Stress-induced development of inflorescence necrosis and bunch-stem necrosis in *Vitis vinifera* L. in response to environmental and nutritional effects

by

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**Summary:** Inflorescences and mature clusters, respectively, of field-grown Müller-Thurgau grapevines (*Vitis vinifera* L.) were immersed in aqueous solutions of the glutamine synthetase (GS) inhibitor phosphinothricin (PPT). Typical symptoms of inflorescence necrosis developed after immersion in 1 or 10 mM PPT, but not in the 0.1 mM treatment. No necrotic symptoms could be induced in mature clusters. In addition, single-node cuttings of field-grown Müller-Thurgau and Pinot noir vines with one cluster, with or without adjacent leaf, were incubated in various solutions with or without PPT at several phenological stages. Necrotic symptoms occurred in all treatments including the H₂O control. However, at early stages of development the symptoms appeared earlier than at later stages, and elevated PPT concentrations reduced the development of necrotic manifestations. The addition of NH₄NO₃ to the solution enhanced the appearance of symptoms both in the presence and absence of PPT, while KNO₃ did not. In incubation solutions without PPT, metallic cations like Mg²⁺, Ca²⁺ and K⁺ reduced the incidence of bunch-stem necrosis. When cuttings were exposed to different environmental conditions, there was no significant light effect, but wind decreased the incidence of both inflorescence necrosis and bunch-stem necrosis. A leaf, attached to the cluster, or the addition of sucrose to the solution effectively delayed the development of necrotic manifestations. These results indicate that GS is present in rachis, flower and berry tissues and that the buildup of toxic NH₄⁺ levels is involved in the development of both inflorescence necrosis and bunch-stem necrosis. However, NO₃⁻ reduction and primary N assimilation appear not to contribute significantly to NH₄⁺ accumulation. This buildup may rather be a secondary effect related to senescence of the tissue due to carbon depletion.

**Key words:** grapevine, phosphinothricin, nitrogen, ammonium, senescence, inflorescence necrosis, bunch-stem necrosis.

**Introduction**

Inflorescence necrosis (IN) and bunch-stem necrosis (BSN) are two physiological disorders in grapevines, developing similar necrotic symptoms on grape pedicels, rachides and peduncles, that are thought to be induced by NH₄⁺ toxicity (Christensen and Boggero 1985, Silva et al. 1986, Jackson and Coombe 1988). Christensen and Boggero (1985) reported a close relationship between N nutrition and BSN, and they proposed a threshold of 1.5% total N and 3000 ppm NH₄⁺ in the rachis tissue for the occurrence of BSN. Jordan (1989) found increased NH₄⁺ concentrations in rachis and berries of shaded vines and a positive correlation between NH₄⁺ content and the severity of IN. In addition, both IN and BSN were related to the level of shading in studies of Pérez Harvey and Gaete (1986) and Jackson (1991).

Ammonia assimilation and NH₃ releasing reactions have been reviewed by Joy (1988). The glutamine synthetase/glutamate synthase (GS/GOGAT) pathway is considered the primary route for NH₃ assimilation in higher plants. Phosphinothricin (glufosinate, PPT), a phosphinic acid analogue of glutamate, is a widely distributed herbicide in grape production. The mode of action of PPT was extensively studied by Wild and Manderscheid (1984), Saüer et al. (1987) and Wild et al. (1987). It is now generally recognized that NH₃ accumulates in tissues treated with PPT due to its selective inhibition of GS, leading to a constriction in photosynthetic activity and senescence of the tissue. Gu et al. (1991, 1994) reported that other GS/GOGAT inhibitors increased NH₄⁺ concentrations in grapevine leaves, flowers, fruit and pedicels but not in petioles and rachis.

Apart from NH₄⁺, metal cations, like K⁺, Ca²⁺ and Mg²⁺ have also been associated with both IN and BSN (Alleweldt and Hipny 1972, Feucht et al. 1975, Steßwag-Kittler 1975, Schaller 1983, Jackson and Coombe 1988). However, the relations between cause and effect have been doubted (Redl 1983, Christensen and Boggero 1985). Nevertheless, foliar treatments with solutions containing Mg and/or Ca are effective in the control of BSN (Perret and Koblet 1972, Rizzotto 1977, Jurgens and Becker 1987).

