A Study of Mineral Nutrition Relationships of Waterberry in Thompson Seedless

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A four-year study was conducted to uncover differences in tissue mineral levels related to waterberry (WB) in Thompson Seedless grapevines and to attempt to alleviate the condition. Clusters were dipped into nutrient solutions with no success in decreasing symptoms. The main effect was an increase in symptoms with the diammonium phosphate treatment. Vineyards of varying WB incidence were surveyed, and differences in mineral content of petiole and rachis tissue were noted and correlated with waterberry incidence. Both petiole and rachis levels of total N and NH_4-N correlated closely with WB incidence, and K rachis levels tended to be lower in the WB symptom samples. Ca and Mg levels did not vary with incidence. In three years of N, P, Ca, and Mg fertilizer trials in vineyards of varying WB susceptibility, the following effects were noted: N and P petiole levels generally increased with N and P treatments, respectively; petiole K was significantly reduced in one location, apparently as an effect of N fertilization, and Ca and Mg treatments did not consistently increase petiole levels of Ca and Mg. None of the fertilizer treatments decreased WB. However, N fertilizer increased WB in some locations, particularly those with histories of low WB incidence and low N fertilization. Regression analysis of all Thompson Seedless rachis mineral analyses, 1979-1982, showed significant correlation only between total N and NH_4-N levels and the occurrence of symptoms. WB symptoms were observed at total N levels of approximately 1.5% and higher and NH_4-N levels of 3000 ppm and above in rachis tissue.

Waterberry is a well-known disorder if grapes in California, especially in certain cultivars. It is especially troublesome to table grape growers whose product is destined for the fresh fruit market (5,6,8,34). However, the problem received little attention by researchers in California until the publication of work on a similar disorder in Europe in the early 1970's.

In Europe, the nomenclature applied to their disorder is descriptive of cluster stem symptoms. Thus, the name "Stiellähme" (Germany, 19,23), "desséchement de la rafle" (France, 28,33), and "disseccamento del rachide" (Italy, 13) appear in the literature. Other descriptive names include "bunch stem die-back" (Australia, 20), "palo negro" (Chile, 6,10), and "shanking" (New Zealand).

In California, the condition of cluster stem necrosis thus described (24,25), is called waterberry. Waterberry refers to the watery, soft, and flabby berries resulting from the interrupted flow of sugars and other ripening constituents into the berries due to the stem necrosis. While the location of the first cluster stem symptoms and their progression to other cluster parts may differ, symptom descriptions are similar (6,8,18,19,23,24). The earliest visual symptom is the development of small (1-2 mm) dark spots on the pedicels and/or other parts of the cluster framework. These spots become necrotic, slightly sunken, and expand to affect larger areas. Various workers report that symptoms may first develop in the primary, secondary, or tertiary portions of the cluster stem structure, depending on cultivar and severity but that usually the cluster tips, shoulders, and upper laterals are most affected (4,6,10,15,16,18,19,23,24,31). The brown-to-black color of the affected cluster stem tissue develops during the berry ripening period and increases in intensity to dark coffee, purplish-black or black (2,5,6,10,15,18, 23,24,31). The dark coloration is attributed to concentrations of oxidized polyphenols (31) and can vary with cultivar (23,31).

As in California, the affected berries are described as metallic, opaque, or dull green in white grapes and red to dark blue in black grapes (5,18,33). As their compositions are affected by the interrupted flow of sugars and other ripening constituents through the cluster stem structure (6,33), the berries become flaccid, wrinkled, withered, and of soft texture (5,6,24,34). In time many of them shrivel and dry completely at a rate dependent upon the extent of stem necrosis and temperature (8,9,24).

Differences in cultivar susceptibility have also been noted by many investigators (5,6,12,18). Some rootstocks, such as Kober 5BB and 125AA which are higher in vigor, contribute to a higher incidence (5,18,23,24,30).

Numerous environmental and cultural factors, such as soil type and fertility, soil moisture conditions, foliage and root damage, vigor, crop levels, and ambient seasonal temperatures (7,21), have been implicated as causes or contributing factors. However, some of the implications reported are contradictory.

In the absence of a pathological cause (4,30), WB is now generally referred to in the literature as a physiological disorder, the two most prevalent theories invovling hormonal or nutritional imbalances (1,7,12,13,19,28,31). The hormonal or auxin theory predicates the existence of an unfavorable auxin gradient between the vegetative and fruiting parts of the vines (15,19,23,30,31) as the auxin level in the clusters decreases at the beginning of ripening. It is suggested that the proportionally higher auxin levels toward the shoot apices cause enzymatic breakdown of the middle lamella of cells in the cluster stems (19,30). Applications of auxin-like compounds and growth regulators have produced inconsistent effects as noted in comparing the results obtained by various European researchers (1,6,13,18,19,22,30,31). In California, treatments with growth regulators have been ineffective in reducing WB symptoms (8,9).

