CHAPTER 2
REVIEW OF LITERATURE

INTRODUCTION

Bud necrosis (BN) in grapes (*Vitis spp.*) is a physiological disorder observed in the compound lateral grape buds. There is no evidence of association of any pathogen or insect pest with BN in grapes in any of the studies conducted so far; therefore it remains an idiopathic disorder. BN occurs in different parts of the world, including Australia (Dry and Coombe, 1994), California (Morrison and Iodi, 1990), Chile (Perez and Kliewer, 1990), India (Bains et al., 1981; Bindra and Chohan, 1975), Israel (Lavee et al., 1981; Lavee, 1987; Ziv et al., 1981), Japan (Naito et al., 1986), and Virginia (Wolf and Warren, 1995). BN or similar disorders affect tulips (*Tulipa fosteriana*) (Czajkowska and Conijn, 1992), lateral buds of black currant (*Ribes nigrum*) (Gill, 1985), almonds (*Prunus amygdalus*) (Kester and Asay, 1978), and poinsettia (*Euphorbia pulcherrima*) (Simons and Smith, 1991). Calcium deficiency, mites (*Rhizoglyphus echinopus*), ammonium toxicity and gibberellic acid application are some of the putative factors responsible for those disorders (Czajkowska and Conijn, 1992; Gill, 1985; Kester and Asay, 1978; Simons and Smith, 1991). Necrosis of the apical meristem of shoot tips and lateral buds followed by subsequent abscission of lateral buds and shoot dieback is observed in almonds (Hellali et al., 1978; Kester and Asay, 1978). The corpus cells in these apical meristems enlarge and cause the rupture of the tunica cells (Hellali et al., 1978). In the case of black currant, the flower primordia within the lateral bud is killed (Gill, 1985). Poinsettias develop necrosis in the bracts and transitional bracts, and is a calcium-deficiency related disorder (Simons and Smith, 1991). Affected poinsettias typically develop small leaves, necrotic lesions and curling of leaf margins. Terminal growing shoot tips fail to develop which result in stunted plants. Calcium sprays (calcium carbonate) prevented bract necrosis in poinsettias (Woltz and Harbaugh, 1986). Necrosis in tulip bulbs is increased by acarid mites and results in the production of deformed leaves and flowers (Czajkowska and Conijn, 1992). Inflorescence necrosis (IN) is another disorder of grapes with some features similar to grape BN. IN is characterized by the development of necrosis in flowers, pedicels and in some instances peduncle tissue of clusters (Jackson and Coombe, 1988; Jordan et al., 1991; Lombard et al., 1993; Keller and Koblet, 1995). The causes of IN, as is the case for BN, are not clear. A positive correlation between high concentrations of ammonium in the rachis and berries and IN severity was observed (Jackson and Coombe, 1988). However, Keller and Koblet (1995) proposed that carbon starvation rather than ammonium toxicity is responsible for IN. Fruit clusters at different stages of development were treated with the glutamine synthetase (GS) inhibitor phosphinothricin (PPT) which caused a reduction in photosynthetic activity and resulted in senescence of the tissue (Keller and Koblet, 1995). Immersion of fruit clusters in PPT solutions increased IN in the clusters and addition of sucrose to the PPT solutions decreased the development of necrotic symptoms. Therefore, IN may be an effect of the senescence process caused by carbon starvation because addition of sucrose prevented IN development in fruit clusters. Glutamine synthetase is responsible for reassimilation of released ammonia (Kamachi et al., 1991). Hence, ammonium toxicity is also

implicated in IN development. High levels of ammonium observed in the tissues might have resulted from carbon depletion in senescing tissues (Keller and Koblet, 1995).

