

Reductions in Yield and Vegetative Growth of Grapevines Due to Eutypa Dieback

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ABSTRACT

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Eutypa dieback of grapevines is a serious canker disease that slowly kills vines, but there is little information on yield reduction of declining vines. The severity of Eutypa dieback on individual grapevines was assessed in 1991 and 1992 as the proportion of the vines' spurs killed or symptomatic. In each year, Eutypa dieback caused a significant reduction in yield of infected vines compared with healthy vines. The linear relationship between disease severity (d) and yield (y), expressed as a percentage of the yield of healthy vines, was $y = 101.0 - 90.26d$ ($r^2 = 0.70$) in 1991, and $y = 100.1 - 98.81d$ ($r^2 = 0.79$) in 1992. The slope of this relationship was not significantly different between years or among five vineyards of two cultivars (Chenin blanc and French Colombard). Yield reduction

was primarily due to a diminished number of clusters per vine, while the effects of disease on mean cluster weight were smaller and not always significant. The effect of disease severity on vegetative growth, measured as pruning weight, was significant in two of three vineyards in 1991 and both of two vineyards in 1992. Linear slopes of the pruning weight-disease severity relationships ranged from -29.5% ($r^2 = 0.06$) to -67.4% ($r^2 = 0.46$) per unit disease severity. Yield reductions for whole vineyards, estimated from the regression models, ranged from 30.1 to 61.9%, depending on mean disease severity. Records for Chenin blanc vineyards in Merced County, CA, revealed a trend of declining yields beginning at 12 yr of age, which closely followed the period of rapid increase of Eutypa dieback. In Barbera vineyards, which are rarely affected by Eutypa dieback, yields increased up to age 10 and then remained constant.

Additional keywords: canker, deadarm, *Eutypa armeniaca*.

Eutypa dieback of grapevines, caused by *Eutypa lata* (Pers:Fr.) Tul & C. Tul. (syn. *E. armeniaca* Hansf. & M. V. Carter), is a lethal, perennial canker disease. Ascospores of the fungus infect through pruning wounds and colonize the xylem tissue, then the cambium and phloem (3,4,6,15,17). After an incubation period of 3 yr or more, a canker forms around the infected wound (4,15,17,18). New shoots arising from spurs near the canker are stunted and have small, distorted, chlorotic leaves. Flowers on affected shoots become desiccated and fail to develop into fruit clusters (15,18,19). These symptoms are believed to be caused by phytotoxic compounds produced by the fungus, one of which has been identified as an aldehyde compound named eutypine (14,23). Once a canker has girdled part of a vine, the affected spurs die, and the fungus grows through the xylem toward the main trunk. If infected wood is not removed, the entire vine is

eventually killed. Symptoms are uncommon until a vineyard reaches 10-12 yr of age (5,18). Thereafter, disease incidence can increase rapidly until nearly every vine is affected at 20 yr of age (5). Eutypa dieback is the primary constraint on vineyard longevity in northern California.

Eutypa dieback is managed primarily by protecting pruning wounds with a fungicide and by delaying pruning until late in the dormant season (16,21). However, both practices are only partially effective. The disease can be managed by sanitation pruning and training new growth to replace diseased spurs (corrective pruning). This practice is labor intensive and expensive. Because of the lack of information on the potential benefits of corrective pruning, growers do not routinely utilize this practice. Corrective pruning is performed more diligently in premium wine-growing areas where the high value of individual vines justifies more intensive management.

Yield reductions due to Eutypa dieback have been investigated in Washington State. Johnson and Lunden (9) reported reductions

of 19–50% and 62–94% for moderately and severely diseased vines, respectively. Because of the qualitative nature of these disease severity ratings, it was not possible to define a quantitative relationship between severity and yield reduction.

While dead spurs and those with severely stunted shoots clearly do not contribute to yield, there is no information on the relationship between diseased spurs (observed during spring) and yield of the apparently healthy portion of the vine. Grapevines are believed to compensate for the loss of fruiting buds by producing more fruit on shoots that arise from remaining buds (1,10,12,13). Compensation might mitigate the yield reduction that would result from loss of spurs to *Eutypa* dieback. Conversely, toxins produced by *E. lata* in diseased wood might also affect the yield of apparently healthy spurs. In severely diseased vines, the loss of photosynthate due to reduced growth might also reduce yield. The net effect of these factors on yield will determine the most appropriate disease assessment system.

The main objective of this research was to determine the quantitative effects of *Eutypa* dieback on grapevine yield and vegetative growth. A supporting objective was to evaluate the effect of *Eutypa* dieback on the yield of apparently healthy spurs on infected vines (compensation vs. yield reduction). Another objective was to examine the relationship between vineyard age and yield for wine grape cultivars susceptible and resistant to *Eutypa* dieback. Preliminary results have been reported (20).