European workers have found lower Ca and Mg levels associated with symptoms and affected tissues and have

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reduced symptoms with Ca and Mg soil and foliar spray treatments (11,13,15,32). They cite the importance of Ca and Mg in cell wall formation and the intergrity of the middle lamella (10,19). Thus, the ratios K/Ca, K/Mg, and K/Ca and Mg are generally considered very important by the researchers (5,11,15,17,18,28,29,31,35), a high ratio of K to Ca and Mg being associated with the disorder.

Much of their work has centered on applications of Ca and Mg of varying concentrations (most commonly $CaCl_2$, $MgCl_2$, and $MgSO_4$ sprays) with varying degrees of success. In some cases, symptom incidence in clusters dipped in $MgSO_4$ solutions (5% as MgO) was < 10% of that in untreated clusters (1,3,5,12,14,16,18,19,27). Response to soil treatments was slower than to sprays (10).

In 1974, reports on these studies prompted us to evaluate the effects of Ca and Mg treatments on the WB problem in California (8,9). Thompson Seedless and Calmeria vineyards with histories of WB were treated with Ca and Mg salts and chelates in repeated foliar sprays and with concentrated placement of high rates in the soil. Vine tissue levels of Ca and Mg increased, but there were no reductions of symptoms in the fruit. In fact, there were significant increases in WB with the $Ca(NO_3)_2$ and $Mg(NO_3)_2$ foliar sprays.

Our work was resumed in 1979 after preliminary cluster rachis tissue analysis studies showed a relationship between N levels and the occurrence of symptoms. The ensuing investigations included evaluations of N, P, K, Ca, and Mg treatments in high and low incidence vineyards in conjunction with tissue analysis comparisons.

Materials and Methods

Three approaches were taken to determine mineral nutrition relationships of WB: nutrient solution cluster dip treatments, 1979-1980; vineyard tissue analysis survey, 1980; and vineyard fertilization trials, 1980-82.

Nutrient solution cluster dip treatments, 1979 and 1980: Cluster dipping treatments were initiated in 1978 by comparing various concentrations of nutrient salts for possible phytotoxicity effects. This made possible the application of high but safe concentrations of N, P, K, Ca, and Mg salts in 1979 and 1980.

The 1979 dips were compared in a Reedley area Thompson Seedless vineyard with a history of high WB incidence. Each cluster was dipped on two dates — 10 and 17 July at early (1% berries soft) and late (85% berries soft) véraison. Five nutrient solution treatments were compared in a randomized complete block design, as follows: 1) calcium sulfate ($CaSO_4$), saturated solution, 29.3 meq/L; 2) calcium nitrate [$Ca(NO_3)_2$], 1% by weight; 3) magnesium sulfate ($MgSO_4$), 5% MgO; 4) diammonium phosphate [$(NH_4)_2HPO_4$], 2%; 5) ammonium sulfate [$(NH_4)_2SO_4$], 1%; 6) check, untreated.

Each of the 32 single-vine replicates contained six individually-tagged data clusters. These clusters were selected for uniformity of size and from shoots of average vigor arising from nodes near the ends of fruiting canes on the same vines. Previous studies have shown higher incidence of WB on shoots arising from near the apices of fruiting canes (author's unpublished data). Each of the five clusters per replicate received one of the dip treatments, and the sixth cluster served as an untreated control. Thus, each dip treatment was repeated on 32 individual clusters.

The surfactant Triton B-1956 was added to all dipping solutions at 0.03% (v/v). The entire cluster was immersed at each dipping. On 3 August, the percent of berries affected was determined by visual estimate in each cluster.

The 1980 treatments included the timing of single dips as well as repeated dips in a Selma area Thompson Seedless vineyard. Clusters were dipped on 7 and 17 July, early (2% berries soft) and late (90% berries soft) véraison, respectively. These dates were chosen for their similarity to those in the aforementioned reported work, predating the onset of symptoms. The trial was designed as a split plot with the dates of treatment as the main plots and the type of dip in single-vine subplots. There was a total of 18 treatments, dipping treatments (5 solutions and an untreated control), as follows: on 7 July, on 17 July, and on both dates combined. There were twenty single-vine replications. Six clusters were tagged and treated individually on each vine as in the 1979 work.

For the 1980 dipping treatments, the solution concentrations were adjusted as follows: 1) calcium chloride ($CaCl_2$), 1% by weight; 2) magnesium sulfate ($MgSO_4$), 5% MgO; 3) potassium sulfate (K_2SO_4), 5%; 4) diammonium phosphate [$(NH_4)_2HPO_4$], 2%; 5) ammonium hydroxide (NH_4OH), 1%; 6) check, untreated.

Calcium chloride replaced the $CaSO_4$ dip used in 1979 because of its higher solubility. A K source (K_2SO_4), and an ammonium source (NH_4OH) not containing PO_4 were added. Triton B-1956 at 0.03% was added to all dipping solutions as in 1979.