The objective of the present investigation was to verify the presence of GS in grape cluster tissues at various phenological stages and to determine the implication of GS inhibition in the occurrence of IN and BSN. In addition, we tested environmental and nutritional effects on the development of IN and BSN, and we also intended to give further evidence for the hypothesis recently proposed by Keller and Koblet (1994), that carbon starvation, rather than excessive N nutrition, is triggering these so-called physiological disorders.
Materials and methods

Immersion experiment: The experiment was conducted in a vineyard at the Swiss Federal Research Station in Wädenswil, Switzerland, with 3 years old Müller-Thurgau grapevines (Vitis vinifera L.), grafted on SO 4 rootstocks. The vines were cane pruned and trained on a vertical shoot trellis (Guyot). Phosphinothricin (PPT) was applied to 10 randomly selected clusters and 10 leaves adjacent to different clusters by dipping them into aqueous solutions containing 0.1, 1 or 10 mM PPT for 5 s. Clusters were treated at anthesis and maturity in 1991. At maturity the berries were removed from the pedicels of 10 additional clusters before immersion.

Incubation experiments: PPT solution experiments were conducted in the greenhouse in 1991, using clusters from the same vines as in the immersion experiment. Clusters were selected for uniformity of growth and phenological stage at anthesis, berry set, veraison (onset of ripening) and maturity. After excision from the vine, the peduncles were put through rubber caps into 25-ml glass vials containing various solutions made with distilled H2O. The solutions used at anthesis were: 0, 0.1, 0.5, 1, 5 and 10 mM PPT. Two further treatments were added at the later stages of development: 10 mM PPT plus either 1 mM NH4NO3 or 1 mM KNO3. Eight replications were used. Light intensities were kept at 1-2 % of full sunlight, which is around the light compensation point of photosynthesis in grapevine leaves (Keller and Koblet 1994).

Three experiments were conducted in 1992 with Müller-Thurgau or Pinot noir (clone M1/17, planted in 1983 on 3309 rootstocks) vines from the same vineyard as used in the immersion experiment. Shoots were selected for uniformity of growth and excised at the base. Single-node cuttings with a cluster and adjacent leaf were cut from the shoots. The cuttings were placed in 100-ml glass vials covered with a rubber cap and containing various solutions made with distilled H2O.

The first experiment was performed with Müller-Thurgau clusters at anthesis and immediately after veraison. The incubation solutions were 0 and 10 mM NH4NO3, combined with 3 additional treatment factors: clusters with or without adjacent leaf, full sunlight or deep shade (1-2 % of full sunlight), with or without wind (separate greenhouse cabinets with two opposing windows either open or closed). The leaf was left attached as a carbon source, and the light treatments were imposed to alter gas exchange activities. The wind treatment was used to stimulate a potential NH3 evolution through the stomata.

The second experiment was conducted with Pinot noir clusters with one leaf at anthesis. The solutions were: 0 or 10 mM NH4NO3, combined with 0 or 2 % sucrose as the major transport compound in the phloem.

The third experiment was effected with Pinot noir clusters with two leaves immediately after veraison. The solutions were: 5 or 10 mM NH4NO3, 10 mM KNO3, 10 mM HNO3, 5 mM Ca(NO3)2 or 5 mM Mg(NO3)2. Both, the second and the third experiment were conducted at 1-2 % of full sunlight without wind. Four replications were used per treatment combination in each experiment. Incubation was continued until symptoms of IN or BSN were visible.

Since NH4 accumulation following PPT applications and the implication of NH4/NH3 in the occurrence of IN and BSN were confirmed by several researchers (see "Introduction"), tissue-NH3 concentrations were not measured in the present investigation. Symptoms of IN were registered as % necrotic flowers per inflorescence and those of BSN as % necrotic rachis tissue per cluster in all experiments. The data were subjected to Bartlett's test to check homogeneity of variance subsequent to square-root transformation. Analysis of variance, F-test and Duncan's multiple range test were used to examine differences between means.

Results

Immersion experiment: In the field, grapevine leaves treated with 1 and 10 mM PPT became necrotic within 4-6 d after immersion and senesced very rapidly. While there were no differences in the severity of necrotic symptoms between these two PPT concentrations, the 0.1 mM PPT treatment only caused irregular necrotic spots on the leaves, implying that this PPT concentration was insufficient to entirely inhibit GS in the leaf. Inflorescences adjacent to treated leaves showed no necrotic symptoms, suggesting that PPT was not translocated from the leaves. Typical symptoms of IN only developed on the flower clusters that had been immersed and first appeared in the 10 mM PPT treatment, 5-6 d after immersion (Fig. 1: A, B). Necrotic symptoms first occurred on flowers and pedicels and only later on rachis and peduncles. After 30 d, the clusters of both the 1 and 10 mM treatments were completely necrotic (Fig. 1: C, D), but no effect could be observed in the 0.1 mM treatment. Some basipetal translocation of PPT or NH4 must have occurred in the 10 mM treatment, since the shoot internode below the immersed cluster became partly necrotic, too (Fig. 1: D). No symptoms were visible, however, 2 weeks after immersion of mature clusters in PPT solutions, regardless of the presence or absence of berries.