MATERIALS AND METHODS

Disease assessment and yield evaluation. During the spring of 1991, five vineyards were surveyed, and the numbers of symptomatic and symptomless spurs were recorded for a sample of vines from each vineyard. Vineyards 1–4 were located in Merced County, CA. Vineyards 1 and 2 were *Vitis vinifera* L. 'French Colombard', and vineyards 3 and 4 were cv. Chenin blanc. Vineyard 5 was a Chenin blanc vineyard in Sacramento County, CA. The five vineyards were established in 1969, 1956, 1967, 1972, and 1969, respectively. Vine spacing was 1.83×3.66 m (1,495 vines/ha) in vineyard 1, 2.14×3.66 m (1,281 vines/ha) in vineyards 2 and 4, 1.52×3.66 m (1,794 vines/ha) in vineyard 3, and 2.44×3.66 m (1,121 vines/ha) in vineyard 5. All vineyards were furrow-irrigated, cordon-trained, and spur-pruned, with two buds per spur. Previous surveys had shown that the spatial patterns of diseased vines in these vineyards were random (5). Subsequent surveys were conducted by enumerating symptomatic and symptomless spurs on each of 50 adjacent vines in four randomly selected rows (200 vines total). Symptoms of dieback were of two types: spurs with typically stunted and distorted shoots, or dead spurs. From the 200 vines, 50 were selected for yield measurement. These vines were selected to represent the entire ranges of both number of symptomless spurs and number of diseased spurs. Vines with obvious symptoms of other diseases or disorders were not selected. When the fruit had matured, all 50 vines in each vineyard were harvested by hand. The number of clusters and the total yield (to the nearest 0.1 kg) were recorded for each vine. Harvest dates in 1991 were 17 September (vineyards 1 and 2), 2 September (vineyard 3), 29 August (vineyard 4), and 26 September (vineyard 5). Vines were pruned during the dormant season, and pruning weights were recorded for each vine as a measure of vegetative growth. Vineyards 1 and 2 were pruned 11 December, and vineyard 5 was pruned 6 January 1992. Pruning weights were not available for vineyards 3 and 4, because the vineyards were destroyed immediately after harvest due to high levels of *Eutypa* dieback.

In 1992, the procedure was repeated in vineyards 1, 2, and 5. The vineyards were surveyed again, and vines used in 1991 were not selected for 1992 experiments. Harvest dates were 15 August for vineyards 1 and 2 and 8 September for vineyard 5. Pruning weights were measured only in vineyards 1 and 2 after the 1992 season. Pruning was done on 8 December.

To confirm the association of *E. lata* with observed symptoms, 60 spurs were collected from one vineyard of each cultivar. Of the 60 spurs, 20 had typical cankers and stunted shoot symptoms,

20 were dead, and 20 were symptomless. In the laboratory, chips of wood were aseptically removed from the canker margins and placed on potato-dextrose agar amended with 100 $\mu\text{g/ml}$ streptomycin sulfate and 50 $\mu\text{g/ml}$ chlortetracycline HCl for the isolation of fungi. For the symptomless spurs, the wood chips were removed from the margins of pruning wounds. Fungi isolated from each spur were identified, and the proportions of diseased and symptomless spurs yielding each fungus were recorded. The association of each fungus with dieback symptoms was measured using odds ratios (7). The odds ratio is the probability of the fungus occurring on a diseased spur divided by the probability of the fungus occurring on a symptomless spur. The significance of the association was tested by the chi-square test, where the expected value of the odds ratio is 1 if there is no association between symptoms and the presence of the fungus. Separate odds ratios were calculated for each combination of vineyard and symptom type. Odds ratios were calculated for the 11 most commonly isolated genera of fungi (*Alternaria*, *Aspergillus*, *Botrytis*, *Cladosporium*, *Diplodia*, *Epicoccum*, *Eutypa*, *Fusarium*, *Penicillium*, *Phomopsis*, and *Rhizopus*).

A disease assessment system was devised based on covariance analysis of the effects of symptomatic and symptomless spurs on yield, using the general linear models procedure of the Statistical Analysis System (SAS)(22). Since the total number of spurs per vine was variable, the number of diseased spurs was not related to the number of symptomless spurs. This facilitated separate evaluations of the effects of the diseased and symptomless spurs. Healthy vines are normally pruned to maintain an optimum number of spurs for balancing high yield and fruit quality. The optimum number of spurs depends on the cultivar, training system, vine spacing, and grower preference. The total number of spurs per vine varied in vines included in our survey, however, because of losses to *Eutypa* dieback or other causes. For the purposes of this study, disease severity on an individual vine was defined as $d_i = 1 - (h_i/h_m)$, where h_i is the number of symptomless spurs on vine i , and h_m is the optimum number of symptomless spurs. The value of h_m for each vineyard was the maximum number of spurs found on any vine in the original 200-vine sample, or the grower's estimate of the optimum number, whichever was lower. For vineyards 1 and 2, $h_m = 12$, for vineyards 3 and 4 $h_m = 16$, and for vineyard 5 $h_m = 18$. The value of d_i is inversely proportional to the number of symptomless spurs on vine i ; it measures the reduction in symptomless spurs, accounting for spurs with symptoms and dead spurs that have been removed or broken off. This assumes that missing spurs were lost due to *Eutypa* dieback.

