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Mechanism for Photoinactivation of PSII by Methyl Viologen at Two Temperatures in the Leaves of Rice (*Oryza sativa* L.)

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We studied the photooxidative effects of methyl viologen (MV) on PSII in rice (*Oryza sativa* L.). Leaves were held at either room temperature (RT) or 4°C. In the presence of MV, the photochemical efficiency of PSII, or F_v/F_m, was more depressed at RT than at the low temperature (LT), but the loss of D1 protein that was detected at RT was not observed at LT. However, the decline in the content of functional PSII, $1/F_0 - 1/F_m$, was similar for MV-treated leaves at either temperature. These results suggest that, at LT, PSII is not protected from MV-induced photooxidation, although degradation of the D1 protein is delayed. The $1/F_0 - 1/F_m$ decreased by MV treatment at RT was significantly recovered during dark incubation for 2 h. Recovery of a small portion of $1/F_0 - 1/F_m$ was also possible, even for tissues treated with MV at LT. Therefore, we believe that MV-induced reversible photoinactivation may exist. This possibility is further discussed in terms of changes in the de-epoxidation state and the rate of PSII-driven electron transport.

Keywords: chlorophyll fluorescence, electron transport rate (ETR), low temperature, methyl viologen (MV), photooxidation, rice, xanthophyll cycle

Methyl viologen (paraquat, 1,1'-dimethyl-4,4'-bipyridylium; MV) has been used to study photooxidative stress (Asada and Takahashi, 1987; Bowler et al., 1994). This potent herbicide mediates a cascade of reactive oxygen species (ROS) by producing superoxide ($\cdot O_2^-$) at PSI sites via the photoreduction of dioxygen. MV accepts electrons from the iron-sulfur cluster Fe-S_A/Fe-S_B of PSI (Fujii et al., 1990), which results in a depletion of NADPH and the inhibition of CO_2 fixation (Dodge, 1971; Preston, 1994). The ROS cascade starts with the dismutation of $\cdot O_2^-$ into H_2O_2 by superoxide dismutase (SOD). When catalytic free metals, e.g., Fe(II) or Cu(I), exist near the production site, the most toxic form of ROS, hydroxyl radical ($\cdot$OH), will be produced from both ROS. These ROS affect the entire photosynthetic machinery, causing membrane leakage because of lipid peroxidation and/or inducing the disassembly of photosystems by the degradation of chlorophylls and proteins.

At low temperatures (LTs), CO_2 fixation and the PSII-driven electron transport rate (ETR) can be significantly depressed (Xu et al., 1999). The decrease in CO_2 fixation is involved in the sensitivity of several key enzymes to low temperatures (Sassenrath et al., 1990; Brüggemann et al., 1992; Byrd et al., 1995), while decreased ETRs may be due to thylakoid phase transitions or an abrupt change in protein/lipid interactions (Brüggemann and Linger, 1994).

Little information has been reported about the action and effect of MV on both PSII and PSI at LT. Sonoike (1996) has proposed that MV could have a protective role as an additional electron acceptor for PSI at LT by relieving the over-reduction of the PSI acceptor side, which is often referred to as the acceptor-side limitation of PSI. Nevertheless, the photooxidative effect of MV on PSII at LT still remains to be investigated.

In the present study, we attempted to elucidate the photooxidation and protection mechanisms of PSII against MV at room temperature (RT) compared with that found at LT. To do so, we investigated the photochemical efficiency of PSII (F_v/F_m), D1 protein contents, the level of functional PSII, the rate of PSII-driven photosynthetic electron transport, and xanthophyll-cycle activity.

MATERIALS AND METHODS

Plant Material

Rice (*Oryza sativa* L. cv. Dongjin-byeo) plants were raised in a growth chamber. A pot-level photosynthetic photon flux density (PPFD) of 100 μmol photons m^{-2} s^{-1} was supplied by metal halide lamps. The growth chamber was maintained at 28/23°C (day/night), with a 14-h photoperiod. For all experiments, 3-cm-long segments were excised from the fully expanded leaves of 4- to 6-week-old seedlings.