Readings on the percent WB in each cluster were taken on 11 August.

Tissue analysis survey, 1980: Thirteen commercial Thompson Seedless vineyards with areas of WB history were each sampled in locations of high and low WB incidence to obtain plant tissue nutrient level differences in the petioles and cluster stem framework. Five different samples were taken in each vineyard as follows.

Low WB incidence area: 1) recently mature leaf petioles; 2) normal clusters.

High WB incidence area: 3) recently mature leaf petioles; 4) normal clusters; 5) symptom clusters.

Sampling was conducted during the early to midstages of cluster stem necrosis symptom development between 29 July and 11 August. Each sample consisted of 75 to 100 petioles or 15 clusters. The cluster laterals and berries were clipped off, leaving the rachis for laboratory analysis. The rachis was used for cluster stem analysis because of its larger amount of tissue as compared to the pedicels. Preliminary cluster tissue analysis comparisons in 1978 showed a very high correlation between the cluster rachis and pedicel total N levels (r = .98), although the pedicel levels were in a lower range than those of the rachis. The same was true when comparing rachis and pedicel NH_4- N levels (r = .97). Therefore, the rachis alone was regarded as sufficiently representative of N compound levels in the cluster stem framework.

The samples were rinsed in distilled H_2O, dried at 42°C (a temperature chosen to minimize the loss of volatile amines and to avoid carmelization of sugars), and analyzed as follows: 1) Total N — standard Kjeldahl digestion distillation; 2) NO_3-N — specific ion electrode on an aqueous extract; 3) NH_4-N — 1 *M* KCl extract with Kjeldahl distillation using MgO; 4) P — oxidation of organic P to PO_4, followed by the mixed vanadate reagent method of PO_4-P analysis; 5) K — emission spectrophotometry on a 2% acetic acid extract; 6) Ca and Mg — ashed samples by atomic absorption spectrophotometry.

Vineyard fertilization trials, 1980-1982: Seven vineyard trials were conducted to determine if the incidence and severity of WB symptoms could be influenced by fertilizer treatments. Two series of trials were established in commercial Thompson Seedless vineyards as follows.

Fertilizer Experiment 1, 1980-1982 (3 WB susceptible vineyards): Three separate trials were established in 1980 with identical fertilizer treatments as follows:

Trial Locations

1. Reedley. Moderate WB history, Hanford fine sandy loam soil, high vine vigor.
2. Selma #1. Moderately high WB history, Delhi loamy sand soil, moderate vine vigor.
3. Selma #2. High WB history, Hesperia sandy loam soil, high vine vigor.

Fertilizer Treatments

1. N — 135 kg/ha N (392 kg/ha ammonium nitrate) in 1980, 1981, and 1982.
2. P — 470 kg/ha P (2242 kg/ha) triple super phosphate [$Ca(H_2PO_4)_2$] in 1980 only.
3. N + P — Combination of above (same years).
4. Ca — 2185 kg/ha Ca [11 210 kg/ha $CaSO_4$ (84% Ca)] in 1980 + 1311 kg/ha Ca (6726 kg/ha $CaSO_4$) in 1982.
5. Mg — 280 kg/ha Mg (2914 kg/ha $MgSO_4$) in 1980 + 168 kg/ha Mg (1748 kg/ha $MgSO_4$) in 1982.
6. None — 0

All of the materials were applied in February. They were banded in furrows on each side of the vine rows, covered with soil, and irrigated in. The four-vine plots were replicated six times in a randomized complete block design. Guard rows separated treated rows.

Bloomtime opposite cluster petiole samples were collected from each plot each year for laboratory analysis. Five symptom and five normal clusters were also taken from each check, untreated plot, and N-treated plot in August and composited according to treatment for each location. The cluster laterals and berries were clipped off, leaving the rachis for laboratory analysis as in the survey work.

The samples were rinsed in distilled H_2O, dried at 40°C, and analyzed for total N, NO_3-N, NH_4- N, P, K, Ca, and Mg.

Visual readings on the percent of WB symptom berries in each cluster on the data vines were made in early August and averaged for each plot.

Fertilizer Experiment 2, 1981-82 (low vs. high incidence WB vineyard areas): Four trials were established to directly compare adjoining low and high WB incidence Thompson Seedless vineyard areas as follows:

Trial Locations

1. Monmouth District: Low incidence vineyard on Delhi loamy sand with history of modest fertilization — steer manure once every three years.

High incidence vineyard on Hesperia sandy loam under a high fertilization program — high rates of manure at least every other year, plus commercial N fertilizer annually.

2. Easton District: Low incidence vineyard on Delhi loamy sand of low N fertilization usage.

High incidence vineyard of very high vigor on Hesperia sandy loam with same fertilizer program as low incidence vineyard.

Fertilizer Treatments

1) N, 2) P, 3) N + P, 4) None - 0. Rates, fertilizer sources, timing, and application methods the same as in Fertilizer Experiment 1.