To evaluate the effect of disease severity on growth and yield, the general linear models procedure was used to conduct linear and polynomial regression analyses for the effects of disease severity on yield, clusters per vine, cluster weight, pruning weight, and the ratio of yield to pruning weight. Regression intercepts (yields of symptomless vines) were standardized to a value of

TABLE 1. Incidence of fungi isolated from healthy and symptomatic spurs and odds ratios for their association with dieback symptoms in two Merced County vineyards

Genus	Incidence ^a	Odds ratio ^b
<i>Alternaria</i>	70.0	1.26
<i>Aspergillus</i>	8.3	0.03
<i>Botrytis</i>	11.7	2.51*
<i>Cladosporium</i>	5.0	0.47
<i>Diplodia</i>	16.7	0.80
<i>Epicoccum</i>	25.0	1.15
<i>Eutypa</i>	55.0	15.07*
<i>Fusarium</i>	65.0	2.05
<i>Penicillium</i>	48.3	1.03
<i>Phomopsis</i>	58.3	1.27
<i>Rhizopus</i>	10.0	0.21

^a All values are for combined data from both vineyards and both symptom types.

^b Values followed by an asterisk are significantly greater than 1.00 ($P \leq 0.05$).

100% (based on estimates from the initial analyses). The linear regression coefficients were compared between vineyards and years by analysis of covariance (22).

Estimated yield reductions. Based on the original 200-vine sample from each vineyard in 1991, mean disease severity within each vineyard was calculated. Yield reduction estimates for each vineyard were calculated by inserting the mean severity into the linear regression model derived from that vineyard. A second estimate was calculated with a regression model derived from combined data from all vineyards. In the second method, data for each year were combined separately.

Yield versus vineyard age. At the Merced County location, yield data from the previous 28 yr were available for several Chenin blanc and Barbera vineyards under the same ownership and management, including those Chenin blanc vineyards used for our yield experiments. *Eutypa dieback* is rarely observed on cv. Barbera. The relationships between yield and vineyard age were described by regression functions estimated using the nonlinear regression (NLIN) procedure of SAS (22).

RESULTS

Of the 11 fungi commonly isolated from the spurs, only *E. lata* was positively associated with dieback symptoms in both vineyards. *Botrytis cinerea* was associated with dieback in one vineyard. *B. cinerea* is saprophytic on the bark of grapevines but does not cause dieback. Combined odds ratios for both vineyards and symptom types were calculated (Table 1).

Disease assessment. The disease assessment system was based on analysis of covariance of the 1991 data. The number of remaining symptomless spurs and the interaction between symptomless spurs and location had significant effects ($P \leq 0.05$) on yield and clusters per vine. Only vineyard location had a significant

effect on cluster weight in 1991 (Table 2). Since there was a significant interaction between the effects of vineyard location and symptomless spurs on yield, analyses were conducted separately for each vineyard. These analyses confirmed that only the number of symptomless spurs significantly affected yield and clusters per vine in each of the five vineyards. The number of diseased spurs did not have a significant effect due to the variable number of spurs per vine. There was no significant effect of symptomless or diseased spurs on cluster weight in 1991. In 1992, analysis of covariance results were very similar, except for the effects on mean cluster weight. There were significant interactions between the effects of vineyard location and symptomless spurs on mean cluster weight in 1992. Also, there were significant effects and interactions involving the effect of diseased spurs (Table 2). Analysis of variance by vineyard revealed that the effect of symptomless spurs on mean cluster weight was significant in vineyards 1 and 2 in 1992.

On the basis of these results, we devised the disease assessment system described in Materials and Methods.

Effects of disease severity on growth and yield. Linear regression provided the best fit for the relationship between disease severity and yield and between disease severity and clusters per vine. The estimates of yield, clusters per vine, and cluster weight for healthy vines for each vineyard (Table 3) were used to standardize variables among the vineyards. This facilitated separate evaluations of the effects of disease, location, and year. When the intercepts were standardized to a value of 100%, regression coefficients for yield and clusters per vine were significant for all five vineyards, and they were not significantly different ($P > 0.05$) among the vineyards in both years. Although linear regression slopes for individual vineyards were somewhat steeper in 1992 than in 1991 (Table 4), they were not significantly different between years for any vineyard ($P > 0.05$). Coefficients of determination for yield

TABLE 2. Sums of squares and *F* tests for the effects of vineyard (VIN), symptomless spurs (SLS), and diseased spurs (DIS) on yield, clusters per vine, mean cluster weight, and pruning weight^a