BN in grapes generally affects the primary bud, and is initially observed as a zone of compressed cells in the primary bud axis, which ultimately results in the death of the primary axis (Lavee et al., 1981; Morrison and Iodi, 1990; Perez and Kliewer, 1990). The term 'primary bud necrosis' is sometimes used to refer to bud necrosis in grapes because only the primary bud aborts in most cases (Dry and Coombe, 1994; Morrison and Iodi, 1990). The smaller secondary buds on either side of the primary bud generally continue to develop after the death of the primary bud (Ziv et al., 1981). Secondary buds are less fruitful than the primary buds (Pratt, 1974) and produce shoots with smaller and fewer fruit clusters than does the primary shoot (Dry and Coombe, 1994). In some cases, twin shoots develop due to the death of the primary bud, and the crushed remains of the primary bud are visible between the twin secondary buds (Dry and Coombe, 1994). Occasionally, secondary buds are also affected (Morrison and Iodi, 1990; Naito et al., 1987; Wolf and Warren, 1995). Therefore, BN reduces crop yields.

Cultivars such as 'Thompson Seedless', 'Flame Seedless', 'Queen of Vineyard', 'Kyoho', 'Campbell Early', 'Pione', 'Shiraz', 'Steuben' and 'Riesling' have been reported to be susceptible to BN (Bains et al., 1981; Bindra and Chohan, 1975; Dry and Coombe, 1994; Hopping, 1977; Lavee, 1987; Lavee et al., 1981; Morrison and Iodi, 1990; Naito et al., 1987; Naito et al., 1989; Perez and Kliewer, 1990; Wolf and Warren, 1995; Ziv et al., 1981).

DEVELOPMENT OF BUD NECROSIS

Bud necrosis usually affects the primary bud (Dry and Coombe, 1994; Perez, 1991; Naito et al., 1989). When viewed under a light microscope, a transverse, compressed zone of cells was observed at the nodes of the primary axis within the compound grape bud (Morrison and Iodi, 1990). The pattern of occurrence of the compressed zone was not uniform (Morrison and Iodi, 1990). In some buds, necrosis occurred at the base of the primary axis, in some from node one to node four on the primary axis, and in others only the apical nodes of the primary axis died (Morrison and Iodi, 1990). A zone of compression with distorted cell walls, followed by cell lysis was observed in this study. BN occurred during the period between bloom and the onset of bud dormancy. Examination under a light microscope revealed that the first visual symptoms of BN described by distorted cells usually became evident between 20 to 40 days after full bloom in 'Queen of Vineyard' (Lavee et al., 1981), 'Thompson Seedless' (Morrison and Iodi, 1990), and 'Kyoho' (Naito et al., 1987). After the primary bud dries, the secondary buds may continue to develop and produce shoots for the next season (Lavee et al., 1981; Naito et al., 1989). Occasionally, under situations of high BN occurrence, secondary buds were also found to be affected by BN (Morrison and Iodi, 1990; Naito et al., 1987; Wolf and Warren, 1995).

The basic pattern of BN occurrence is therefore described by development of necrosis in the primary axis of the compound grape bud after anthesis followed by primary bud death. Reports in BN literature are similar regarding this basic pattern of BN occurrence within the primary axis of

the grape bud. However, there are some differences in the occurrence of BN along the nodes of the current season shoot. Studies conducted on 'Thompson Seedless' (Morrison and Iodi, 1990; Perez and Kliewer, 1990), 'Flame Seedless' (Morrison and Iodi, 1990) and 'Queen of Vineyard' (Lavee et al., 1981) reported a higher percentage of BN is in the basal nodes than in the more distal nodes. Conversely, 'Kyoho' (Naito et al., 1989) and 'Riesling' (Wolf and Warren, 1995) expressed a higher percentage of BN in nodes six to fifteen than in the more basal nodes. The cultivar 'Queen of Vineyard' has fruitful buds beyond node seven (Lavee et al., 1981). Lavee et al. (1981) reported that buds (beyond node seven) with a greater number of inflorescence primordia have a lower tendency to develop necrosis than do the basal (1-7) nodes. Imbalance of endogenous hormones, particularly high levels of gibberellins were suggested as a possible reason for both the poor inflorescence differentiation and the high BN occurrence in the basal seven nodes of 'Queen of Vineyard' (Lavee et al., 1981). Morrison and Iodi (1990) by contrast, found no correlation between cluster differentiation and BN in 'Thompson Seedless'; BN occurred irrespective of the presence of differentiated clusters or tendrils. There are also reports of a greater frequency of BN in the more distal nodes (nodes 5-20) than in the basal nodes (nodes 1-4) (Wolf and Warren, 1995). Naito et al. (1989) reported high levels of BN from nodes one to twelve in the grape cultivars 'Campbell Early', and 'Muscat Bailey'. However, 'Pione', and 'Kyoho' had higher levels of BN from nodes five to twelve than nodes one to four. These inconsistencies suggest a cultivar or regional effect on BN incidence.

