FEBS Letters 430 (1998) 288–292 FEBS 20445

Temperature/light dependent development of selective resistance to photoinhibition of photosystem I

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Received 25 April 1998; revised version received 24 May 1998

Abstract Exposure of winter rye leaves grown at 20°C and an irradiance of either 50 or 250 μmol m^{-2} s^{-1} to high light stress (1600 μmol m^{-2} s^{-1}, 4 h) at 5°C resulted in photoinhibition of PSI measured in vivo as a 34% and 31% decrease in $\Delta A_{820}/A_{820}$ ($P700^{+}$). The same effect was registered in plants grown at 5°C and 50 μmol m^{-2} s^{-1}. This was accompanied by a parallel degradation of the PsaA/PsaB heterodimer, increase of the intersystem e^{-} pool size as well as inhibition of PSII photochemistry measured as F_v/F_m. Surprisingly, plants acclimated to high light (800 μmol m^{-2} s^{-1}) or to 5°C and moderate light (250 μmol m^{-2} s^{-1}) were fully resistant to photoinhibition of PSI and did not exhibit any measurable changes at the level of PSI heterodimer abundance and intersystem e^{-} pool size, although PSII photochemistry was reduced to 66% and 64% respectively. Thus, we show for the first time that PSI, unlike PSII, becomes completely resistant to photoinhibition when plants are acclimated to either 20°C/800 μmol m^{-2} s^{-1} or 5°C/ 250 μmol m^{-2} s^{-1} as a response to growth at elevated excitation pressure. The role of temperature/light dependent acclimation in the induction of selective tolerance to PSI photoinactivation is discussed.

Key words: Photosystem I; Photoinhibition; P700; Intersystem electron pool; Excitation pressure; Low temperature

1. Introduction

Photoinhibition has been defined as a decrease of photosynthetic efficiency under light conditions that exceed the photon requirement for photosynthesis [1,2]. It is well established that low temperatures in combination with moderate or even low photon fluency rates increase the susceptibility of photosynthesis to photoinhibition [3,4]. Until recently, PSII has been generally thought to be the primary target for photoinhibition although photoinactivation of PSI was also reported in in vitro systems [5–7]. Since the initial reports on PSI photoinhibition in vivo in algal systems [8,9] and intact spinach leaves [10], there have been several additional reports of selective inhibition of PSI related photochemical activities under either weak illumination in chilling sensitive plants [11–13] or under high light in potato leaves [14]. In these reports, it was demonstrated that during light stress at low temperatures the maximum quantum yield of electron transport through PSI, the pool of photooxidizeable reaction center pigment of PSI (P700), and the efficiency of P700 oxidation were dramatically reduced [11–14]. Decreased levels of EPR-detectable $P700^{+}$ [10] and inhibition of PSI electron transport [15,16] were also shown to occur in vivo under high light at ambient temperatures. Selective photoinactivation of PSI in isolated thylakoid membranes has been also reported at chilling temperatures and at 25°C in thylakoids isolated from either chilling sensitive or chilling tolerant plants [17]. Chilling and concomitant oxidative stress have been implicated as major requirements for the PSI photoinactivation in vivo [18]. The extent of photoinhibition of PSI was also shown to be highly dependent on the growth irradiance of plants [13].

It has been proposed that the photoinhibitory process in the reaction center of PSI involves at least three steps [12,18]: (1) inactivation of the acceptor site, (2) subsequent destruction of the reaction center and (3) specific degradation of the PsaB gene product, one of the two subunits of the PSI heterodimer [19]. The primary site of the photoinactivation of PSI at chilling temperatures in vivo appears to be the iron-sulfur centers [20]. More recently, excessive linear electron transport has been suggested to be a primary cause of the loss in PSI activity [16].

It has been documented that the susceptibility of photosynthesis to photoinhibition strongly depends on the growth temperature and growth light regimes to which the organism is exposed [4,21–23]. The development of tolerance to photoinhibition of PSII has been reported for organisms grown under elevated excitation pressure estimated as $1-qP$, where qP is the coefficient of photochemical quenching. The enhanced tolerance to photoinhibition of PSII in vivo in winter wheat and winter rye appears to be primarily a consequence of an enhanced capacity to utilize the absorbed light energy through photosynthesis and ultimately growth, with minimal changes in either pigment content and composition, Lhcb abundance or leaf absorbance, whereas in the green alga *Chlorella vulgaris*, it appears to be primarily due to a reduction in light harvesting capacity coupled with an enhanced capacity to dissipate excess light non-photochemically [21–23].

