduncle end of the fruit is a common location for maximum lesion density (Keitt 1917; Scherm and Brannen 2005), and the results of this study indicate that >60% of fruit had most disease on the upper hemisphere. However, peaks in lesion density can often occur on an axis different to the peduncle-flower scar axis (a few fruit of each cultivar had the peak in lesion density 180° from the peduncle end). The observation that many fruit do not have the highest density of lesions associated with the peduncle zone indicates that the symptom side of those peach fruit was uppermost at time of infection when sufficient wetting of the hirsute fruit surface would favor successful colonization and infection (Keitt 1917). The fruit presentation near time of maturity may be different to that during the first few weeks after fruit-set, when the developing fruit are subject to peaks in conidia dispersal (Lawrence and Zehr 1982; Scherm et al. 2008). Zones exposed to wetting may change over time due to fruit weight, but whether waves of infection can result in different areas of disease on fruit has not been ascertained. Marking fruit shortly after calyx shed and monitoring them throughout the season for angle relative to the peduncle axis, inoculum availability and disease development will facilitate establishment of factors that influence the location of disease development on the fruit surface. In certain cases disease might develop on unexposed locations on the fruit surface that are subject to microclimatic conditions conducive to infection (e.g., if two fruit are in physical contact).

Existing fungicides can be effectively used to manage peach scab (Scherm and Savelle 2001; Lan et al. 2003; Schnabel and Layne 2004; Scherm and Brannen 2005; Schnabel et al. 2007). These data indicate that the uppermost zones of the fruit are at the greatest risk of infection during periods suitable for dispersal of *F. carpophilum* conidia, and the uppermost zone is not necessarily the peduncle end of the fruit, either when fruit are young or mature. Novel management approaches might be used to help protect the fruit on trees during the period of susceptibility—with particular attention to the upper zones of the peach fruit. Minimizing inoculum

by pruning and good hygiene, and reducing wind and splash should help minimize infection of these vulnerable fruit surfaces. Furthermore, treatments specifically targeted to protect the uppermost surfaces of developing fruit would protect the fruit and reduce disease, thereby increasing the quality and proportion of fruit suitable for trade in the fresh market.

**Acknowledgements** The authors express their thanks to Keith Hough and Emma Cutchens for their help in counting peach scab lesions on peach.

## References

- Anonymous. (1995). United States Standards for Grades of Peaches. U.S. Department of Agriculture, Agricultural Marketing Service, Fruit and Vegetable Division, Fresh Products Branch, Washington, DC.
- Bock, C. H., Parker, P. E., & Gottwald, T. R. (2010). Distribution of canker lesions on grapefruit in Florida. *Phytopathology*, 100, S15.
- Gottwald, T. R. (1983). Factors affecting spore liberation by *Cladosporium carpophilum*. *Phytopathology*, 73, 1500–1505.
- Keitt, G. W. (1917). Peach scab and its control. *United States Department of Agriculture, Bulletin*, 395, 1–66.
- Lan, Z., & Scherm, H. (2003). Moisture sources in relation to conidial dissemination and infection by *Cladosporium carpophilum* within peach canopies. *Phytopathology*, 93, 1581–1586.
- Lan, Z., Scherm, H., & Horton, D. L. (2003). Reduced midseason pesticide program for control for scab and plum curculio in peach. *Plant Disease*, 87, 699–706.
- Lawrence, E. G., Jr., & Zehr, E. I. (1982). Environmental effects on the development and dissemination of *Cladosporium carpophilum* on peach. *Phytopathology*, 72, 773–776.
- Okie, W. R. (1998). *Handbook of peach and nectarine cultivars: performance in the southeastern United States and index of names*. United States Department of Agriculture, Agriculture Handbook No. 714, pp 814.
- Quilot, B., Kervella, J., & Genard, M. (2004). Shape, mass and dry matter content of peaches of cultivars with different domestication levels. *Scientia Horticulturae*, 99, 387–393.
- Scherm, H., & Brannen, P. M. (2005). Peach scab. In D. Horton & D. Johnson (Eds.), *Southeastern peach growers' handbook* (pp. 134–136). Athens: University of Georgia, College of Agricultural and Environmental Sciences.
- Scherm, H., & Savelle, A. T. (2001). Control of peach scab with reduced mid-season fungicide programs. *Plant Disease*, 85, 706–712.
- Scherm, H., Savelle, A. T., Boozer, R. T., & Foshee, W. G. (2008). Seasonal dynamics of conidial production potential of *Fusicladium carpophilum* in southeastern peach orchards. *Plant Disease*, 92, 47–50.

- Schnabel, G., & Layne, D. R. (2004). Comparison of reduced-application and sulfur based fungicide programs on scab intensity, fruit quality, and cost of disease control on peach. *Plant Disease*, 88, 162–166.
- Schnabel, G., Layne, D., & Holb, I. (2007). Micronised and non-micronised sulphur applications control peach scab equally well with negligible differences in fruit quality. *Annals of Applied Biology*, 150, 131–139.
- Zanchin, A., Bonghi, C., Casadoro, G., Ramina, A., & Rascio, N. (1994). Cell Enlargement and cell separation during peach fruit development. *International Journal of Plant Sciences*, 155, 49–56.