Factors previously identified that might promote BN are excessive irrigation, carbohydrate level, shade and gibberellin-like activity (Bindra and Chohan, 1975; Dry and Coombe, 1994; Hopping, 1977; Lavee, 1987; Lavee et al., 1981; Morrison and Iodi, 1990; Naito et al., 1987; Naito et al., 1989; Perez and Kliewer, 1990; Wolf and Warren, 1995; Ziv et al., 1981).

VEGETATIVE VIGOR

Shoot vigor, which can be quantified as cane diameter, internode length, and growth rate, is often positively correlated with BN (Dry and Coombe, 1994; Lavee et al., 1981; Wolf and Warren, 1995). Working with the cultivar 'Queen of Vineyard' in Israel, Lavee et al. (1981) reported that cane diameter >10 mm typically had 15 to 50% more BN than did canes of < 10 mm diameter. Similarly, Dry and Coombe (1994) in Australia working with 'Shiraz' also reported that thicker shoots (>12mm) had 15 to 40% more BN than did thinner shoots (<12mm) from nodes 2 to 7. Additionally, Wolf and Warren (1995) found that BN incidence of 'Riesling' was positively correlated with specific growth rate of shoots measured in the three-week period after bloom. It should be emphasized, however, that these reports have only shown *correlations* with BN incidence, that cause and effect relationships are lacking, and that conflicting data also exist. For example, Naito et al. (1989) working with 'Kyoho' were unable to establish any correlation between BN incidence and shoot length, shoot thickness or bud size. These variable results illustrate that though shoot vigor and BN incidence are positively correlated in some situations, that relationship is not uniform in all the varieties susceptible to BN.

Shoot thinning increased the incidence of BN of 'Shiraz' in Australia (Dry and Coombe, 1994). Dry and Coombe (1994) hypothesized that removal of shoots from vines increased the vigor of the remaining shoots on the vine, and thereby increased BN incidence. But there did not appear to be any significant difference in the percentage of BN between the control and the treatment vines where 75 percent of the shoots were removed. The treatments involving 85 percent shoot removal did however, almost double the BN percentage compared to the control. But, the study does not mention how many shoots were retained on the vine. There are conflicting reports regarding effect of shoot thinning on BN incidence. For instance, inconsistent results were obtained in the study conducted in Virginia, USA by Wolf and Warren (1995). 'Riesling' vines thinned to 10 shoots per meter of canopy were compared with vines thinned to 20 shoots per meter of canopy in two different years. No significant difference was observed in one year while, in the second year, vines thinned to 20 shoots per meter of canopy had 44 percent BN compared to 30 percent BN in vines thinned to 10 shoots per meter. Similarly, another study conducted in Chile on 'Thompson Seedless' vines demonstrated that shoot thinning and shoot positioning reduced BN (Perez and Kliewer, 1990). Shoot thinning was done by removing about 8 to 10 shoots around the head region where the arms of the vines separated. Perez and Kliewer (1990) claimed that the process of shoot thinning increased light penetration within the vine canopy thereby reducing BN. But the basal nodes (1-8) still showed approximately 54 percent BN as opposed to the controls which had approximately 70 percent BN. Shoot thinning and/or positioning only reduced BN levels by approximately 15 percent at all node positions.

