Effects of KCN and Salicylhydroxamic Acid on the Root Respiration of Pea Seedlings

TREVOR WEBB AND WILLIAM ARMSTRONG

Department of Plant Biology, University of Hull, Hull HU6 7RX, England

Received for publication September 30, 1982 and in revised form February 3, 1983

ABSTRACT

Polarography, using cylindrical platinum electrodes, proved suitable for measuring changes in the internal apical O$_2$ concentration of the primary root of pea (Pisum sativum L. cv Meteor) effected by KCN and/or salicylhydroxamic acid (SHAM) in the bathing medium. An electrical root-aeration analog was used to help evaluate some of the results. Concentrations of KCN <0.05 millimolar had no significant effect. In response to 0.1 millimolar KCN, the O$_2$ concentration rose substantially for approximately 2 hours, then declined, and after 10 hours had frequently fallen below the pretreatment level. Such changes suggest an initial inhibition of cytochrome oxidase-mediated O$_2$ uptake followed by an induction of the alternative, cyanide-resistant respiratory pathway. These treatments proved nonlethal. Changes in O$_2$ concentration similar to those described for 0.1 millimolar KCN were observed in response to 1 and 10 millimolar KCN but these treatments were lethal and the root apex became soft and often appeared flooded. Roots survived and showed no significant responses when treated with SHAM at concentrations <5 millimolar. However, when the alternative pathway had been (apparently) induced by 0.1 millimolar KCN, the addition of 5 millimolar SHAM to the bathing medium caused a substantial and persistent rise in the root apical O$_2$ concentration, suggesting that this (nonlethal) concentration of SHAM could indeed inhibit O$_2$ uptake via the cyanide-resistant pathway.

It is concluded that while O$_2$ uptake normally occurs by the cytochrome pathway in the primary pea root, the alternative, cyanide-resistant pathway can be induced by 0.1 millimolar KCN.

Cyanide is a known inhibitor of Cyt oxidase yet the capacity to respire in its presence appears to be widespread throughout the plant kingdom (3, 5). There is now general agreement that cyanide-insensitive respiration is the result of an electron flow which bypasses the classical, Cyt pathway at the position of ubiquinone (20, 21), O$_2$ being ultimately reduced by an 'alternative' oxidase. Although the nature of the alternative oxidase has yet to be identified, it is known that electron flow to this terminal oxidase is insensitive to cyanide but sensitive to the hydroxamic acid group of compounds, e.g. SHAM (2) (16).

Despite the apparently widespread distribution of cyanide-insensitive respiration, its physiological significance remains uncertain. Several areas in which such respiration might be of importance have been reviewed by Lambers (7) and these include roles in heat production, fruit ripening, respiration of plants that contain high levels of cyanogenic glycosides producing HCN upon wound-oxidation of NADH, and ion transport and osmoregulation.

Also, Lambers suggests that cyanide-insensitive respiration might play an important role (at least in roots) in the oxidation of sugars that have been translocated to the roots in excess of their demand for energy production, carbon skeletons for structural growth, storage, and osmoregulation.

Reports in which O$_2$ uptake by intact organs has been measured are limited yet Lambers has stressed the importance of such measurements when attempting to elucidate the physiological significance of the alternative pathway. In those cases where whole root respiration has been considered, the effect of inhibitors on O$_2$ uptake from the root-bathing medium has been monitored (8, 9, 11). However, because some of the O$_2$ respired by the roots of whole plants is received from the shoot, changes in O$_2$ concentration of the bathing medium can be misleading; the diffusional impedances of suberized (O$_2$-impermeable) root surfaces and unstirred boundary layers at these surfaces can also considerably influence O$_2$ exchange. Consequently, in the present investigation, changes in respiratory activity in vivo in the primary pea root were observed as changes in internal apical O$_2$ concentration. Pea seedlings were arranged with their roots bathed in a deoxygenated liquid:agar medium and the root apex received O$_2$ from the shoot by gaseous diffusion through the interconnecting gas-space continuum of the cortical ground tissues. A cylindrical platinum electrode (cathode) ensheathed the primary root apex and ROL to the electrode was monitored polarographically; the corresponding apical O$_2$ concentration of the root gas-space was calculated from the ROL (1). Using this technique, a reduction in O$_2$ uptake is reflected by an increase in ROL; conversely, an increase in respiratory rate causes a decrease in ROL. Data from an electrical root aeration analog (1, 2) helped in evaluating the experimental results.