Bloomtime petiole samples and early August cluster rachis samples were taken for laboratory analysis as in Fertilizer Experiment 1. Percent WB readings of each cluster were also taken in the same manner as in Fertilizer Experiment 1.

All of the replicated trial data were analyzed for statistical significance by two-way analysis of variance and Duncan's multiple range test.

Results and Discussion

The WB readings in these trials will be expressed as follows: Waterberry incidence — a calculation of the percent clusters with WB symptoms (number of clusters

Table 1. Waterberry severity in cluster dipping treatments, 1979 and 1980.

1979

Dipping compounds	Mean % berries with WB symptoms
Calcium sulfate	9.9 b[1]
Calcium nitrate	10.0 b
Magnesium sulfate	5.8 b
Diammonium phosphate	18.2a
Ammonium sulfate	8.6 b
Check, untreated	10.3 b

1980

	Mean % berries with WB symptoms		
	7 July dip	17 July dip	both dips
Calcium chloride	8.7 b	17.1ab	15.1[1]
Magnesium sulfate	11.0 b	13.9 b	10.5 b
Potassium sulfate	12.1 b	17.3ab	8.8 b
Diammonium phosphate	15.3ab	22.0a	27.0a
Ammonium hydroxide	20.1a	13.1 b	15.6 b
Check, untreated	13.4ab	12.1 b	11.6 b

[1]Mean separation within columns by Duncan's multiple range test, .05.

Table 2. Recently mature leaf petiole laboratory analysis, 1980 waterberry survey vineyards (means of 13 vineyard locations, dry wt. basis).

	Total-N %	NO_3-N ppm	NH_4-N ppm	P %	K %	Ca %	Mg %	Ratio K:Ca + Mg
Low incidence areas	0.84 b[1]	646 b	555a	0.39a	0.87a	2.32a	1.36a	.24a
High incidence areas	0.97a	1088a	784a	0.38a	0.88a	2.36a	1.38a	.24a

[1]Means separation within columns by Duncan's multiple range test, .05.

showing WB symptoms ÷ total number of cluster examined) × 100; Waterberry severity — the mean percent of affected berries in all of the clusters per lot or treatment (total WB readings of all clusters ÷ total number of clusters examined).

Nutrient solution cluster dip treatment — 1979 and 1980: None of the dipping treatments reduced WB in either year (Table 1). Thus, inexplicably, we are not able to duplicate the results of the aforementioned European studies in which cluster dips of varying concentrations of $CaSO_4$ and $MgSO_4$ reduced WB symptoms (1,3,5,12,14,16,18,19,27). Note that the diammonium phosphate dip significantly increased WB in both years (Table 1). This effect, coupled with data from a previous study that $Ca(NO_3)_2$ and $Mg(NO_3)_2$ foliar sprays had also increased WB symptoms (8), implicated N with higher WB. This led to expansion of this investigation to include N treatments.

Vineyard tissue analysis survey — 1980: The petiole analyses show the high-incidence areas to be significantly higher in both total N and NO_3-N than the low incidence areas (Table 2). None of the other nutritional elements (P, K, Ca, and Mg) show differences between high and low WB incidence areas. Again, this differs from previous reports of a direct relationship between K and Ca and/or Mg in petioles and manifestation of WB symptoms (11,13,15,32).

Rachis tissue frequently shows symptoms of WB and, therefore, was used for chemical analysis comparisons. The rachis analyses in Table 3 show a direct relationship between total N and NH_4-N tissue levels and WB incidence. There were no significant differences in levels of NO_3-N, P, Ca, and Mg levels between normal and symptom clusters. Thus, the correlation between samples and N levels is manifested with the reduced forms of N and not with NO_3-N. Again, differences in Ca and Mg levels between apparently healthy and WB cluster rachises did not differ significantly.

However, K levels were significantly lower in the WB symptom cluster rachises than in those of normal clusters. Because of the relative stability of Ca and Mg levels, the reduced K concentrations lowered the values of the

Table 3. Cluster rachis laboratory analysis, 1980 waterberry survey vineyards (means of 13 vineyard locations, dry wt. basis).

Description	Total-N %	NO_3-N ppm	NH_4-N ppm	P %	K %	Ca %	Mg %	Ratio K:Ca + Mg
Low incidence areas								
Normal clusters	1.27 c[1]	554a	1498 c	0.37a	2.15a	1.09a	0.34a	1.50a
High incidence areas								
Normal clusters	1.85 b	550a	2581 b	0.37a	2.10a	0.90a	0.28a	1.78a
Symptom clusters	2.50a	558a	4627a	0.37a	1.33 b	0.93a	0.28a	1.10 b

[1]Means separation within columns by Duncan's multiple range test, .05.

Table 4. Fertilizer Experiment 1: Bloomtime petiole analysis (3-year means, 1980-82, dry wt. basis).