	1991					1992				
	df	Sum of squares ^b	Mean square ^b	<i>F</i> value	Pr > <i>F</i>	df	Sum of squares	Mean square	<i>F</i> value	Pr > <i>F</i>
Yield										
VIN	4	73.0	18.2	0.63	0.6411	2	0.7	0.4	0.03	0.9748
SLS	1	3,125.9	3,125.9	108.04	0.0001	1	1,454.2	1,454.2	104.42	0.0001
DIS	1	36.8	36.8	1.27	0.2607	1	1.8	1.8	0.13	0.7172
SLS*VIN	4	397.3	99.3	3.43	0.0096	2	5.2	2.6	0.19	0.8311
DIS*VIN	4	46.2	11.5	0.40	0.8092	2	40.2	20.1	1.44	0.2393
SLS*DIS	1	4.9	4.9	0.35	0.5536
Error	210	6,075.9	28.9	145	2,019.3	13.9
Clusters per vine										
VIN	4	1,096.3	274.1	0.44	0.7784	2	54.9	27.4	0.11	0.8954
SLS	1	64,440.5	64,440.5	103.83	0.0001	1	27,291.0	27,291.0	109.96	0.0001
DIS	1	1,859.6	1,859.6	3.00	0.0849	1	234.6	234.6	0.95	0.3325
SLS*VIN	4	11,693.7	2,923.4	4.71	0.0012	2	728.4	364.2	1.47	0.2339
DIS*VIN	4	959.0	239.7	0.39	0.8183	2	379.0	189.5	0.76	0.4679
SLS*DIS	1	40.5	40.5	0.16	0.6869
Error	210	130,338.7	620.7	145	35,986.9	248.2
Mean cluster weight										
VIN	4	0.02	0.00	2.61	0.0364	2	0.09	0.05	20.47	0.0001
SLS	1	0.00	0.00	0.38	0.5375	1	0.00	0.00	0.69	0.4076
DIS	1	0.01	0.01	3.13	0.0782	1	0.02	0.02	8.09	0.0051
SLS*VIN	4	0.00	0.00	0.54	0.7081	2	0.02	0.01	3.70	0.0270
DIS*VIN	4	0.00	0.00	0.33	0.8603	2	0.00	0.00	1.49	0.2283
SLS*DIS	1	0.01	0.01	4.15	0.0435
Error	210	0.39	0.00	145	0.33	0.00
Pruning weight										
VIN	2	4.35	2.17	4.62	0.0119	1	0.50	0.50	1.81	0.1818
SLS	1	10.16	10.16	21.59	0.0001	1	5.81	5.81	20.91	0.0001
DIS	1	2.67	2.67	5.67	0.0190	1	1.18	1.18	4.25	0.0418
SLS*VIN	2	4.58	2.29	4.88	0.0328	1	0.12	0.12	0.44	0.5088
DIS*VIN	2	5.28	2.64	5.61	0.0048	1	0.26	0.26	0.93	0.3378
SLS*DIS	1	0.01	0.01	0.03	0.8732
Error	108	50.82	0.47	100	27.79	0.28

^a Interactions not shown were not significant.

^b Sums of squares and mean squares have been rounded.

and clusters per vine ranged from 0.53 to 0.82 (Table 4). Linear regression of the effect of disease severity on mean cluster weight resulted in slight negative slopes, which were not significant ($P > 0.05$) for any vineyard in 1991. In 1992, disease severity had a significant effect ($P > 0.05$) on mean cluster weight in vineyards 1 and 2 (Table 4). Growth and yield were lower for the French Colombard vineyards in 1992 than in 1991 (Table 3). Since there were no significant differences in the effect of disease on yield among the vineyards, combined models were developed based on data from all five vineyards in 1991 and three vineyards in 1992 (Figs. 1 and 2). Slopes of the combined models were not significantly different ($P = 0.1341$) between years.

Eutypa dieback reduced vegetative growth (measured as pruning weight), but the effect was smaller than the effect on yield. Analysis of covariance showed significant effects of location, symptomless and diseased spurs, and significant interactions on vegetative growth in 1991 (Table 2). In 1992, only symptomless and diseased spurs had significant effects on pruning weight (Table 2). Linear regression coefficients for the effect of disease severity on pruning weight were significant for two of the three vineyards in 1991, but the coefficients of determination were quite low (0.06 to 0.38). In 1992, the effect of disease severity on pruning weight was significant for both vineyards (Table 4, Fig. 2). Disease severity had a significant effect on the ratio of yield to pruning weight in four of the five vineyard-year combinations for which pruning weight was measured (Table 5).

Estimated yield reduction. Mean disease severity for the vineyards ranged from 0.405 to 0.662 in 1991 and from 0.335 to 0.478 in 1992. Yield loss estimates ranged from 30.1 to 61.9% (Table 6). The individual and combined models produced similar estimates.