To date, little or no information has been published regarding the potential influence of various temperature/light growth regimes on the development of either tolerance or susceptibility of PSI to excessive radiation. In this report, we examine this possibility and show for the first time that photosynthetic

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Abbreviations: EPR, electron paramagnetic resonance; F_m, maximal chlorophyll fluorescence with all PSII reaction centers closed in dark-adapted state; F_o, minimal fluorescence with all PSII reaction centers open in dark-adapted state; F_v, variable fluorescence (F_m-F_o); PsaA/PsaB, heterodimer of PSI reaction center; PSI, PSII, photosystem I and photosystem II, respectively; P700, reaction center pigment of PSI; $P700^{+}$, oxidized form of the reaction center pigment of PSI; qP, photochemical quenching of chlorophyll fluorescence

PII: S0014-5793(98)00681-4

adjustment of winter rye to elevated excitation pressure protects against photoinhibition of PSI in vivo.

2. Materials and methods

Winter rye (*Secale cereale* L. cv Musketeer) was germinated from seeds in course vermiculite either at a temperature of 5/5°C or 20/16°C (day/night) and different light intensities as described in [23]. Photoinhibitory treatment of rye leaves was performed at 5°C and irradiance of 1600 μmol m^{-2} s^{-1} as in [24]. Thylakoid membranes from control and high light treated leaves were isolated as described previously [23]. Total Chl concentration was measured according to [25].

Chlorophyll *a* fluorescence of dark-adapted (30 min) rye leaves was measured using a PAM-101 chlorophyll fluorescence measuring system (Walz, Effeltrich, Germany) as described in [24]. Instantaneous (dark) chlorophyll fluorescence at open PSII centers (F_o) was excited by non-actinic modulated measuring beam (650 nm, 0.12 μmol m^{-2} s^{-1}). Maximum fluorescence at closed PSII centers (F_m) was induced by saturating white light pulses (800 ms, 2800 μmol m^{-2} s^{-1}) provided by a Schott lamp (KL 1500, Schott Glaswerke, Mainz, Germany) and controlled from a Walz PAM-103 Trigger Control Unit.

The redox state of P700 was determined in vivo under ambient CO_2 conditions using a PAM-101 modulated fluorometer equipped with ED-800T emitter-detector and PAM-102 units as described in [26]. Far red light (FR) (λ_{max} = 735 nm) was provided by the 102-FR light source. MT (multiple turnover 50 ms) and ST (single turnover-half peak width 14 μs) saturating flashes were applied with XMT-103 and XST-103 power/control units respectively. The redox state of P700 was evaluated as the absorbance change around 820 nm at the growth temperature (20°C or 5°C). The signals were recorded using an oscilloscope card (PC-SCOPE T6420, Intelligente Messtechnik GmbH, Backnang, Germany) installed in an IBM-PC. The complementary area between the oxidation curve of P700 after ST and MT excitation and the stationary level of $P700^+$ under FR represent the ST- and MT-areas respectively and were used for calculations of the functional pool size of intersystem electrons on a P700 reaction center basis which was determined as [27]: e^-/P700 = MT-area/ST-area.

The amount of $P700^+$ was determined by the size of the associated light-induced EPR signal I at g = 2.0025 region [28] as described previously [29]. EPR spectra were recorded at 20°C using an ESR 220 spectrometer with 100 kHz field modulation equipped with a custom designed data acquisition system and an IBM-PC. Actinic light (320 μmol m^{-2} s^{-1}) was supplied to the front of the cuvette via fiber optic light source (1000 W halogen lamp, NARVA, Germany). The dark signal was subtracted from the light-induced EPR signal I and signal area was calibrated by using a Cr^{3+}/MgO standard containing 1.4×10^{16} spins/g. All other experimental conditions are given in the text.

Thylakoid membranes from control and photoinhibited leaves were solubilized at a Chl:SDS ratio of 1:20 (w/w) and loaded on an equal Chl basis of 6 μg $lane^{-1}$. SDS-PAGE was performed with a 12% (w/v) resolving and a 8% (w/v) stacking gel on a Mini-Protean II apparatus (Bio-Rad) using a Laemmli buffer system [30]. Separated polypeptides were transferred electrophoretically to nitrocellulose membranes (0.2 μm pore size, Bio-Rad). The membranes were preblocked and probed with the PsaA/PsaB heterodimer antibody (1:750 dilution). After incubation with the secondary antibody conjugated with horseradish peroxidase (1:20 000 dilution, Sigma), the antibody complexes were visualized by incubation of the blots in ECL detection reagents (Amersham) and developed on X-Omat XRP5 film (Eastman Kodak). Polyclonal antibodies against the PsaA/PsaB heterodimer were raised for immunoblotting essentially as described in [31].