Location	Fertilizer[1] treatment	Total-N %	NO_3-N	P %	K %	Ca %	Mg %	K:Ca + Mg
Reedley	none-0	1.24 b[2]	1600 b	.62 b	1.38a	1.20 b	.68a	.73a
	N	1.42a	3128a	.61 b	1.43a	1.27ab	.70a	.73a
	P	1.28 b	1894 b	.67ab	1.73a	1.27ab	.69a	.88a
	N + P	1.61a	3517a	.73a	1.47a	1.31ab	.78a	.70a
	Ca	1.34 b	2028 b	.66ab	1.69a	1.38a	.66a	.82a
	Mg	1.24 b	2058 b	.62 b	1.44a	1.33ab	.71a	.71a
Selma #1	none-0	0.93a	575 b	.26 b	2.30a	1.05a	.58a	1.41a
	N	0.96a	800a	.27 b	2.45a	0.94a	.54a	1.66a
	P	0.90a	675ab	.35a	2.02a	1.15a	.60a	1.15a
	N + P	0.97a	750ab	.35a	2.00a	1.02a	.57a	1.26a
	Ca	0.91a	675ab	.26 b	2.05a	1.09a	.58a	1.23a
	Mg	0.94a	625 b	.27 b	1.90a	1.02a	.59a	1.18a
Selma #2	none-0	1.20a	1400ab	.37 b	2.45a	1.04a	.63a	1.47a
	N	1.17a	1650a	.37 b	2.50a	1.07a	.65a	1.45a
	P	1.14a	1275 b	.53a	2.60a	0.94a	.65a	1.64a
	N + P	1.13a	1550ab	.48a	2.75a	1.05a	.63a	1.64a
	Ca	1.15a	1200 b	.28 b	2.10a	1.08a	.62a	1.24a
	Mg	1.13a	1450ab	.39 b	2.75a	1.02a	.66a	1.64a

[1]Means separation within columns by Duncan's multiple range test, .05.
Each location analyzed separately.
[2]Refer to **Materials and Methods** for explanation of treatments.

ratio K:Ca + Mg significantly in the symptom cluster rachis samples. This is inverse to the reports of several European studies in which lower Ca and Mg levels (11,13,15,32) increased the values of the ratio K:Ca + Mg (5,11,15,17,18,28,29,31,35). These dissimilar results were unexpected and, at this point, are unexplainable.

Vineyard fertilization trials, 1980-82: *Fertilizer Experiment 1.* In Table 4 the means of three years of bloomtime petiole analyses show significantly higher total N levels from N treatment in the Reedley location only. However, the NO_3-N levels reveal a significantly higher vine N status from the N treatment in all three locations and from the N + P treatment in Reedley. P levels were also significantly increased from the P and NP treatments in all locations with the exception of the P only treatment at Reedley. The Ca treatment was less effective in influencing petiole levels significantly, with higher Ca in the Reedley location only. The Mg treatment did not increase Mg levels in any location.

The cluster WB readings in Table 5 represent the percent of clusters with symptoms (incidence) and, in Table 6, the percent of affected berries in all clusters (severity). The incidence (Table 5) was significantly increased by the N and/or N + P treatments in two locations in one year — 1980. The mean for all three locations was also significantly greater in the N + P treatment in 1980. The P, Ca, and Mg treatments had no effect in any location. The 1981 and 1982 data are not shown due to lack of treatment differences.

At Reedley, the severity of WB symptoms (Table 6) was increased by application of N in 1981 and N + P in 1982. None of the fertilizer treatments decreased WB severity as compared to control.

It should be noted that 1981 was a year of very low WB incidence in Thompson Seedless in the San Joaquin Valley, even in vineyards with a history of WB problems. This can be seen in the very low readings in all locations in 1981.

Fertilizer Experiment 2: The bloomtime petiole analysis in Table 7 shows the high N status with most of the N treatments. While the greatest increase in NO_3-N from fertilization with N appeared in the N-deficient, low-incidence vineyard at Easton, the high-incidence trial areas at each location also had higher NO_3-N levels than the low-incidence areas.

The petiole P levels were significantly increased with most of the fertilizer treatments containing P. Petiole K

Table 5. Fertilizer Experiment 1: Percent clusters with waterberry symptoms (incidence).

	1980[3]			
Treatment[2]	Reedley	Selma #1	Selma #2	Mean, 3 locations
none-0	20.2 c	66.2ab[1]	38.8a	41.8 b
N	40.0a	56.7 b	39.7a	45.4 b
P	26.4 bc	62.4ab	39.7a	42.9 b
N + P	35.9ab	72.4a	46.2a	51.5a
Ca	29.6abc	56.9 b	40.7a	42.4 b
Mg	18.9 c	67.1ab	37.1a	41.0 b

[1]Mean separation by Duncan's multiple range test; like letters within a column are not significantly different at the 5% level.
[2]Refer to **Materials and Methods** for explanation of treatment.
[3]1981 and 1982 data omitted as no differences were shown.

