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Anatomical Study of Primary Bud Necrosis in *Vitis vinifera* L. cv. Askari in Winter Dormant Bud

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Abstract

Primary bud necrosis in grapevine is a physiological disorder observed in the compound buds. This study was conducted to investigate the anatomical changes in cv. Askari grapevine in winter dormant buds. Bud samples were taken from Askari grapevine at Sisakht vineyards, south-western of Iran. Compound buds were dissected and assessed under a binocular microscope at 10-40x magnification and a digital microscope (Dinolite-AM413T) was used for taking photos. The results showed that the bud necrosis incidence was observed in primary buds more than in the secondary buds. Anatomical observations showed that PBN disorder occurred in central buds as brown small spot and gradually developed in the whole bud and bud necrotic remained on canes and did not abscission. On the results of present study, dissecting buds in autumn can raise awareness of PBN and fruitfulness of bud in the following spring, hence, growers should determine the level of bud necrosis prior to winter pruning so that pruning techniques might be adjusted to account for the bud damage.

Keywords: Askari grape, PBN, Disorder, Primary, Secodary bud.

INTRODUCTION

A large number of grapevine cultivars are cultivated in Iran. Askari is one of the major table grape cultivars and the yield of this cultivar is variable in different region. Bud necrosis with its significant effect on yield reduction is a common phenomenon in some Iranian vineyards and also within several cultivars in different parts of the world (Bindra and Chohan, 1975; Bains *et al.,* 1981; Lavee *et al.*, 1981; Naito *et al.*, 1986; Morrison and Iodi., 1990; Perez and Kliewer, 1990; Dry and Coombe, 1994; Wolf and Warren, 1995). The grapevine compound bud contains three or more individual buds. The main central bud is termed 'primary' and on either side of this bud are the 'secondary' and 'tertiary' buds. Generally, the primary bud develops into a new fruiting shoot in spring, while the secondary and tertiary buds remain dormant. If the shoot of the primary bud is damaged or dies, the secondary buds may develop a shoot to compensate for the loss. The death of the primary bud is termed primary bud necrosis (PBN). In this situation, secondary buds may burst, they often bear no fruit or produce smaller bunches resulting in yield loss. The disorder usually affects the primary buds, but occasionally the secondary buds will also abort (Rawnsley, 2003).

The time that PBN develops is dependent on cultivar. It has been reported that PBN commences soon after flowering and may be developed up to 10 weeks after full bloom (Morrison and Iodi, 1990) and continued to dormant stage. High shoot vigour, (Lavee *et al.*, 1981; Dry and Coombe, 1994), high level of soil nitrogen (Kliewer *et al.*, 1994), canopy shading (Perez and Kliewer, 1990; Wolf and cook, 1992) and exogenous application of gibberellic acid (Zive *et al.*, 1981) have all been shown to increase th incidence of PBN. Morrison and Iodi (1990) found that necrosis occurred at the base of the primary axis and in other buds, only apical nodes of the primary axis died. Zive *et al.*, (1981) showed that in young undifferentiated buds, necrosis developed below the apex causing death of the primary bud.

Vasudevan *et al.* (1998) found that the zone of compressed cell began at the base of the primary bud and advanced to the leaf primordial. PBN can be start in the zone of leaf primordial in some primary bud, not just at the base (Rawnsley and Collins, 2005). Collins *et al*. (2006) showed that cell breakdown was observed in all buds where PBN was visible. Collapse and thickening of cell walls was observed in a region of necrotic tissue and severity of PBN appeared to increase over time. However, there is a little information about the extent and distribution of this disorder in Iran. The aim of this research was to investigate different severity of PBN leading to further insight on the progression of this physiological disorder in cv.Askari.