Shoot thinning in the Australian study might have increased vigor in the remaining shoots because under conditions of severe stress, like defoliation, the trunk serves as a pool from which carbohydrates can be mobilized (Koblet et al., 1993). Photosynthesis and mobilization of stored carbohydrates from roots, trunks and canes are the two sources of carbohydrates. Defoliation is known to increase net photosynthetic rate of remaining leaves (Koblet et al., 1993; Edson et al., 1995). Therefore, both photosynthesis and remobilization, might have increased the vigor of the remaining shoots. But as it is not clear how many shoots were remaining after the shoot thinning treatment in the Australian work, it is not possible to arrive at any conclusions regarding the effect of shoot thinning on BN occurrence.

SHADE

Canopy shade, especially mutual leaf shading, is also associated with BN (Perez and Kliewer, 1990; Wolf and Warren, 1995). The earliest report of shade effects on BN was a study conducted by Perez and Kliewer (1990) in Chile on 'Thompson Seedless'. Application of 48 percent artificial shade over a fifteen-day period at different times between flowering and veraison increased BN to 72 percent, compared to the non-shaded vines which had 42 percent BN. Increasing the duration or level of artificial shade did not result in any further increase in BN (Perez and Kliewer, 1990). Nevertheless, Perez and Kliewer (1990) could not totally eliminate BN after increasing the light penetration within the canopy by removing eight to ten shoots from near the head region. Improvement in light microclimate within the vine canopy decreased BN only very slightly. The shoot thinned vines had 53 percent BN, while the controls had 70 percent

BN. Presence or absence of the leaf subtending a bud, a lateral closest to the bud, or a cluster opposite a bud had no significant effect on BN incidence (Perez and Kliewer, 1990). However, there are two other shade studies that yielded different results. Morrison and Iodi (1990) conducted a shading trial in California with 'Thompson Seedless'. The vines were shaded at fruit set using shoots from adjacent vines to provide two leaf layers above the experimental shaded vines. No effect of shading was observed on BN occurrence (Morrison and Iodi, 1990). Similarly, no significant correlation was observed between artificial shading and BN occurrence in 'Riesling' (Wolf and Warren, 1995). Nevertheless, Wolf and Warren (1995) observed that usually buds collected from well exposed parts of the vine had around 20 percent less BN than those collected from the shaded parts of the vine. Shading of individual buds was also done using muslin cloth, leaving the leaf blades and petioles subtending the buds unshaded. No significant effect of individual bud shading on BN incidence was observed (Wolf and Warren, 1995).

Shoot thinning can have two effects on plant canopy: firstly, it can increase light penetration within the canopy and secondly, it can increase the vigor of the shoots remaining on the plant because competing sinks are removed, increasing translocation of carbon resources into the remaining shoots. There are conflicting reports regarding the effect of shoot thinning. Shoot thinning decreased BN in 'Thompson Seedless' (Perez and Kliewer, 1990; Wolf and Warren, 1995), and conversely, increased BN in 'Shiraz' (Dry and Coombe, 1994). Perez and Kliewer (1990) suggested that shoot thinning reduced BN in 'Thompson Seedless' vines due to increased light penetration within the vine canopy. Alternatively, Dry and Coombe (1994) suggested that shoot thinning increased the vigor of the shoots remaining on the 'Shiraz' vines resulting in increased BN. Hence, the canopy shade effects must be separated from the vigor effects to arrive at any conclusion about the cause of BN. Wolf and Warren (1995) conducted studies where they attempted to separate the shade effects from the vigor effects. The percentage of BN increased under both shaded and unshaded vines when the shoot number was increased from 10 to 20 shoots per meter of canopy. But no significant difference was observed between shaded and control vines. These results indicate that there is a positive correlation between vigor and BN, but the role of transient periods of shade is not clear. Perez and Kliewer (1990) suggested that shade acts as a direct stimulus for the development of necrosis because shade reduces the light intensity, thereby retarding bud development. However, shade could also change the levels of carbohydrate or proteins which could cause BN. At low light intensities, the rate of photosynthesis is reduced because enzymes involved in the TCA cycle are inhibited (Taiz and Zeiger, 1991). Light also regulates protein synthesis and degradation (Turpin and Weger, 1990). Reduced photosynthesis decreases the supply of carbon skeletons for the synthesis of amino acids, and under low light intensities many of the enzymes for protein synthesis are not activated (Turpin and Weger, 1990).