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Chemical Treatment

To investigate the photooxidative effect of MV, we floated the leaf segments abaxial-side down on either 10 μM MV or water. Afterward, the segments were exposed to 50 μmol light $m^{-2} s^{-1}$ at RT for 1 h, then transferred to darkness for 1 h (a total of 2 h for pre-incubation). The segments were next divided into two groups, which were further exposed to 150 μmol light $m^{-2} s^{-1}$ for 3 h at either RT or LT, 4°C. This served as our photooxidative treatment. During the entire experimental period (i.e., pre-incubation and photooxidation), the leaf segments remained atop the MV or water.

Measurement of Chlorophyll (Chl) Fluorescence

To monitor the photooxidative effect of MV on PSII, we measured the ratio of variable fluorescence (F_v) to the maximum yield of fluorescence (F_m), using a portable fluorometer (Plant Efficiency Analyzer; Hansatech Instrument, Norfolk, UK). Readings were taken after the samples were dark-adapted for 10 min at RT. Variable fluorescence was obtained by subtracting the initial Chl fluorescence (F_0) from the maximum yield of fluorescence. So that $1/F_0 - 1/F_m$ would serve as the measure of functional PSII units, all fluorescence yields (F_0 and F_m) were normalized to the mean F_0 value of the controls, according to the method of Kim et al. (2001). The physical meaning of this value is true only when the two parameters were measured after a dark-recovery period.

The relative PSII-driven ETR was calculated as $(1 - F_s/F_m') \times 0.5 \times PPFD \times$ leaf absorptance, according to Genty et al. (1989), where F_s is the steady-state fluorescence yield, F_m' is the maximum yield of fluorescence in light-acclimated leaves, and 0.5 is a constant assuming an equal distribution of absorbed photons between PSII and PSI. Leaf absorptance was taken as 0.85. For the ETR analysis, Chl fluorescence was measured with a pulse-amplitude modulated fluorometer (PAM-2000; Walz, Effeltrich, Germany).

Isolation of Thylakoid Membranes and Western Blot Analysis

Thylakoid membranes were isolated from the leaf segments according to the procedures of Rintamäki et al. (1995, 1996), with some modifications. After Chl contents were determined by the method of Arnon (1949), the membranes were solubilized in 100 mM Tris-HCl (pH 6.8), 17% (w/v) glycerol, 3.5% (w/v) SDS, 6 M urea, and 10% (v/v) β-mercaptoethanol for 30 min at 70°C. They were then subjected to SDS-PAGE using a gel containing 6 M urea. The polyacrylamide concentration in the separation gel was 15%. Each lane was loaded with thylakoid-membrane proteins equivalent to 0.5 μg of chlorophyll. After electrophoresis, the separated proteins were immunoblotted with rabbit antibodies raised against the DE loop (Residues 225 to 249) of the spinach D1 protein.

Pigment Analysis

Photosynthetic pigments were analyzed according to the technique of Gilmore and Yamamoto (1991). Five leaf segments were frozen in liquid nitrogen and ground with a mortar and pestle in ice-cold 100% acetone. The pigment extracts were filtered through a 0.2-μm syringe filter. Pigment separation was performed in an HPLC system (HP 1100 series; Hewlett Packard, Waldbronn, Germany) on a Spherisorb ODS-1 column (Alltech, USA), as described by Gilmore and Yamamoto (1991). Concentrations of the pigments were estimated by using the conversion factors for peak area (in nanomoles) that were calculated for this solvent mixture by Gilmore and Yamamoto (1991). The de-epoxidation index (D_i) was calculated as a percentage of $A \times 0.5 + Z$ of $V + A + Z$, where A is antheraxanthin, Z is zeaxanthin, and V is violaxanthin. This parameter was then used to indicate the degree of de-epoxidation in the xanthophyll cycle.