3. Results and discussion

The effects of exposure to high light treatment at 5°C on the photochemical efficiency of PSII (F_v/F_m) in winter rye leaves grown at different temperature/light regimes are presented in Fig. 1. As expected [21,23], the F_v/F_m gradually decreased in all samples examined over the 8-h exposure to high light. However, leaves of plants grown at 20/800 were significantly less susceptible to the photoinhibitory treatment than leaves of plants grown at either 20/250 or 20/50 as indicated by the differential decrease in F_v/F_m during exposure to high light stress. Furthermore, leaves of rye grown at 5/250 exhibited a susceptibility to photoinhibition comparable to rye leaves

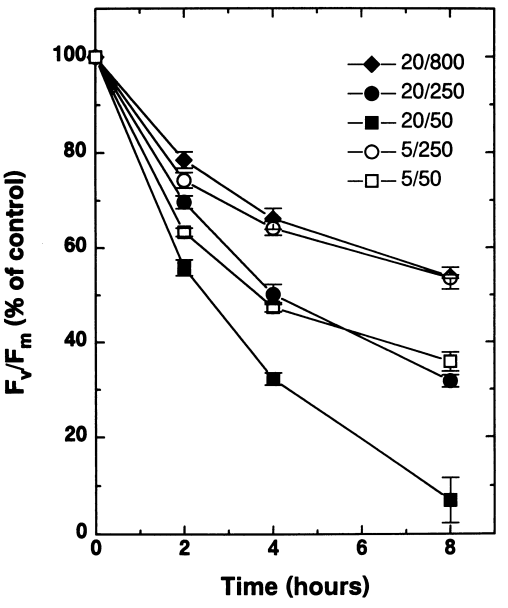

Fig. 1. Effect of growth regime on tolerance to photoinhibition of PSII measured as F_v/F_m as a function of photoinhibition time in winter rye. Photoinhibition occurred at 5°C with PPFD of 1600 μmol m^{-2} s^{-1}. Growth regimes were 20°C (closed symbols) and 5°C (open symbols) at 50 (■, □), 250 (●, ○), and 800 μmol m^{-2} s^{-1} (◆). Results are means ± S.E., n = 3.

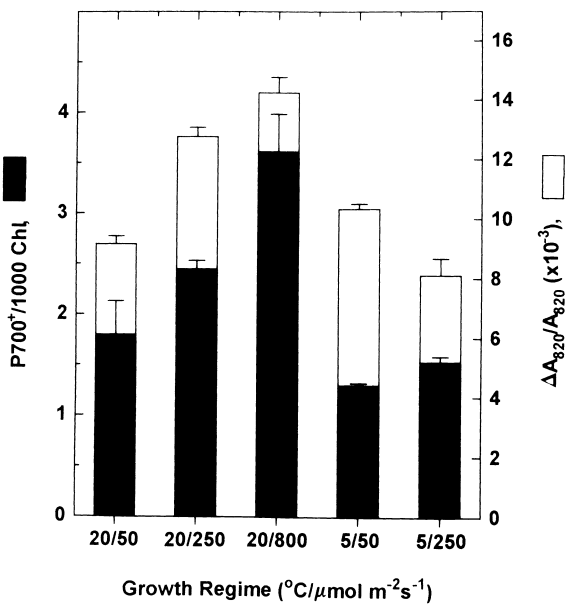

Fig. 2. Effects of growth regimes on the in vitro and in vivo measured relative amounts of light-induced $P700^+$ in isolated thylakoid membranes and rye leaves. The amount of $P700^+$ in isolated thylakoids was determined by the associated light-induced EPR signal I and the data are presented as $P700^+$/1000 Chl. The amount of $P700^+$ in intact leaves was measured by the absorbance changes at 820 nm and the data are presented as $\Delta A_{820}/A_{820}$. All experimental conditions were as in Section 2.