The aims of this study were to establish the suitability of the polarographic technique for determining root respiratory changes in vivo effected by inhibitors in the bathing medium and to assess the potential for cyanide-resistant respiration in the primary pea root.

MATERIALS AND METHODS

Plant Material. Seeds of Pisum sativum L. cv Meteor were surface sterilized, soaked in sterile distilled H$_2$O, and germinated in the dark, at 25°C, under aseptic conditions. Germinated seeds (radicle length, 1 to 2 cm) were aseptically transferred to Pyrex cylinders containing sterilized 1% (w/v) agar prepared in a nutrient solution one-quarter the strength of that described by Hoagland and Snijder (6). Black polythene excluded light from the growth medium and seedlings were allowed to develop in diffuse light, at 25°C, until the primary root was about 5 cm in length (i.e. prior to lateral root appearance).

Inhibitors. Solutions of KCN and SHAM were prepared immediately prior to use in an anaerobic 0.05% (w/v) agar medium. It was necessary to heat the agar medium to dissolve the SHAM at concentrations above 5 mM; the solution was then cooled.

1 To whom reprint requests should be sent.

2 Abbreviations and trivial name: SHAM, salicylhydroxamic acid; ROL, radial oxygen loss; disulfiram, tetraethylthiuram disulfide.
SHAM was obtained from Sigma Chemical Co.

Arrangement for ROL Assay. Seedlings were supported in rubber stoppers using moist cotton wool, with the primary root bathed in 100 ml of the anaerobic agar medium (minus inhibitor) in shortened boiling tubes. The 1% (solid) agar in which the seedlings were cultivated was completely removed to ensure that the whole of the root was accessible to any added inhibitor. An Ag/AgCl anode and a small glass tube projected through the rubber stopper to the bottom of the experimental vessel. Moist air was applied to the shoot via a glass chamber which screened the aerial regions of the plant, and possessed gas inlet and outlet tubes. A cylindrical Pt cathode was arranged to ensheathe the root apex above the meristem and the ROL and internal apical O2 concentration were determined as described by Armstrong (1). One-half of the agar solution was then carefully removed via the glass tube and replaced by an equal volume of anaerobic medium in which twice the required concentration of inhibitor had been dissolved. This concentration was such as to give the desired level of inhibitor when diluted with the anaerobic solution remaining in the vessel.

Inasmuch as the anode and cathode were positioned at the bottom of the experimental vessel, the removal of only one-half of the agar solution maintained the electrical contact necessary for O2 reduction. In this way, a continuous polarographic trace of radial O2 loss from the root apex could be maintained and the inhibitor effects constantly observed.

At the conclusion of the experiments, plants were removed and the root system was washed repeatedly with distilled H2O. The length and apical radius of the experimental roots were recorded and the plants were arranged with their roots bathed in aerated, quarter-strength Hoagland solution (6); the shoots were aerated with moist air. Roots which subsequently increased in length were considered to have survived the inhibitor treatment; conversely, a failure to elongate was considered as evidence of irreversible inhibitor toxicity.

Presentation of Results. In the literature, changes in O2 uptake in response to respiratory inhibitors are normally expressed as a percentage of the pretreatment (uninhibited) respiratory rate. However, inasmuch as the changes in apical O2 concentration measured by the polarographic technique are only a reflection of respiratory changes, the results from this technique need to be presented in a different manner. One possibility is to express the change in O2 concentration as a percentage of the original, pretreatment value. Unfortunately, due to differences in respiratory demand, length, and other diffusion characteristics, the initial apical concentration varied between 2 and 8% O2 and changes expressed as a percentage of the original value were so exaggerated as to make comparison between plants extremely difficult. Consequently, the changes in O2 concentration effected by the inhibitors were expressed as a numerical rise or fall in the percentage of apical O2 concentration relative to the initial concentration; this method was considered to bring the changes within reasonable bounds and allow more meaningful comparisons to be made between individual plants.