MATERIALS AND METHODS

Bud samples were taken from Askari grapevine at Sisakht vineyards, south-western of Iran (Lat. 30^{o}, 51$^{/}$, 57$^{//}$ N, Long. 51^{o}, 27$^{/}$, 24$^{//}$ E, Alt. 2200 m) from November 2007 to March 2008. The mean daily maximum and minimum temperature for Sisakht zone were 28.8 ^{o}c and -13.6^{o}c, respectively. The vines were 18-year-old on own roots and with spaced 2.5-3 m apart on rows 3 m apart. All cultural practices were applied uniformly across blocks and in accordance with standard commercial practices. The sampling dates were 21- Nov., 21- Dec., 20- Jan., 19- Feb. and 19 March. The samples were placed in sealed plastic bags and stored in a cool place (4 ^{o}c) to minimize water loss. Compound buds at node 1 to 20 from the mature canes were grouped: 1-5, 6-10, 11-15 and 16-20 nodes, dissected and assessed for the presence of PBN. The presence of PBN was readily determined by making a transverse cuts with a scalpel at half the height of the bud; additional cuts were used to check the state of the secondary buds. The cuts were made deep enough to ensure that the three buds (primary, secondary and tertiary) to be exposed. Buds were dissected under a Binocular microscope at 10-40x magnification and a digital microscope (Dinolite-AM413T) was used for taking photos. A bud with no damage would show green tissue for the primary, secondary, and tertiary bud, indicating that these buds are alive. In contrast, a damaged bud will show a dark/brown discoloration (Fig. 1).

RESULTS AND DISCUSSION

The assessment of PBN showed that the highest incidence of PBN (18.09% and 18.78) was observed in February and March, respectively and the lowest incidence of PBN (6.28%) in November. Also the nodes of 1 to 5 had a higher incidence of PBN (39.57%) whereas, lowest of PBN (0.2%) were observed in nodes 16 to 20. Healthy primary bud displayed an uniform tissue and there is no symptom of cell breakdown (Fig. 2).

Anatomical observations of Askari grapevine dormant buds, showed that PBN occurred in central buds as brown small spot and gradually developed in the whole bud and bud necrotic remained on canes and did not abscission. Necrotic bud was observed in Primary bud and sometimes the disorder occurred in secondary buds (Fig. 3).

In healthy primary and secondary buds there was extensive damage, distorted tissue and finally case a splitting in PBN state. In most cases, tissue above of necrotic zone become completely brown and dry, while the tissue below that bud remained green color and health (Fig. 4).

The incidence of PBN on *Vitis vinifere* L. cv. Askari in Southwest of Iran was identical to that described by Lavee et al., (1981), Morrison and Iodi (1990), Perez and Kliewer (1990) and Collins and Belinda (2004). In recent years, the use of bud dissection analysis has shown that some vineyards experience high levels of PBN which can ultimately reduce yield potential. Significantly high levels of PBN have also been observed in cultivars such as Cabernet Sauvignon, Riesling, Viognier and Chardonnay (Dry *et al.,* 2003; Dry and Coombe, 1994; Rawnsley, 2003). Vigorus growth, expressed as cane diameter, internode length and growth rate, has been associated with a high incidence of PBN. For example, 'Shiraz' cultivar is a highly vigorous cultivar and is prone to PBN. Also, yield reduction can be due to increasing the secondary to primary shoot ratio in vigorous vineyards. Previous reports indicated that PBN increased to the onset of bud dormancy (Morrison and Iodi, 1990; Lavee *et al.,* 1981; Vasudevan *et al.,* 1998). Although our results supported this assumption, sampling throughout the entire season revealed the incidence of PBN could increase later. There is a probability that climatic and cultural conditions cause variability PBN incidence in vineyards.