Yield versus vineyard age. Yields of Chenin blanc vineyards increased with age until the vineyards reached about 12 yr, and then yields decreased with age. The relationship was best described by the polynomial function $y = 1.3696 + 3.6490a - 0.15097a^2$, where y is yield in metric tons per hectare, and a is age in years (Fig. 3). Yields of Barbera vineyards also increased with age until about 12 yr, but then yields remained steady for this cultivar. This relationship was best described by the function $y = 32.9(1 - \exp[-0.35a]) - 11.6$.

DISCUSSION

Yield effects for perennial canker diseases have rarely been reported. This is partially due to the difficulty in establishing experimental plots. In addition to the long periods involved in establishing plants and allowing the disease to develop, it is very difficult to impose and measure different disease levels on populations of perennial plants. Traditional methods of imposing different disease levels, such as inoculation time, fungicide sprays, or genetic resistance are often impractical or unavailable. Another approach is to "impose" different disease levels through careful

TABLE 3. Estimates of intercepts (values for symptomless vines) from linear regression of yield, clusters per vine, and mean cluster weight

Vineyard	Yield (kg)		Clusters per vine		Mean cluster wt (kg)		Pruning wt (kg)	
	1991	1992	1991	1992	1991	1992	1991	1992
1	33.63	22.34	168.7	109.5	0.207	0.221	3.10	2.19
2	40.40	23.15	196.0	113.9	0.227	0.231	2.73	2.20
3	31.42	...	127.7	...	0.261
4	29.46	...	124.6	...	0.256
5	27.49	29.90	109.1	101.4	0.261	0.294	3.59	...

^a No data collected.

TABLE 4. Regression coefficients and coefficients of determination for linear regression of yield, clusters per vine, mean cluster weight, and pruning weight over disease severity^a

	1991			1992		
	Slope	<i>P</i> value	<i>r</i> ²	Slope	<i>P</i> value	<i>r</i> ²
Vineyard 1						
Yield	-88.9	0.0001	0.55	-104.5	0.0001	0.82
Clusters/vine	-81.5	0.0001	0.59	-96.0	0.0001	0.81
Mean cluster wt	-23.6	NS ^b	0.12	-45.7	0.0001	0.41
Pruning wt	-63.6	0.0001	0.38	-67.4	0.0001	0.46
Vineyard 2						
Yield	-87.8	0.0011	0.67	-100.4	0.0001	0.78
Clusters/vine	-80.5	0.0002	0.64	-88.8	0.0001	0.79
Mean cluster wt	-34.3	NS	0.17	-61.9 ^c	0.0001	0.51
Pruning wt	-29.5	NS	0.06	-64.9 ^c	0.0001	0.34
Vineyard 3						
Yield	-89.0	0.0001	0.77
Clusters/vine	-81.7	0.0002	0.60
Mean cluster wt	-22.3	NS	0.09
Pruning wt
Vineyard 4						
Yield	-93.5	0.0001	0.67
Clusters/vine	-87.7	0.0001	0.66
Mean cluster wt	-28.6	NS	0.11
Pruning wt
Vineyard 5						
Yield	-79.9	0.0001	0.67	-89.8	0.0001	0.80
Clusters/vine	-78.9	0.0001	0.53	-88.1	0.0001	0.74
Mean cluster wt	-4.2	NS	0.01	3.8	NS	0.00
Pruning wt	-42.1	0.0077	0.23

^a Intercepts were standardized to a value of 100%.

^b Not significantly different from zero ($P > 0.05$).

^c The 1992 value was significantly different from that in 1991 ($P \leq 0.05$).

^d No data were collected.

selection of existing plants that are naturally affected with various levels of disease (9). This was the method employed in this study. In this approach, developing a disease assessment system that is related to yield can be troublesome, particularly for canker diseases (2). However, the system used in this study could successfully predict yield reduction. Disease severity may be better related to yield for Eutypa dieback than other canker diseases because Eutypa dieback causes a direct loss of fruiting units (spurs). Yield was well correlated to the number of symptomless spurs per vine in this study. This is somewhat analogous to the reported relationships between yield and healthy leaf area duration (HAD) for several crops (24). However, because of the demonstrated inconsistent relationship between vegetative growth and yield of grapevines, it seems unlikely that HAD would provide a good prediction of yield for grapevines with Eutypa dieback. It also should be noted that the relationship between symptomless spurs and yield would not be expected to hold up at very high spur numbers (1,10,12,13), so any yield prediction based on this variable must consider the optimum number of spurs for the cultivar and site.

In this study, the primary effect of Eutypa dieback was to reduce the number of buds on diseased vines by reducing the number of functional spurs. The resulting decrease in clusters accounted for 88.4 to 98.7% of the yield reduction. Decreased cluster size accounted for less than 12% of the yield reduction. Vegetative growth was less affected by Eutypa dieback than yield.