Table 6. Fertilizer Experiment 1: Mean percent berries with waterberry symptoms (severity).

Fertilizer[2]	1980				1981				1982			
treatment	Reedley	Selma #1	Selma #2	Mean, 3 locations	Reedley	Selma #1	Selma #2	Mean, 3 locations	Reedley	Selma #1	Selma #2	Mean 3 locations
none-0	3.4a	13.4a[1]	10.0a	8.9a	0.80 bc	0.16a	0.15a	0.37 b	3.43 b	1.83a	0.37a	1.88a
N	5.3a	9.1a	7.4a	7.3a	2.20a	0.04a	0.14a	0.80a	4.52 b	1.44a	0.18a	2.05a
P	2.5a	11.7a	7.1a	7.1a	1.45 b	0.11a	0.10a	0.55ab	4.75 b	2.80a	0.45a	2.66a
N + P	4.1a	11.1a	5.6a	6.9a	0.69 bc	0.06a	0.21a	0.37 b	7.00a	1.13a	0.27a	2.80a
Ca	3.3a	8.5a	8.2a	6.7a	1.02 b	0.11a	0.02a	0.38 b	4.72 b	1.62a	0.30a	2.22a
Mg	2.6a	10.9a	5.8a	6.4a	0.14 c	0.15a	0.10a	0.13 b	5.15 b	1.97a	0.05a	2.39a

[1]Mean separation by Duncan's multiple range test; like letters within a column are not significantly different at the 5% level.
[2]Refer to **Materials and Methods** for explanation of treatments.

Table 7. Fertilizer Experiment 2: 1981-82 mean bloomtime petiole analysis (low vs. high incidence trial locations).

Location	Fertilizer treatment	Total-N %	NO_3-N ppm	P %	K %	Ca %	Mg %
Monmouth	0-none	1.00 b[1]	950 ab	0.30 b	1.85a	1.30a	0.61a
Low-	N	1.56a	950ab	0.33ab	1.85a	1.35a	0.59a
incidence	P	1.00 b	825 b	0.39a	1.85a	1.45a	0.64a
	N + P	1.60a	1075a	0.35ab	1.85a	1.38a	0.61a
High	0-none	1.21 b	2050 b	0.39 b	2.10a	1.30a	0.58a
incidence	N	1.96a	2475ab	0.40 b	2.13a	1.25a	0.60a
	P	1.27 b	2625a	0.53a	2.28a	1.28a	0.61a
	N + P	1.29 b	2600a	0.53a	2.10a	1.28a	0.61a
Easton	0-none	0.78a	288 b	0.42ab	1.85ab	1.17a	0.74a
Low-	N	0.85a	825a	0.29 b	1.85ab	1.15a	0.77a
incidence	P	0.76a	288 b	0.50a	2.05a	1.16a	0.74a
	N + P	0.87a	613a	0.44a	1.63 b	1.18a	0.78a
High	0-none	0.99 b	850 b	0.35ab	1.35a	1.18a	0.59a
incidence	N	1.11a	1188a	0.31 b	1.23a	1.03a	0.59a
	P	0.98 b	725 b	0.40a	1.31a	1.13a	0.59a
	N + P	1.09a	1238a	0.33ab	1.29a	1.20a	0.62a

[1]Mean separation within columns by Duncan's multiple range test, .05. Each of four trial areas analyzed separately.

Table 8. Fertilizer Experiment 2: Percent clusters with waterberry symptoms (incidence).

Fertilizer[2] treatment	1981 Monmouth low	1981 Monmouth high	1981 Easton low	1981 Easton high	1982 Monmouth low	1982 Monmouth high	1982 Easton low	1982 Easton high	Mean of locations and years low	Mean of locations and years high
0-none	1.2a[1]	36.3a	0.3a	10.7a	38.7a	63.5a	68.9ab	46.6a	27.3 b	39.3a
N	2.2a	40.4a	14.1 b	9.7a	45.4a	65.5a	80.8a	54.1a	35.6a	42.4a
P	2.1a	30.7a	1.2a	12.0a	45.9a	63.2a	59.1 b	48.6a	27.1 b	38.6a
N + P	3.2a	27.4a	12.6 b	16.1a	41.4a	71.7a	79.3a	54.9a	34.9a	42.5a

[1]Mean separation by Duncan's multiple range test; like letters within a column are not significantly different at the 5% level.
[2]Refer to **Materials and Methods** for explanation of fertilizer treatment.

Table 9. Fertilizer Experiment 2: Mean percent berries with waterberry symptoms (severity).