Basal buds on the canes have a higher vigour potential and a lower reproductive differentiation rate, and these tend to develop a higher PBN incidence. Low fertility of basal buds is common in many cultivars in vigorous situation. There is many possible causes of PBN incidence, that high shoot vigour (Lavee *et al*., 1981; Dry and Coombe, 1994), canopy shading (Perez and Kliewer, 1990; Wolf and Cook, 1992), frost stress, low bud carbohydrate reserve, high GA3 level (Zive *et al.,* 1981), high level of soil nitrogen (Kliewer *et al.,* 1994), have all been shown to increase the incidence of PBN. Visually a bud with PBN appear similar to that of a healthy bud and, therefore, difficult to detect by eye (Dry and Coombe, 1994). Although it is possible to see PBN in the field with a hand lens, bud dissection are need to accurately detect PBN and a assess bud fruitfulness. Microscopic bud dissection is being used to assess bud fertility and predict potential yield in vineyards. PBN disorder can be easily detected in dissected buds and also whether partial or complete necrosis has occurred. Similar to the observation by Vasudevan *et al*. (1998a), the first visible symptom of PBN was indicated by the presence of distorted and compressed cells with irregular cell walls. There were, however, some differences in the location of PBN. Vasudevan *et al*. (1998a) found that the zone of compressed cells began at the base of the primary bud and advanced to the leaf primordia. Our observations indicated that PBN start in the central zone of leaf primordia in some primary buds, not just at the base. Morrison and Iodi (1990) also observed the random distribution of PBN in the early stages of development. In Thompson Seedless, PBN is characterized by the formation of a distinct necrotic zone most commonly located at the fourth leaf primordia (Perez and Kliewer, 1990). Collins *et al*. (2006) showed that the location of cell breakdown due to PBN appeared to be random and was not isolated to one region within the primary bud.

The formation of necrotic cells in the primary bud caused a rupture or separation between the basal part of the bud and the apical meristem, resulting in death of the primary bud. PBN

stopped further primordial growth, so that cells matured more rapidly without forming fully developed leaf primordia. This entire cell region of the primary bud then breaks down and, if severe, necrosis extended into the secondary buds. Cell separation at the necrotic zone was due to cell breakage, rather than formation of an abscission zone.

CONCLUSIONS

The incidence of PBN is serious in Iran, and Askari is a cultivar which is one of the main table grapes grown in many areas is very susceptible to physiological disorder of PBN. This study which is reported for the 1st time in Iran, clearly points out the importance of this problem. The number of buds retained on the grapevine after pruning has a considerable impact on canopy extension and vine yield in the following season. If too few buds are retained due to PBN, yield may be reduced to below what the vine would otherwise have the capacity to ripen. Also, shoot grow may be excessively vigorous because of a lack of competition with the fruit and other shoots.

It is concluded that primary bud necrosis is one of important factors of low fruitfulness in vineyards under consideration. Also it was conclusion that PBN incidence and distribution of buds on thick and thin canes were correlated. However when buds along canes are detected prior to onset of spring, severe PBN can be observed. PBN disorder appears to be widespread throughout the Askari grapevine in some of vineyards under consideration.

In general, < 20% PBN may not cause significant yield loss. Although it was expected that PBN levels would increase during the season, the incidence of PBN fluctuated. This verifies that, regardless of cultivar, bud fruitfulness must be assessed as close to pruning as possible to ensure pruning levels are modified accordingly. Therefore, fruitfulness can be estimated prior to pruning by dissecting of a sample of buds which allows a grower to optimize crop load by leaving the best combination of cane and spur length.

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Figures

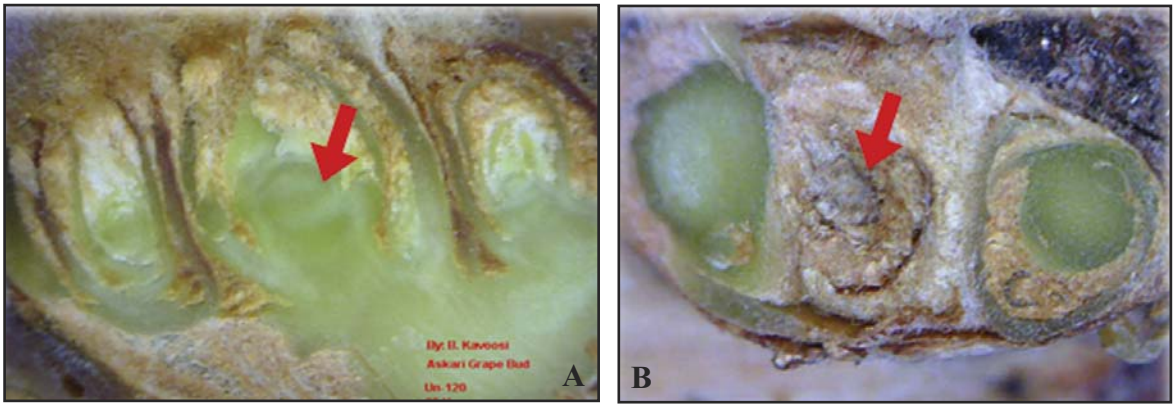

Fig.1. Cross sectioned compound bud showing all buds are alive (**A**), and damaged primary bud (**B**) in Askari grapevine.