Light and temperature affect bud fruitfulness in plants. At light intensities of 180 $\mu mol \cdot m^{-2} \cdot s^{-1}$ the bud fruitfulness, indicated by small or totally absent flower primordia, was low but increased at higher light intensities over a temperature range of 25C to 30C (Buttrose, 1969). The optimum temperature range for flower primordia differentiation during daytime is 30C to 35C for a four-hour period per day and night temperature is 25C for 8 hours per day (Buttrose, 1969). Basal buds were more fruitful when initial temperatures of 30C declined to 20C when compared to a

constant 30C (Buttrose, 1969). This could be because shoot development is slower at 20C than at 30C, and therefore basal buds might have developed due to less competition from the distal nodes. When shoot development is slow, fewer nodes develop than when compared to a relatively faster rate of growth. Therefore, the photosynthates required to mature the nodes and the buds in a slow developing shoot is much lower than in a fast developing shoot (Taiz and Zeiger, 1991). Hence, a possible role of carbohydrate deprivation in BN incidence is indicated. Vigorous vines might be experiencing carbohydrate deprivation in their shoots causing BN in the axillary buds.

FERTILIZER AND IRRIGATION

Studies conducted in India have reported that excessive fertilizer application and irrigation resulted in flower bud killing in 'Anab-e-Shahi' grapes (Bains et al., 1981; Bindra and Chohan, 1975). Bindra and Chohan (1975) observed an increase in the number of dead buds (87% dead buds) in vineyards that were overmanured with nitrogen, phosphorus, potassium and organic manure compared to the vineyards which had restricted fertilization and irrigation (12% dead buds). Similarly, Bains et al. (1981) working with 'Anab-e-Shahi' grapes in India examined "bearing", "overvigorous", and "weak" vines. Yields from the overvigorous, and weak vines were lower than from the bearing vines. Nitrogen, phosphorus, and potassium levels in old wood and current season shoots were analyzed. Bains et al. (1981) observed that nitrogen and potassium levels were significantly higher in overvigorous vines followed by bearing and weak vines respectively. Analysis of phosphorus however, did not reveal any significant difference. Bains et al. (1981) concluded that any deviation from optimum levels of nitrogen should be avoided in order to maintain vine fruitfulness. However, Perez (1991) did not find any significant effect of nitrogen and potassium fertilizers on BN incidence. Perez (1991) did find a significant effect of irrigation on BN incidence. 'Thompson Seedless' vines were irrigated at the rate of 25 to 30 cm water per irrigation at three-week intervals. Irrigation increased the vine size and canopy shade, which led to increased BN and decreased bud fruitfulness. Photosynthetic photon flux in the irrigated vines was lower (163 $\mu mol \cdot m^{-2} \cdot s^{-1}$) than in the non-irrigated vines (558 $\mu mol \cdot m^{-2} \cdot s^{-1}$). Additionally, in another work conducted in Japan with 'Kyoho', nutrient elements such as nitrogen, phosphorus, potassium, calcium, magnesium, manganese, and boron of lateral buds were analyzed and no significant correlation was observed between any nutrient element and BN (Naito et al., 1987). There are conflicting reports in the literature regarding the effect of nutrients on BN. From these reports, it is not possible to conclude whether the nutrient factor is cause, effect, or independent of the BN problem.