In the figures, the apical O2 concentration immediately prior to the addition of the anaerobic agar ± the respiratory inhibitor was taken as the baseline and given a numerical value of zero (time 0 h). The subsequent changes in the percentage of O2 concentration after varying incubation periods are related to this baseline. In the majority of cases, the figures show the initial (0.25 h) changes in O2 concentration, and those after 1, 2, 5, and 10 h of a particular treatment. The electrical current values from which these O2...
Fig. 2. The numerical rise (+) or fall (−) with time in the percentage of apical \( O_2 \) concentration of pea roots, relative to that at time 0, in response to A, 0.01 and 0.05 mM KCN, and B, 0.1 mM KCN (five replicates). The broken line indicates the mean (and range) of the control pea results in which one-half of the root-bathing medium was replaced with anaerobic agar solution minus inhibitor (see Fig. 1C).

Table 1. Effect of 1 and 10 mM KCN on Apical Oxygen Concentration

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mean Numerical Increase in the Percentage ( O_2 ) Concentration Relative to That at Time 0</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.25 h</td>
</tr>
<tr>
<td>1 mM KCN</td>
<td>3.25</td>
</tr>
<tr>
<td>10 mM KCN</td>
<td>3.55</td>
</tr>
<tr>
<td>1 mM KCN*</td>
<td>0.50</td>
</tr>
<tr>
<td>10 mM KCN*</td>
<td>1.03</td>
</tr>
</tbody>
</table>

* Preincubation in 0.1 mM KCN (see Fig. 2B for general response to 0.1 mM KCN).

Concentrations were calculated were obtained directly from the polarographs and are accurate to two decimal places. Continuous and, hence, smooth records of the currents (equivalent to internal \( O_2 \) concentration) were also monitored on chart recorders, but for convenience, the changes in \( O_2 \) concentration expressed in the figures are linked by straight lines. It must be emphasized that only the experimental points shown in the figures are therefore truly representative of the change in apical \( O_2 \) concentration after a given time period; nevertheless, changes at intermediate time intervals could be extrapolated from the chart records.

Controls. The physicochemical effect of KCN and SHAM on the electrolytic reduction of \( O_2 \) was determined by observing changes in the polarographic measurement of ROL (and, hence, apical \( O_2 \) concentration) from artificial silicone-rubber roots (1). Neither 0.15 mM KCN nor 1 mM SHAM caused a significant change in the \( O_2 \) concentration of such roots over a 20-h period (Fig. 1A), suggesting that these chemicals had little effect per se on the reduction of \( O_2 \) by the Pt cathode.

The physical effect of the solution change on the ROL was determined in plant controls in which the anaerobic root bathing medium was replaced by anaerobic medium minus inhibitor. The results for nine replicates are shown in Figure 1B; the mean (and the range) of these changes after a given time interval is shown in Figure 1C. A slight increase in the pea root apical \( O_2 \) concentration occurred during the first 0.25 h, but after 1 h, the concentration had almost approached the baseline value, and remained so for at least 2 h. Consequently, it would appear that the physical effect of the solution change on the apical \( O_2 \) concentration was virtually negligible. In the longer term (2 to 10 h) the apical concentration increased slightly in some cases, but decreased in others; the mean
RESULTS

Effect of KCN. When compared with the controls (Figs. 1A and 2A, curve c), the apical \( \text{O}_2 \) concentration varied little during a 10-h incubation in 0.01 and 0.05 mM KCN (Fig. 2A, curves a and b). Roots survived at these cyanide concentrations, elongating during the treatments and in the postincubation in aerated Hoagland solution. In contrast to the above, there was an immediate increase in the root apical \( \text{O}_2 \) concentration in response to 0.1 mM KCN and, after 2 h, the rise in concentration was between 3.39 and 6.32% \( \text{O}_2 \) (Figs. 2B and 5); an analog simulation showed that a rise of this magnitude accords with a 50% reduction in the pretreatment respiratory rate. Thereafter, the concentration fell steadily, and frequently declined to a level below the pretreatment concentration (Fig. 2B, curves a–d): the analog showed that such changes could be brought about by a rise of 15% in the pretreatment respiratory rate. The response to 0.1 mM KCN was clearly in excess of that exhibited by the control pea roots (Fig. 2B, curve f) and roots survived this cyanide treatment.