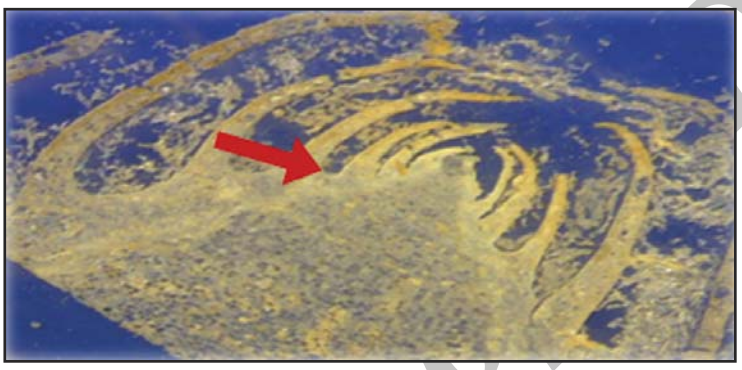

Fig. 2. Longitudinal section of healthy primary grapevine cv. Askari.

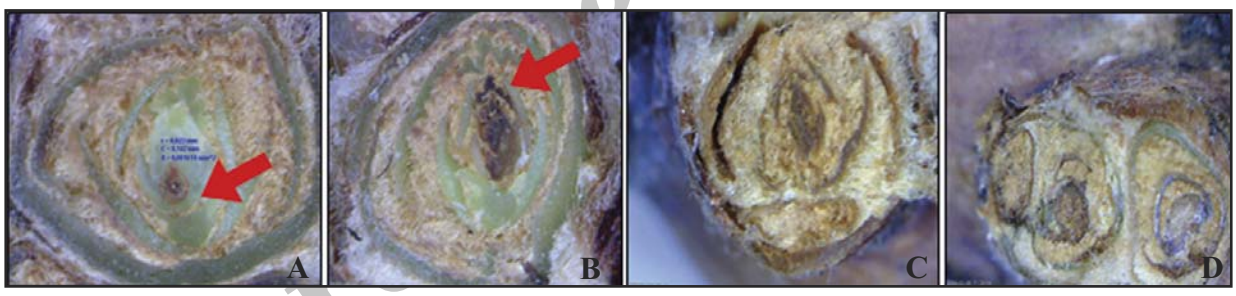

Fig. 3. Cross section through a mature Askari grapevine dormant bud, Start of PBN (**A**), developing PBN (**B**), Full PBN (**C**) and primary and secondary and tertiary buds necrosis (**D**).

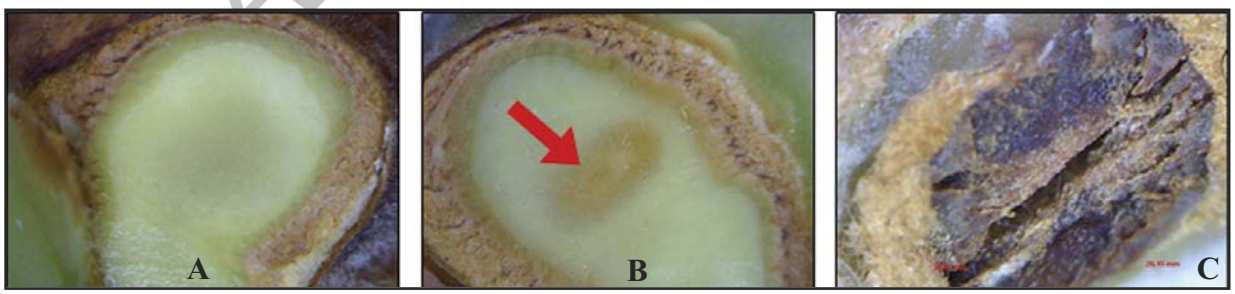

Fig. 4. Cross sub section of both health (**A**) and death (**B**) primary bud with separation tissues(**C**) in Askari grapevine dormant bud.