RESULTS

MV Effect on Photochemical Efficiency of PSII

When rice leaf segments were treated at RT, MV caused significant photooxidative damage to PSII activity. This was demonstrated by the decrease in photochemical efficiency, or F_v/F_m (Fig. 1). Although PSII activity rapidly decreased to just 34% of that in the control samples at RT, activity at LT (4°C) was only mildly reduced, to 83% of the control level. In both cases, PSII activity decreased in a time-dependent manner over the 3-h treatment. At LT, the decrease in activity in the controls was thought to be associated with photoinhibition, because the light intensity during treatment was higher than that used in the growth chamber. Even moderate light levels are known to damage photosystems at LT. However, MV seemed to protect PSII against such photooxidative damage when the PPFD was set at 150 μmol $m^{-2} s^{-1}$.

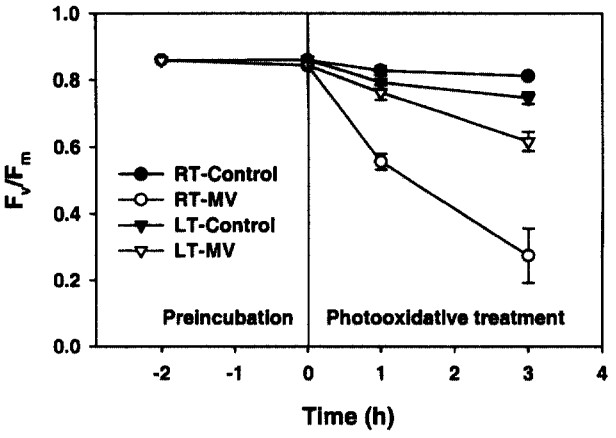

Figure 1. Decrease in photochemical efficiency of PSII, F_v/F_m, by MV at two temperatures. Leaf segments were pre-incubated with 10 μM MV under a PPFD of 50 μmol m^{-2} s^{-1} for 1 h at RT, then held in darkness for 1 h. For photooxidative treatment, segments were exposed to a PPFD of 150 μmol m^{-2} s^{-1} for 3 h at either RT or 4°C. F_v/F_m was measured after 10 min of dark-adaptation at RT. Error bars indicate SE (n = 3 - 5).

MV Effect on Changes in Chl Fluorescence Parameters

MV treatment at LT caused an increase in the initial Chl fluorescence (F_0) and a decrease in the maximal yield of fluorescence (F_m), finally resulting in a reduced value for F_v/F_m (Fig. 2). In contrast, this compound caused both F_0 and F_m to decrease at RT, with the effect being more prominent with F_m. This action was the main contributor to the rapid decrease in F_v/F_m (see MV of Fig. 2). However, when leaf segments were treated with MV at LT, its negative effect on F_m was considerably inhibited, and F_0 was rather increased. We interpret this to mean that the decline in PSII activity caused by MV at RT is probably mediated by a different mechanism than that involved in MV-induced photooxidation at LT.

Relationship of the Decrease in PSII Activity to the Loss of the D1 Protein

The decrease in PSII activity during photoinhibition at LT (as well as its inhibition at RT) is frequently attributed to the loss of the D1 protein, a core component of PSII (Aro et al., 1990, 1993; Eu et al., 1996). In the present study, however, the damage incurred by LT treatment for 3 h under 150 μmol photons m^{-2} s^{-1} did not cause significant protein loss. This was indicated by only a slight decrease in values for the Chl fluorescence parameters (Fig. 2). In contrast, MV treatment at RT prompted the D1 protein to be lost in a concentration-dependent