Fertilizer[2] treatment	1981 Monmouth low	1981 Monmouth high	1981 Easton low	1981 Easton high	1982 Monmouth low	1982 Monmouth high	1982 Easton low	1982 Easton high	Mean of locations and years low	Mean of locations and years high
none-0	0.19a[1]	2.04a	0.01 b	1.53a	4.2a	11.7a	20.2 b	9.3a	6.2 b	6.1a
N	0.66a	2.20a	1.19a	1.27a	6.7a	12.2a	34.4a	12.6a	10.7a	7.1a
P	0.10a	1.16a	0.02 b	1.14a	6.2a	11.6a	14.4 b	9.0a	5.2 b	5.7a
N + P	0.77a	2.36a	0.81a	1.49a	5.9a	13.8a	24.2ab	11.4a	7.9ab	7.2a

[1]Mean separation by Duncan's multiple range test; like letters within a column are not significantly different at the 5% level.
[2]Refer to **Materials and Methods** for explanation of fertilizer treatment.

levels were significantly affected only in the Easton low-incidence area, where in the N + P treatment there was a reduction in K as compared to the P treatment. This suggests N as the possible cause of the reduction in K, an effect which is occasionally encountered in fertilizer trials. Only the Ca and Mg levels were unaffected by fertilizer treatment.

WB symptom data are given in Tables 8 and 9. The incidence (Table 8) and severity (Table 9) of WB increased with N and N + P treatments in the low-incidence Easton vineyard. The Monmouth vineyard was unaffected by fertilizer treatment. However, the overall means of the low-incidence areas of both vineyards show a significant increase in WB from the N and N + P treatments. There were no fertilizer treatment effects on WB in the high-incidence areas or from P applications in either of the trial locations. Thus, of the variables measured, N fertilizer alone appears to have influenced WB symptoms and only in the low-incidence, lower N status vineyard.

Table 10 shows cluster rachis laboratory analyses from untreated and N-only treated vines and the analysis of variance of the results of all of the seven trial locations during the years of study. The data from the high- and low-incidence trial locations are not statistically analyzed as the samples were composited by treatment for laboratory analysis.

The statistically analyzed data reveal several significant differences in the chemical constituents of the rachis related to the presence of WB symptoms. Symptom clusters have higher total N levels in the non-fertilized vines; ammonium-N levels were higher in both N fertilized and non-fertilized vines, whereas K levels were lower. This may illustrate competition or antagonism between K and NH_4 ions in grape tissues as previously reported in other crops (27).

P levels are significantly lower, and the Ca levels are higher in the N-treated symptom clusters as compared to

Table 10. Fertilizer Experiments 1 and 2: 1982 Cluster rachis analysis. Means of high- and low-incidence trial comparisons and all 7 trial locations of Experiments 1 and 2.

Trial locations	Fertilizer[2] treatment	Cluster description	Total-N %	NO_3-N ppm	NH_4-N ppm	P %	K %	Ca %	Mg %	Ratio K:Ca + Mg
High incidence	0-None	normal	1.74	750	2488	0.33	2.90	0.60	0.19	3.67
		sympton	2.28	600	4400	0.22	1.01	0.76	0.19	1.06
	N	normal	2.22	900	3325	0.34	2.65	0.60	0.18	3.98
		symptom	2.09	775	4925	0.21	0.92	0.77	0.23	.92
Low incidence	0-none	normal	1.48	475	1813	0.28	2.45	0.60	0.22	2.99
		symptom	1.55	425	2450	0.24	1.15	0.69	0.23	1.25
	N	normal	1.18	775	2488	0.24	1.40	0.69	0.28	1.44
		symptom	1.83	600	3450	0.19	0.79	0.72	0.21	.85
All 7 trial locations	0-none	normal	1.77 b	[1]550a	2575 b	0.38a	2.70a	0.61 b	0.18a	3.42a
		symptom	2.25a	525a	4117a	0.32ab	1.24 b	0.69ab	0.19a	1.41 b
	N	normal	2.02ab	692a	3088 b	0.34ab	2.22a	0.63ab	0.19a	2.71a
		symptom	2.19a	667a	4383a	0.28 b	1.12 b	0.73a	0.28a	1.11 b

[1]Mean separation within columns by Duncan's multiple range test, .05.
[2]Refer to **Materials and Methods** for explanation of fertilizer treatment.