The initial response to 1 and 10 mM KCN (Table I) was similar to that effected by 0.1 mM KCN (Fig. 2B), i.e. a significant increase in the apical \( \text{O}_2 \) concentration, followed in some cases by a decline. This decline, however, was much less accentuated than for 0.1 mM...
KCN. These higher concentrations of cyanide also caused root death. At the conclusion of these treatments, the primary root was soft and became flooded during the postexperimental incubation in aerated Hoagland solution. Cyanide, at concentration of 1 and 10 mM also caused a substantial, and sustained, increase in the apical O₂ concentration of roots which had been pre-incubated in 0.1 mM KCN (Table I); root death again ensued in all cases.

Effect of SHAM. The changes in O₂ concentration in response to 0.1, 1, and 5 mM SHAM (Fig. 3A, curves a, b, and c, respectively) were insignificant when compared with those exhibited by the control, artificial root (Fig. 1A), and the mean of the control pea roots (Fig. 3B, curve d). Growth continued during and after the treatments.

Although the initial (0 to 1 h) response to 10 and 25 mM SHAM was variable, the apical O₂ concentration then increased in all cases and after 10 h was substantially greater than the initial concentration (Fig. 3B, curves a-c). Root death resulted in all cases; at the end of the experiments, all roots were soft and the apical several centimeters became flooded during the postexperimental incubation in Hoagland solution.

Effect of Combinations of KCN and SHAM. The results in Figure 4 show the effect of 0.1 mM KCN in combination with 0.1, 1, or 5 mM SHAM. The responses to 0.1 mM KCN + 0.1 mM SHAM and 0.1 mM KCN + 1 mM SHAM (Fig. 4, curves a and b, respectively) were similar to that effected by 0.1 mM KCN alone (Fig. 2B), i.e. an initial increase in apical O₂ concentration followed by a decline after about 2 h, and roots survived these treatments. The response to 0.1 mM KCN + 5 mM SHAM was clearly different, however, in that the apical O₂ concentration rose substantially (viz. >8% O₂) and remained at this level over a 10-h period (Fig. 4, curve c); also this treatment proved lethal.

In further tests, pea roots were pre-incubated in 0.1 mM KCN and this was followed by a 0.1 mM KCN + 5 mM SHAM treatment; a representative example of four results is shown in Figure 5, curve a. In response to the 0.1 mM KCN pretreatment, the O₂ concentration increased and then declined. When the combined inhibitors were added (at 8 h in Fig. 5a), the apical O₂ concentration increased almost immediately and by 6.2 to 9.2% O₂ after 3 h (i.e. at 11 h in Fig. 5a), relative to the concentration at time zero; the concentration then approximately stabilized at this level during the following 12 h. Roots failed to survive this treatment. In control roots, only a slight change in the apical O₂ concentration was effected by a second exposure to 0.1 mM KCN (minus SHAM) (Fig. 5, curve b from 8 to 22 h) and by the addition of 5 mM SHAM in the absence of cyanide (Fig. 5, curve c from 8 to 22 h, and Fig. 5, curve e from 0 to 8 h). Also, when the root bathing medium was first replaced with anaerobic medium alone (Fig. 5, curves c and d at time 0) or 5 mM SHAM (Fig. 5, curve e at time 0), an insignificant change in the apical O₂ concentration was apparent when compared with the control pea roots (Fig. 1B and C) and the artificial control root (Fig. 1A). However, further changing the medium to 0.1 mM KCN + 5 mM SHAM (Fig. 5, curves d and e at 8 h) caused an almost immediate and substantial increase in the apical O₂ concentration which, after 3 h (i.e. at 11 h in Fig. 5, d and e) could be as great as 6 to 8% O₂; again, this treatment proved lethal.
A significant net increase in the apical \( O_2 \) concentration (by up to 5.10 and 4.16% \( O_2 \)) was apparent in response to a 10-h incubation in 1 mM KCN + 25 mM SHAM and 10 mM KCN + 25 mM SHAM, respectively. Without exception, root apices were soft at the end of these treatments and death resulted; some contraction of the root was also evident.

**DISCUSSION**

The most striking response to cyanide was that effected by 0.1 mM KCN. The substantial initial increase in apical \( O_2 \) concentration (by 3.39 to 6.32% \( O_2 \); Figs. 2B and 5) almost certainly reflects an inhibition of Cyt oxidase activity and the analog studies suggest that such changes could result from a 50% reduction in the initial respiratory rate. However, the subsequent marked decline in \( O_2 \) concentration (Fig. 2B from 2 to 10 h) strongly suggests a renewal of respiration, and presumably represents a stimulation of the alternative, cyanide-resistant pathway. The net decline in \( O_2 \) concentration (Fig. 2B, curves a–d) could be realized in the analog root by stimulating the initial respiratory rate by 15% and it is possible, therefore, that \( O_2 \) uptake by the alternative pathway ultimately exceeded that of the initial Cyt respiration.