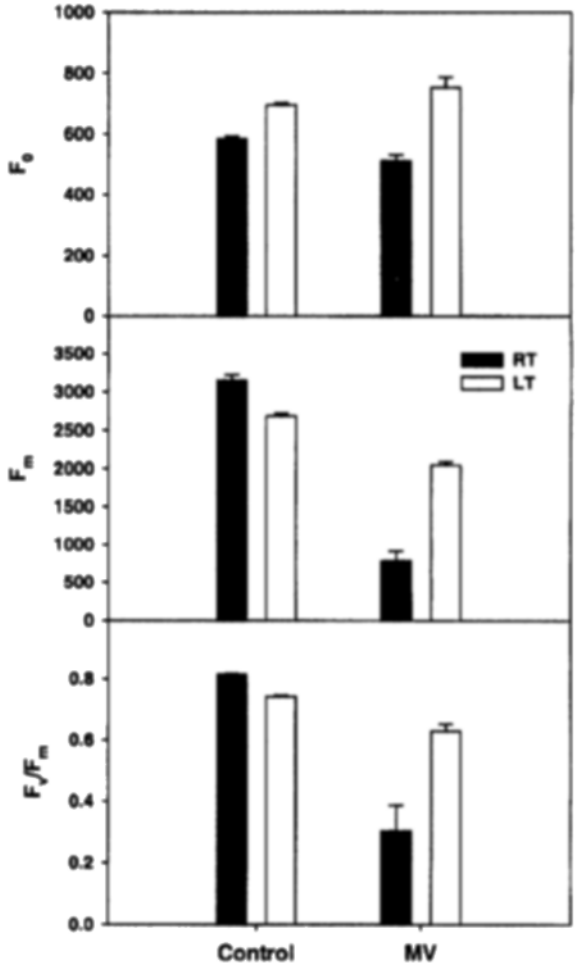

Figure 2. Changes in Chl fluorescence yields, F_0 and F_m, by MV at two temperatures. Experimental conditions are as described with Figure 1. Initial Chl fluorescence (F_0) and maximum yield of fluorescence (F_m) were measured after dark-adaptation for 10 min at RT. Error bars indicate SE (n = 5).

manner; 10 μM MV, a level which had also promoted significant changes in PSII activity, was sufficient to cause a significant decline in protein content (Fig. 3A). However, this loss was not observed in leaf segments exposed to MV at LT (Fig. 3B).

MV Effect on Changes in the Content of Functional PSII

The decrease in PSII activity because of MV was significantly less at LT than at RT (Fig. 1), a trend that was well correlated with the loss of D1 protein (Fig. 3). However, this observation does not imply that the D1 protein, as protected from degradation in MV-treated leaves at LT, was necessarily in the functional state. Moreover, one cannot exclude the possibility that other

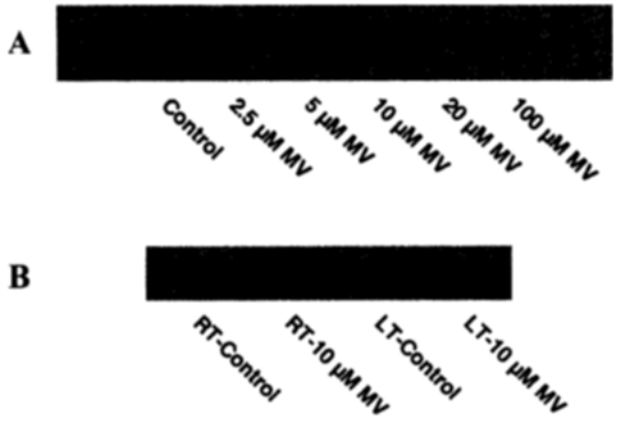

Figure 3. Difference in the loss of D1 protein by MV at two temperatures. **A,** Rice leaf segments treated with various concentrations of MV (2.5, 5.0, 10.0, 20.0, and 100.0 μM) at RT. **B,** Segments treated with 10 μM MV at RT and at LT. For both **A** and **B,** experimental conditions are the same as described with Figure 1.