Fig. 1: Occurrence of inflorescence necrosis in response to immersion of Müller-Thurgau grape clusters in phosphinothricin (PPT) solutions: 6 days after immersion in 1 mM PPT (A) or 10 mM PPT (B), and 30 days after immersion in 1 mM PPT (C) or 10 mM PPT (D), respectively.
Incubation experiments: When the peduncles of excised bunches were placed in various PPT solutions, necrotic symptoms and sequential senescence occurred in all treatments (even in the H$_2$O control) at all developmental stages (Fig. 2). However, in the 5 and 10 mM PPT treatments (Fig. 2: A, C), particularly in combination with NH$_4$NO$_3$ but not KNO$_3$ addition (Fig. 2: B, D, F), the symptoms appeared much earlier and were more intense than at lower PPT concentrations and the H$_2$O control. The first symptoms were visible after 1 d at anthesis and berry set, after 2 d at veraison and after 5 d at maturity. In contrast to the immersion experiment, symptoms first occurred on peduncles and rachis and only later on flowers or berries (Fig. 2: A, B). At veraison and maturity only peduncles and rachis developed necrotic symptoms, whereas the berries remained green, although a loss of turgor with concomitant dehydration could be observed in all treatments, in particular at veraison (Fig. 2: C - F).

Symptoms of IN or BSN also occurred in all incubation solution treatments conducted with single-node cuttings. Inflorescence necrosis of Müller-Thurgau cuttings was most severe at the combinations +N/-light/-wind/-leaf and least severe at the combination -N/+light/+wind/+leaf (Table). There were, however, significant interactions among treatment factors. When the adjacent leaf had been excised before imposition of the additional treatments, IN severity was always very high. Only if the leaf was left attached, the effects of the other factors became apparent: incubation in water led to low levels of IN, regardless of the presence or absence of wind. If NH$_4$NO$_3$ was added to the solution, IN was significantly increased, but wind reduced the N effect (Fig. 3). Bunch-stem necrosis on clus-

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Table

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Fig. 2: Occurrence of necrotic symptoms in response in incubation of Müller-Thurgau grape clusters in phosphinotrin (PPT) solutions: 1 day in H$_2$O or 0.1, 0.5, 1, 5, 10 mM PPT at anthesis (A). 1 day in 10 mM PPT plus 1 mM KNO$_3$ or NH$_4$NO$_3$ at berry set (B). 9 days in H$_2$O or 10 mM PPT at veraison (C). 9 days in H$_2$O or 10 mM PPT plus 1 mM NH$_4$NO$_3$ or KNO$_3$ at veraison (D). 6 days in H$_2$O or 0.1, 0.5, 1 mM PPT (E), and in 5 or 10 mM PPT or 10 mM PPT plus 1 mM NH$_4$NO$_3$ or KNO$_3$, respectively, at maturity (F).

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Figs. 3 and 4: Effect of N (10 mM NH$_4$NO$_3$), light, wind and adjacent leaf on inflorescence necrosis (3, above) resp. bunch-stem necrosis (4, below) of single-node Müller-Thurgau grape cuttings. Left bar of each pair: treatment factor present; right bar: treatment factor absent. Values are means ± se (n = 32). n.s., * non-significant and significant at P < 5 % and at P < 1 %, respectively.
ters from Müller-Thurgau cuttings taken after veraison developed more rapidly than IN. On an average of all treatments, 81% of the rachis tissues were necrotic after 7 d of incubation, as compared to 58% of the inflorescences after 10 d (Table). However, these clusters had been collected in the vineyard during a period of cool and rainy weather, and only 8 d later a severe incidence of BSN occurred in the field. Therefore, BSN may already have been induced when the cuttings were taken. Nevertheless, as with IN, the percentage of BSN was always very high if the adjacent leaf was excised. When the leaf was left attached, the severity of BSN was increased in the shade, except for the treatment combination +N/-light/+wind/+leaf, where the leaves remained green for a prolonged period. This combination also fully accounted for the statistical significance of the N effect on BSN (Fig. 4).

As shown in Fig. 5 for Pinot noir cuttings at anthesis, the adjacent leaf could be replaced by the supply of 2% sucrose as carbon source to the incubation solution. After 14 d of incubation, sucrose added to the NH$_4$NO$_3$ solution reduced IN to the level of the H$_2$O control and almost prevented the development of IN in the absence of NH$_4$NO$_3$.

In this latter treatment, berry development could be sustained until the pea-size stage without further sucrose supply. On Pinot noir cuttings taken after veraison, all leaves senesced and were abscised within one week, while BSN only developed after leaf abortion. The N concentration in the incubation solution did not significantly alter the severity of BSN, while the substitution of NH$_4$ for different cations reduced BSN, although the difference between NH$_4$ and H$^+$ was not significant. Metal cations however, in particular Mg$^{2+}$, effectively prevented the development of BSN after 17 d of incubation (Fig. 6).