The induction of the alternative pathway by 0.1 mM KCN is clearly supported by the responses to combinations of 0.1 mM KCN + 5 mM SHAM. When these inhibitors were added to previously untreated roots, the initial increase in \( O_2 \) concentration again occurred but was sustained (Fig. 4, curve c), suggesting that the presence of 5 mM SHAM had prevented the induction of the alternative pathway. The addition of 0.1 mM KCN + 5 mM SHAM following 0.1 mM KCN pretreatment (during which the alternative pathway had been induced; Fig. 5, curve a from 0 to 8 h) also caused a substantial and sustained increase in the apical \( O_2 \) concentration (Fig. 5, curve a from 8 to 22 h). A control in which the 0.1 mM KCN pretreatment was followed by a second exposure to 0.1 mM KCN (minus SHAM) indicated that the solution change per se had little effect (Fig. 5, curve b). Consequently, the substantial increase in \( O_2 \) concentration effected by 0.1 mM KCN + 5 mM SHAM (Fig. 5, curve a) appears to have been caused solely by the hydroxamic acid. Inasmuch as 5 mM SHAM alone caused an insignificant change (Fig. 5, curve c from 8 to 22 h, and Fig. 5, curve e from 0 to 8 h), we conclude that alternative respiration is not normally active in the primary pea root but may be induced by 0.1 mM KCN, and that respiration under physiological conditions is predominantly supported by Cyt oxidase.

Whereas treatments of 0.1 mM KCN and 5 mM SHAM alone proved nonlethal, in combination they caused a softening of the root apex and a failure to regrow during the postexperimental incubation in aerated Hoagland solution. This was not altogether surprising, however, considering that both the cyanide-sensitive and cyanide-resistant respiratory pathways were presumably inhibited by the combined inhibitors.

The stabilization of the apical \( O_2 \) concentration in response to 0.1 mM KCN + 5 mM SHAM, indicating an increase of >8% \( O_2 \) (Fig. 4, curve c), correlates well with the rise in the analog root apex (by 7.73% \( O_2 \)) when respiration was totally inhibited, and presumably represents the maximum inhibition of Cyt oxidase effected by the 0.1 mM KCN. The relatively lower initial increase in response to 0.1 mM KCN alone (Fig. 2B) presumably reflects the balance between inhibition of Cyt oxidase (by 0.1 mM KCN) and the degree to which respiration via the alternative pathway is stimulated.

The insignificant response to 0.01 and 0.05 mM KCN (Fig. 2A,
curves a and b) suggests that these low cyanide concentrations are ineffective in inhibiting Cyt oxidase activity. The failure of 0.1 and 1 mM SHAM to prevent the induction (by 0.1 mM KCN) of the alternative pathway (Fig. 4, curves a and b) suggests also that these concentrations of SHAM are too low to inhibit the alternative oxidase when applied to the intact pea root.

Although an overall increase in the apical O2 concentration was effected by individual treatments of 1 and 10 mM KCN (Table I), and 10 and 25 mM SHAM (Fig. 3B), in view of the lethal response to these concentrations, it is not possible to link any changes in O2 concentration with changes in the activity of a specific respiratory pathway.

**FINAL COMMENTS**

Lipoxygenase, an enzyme which catalyzes the dioxygenation of certain unsaturated fatty acids, exhibits a similar insensitivity to cyanide and inhibition by SHAM as the alternative oxidase (14, 17). When investigating O2 uptake by the alternative pathway, it is necessary, therefore, to quantify or eliminate the possible contribution of lipoxygenase-mediated O2 consumption. Disulfiram, which inhibits the alternative oxidase but not lipoxygenase (4), has recently been featured in studies on respiratory activity in mitochondrial preparations (13, 17) and may prove to be a useful tool to distinguish between alternative respiration and lipoxygenase activity in *vivo*. Unfortunately, the use of disulfiram in *vivo* is restricted, due to either a limited penetration into the tissue or its dissipation in the cytosol (4). At this stage, it is not possible to comment on the activity of lipoxygenase in the intact pea root although there are grounds for considering it to be insignificant in intact tissue (12). However, 5 mM SHAM apparently inhibited O2 uptake only in the presence, and not in the absence, of 0.1 mM KCN, suggesting that neither the alternative oxidase nor lipoxygenase is significantly active under normal circumstances in the primary pea root. Consequently, unless lipoxygenase activity is itself stimulated by cyanide, then the results presented strongly support the thesis that the large increase in apical O2 concentration effected by 5 mM SHAM in the presence of 0.1 mM KCN (Fig. 4, curve c, and Fig. 5, curves a, d, and e) is due entirely to an inhibition of both Cyt oxidase and the alternative oxidase.