components of the PSII complexes, including the peripheral proteins, may have been involved in this decreased activity. Therefore, to determine the actual damage to PSII, the content of functional PSII was quantified by a Chl fluorescence parameter, $1/F_0 - 1/F_m$. This value is regarded as a reliable indicator of functional PSII units when measured after a dark-recovery period of about 30 min (Kim et al., 2001). However, our preliminary test showed that the decreased parameter after the photooxidative treatment at RT was on the way of recovery after 30 min but reached a plateau after a 2-h dark-incubation period. For leaves treated with MV at LT, the decrease in $1/F_0 - 1/F_m$ was less than at RT, with full recovery to the control level being attained after about 2 h (Fig. 4). Based on the value of $1/F_0 - 1/F_m$ after the 2-h dark-recovery, we believe that MV treatment at either temperature caused similar effects on the contents of functional PSII (Fig. 4).

MV Effect on PSII-Driven Electron Transport Rate

The changes in $1/F_0 - 1/F_m$ after MV treatment (Fig. 4) was closely related to a decrease in the F_m value (Fig. 2). Because this change was recoverable, we can assume that the down-regulation mechanisms of PSII activity were also involved in the MV-treated leaves. Most of these mechanisms are tightly associated with the rate of photosynthetic electron transport activity in PSII. Xu et al. (1999) have reported that the relative PSII-driven ETR is significantly reduced in rice leaves at LT. We also noted a similar effect (albeit to a lesser degree) in the control leaves at LT (Fig. 5). In the presence of

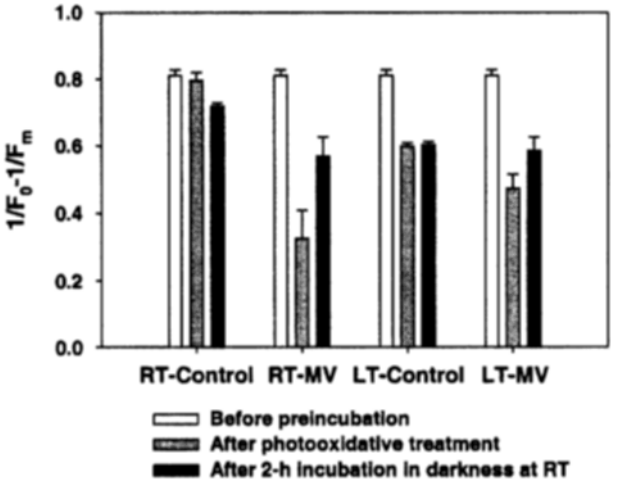

Figure 4. Changes in a Chl fluorescence parameter, $1/F_0 - 1/F_m$, by MV at two temperatures. After pre-incubation and photooxidative treatments, as described with Figure 1, leaf segments were allowed to recover in darkness at RT for 2 h. All the fluorescence yields, F_0 and F_m, were measured after 10 min of dark-adaptation at RT, and were normalized to the mean F_0 value in untreated control leaves before pre-incubation. Error bars indicate SE (n = 5 - 7).

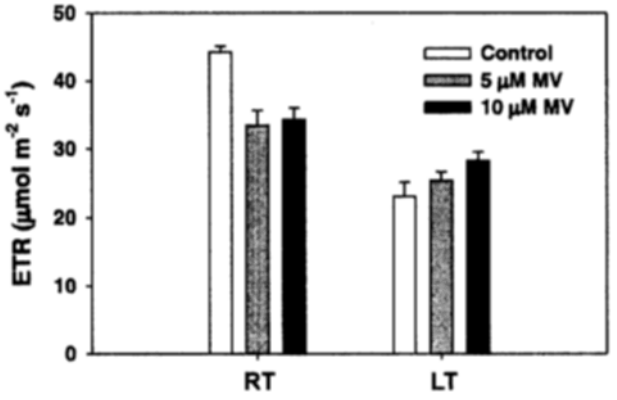

Figure 5. Effect of MV on the PSII-driven electron transport rate (ETR) at two temperatures. Experimental conditions (pre-incubation and photooxidative treatment) are as described with Figure 1. After the latter, ETR was measured under the same conditions (150 μmol m^{-2} s^{-1} at RT or LT) without dark-adaptation, using a pulse-amplitude modulated fluorometer (PAM-2000). Error bars indicate SE (n = 5).