There was no evidence for yield compensation by healthy shoots on infected vines in this study. Eutypa dieback caused a linear reduction in yield and number of clusters per vine. If compensation had occurred, the expected relationship between disease severity and yield would be a convex curve (8). Compensation might be manifested as more or larger clusters on symptomless spurs on severely diseased vines. The linear relationship between disease severity and clusters per vine indicates that the number of clusters per spur was independent of disease severity. Regression of disease severity on mean cluster weight showed that clusters were actually smaller on severely diseased vines. In general, compensation by grapevines has been documented only when the number of buds was reduced from a very excessive level to a less excessive or normal level (1,10,12,13). The numbers of buds retained on the

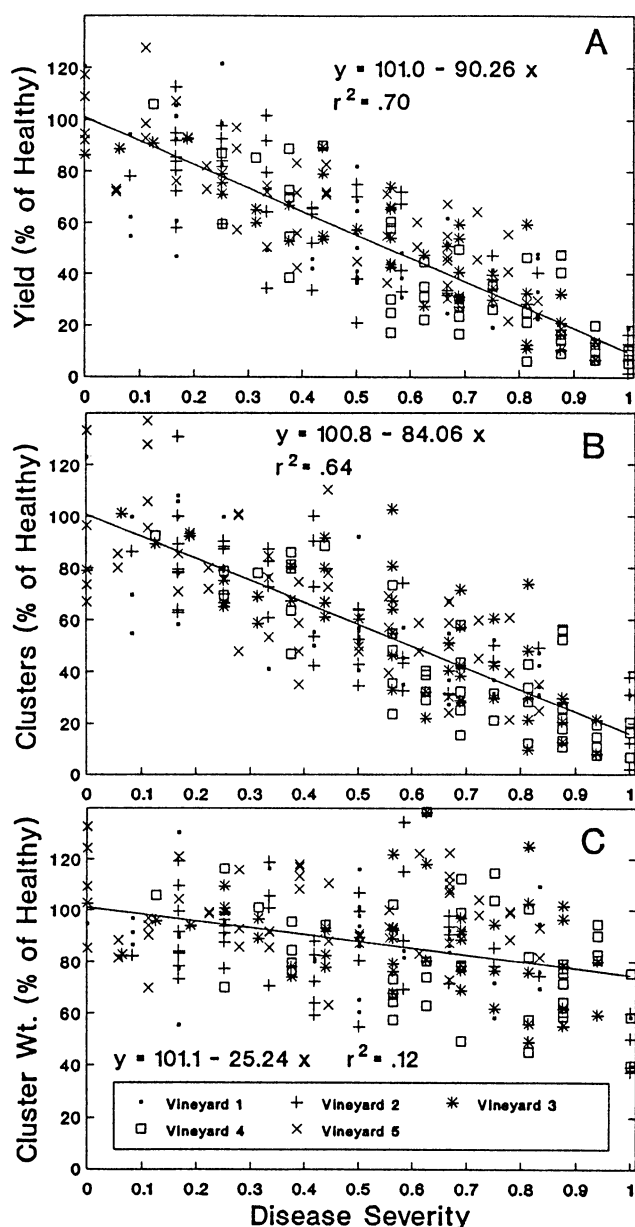


Fig. 1. Regression analyses of the relationship between Eutypa dieback severity and A, yield of individual vines; B, number of clusters per vine; and C, mean cluster weight in 1991. Dependent variables are expressed as a percentage of the estimates for healthy vines.