CARBOHYDRATE STATUS OF NECROTIC BUDS

Flowers are formed by specialized axillary meristems or latent buds in the grapevines (Botti and Sandoval, 1990). An increase in the size of the cell, nucleus and nucleolus of stem apices during flower induction is noticed. The amount of starch granules also increases in latent buds during this period. Carbohydrates are essential for mitochondrial growth and multiplication during the vegetative and floral induction processes in the axillary bud (Botti and Sandoval, 1990). But low

light intensities can cause a reduction in carbohydrate resources. Under low light situations, the net assimilation rate of source leaves is reduced, reducing carbohydrate supply to axillary buds. This may impair axillary bud development (Hopping, 1977). Naito et al. (1987), in Japan, where sugars extracted from strong and weak shoots of 'Kyoho' vines were analyzed. Strong shoots were characterized as those that were longer (200-350cm), had larger internodal cross sectional area (80-118 mm^2), and bud size (600-700 mm^3) than the weak shoots (115-140 cm, 22-50 mm^2, 165-425 mm^3). The Japanese study revealed a positive correlation between reducing and non-reducing sugars in lateral buds and BN incidence in 'Kyoho' grapevines in Japan. Starch, conversely, was higher in the weaker shoots with low levels of BN. However, there are conflicting reports with regard to the role of carbohydrates in BN incidence. Work done in India on 'Anab-e-Shahi' vines demonstrated a higher level of total nonstructural carbohydrates such as starch, reducing sugars, and non-reducing sugars in 'bearing' vines of moderate vigor, compared to highly vigorous, unfruitful vines (Bains et al., 1981). Morrison and Iodi (1990) found almost no starch within the primary bud or at the base of the compound bud during the early stages of BN in 'Flame Seedless' and 'Thompson Seedless' when compared to healthy buds. Morrison and Iodi (1990) questioned whether low starch levels in the necrotic buds were an *effect* of altered metabolic processes during senescence, or were the *cause* of BN. These variable findings again suggest that there may be a cultivar as well as an environmental influence on the pattern of BN.

Removal of shoot tips temporarily eliminates the growing tips as a competing sink for carbohydrates resulting in preferential translocation of photosynthates to the fruit clusters and the basal nodes of the shoot (Candolfi-Vasconcelos and Koblet, 1990). Accumulation of high carbohydrate reserves is necessary just prior to and during the period of flower bud formation because carbohydrates are essential for differentiation of flower primordia within buds (Winkler et al., 1974). During active growth, the percentage of carbohydrate is higher in the shoots than in old wood because the carbohydrates present in the old wood are utilized by the shoots during their early growth and are replenished only after shoot growth slows (Koblet et al., 1993). Vigorously growing vines produce a large number of shoots, the growing points of which serve as competing sinks (Candolfi-Vasconcelos and Koblet, 1990, Koblet et al., 1993). There is preferential translocation of photosynthates towards these growing shoot tips. This might result in carbohydrate deprivation in the lateral buds resulting in BN, which might have been the case with the 'Anab-e-Shahi' buds. However, the Japanese study, where a positive correlation between reducing and non-reducing sugars in lateral buds and BN was observed, is different from the studies conducted in California and India where a negative correlation between carbohydrates and BN incidence was indicated. This again suggests a cultivar effect on BN occurrence.

GIBBERELLIN-LIKE ACTIVITY

The growth regulator, gibberellic acid has also been cited as one of the factors that increased BN especially in the cultivars, 'Queen of Vineyard', 'Alphonse Lavallee', 'Early Panse' and 'Kyoho' (Lavee, 1987; Naito et al., 1986; Ziv et al., 1981). Exogenous application of gibberellic acid (GA_3) (100 $mg \cdot L^{-1}$) nine days before or seven days after flowering increased BN incidence on nodes that ranged from 1 to 25 in the cultivar, 'Kyoho' (Naito et al., 1986). Application of GA_3