MV, ETR decreased at RT but increased slightly at LT. MV may have increased the transport rate by releasing the acceptor-side limitation of PSI caused by a shortage of reducing power at the lower temperature. In contrast, the rate decrease at RT suggests possible damage to PSII. However, the extent of the decline was much less than that observed in the control leaves at LT.

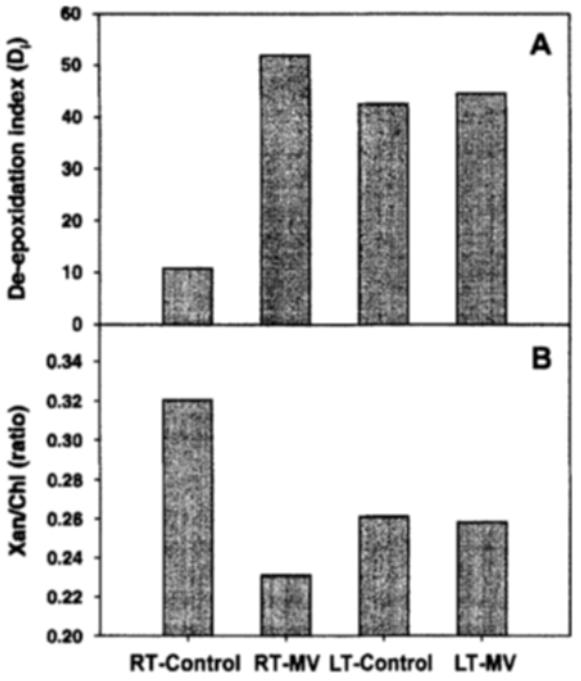

Figure 6. Difference in the de-epoxidation index (D_i) **(A)** and the ratio of xanthophyll to chlorophyll (Xan/Chl) **(B)**. D_i was calculated as a percentage of A × 0.5 + Z of V + A + Z, where A is antheraxanthin, Z is zeaxanthin, and V is violaxanthin, and Xan/Chl as V + A + Z of Chl *a* + Chl *b*. Experimental conditions are described with Figure 1.

MV Effect on Changes in the De-Epoxidation Index (D_i) and the Relative Ratio of Xanthophyll to Chl

A high de-epoxidation state generally is thought to have a protective role by down-regulating PSII. D_i was significantly increased in control leaves that were kept in the light at LT (Fig. 6A). MV treatment at RT also caused rapid de-epoxidation of xanthophylls through the acidification of the thylakoid lumen, possibly due to an accelerated ETR in the presence of MV. At LT, MV may have induced a slight additional increase in D_i compared with the LT-Control (Fig. 6A). This result is consistent with a similarly observed acceleration in ETR by MV at LT (Fig. 5). Changes in D_i were not proportional to the accumulated amounts of de-epoxidized xanthophylls (Fig. 6B), but the pool size of the xanthophylls (relative to Chl) was probably inversely proportional to the accumulation of zeaxanthin.

DISCUSSION

Based on the results reported here, we suggest that the photooxidative effect of MV differs substantially between RT and LT (4°C). MV treatment at the lower temperature caused the initial Chl fluorescence (F_0) to increase while the maximal yield of fluorescence (F_m) was decreasing. This finally resulted in reduced efficiency (Fig. 2), as had also been reported by Kim et al. (1997). The decrease in the photochemical efficiency of PSII was not as significant as that measured at RT, and the high F_v/F_m calculated at LT was mainly the result of a relatively high F_m value. In parallel to this decrease in efficiency, the decline in D1 protein content was much less at LT than at RT (Fig. 3). Therefore, we believe that MV protects PSII at LT.