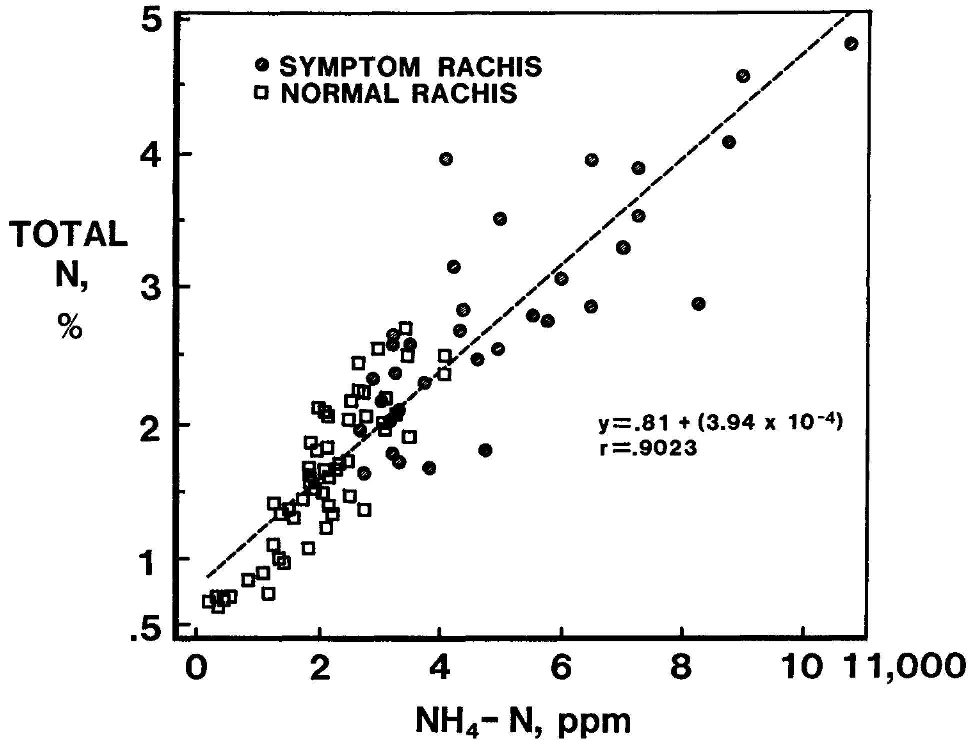

Fig. 1. Relationship between total N and NH_4-N in normal and symptom rachis tissue.

normal clusters with no N treatment. The ratio of the K:Ca + Mg is consistently significantly lower in the clusters with symptoms than in the clusters without symptoms. This appears to be largely an effect of the decreased K levels.

Cluster rachis analysis, 1979-82 (all samples): Regression analysis was performed on the tissue analysis data from all of the normal and symptom cluster rachis samples taken from all of the trials, 1979-82. Comparisons include total N *vs.* NH_4-N, K *vs.* total N, K *vs.* NH_4-N, and K *vs.* Ca + Mg. All had low coefficients of correlation except for total N *vs.* NH_4-N which is presented in Figure 1. Figure 1 also shows a relationship between total N and NH_4-N levels and the occurrence of symptoms. WB symptoms occurred when rachis total N levels were above 1.5% and NH_4-N levels were 3000 ppm and above. The results may indicate critical concentration limits above which levels WB symptoms become manifest and below which levels such symptoms do not usually appear.

The degree of correlation between total N and NH_4-N is remarkbly good, considering that the data represent samples from various vineyards of high and low WB incidence, varying degrees of vigor, different soil types, varied cultural practices and fertilizer programs over a four-year period of varied weather conditions.

While close correlation of total N concentration with symptom development is implied, the relationship of symptom presence to NH_4-N concentration may be coincidental. High NH_4-N levels can be the by-product of enzymatic reduction of N to amino radicals beyond the amounts required in amino acid and protein synthesis. Further reduction of surplus amino groups produces NH_4 and free NH_3 as does the natural degeneration of proteins into phenols by deamination and oxidation (2). Also, necrotic tissue can result from causes other than NH_4 toxicity.

The process which produces WB is probably quite complex, and high total N may be a necessary factor. If so, control of N levels through fertilization practices may serve to decrease the incidence and severity of the condition.

Conclusion

This investigation has shown WB to be related to a higher N status of the vines and higher cluster rachis tissue levels of N and NH_4. High WB incidence areas all had a higher N status than low incidence areas within a given vineyard. However, some high-incidence areas had a lower N status than did low-incidence areas in other vineyards as indicated by petiole analysis. N fertilization was also shown to increase WB in certain vineyard locations. This was especially true of low-incidence areas with a lower N status. This suggests that growers should consider lowering N usage in vineyards prone to WB.

Other important nutritional relationships of the WB problem that exists in the San Joaquin Valley of California were also found. In California, the incidence or severity of WB could not be related to Ca or Mg levels in the tissue or to elevated K:Ca + Mg ratios as reported in the European literature (1,3,5,12,14,16,18,19,27). In fact, K:Ca + Mg was lower in high incidence vineyard areas and symptom tissue. Also, application of Ca or Mg salts in solution to clusters or as a fertilizer to the soil did not reduce symptoms in contrast to findings reported in Europe (11,13,15,32).

Analysis of the cluster rachis was found to be useful in studying mineral nutrition relationships of WB. It demonstrated a close relationship between total N and NH_4-N levels in symptom and normal cluster framework tissue as well as a lack of Ca and Mg relationships. Levels above

1.5% N and 3000 ppm NH_4-N in the rachis were associated with WB development. Ammonium in plant tissues has been shown to cause necrosis (26). However, the role of N and/or NH_4 in WB needs more detailed study to determine if they are directly or indirectly invovled in the induction of WB symptoms.

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