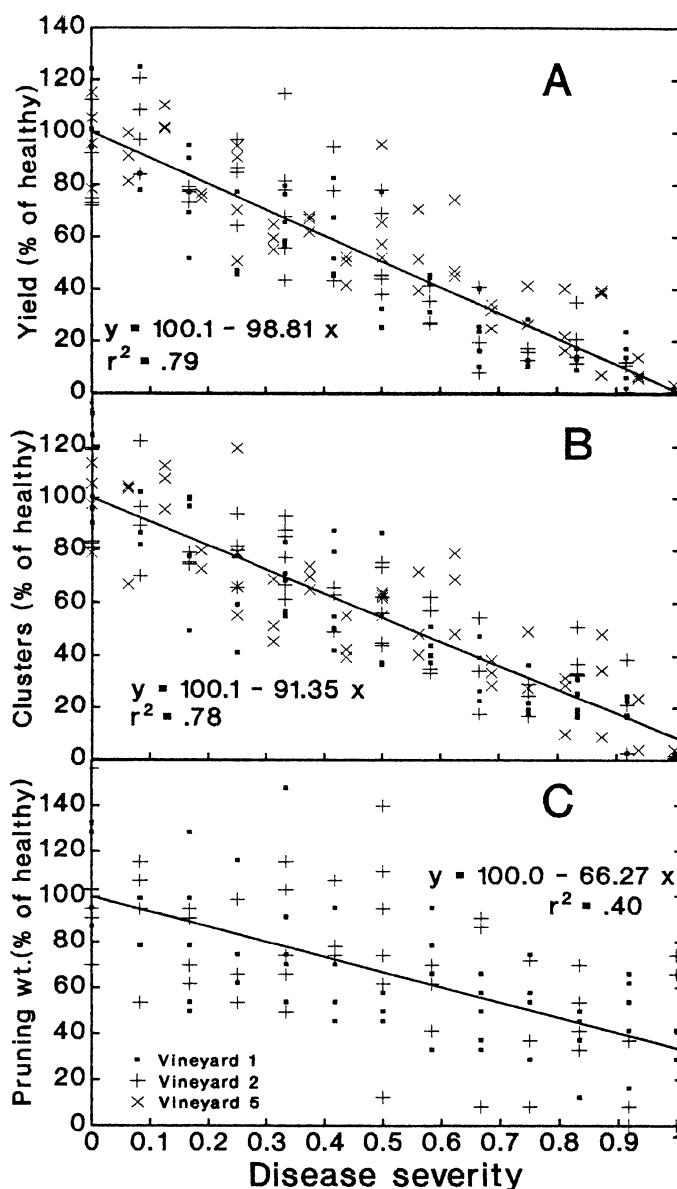


Fig. 2. Regression analyses of the relationship between Eutypa dieback severity and A, yield of individual vines; B, number of clusters per vine; and C, pruning weight per vine in 1992. Dependent variables are expressed as a percentage of the estimates for healthy vines.

TABLE 5. Linear regression intercepts, slopes, coefficients of determination, and *P* values for the effect of disease severity on yield-to-pruning weight ratios

Vineyard	1991				1992			
	Intercept	Slope	<i>r</i> ²	<i>P</i>	Intercept	Slope	<i>r</i> ²	<i>P</i>
1	11.5	-4.3	0.08	0.0737	11.5	-8.6	0.40	0.0001
2	17.2	-13.0	0.26	0.0004	12.4	-8.2	0.19	0.0015
5	8.2	-3.7	0.16	0.0282	... ^a

^a No data were collected.

TABLE 6. Mean disease severity (proportion) and yield losses for five vineyards estimated by models derived from individual vineyards or from combined data

Vineyard	Mean severity		Yield loss (%)			
	1991	1992	Separate model		Combined model	
			1991	1992	1991	1992
1	0.465	0.478	41.3	50.0	42.0	47.2
2	0.444	0.495	39.0	49.7	40.1	48.9
3	0.619	... ^a	55.1	...	55.9	...
4	0.662	...	61.9	...	60.0	...
5	0.405	0.335	32.3	30.1	36.6	33.1

^a No data were collected.

vines in this study were always at or below what would be considered a normal level.

On grapevines, a small portion of the crop is produced from suckers, which are shoots arising from latent buds on the vine trunk and arms. This might be considered a form of compensation. The disease severity rating does not account for effects on sucker growth. These shoots, however, contributed very little to yield. This is reflected in the fact that the slopes of the yield reduction models approached or exceeded -100 (Table 4). This was due to the reduction in mean cluster weight in combination with the reduction in number of clusters and is another indication of the lack of compensation.

Mean cluster weight was significantly reduced in 1992 but not in 1991. This reduction in cluster size of apparently healthy spurs may have been due to phytotoxic fungal metabolites from the infected spurs. The 1992 results support the findings of Johnson and Lunden (9), who reported significant reductions in mean cluster weight due to *Eutypa* dieback. Apparently, some spurs that appeared healthy during the spring suffered a reduction in cluster size. In our study, this effect was smaller in magnitude and less consistent than the reduction in number of clusters per vine. Although there were differences in yield among the vineyards, the effect of *Eutypa* dieback on yield did not depend on vineyard location. Differences in yield among the vineyards were due to factors other than *Eutypa* dieback.

The yield loss estimates reported here (Table 6) are consistent with those reported in Washington State (9). Lower disease severity in 1992 was partially due to the omission of two severely diseased vineyards that were destroyed after the 1991 season. Removal and replacement of diseased spurs in vineyard 5 also contributed to diminished disease severity in 1992. Our models derived from combined data fit as well as the models derived from individual vineyards, and loss estimates were very similar between combined and individual models.

The reduction in vegetative growth due to *Eutypa* dieback was less pronounced than the reduction in yield. Interactions involving the effect of diseased spurs were statistically significant but of little practical importance. Vegetative growth is strongly affected by vine vigor and is not strongly affected by bud number (1,10,12,13). When bud number is low, vegetative growth is maintained by suckers arising from latent buds on the trunk of the vine. These suckers are less fruitful than shoots arising from the previous year's buds. Extensive suckering leads to an undesirable ratio of yield to vegetative growth.