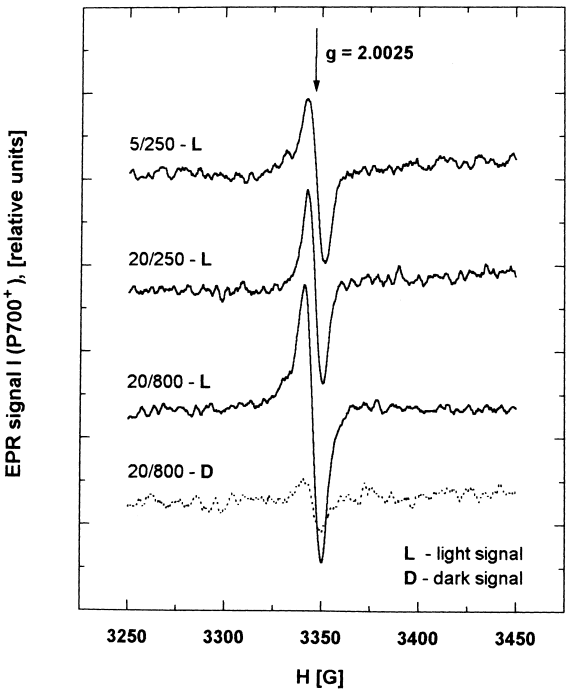

Fig. 3. EPR spectra of the light-induced EPR signal I ($P700^+$) in thylakoids isolated from rye seedlings grown under different temperature (°C)/light (μmol m^{-2} s^{-1}) regimes. The spectra were recorded at 20°C in g = 2.0025 region with the following instrumentation setting: frequency 9.5 GHz, microwave power 12.5 mW, time constant 0.2 s, modulation amplitude 5.0 G. The reaction medium contained: 50 mM Tricine-NaOH buffer (pH 7.8), 0.1 M sorbitol, 10 mM NaCl, 5 mM $MgCl_2$, 0.1 M MV, 0.2 mM DCMU and 3 mg Chl ml^{-1}.

grown at 20/800, whereas leaves of rye grown at 5/50 exhibited a sensitivity to photoinhibition comparable to that in 20/250 plants (Fig. 1). The increased tolerance to photoinhibition of PSII exhibited by plants grown at either 20/800 or 5/250 is correlated with an increased photosynthetic capacity in response to growth at elevated PSII excitation pressure measured as 1−qP [21]. Although rye grown at either 5/250 and 20/800 were exposed to different light/temperature regimes, they were exposed to growth conditions that resulted in a comparable reduction state of PSII (1−qP = 0.33). Similarly, rye grown at either 5/50 or 20/250 exhibited comparable values of 1−qP (0.14 and 0.19, respectively), whereas rye grown at 20/50 exhibited a 1−qP equal to 0.07 [23].

The extent of the absorbance decrease at 820 nm ($\Delta A_{820}/A_{820}$) of rye leaves developed under the same conditions was used to estimate the extent of PSI photooxidation ($P700^+$) [26,32]. Fig. 2 illustrates that the relative amount of $P700^+$ in plants grown at 20°C measured in vivo depends on the growth irradiance with the highest value of $\Delta A_{820}/A_{820}$ being observed in plants grown at 20/800 (Figs. 2 and 4). However, plants acclimated to either 5/50 or 5/250 exhibited values of $\Delta A_{820}/A_{820}$ similar or lower than leaves of rye plants acclimated to 20°C and same irradiance.

Although monitoring the absorbance changes around 830 nm has been widely used for measuring the redox state of P700 in various photosynthetic systems [14,26,27,32], possible accumulation of other excited triplet state species could contribute to the absorbance changes in this wavelength region [26]. To assess the reliability of the $\Delta A_{820}/A_{820}$ as a measure of P700 content, EPR measurements of the light inducible EPR signal I, which has been recognized to reflect the oxidized form of the pigment P700 ($P700^+$) [28], were performed in thylakoid membranes isolated from plants grown at all temperature/light conditions. Typical EPR spectra of thylakoids isolated from rye leaves grown at 20/250, 20/800 and 5/250 temperature/irradiance growth regimes in the g = 2.0025 region are presented in Fig. 3. The amplitude of the light-induced EPR signal I per total chlorophyll basis was 150% higher in thylakoids from 20/800 plants but 52% lower in thylakoids of plants acclimated to 5/250 compared to the signal registered in thylakoids isolated from 20/250 plants.

Although the data of in vivo and in vitro measurements of $P700^+$ differ slightly due to the different methods of estimation, the values of $P700^+$ estimated either by $\Delta A_{820}/A_{820}$ or by the EPR signal I exhibited similar trends as a function of the various growth regimes (Fig. 2). The growth light dependent increase of P700 presented above is in agreement with earlier data indicating a larger proportion of Chl a-binding proteins of PSI in high light plants [33]. The differences in P700 content between rye grown at 20/250 and 5/250 are consistent with results reported previously [34].