Sonoike (1996) and Kim et al. (2001) have reported that MV may also provide protection for PSI during low-temperature photoinhibition. In the current study, however, the degree of irreversible photooxidative damage (in terms of non-functional PSII) was the same in the MV-treated leaves (both at RT and at LT) or in the control leaves at LT. Therefore, we assume that some of the D1 proteins present in the MV-treated leaves at LT (see Fig. 3) possibly were damaged, but not degraded. Ottander et al. (1993) have demonstrated that the repair cycle of the D1 protein is largely inhibited at LT.

Because MV has generally been used as a photooxidative stress inducer, little attention has been paid to a system's recovery from this MV stress. However, the value for $1/F_0 - 1/F_m$, which was decreased by photooxidation at RT, was found to be considerably reversible after tissues were incubated for 2 h in darkness (Fig. 4). Some photosystems could be reactivated in leaves treated with MV at LT, too. This finding implies that a significant portion of PSII activity could be reversibly recovered after being decreased by MV. The reactivation process of photoinactivated or down-regulated PSII may be related to the reversible reactivation of phosphorylated and functional D1 proteins (Rintamäki et al., 1996; Kim et al., 1997). Nonetheless, the remaining portion of the photoinactivated PSII was considered irreversibly damaged, because those particular D1 proteins can be replaced only by de-novo synthesis in the light (Rintamäki et al., 1996). Damaged D1 protein is phosphorylated and remains intact at LT, a fact that probably explains why protein loss was not observed in our leaves treated with MV at LT.

Excessive excitation energy can be dissipated either photochemically or non-photochemically (Xu et al., 1999), and both routes are dependent on ETR. In our experiments, ETRs were measured under the same conditions as those used for the photooxidative treatment at RT or LT. At the lower temperature, ETR dropped significantly (Fig. 5). This response, also reported by Xu et al. (1999), may have resulted from lipid-phase tran-

sitions in the thylakoid membrane or because of an abrupt change in the protein/lipid interaction upon sudden exposure to LT, as has been suggested by Brüggemann and Linger (1994). In addition, the activities of several key enzymes for CO_2 fixation are significantly decreased by low-temperature photoinhibition (Sassenrath et al., 1990; Brüggemann et al., 1992; Byrd et al., 1995). Such an inhibition of carbon metabolism can result in a decrease in $NADP^+$, i.e., the last electron acceptor of PSI, thereby finally producing the acceptor-side limitation of PSI. Therefore, in our leaves treated with MV at LT, MV was thought to be releasing the acceptor-limitation, working as a strong electron acceptor and producing an increase in ETR (Fig. 5). In contrast, the decrease in ETR for leaves treated with MV at RT may have been a result of PSII down-regulation and/or photooxidative damage to the PSII complexes.

The increased efficiency of electron capture enhances ETR and the acidification of the thylakoid lumen, providing favorable conditions for xanthophyll-cycle de-epoxidation (Büch et al., 1994; Pfündel and Bilger, 1994; Thiele and Krause, 1994). According to Xu et al. (1999), zeaxanthin is accumulated in barley and rice leaves during chilling in the light. Interestingly, MV could also have induced the de-epoxidation of xanthophylls at RT in our study (Fig. 6), even though the ETR was diminished (Fig. 5). Although that decrease may have been due to photooxidative damage to PSII, our preliminary results showed that changes in the D_i were reached up to 30 min before photooxidative damage could prompt a decrease in the transport rate. As had also been observed by Xu et al. (1999), we found that the D_i was significantly increased in control leaves kept in the light at LT (Fig. 6A).

In the present study, we demonstrated that substantial temperature-dependent differences exist in the photooxidative effect of MV on PSII activity, D1 protein levels, functional PSII content, ETR, and the xanthophyll cycle. We should emphasize that MV causes reversible photoinactivation of PSII as well as irreversible photooxidative damage. The xanthophyll cycle plays an important role in this mechanism for reversible photoinactivation. However, we note that the pool size of xanthophylls was inversely proportional to the accumulation of zeaxanthin, suggesting that xanthophylls are very sensitive to photooxidative treatment. That phenomenon requires further investigation.

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