Discussion

Our results suggest that GS is present and NH$_4^+$ assimilation may occur in all organs of the grape cluster at any stage of development between anthesis and maturity. Both IN and BSN could be induced by using PPT as GS inhibitor. Thus, the implication of NH$_4^+$ accumulation in the development of these disorders was confirmed indirectly. When attached grape inflorescences were immersed in PPT solutions, necrotic symptoms first developed on flowers and pedicels, while they first occurred on peduncles and rachis during incubation of detached clusters in PPT solutions. This is not surprising, since PPT is relatively immobile in plants, and it must have been taken up through the stomata and cuticula in the immersion experiment or through the cut end of the peduncle in the incubation experiments.

Necrotic symptoms developed earlier when NH$_4$NO$_3$ was added to PPT solutions as compared to KNO$_3$ plus PPT or PPT alone. Thus, the N effect was due to NH$_4^+$ supply, and the NO$_3^-$ reduction appears not to contribute significantly to NH$_4^+$ accumulation, regardless of the light conditions, even if GS is inhibited. This is consistent with our earlier results (Keller and Koblet 1994) and with data obtained by Wild et al. (1987), Gu et al. (1991) and Lohnertz (1991). Even the glycolate pathway, which was proposed by Wild et al. (1987) as the major source for NH$_4^+$ after PPT application, may have provided substantial amounts of NH$_4^+$ in inflorescence tissues only in the immersion experiment, but not in the incubation experiments conducted at light intensities close to the compensation point of photosynthesis, since photorespiration is reduced in proportion to net photosynthesis at low irradiance (Sharkey 1988). Therefore, the question remains, where the NH$_4^+$ that is accumulated is derived from.

The leaf area to fruit ratio is a widely used indicator of the source-sink balance, and separation of isolated sink organs from the parent source plant causes a stress itself due to carbon starvation, since interactions with other plant parts are no longer possible. Srivastava and Singh (1987) suggested that glutamate dehydrogenase (GDH) may be responsible for NH$_4^+$ assimilation under conditions of stress or high NH$_4^+$ levels, and both GS and GDH were reported to be active in grape berries by Ghisi et al. (1984). In our
incubation experiments, the inhibition of GS invariably resulted in senescence of the tissue, while in the absence of PPT, i.e. when GS was active, the addition of sucrose to NH$_4$NO$_3$ solutions delayed the occurrence of IN, although sucrose has been shown to inhibit GDH (SRIVASTAVA and SINGH 1987) and to increase NO$_3$ reduction (ASLAM and HUFFAKER 1984) under experimental conditions similar to those used in our study.

Thus, GDH does not appear to contribute to NH$_4^+$ assimilation or detoxification, as described by WALLS-GROVE et al. (1983) and GU et al. (1994). On the contrary, ROBINSON et al. (1992) reported that GS activity decreased and GDH activity increased during periods of carbon starvation, due to protein degradation to supply carbon skeletons to the tricarboxylic acid (TCA) cycle. Protein breakdown is related to NH$_3$ release and senescence (JOY 1988), and RABE (1990) assumed that any stress causing glucose depletion and/or reduced growth results in NH$_3$ accumulation. Therefore, we suppose that both IN and BSN are the visible manifestations of senescence processes induced by stress situations leading to carbon starvation. The accumulation of NH$_3$ may, hence, be a secondary effect of senescence. This hypothesis is supported by the fact that sucrose was equally effective in preventing the development of IN as was the presence of a leaf. Under non-stress conditions, as it was the case in the experiment using attached clusters in the field, there seems to be only insignificant NH$_4$/NH$_3$ production, since ripe grapes failed to develop necrotic symptoms after immersion in PPT solutions. On the other hand, the large variance within treatments in the incubation experiments may partly be due to different numbers of flowers or berries per unit leaf area. In addition, the flowers of the same inflorescence do not reach the same stage of development simultaneously (SWANEPOEL and ARCHER 1988), and thus, some flowers may have gained competitive advantage over the other flowers.

Incubation of excised clusters in PPT solutions took 1 d at anthesis, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms. Between berry set and veraison, 9 d at veraison and 5 d at maturity for development of necrotic symptoms.

In addition to refixation, NH$_3$ may be emitted from the sub-stomatal cavity to the atmosphere (FARQUHAR et al. 1990, SCHÖNERING et al. 1991). The beneficial effect of the wind treatment in the incubation experiments may have been brought about by stimulation of NH$_3$ emission due to reduced boundary layer resistance and increased NH$_3$ vapor pressure gradient. This suggests that the severity of BSN may be reduced by removal of old leaves in the cluster zone to facilitate air movement around grape bunches, which is a common canopy management practice in temperate climate zones.

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