before and after full bloom increased BN to almost 100 percent in nodes 5 to 20, while the controls, which received no GA_3 sprays, showed only 0 to 30 percent BN (Naito et al., 1986). Another study conducted in Israel, also reported that GA_3 application (20 $mg \cdot L^{-1}$) at flowering increased BN incidence in nodes 6 to 12 (80% BN) compared to the basal five nodes (50% BN) (Ziv et al., 1981). Ziv et al. (1981) offered two possible explanations for variable node effect. One possibility was that basal buds were less sensitive to GA_3 because they constituted a weak sink for GA_3 at the time of the experiment. The other possibility was that basal buds are more developed than the distal buds and have stopped their growth and are therefore, less sensitive to GA_3 activity. Spraying GA_3 21 days after full bloom did not cause BN even though the concentration was increased to 100 $mg \cdot L^{-1}$ (Ziv et al., 1981). Young buds elongated followed by the development of a necrotic layer at the base of the primary axis. The method of GA_3 application also influenced the level of BN incidence. More necrotic buds were observed when GA_3 was applied by petiole feeding than by laminar spray (Ziv et al., 1981). However, in California, exogenous application of GA_3 was ineffective in causing BN in a 'Thompson Seedless' vineyard (Morrison and Iodi, 1990). In the Californian study, GA_3 was applied at full bloom (10 $mg \cdot L^{-1}$), and then again (100 $mg \cdot L^{-1}$) at nine days and 17 days after bloom.

Lavee et al. (1981) observed a higher incidence of BN at the basal nodes than at distal nodes (above the seventh node). Analysis of the levels of endogenous gibberellin-like activity (GLA) by ascending paper chromatography in buds from nodes three to seven showed that the lateral buds from vigorous vines had about two to three times more GLA than did buds from less vigorous vines, especially at five and 15 days after full bloom. The level of bound GLA was also higher in the buds of vigorous vines than in the normal vines, particularly at 30 days after full bloom (Lavee, 1987). GLA usually binds over time and becomes inactive. It was suggested that the low levels of free GLA are the reason for lack of necrosis in the younger, distal buds. Lavee (1987) arrived at the conclusion that gibberellin-like substances are mainly responsible for BN. Application of the growth retardants, succinic acid-2,2-dimethylhydrazine (SADH) and β-[(4-chlorophenyl)methyl]-a-(1,1-dimethyl-ethyl)-1H-1,2,4-triazole-1-ethanol (paclobutrazol) reduced the rate of shoot growth as well as the level of BN (Naito et al., 1986; Wolf and Warren, 1995). SADH and paclobutrazol are inhibitors of gibberellin biosynthesis and their effect on BN suggests a positive correlation between GLA and BN.

CONCLUSION

BN is correlated with numerous factors; however, no clear cause and effect relationships have been established. Natural or artificial shade has been suggested as a cause of BN (Perez and Kliewer, 1990). However, BN was not completely eliminated by improving the light environment within the vine canopy. Conversely, Wolf and Warren (1995) did not observe any correlation between shade applied prior to veraison and BN. Some studies reported that vigorous, necrosis-prone vines had a lower percentage of total carbohydrates than did the weak vines (Bains et al., 1981; Naito et al., 1987). Thus studies of carbohydrate distribution and accumulation are warranted to determine correlations with BN susceptibility. Fertilizer application and irrigation have also been reported to cause BN (Bindra and Chohan, 1975). Yet, other studies conducted in

Chile, Japan, and Virginia have not found any correlation between nutrients and BN (Naito et al., 1987; Perez; 1991; Wolf and Warren, 1995). Gibberellin-like activity has also been found to be positively correlated with BN occurrence (Lavee et al., 1981; Lavee, 1987; Naito et al., 1986; Ziv et al., 1981). Therefore, further research aimed at determining relationships if any, between nutrient elements and carbohydrates with BN should be conducted.

The basic pattern of necrosis is similar in all the BN-prone cultivars reported so far, but with some differences. For instance, the necrosis appears as cell compression followed by cell rupture and finally primary bud death. In some regions like Australia, Israel, California and Chile, the basal buds have been found to be prone to BN while in Virginia and Japan, the distal (above 5th node) buds are more susceptible. The effect of the different stresses such as shade and carbohydrate and mineral nutrient deprivation as well as effect of growth regulators on BN incidence varies with cultivar and environment. Some cultivars are able to tolerate these stresses and therefore might be less sensitive to BN.

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