A second point of interest to emerge from the results concerns the site of action of SHAM. Although Schonbaum et al. (16) consider the substituted hydroxamic acids to be specific inhibitors of the alternative oxidase, Rich et al. (15) suggest that they may act on a portion of the electron transport chain common to both the Cyt and alternative pathways. Again, however, the failure of 5 mM SHAM to affect significantly the pea root apical O2 concentration in the absence of cyanide (Fig. 5, curve e between 0 and 8 h) compared with the substantial response in the presence of 0.1 mM KCN (Fig. 5, curve e between 8 and 22 h) favors the view of Schonbaum et al. that SHAM (5 mM) specifically inhibits the alternative oxidase. In the present study, only at concentrations ≥10 mM is there any indication that SHAM may act to effectively curtail electron transport through both the Cyt and alternative pathways. Consequently, one must be very guarded when correlating the apparent changes in O2 concentration effected by lethal concentrations of inhibitor with changes in the activity of a specific respiratory pathway; also, the results might equally be explained in terms of changes in root wall permeability and/or flooding of the gas-space continuum arising from root death. The relevance of reports on O2 uptake by root systems bathed in what have proved to be lethal concentrations of inhibitor (at least in pea) must therefore be seriously questioned. In particular, Lammers and his colleagues have repeatedly employed 25 mM SHAM as a specific inhibitor of the alternative pathway (8–11, 19) yet they have failed to comment upon the effect of the inhibitor on root viability and integrity.

The results of this study are of further interest in that the respiration inhibitors generally curtailed root elongation. In response to a 10-h incubation in 0.1 mM KCN, Cyt oxidase was initially inhibited, followed by an induction of the alternative pathway, and root elongation never exceeded 2 mm. In the control (no inhibitor) treatment, elongation during this period could approach 9 mm. Elongation also varied between 4 and 8 mm when roots were incubated for 10 h in 0.1 and 1 mM SHAM and in 0.01 and 0.05 mM KCN (i.e. treatments in which the alternative pathway was neither operative initially nor induced). Presumably, the inability to extend significantly during treatments in which O2 uptake was predominantly via the alternative pathway (i.e. after an approximately 2-h incubation in 0.1 mM KCN) reflects the nonphosphorylative nature of this pathway beyond the branching point (viz. ubiquinone) (18). Roots could nevertheless survive a 20-h incubation in 0.1 mM KCN, indicating that the Cyt pathway may be inhibited without undue damage to the root. Consequently, one may speculate that a possible physiological role for alternative respiratory activity in roots might be to prolong viability during times when the activity of the Cyt respiratory pathway may be temporarily suppressed.

**Acknowledgments**—We thank the Science and Engineering Research Council (U.K.) for their financial support. We are grateful to Miss E. M. Sharpe and Mrs. J. Minto for typing the manuscript.

**LITERATURE CITED**

2. ARMSTRONG W, EJ WRIGHT 1976 An electrical analogue to simulate the oxygen relations of roots in anaerobic media. Physiol Plant 36: 383–387
7. LAMBERS H 1980 The physiological significance of cyanide-resistant respiration in higher plants. **Plant Cell Environ** 3: 293–302
12. LATIES GG 1982 The cyanide-resistant, alternative path in higher plant respiration, **Annu Rev Plant Physiol** 33: 519–555
13. MILLER MG, RL OEDENBO 1981 Use of tetrathyiylthiiriam diisulfide to discriminate between alternative respiration and lipoxygenase. Physiol Plant 67: 962–964

Downloaded from on November 11, 2017 - Published by www.plantphysiol.org
Copyright © 1983 American Society of Plant Biologists. All rights reserved.