Eutypa dieback not only reduces yields but may affect wine quality. The quality of wine grapes is affected by the balance

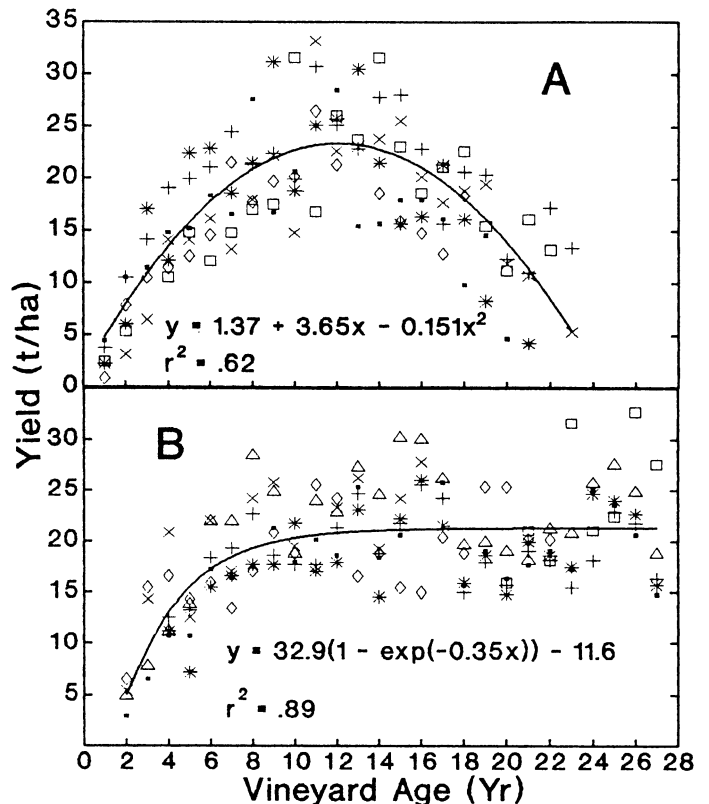


Fig. 3. Relationship between vineyard age and yield for A, six Chenin blanc vineyards and B, eight Barbera vineyards in Merced County, CA. Symbols represent the different vineyards. Data are from 1964 to 1990.

between crop size and amount of vegetative growth, measured by the ratio of yield to pruning weight. High yield-to-pruning weight ratios are associated with delayed maturity, while low ratios are associated with undesirable wine flavors (1,10,12,13,25). The optimum ratio depends on cultivar, rootstock, training system, and location. It is also desirable to achieve a uniform yield-to-pruning weight ratio within a vineyard to promote even maturity and juice quality. The differential effect of *Eutypa* dieback on yield versus pruning weight caused a reduction in the yield-to-pruning weight ratios on infected vines (Table 5). Variability in this ratio within a vineyard could result in uneven berry maturity and a reduction in wine quality.

Results of the canker isolations confirmed that *E. lata* was the only canker pathogen associated with dieback symptoms in these vineyards. Stunted and distorted shoot symptoms of *Eutypa* dieback are not caused by any other pathogen. Dead spur symptoms have been attributed to other fungi, such as *Phomopsis viticola* and *Botryodiplodia theobromae*, but their pathogenicity has not been shown (11,17). Regardless of their pathogenicity, no other purported canker fungi were associated with the presence of dieback symptoms in the vineyards included in this study (Table 1).

Yield versus vineyard age. The destructive potential of *Eutypa* dieback was demonstrated by the relationship between age and yield of Chenin blanc vineyards; in some vineyards over 20 yr old, yield had declined by as much as 83% compared with their

peak years. The decrease in yield that occurs after these vineyards reach approximately 12 yr of age is associated with the period of rapid increase of *Eutypa* dieback. In Merced County Chenin blanc vineyards, *Eutypa* dieback is rare until the vineyards reach 10–12 yr of age. Between 12 and 20 yr of age, disease increases each year, until nearly all vines are infected at 20 yr (5). Healthy vineyards generally experience increased yields up to 10 yr of age and subsequently maintain a stable yield for several decades. This is demonstrated by the Barbera vineyards, which are rarely affected by *Eutypa* dieback (Fig. 3).

It is generally recognized that *Eutypa* dieback can be a costly disease because of the expense of vine removal, replanting or re-grafting, and the delay in productivity of new vines, but there is little recognition of the magnitude of yield losses that can occur. The cumulative yield loss over the life of the infected vineyards represents a very substantial monetary loss. The results of this research show that substantial yield reduction occurs annually due to *Eutypa* dieback, even on vines with relatively low disease severity. The reduction in yield of individual vines is reflected in yield losses at the vineyard level (Table 6, Fig. 3). The economic cost of this yield reduction may justify a change in management practices toward more diligent corrective pruning to maintain a higher number of symptomless spurs.

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