The apparent size of the electron donor pool to PSI was also assessed in vivo by measuring a flash-induced ΔA_{820} under steady state oxidation of PSI by FR light [27]. Typical traces of illumination cycles representing the redox state of P700 upon application of single turnover (ST) and multiple turnover (MT) saturating light pulses are shown in Fig. 4. The results indicate that plants grown at 20°C exhibit a 2.7-fold

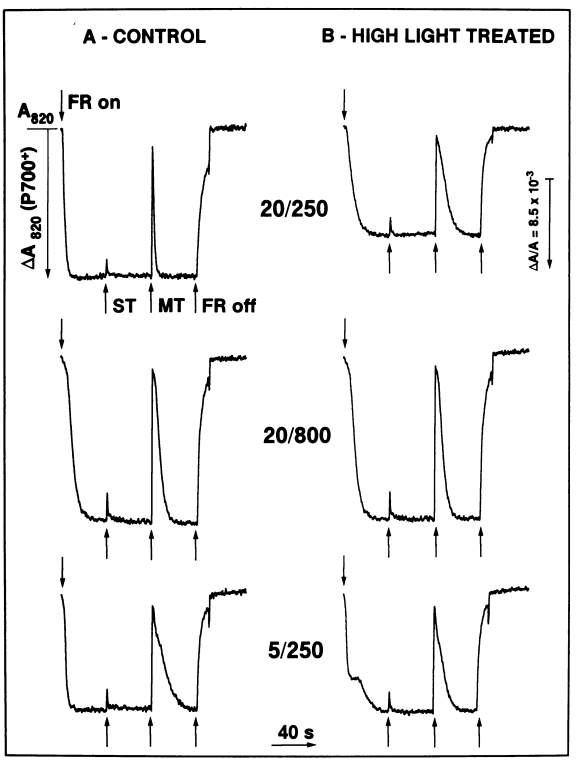

Fig. 4. In vivo measurements of the redox state of P700 in winter rye grown under different temperature (°C)/light (μmol m^{-2} s^{-1}) regimes. The measurements were performed at the growth temperature. After reaching a steady state level of $P700^+$ by FR, ST and MT pulses were applied. A: Control leaves. B: High light (1600 μmol m^{-2} s^{-1}, 4 h, at 5°C) treated leaves.

higher electron donor pool size to PSI as a function of increasing growth irradiance from 50 to 800 μmol photons m^{-2} s^{-1} (Table 1). A similar 2.3-fold increase was also found in plants acclimated to 5°C upon increasing the growth irradiance from 50 to 250 μmol photons m^{-2} s^{-1}. It appears that rye plants grown at 5/250 exhibit an e^-/P700 comparable to that of rye acclimated to 20/800, whereas rye grown at either 20/250 or 5/50 show comparable e^-/P700 values.

Along with photoinhibition of PSII photochemistry (Fig. 1), exposure of intact rye leaves acclimated to various growth regimes to high light treatment at 5°C caused a differential reduction of PSI measured by $\Delta A_{820}/A_{820}$. The original traces presented in Fig. 4 and the changes in $\Delta A_{820}/A_{820}$ quantified in Table 1 clearly indicate that the steady state amount of $P700^+$ is reduced by 30 to 40% as a result of photoinhibitory treatment in plants grown at either 20/50, 20/250 or 5/50. In contrast, plants grown at either 5/250 or 20/800 exhibited minimal changes in P700 photooxidation (Table 1), even though PSII photochemistry was reduced by 45% (Fig. 1) under the same photoinhibitory conditions. The differential resistance of 5/250 and 20/800 plants to PSI photoinhibition was accompanied by minimal changes in the apparent electron pool size, whilst a dramatic increase of the e^-/P700 values was observed in 20/50, 20/250 and 5/50 plants upon exposure to photoinhibitory irradiance (Table 1).

Since the specific degradation of the PsaB gene product has been recognized as a main target of PSI under photoinhibitory conditions [12,19], the effect of high light treatment on the abundance of PsaA/PsaB heterodimer was examined using immunoblotting. As can be seen from the immunoblots presented in Fig. 5, thylakoids isolated from 5/250 and 20/800 plants exhibited minimal changes in the abundance of the PSI heterodimer after the photoinhibitory treatment (HL) compared to non-photoinhibited controls (C). In contrast, thylakoids isolated from plants grown at either 20/250 or 5/50 (Fig. 5) exhibited reductions of PSI reaction center polypeptides in response to high light stress (HL) compared to non-photoinhibited controls (C).

It has been demonstrated that photoinactivation of PSI could be prevented in the presence of DCMU [14,16], and excessive linear electron transport has been suggested to be a major cause of the loss in PSI activity [16]. Assuming this, it seems reasonable to suggest that the partial down regulation of PSII upon exposure to photoinhibitory conditions may induce certain resistance to PSI photoinhibition. In fact, plants acclimated to 20/800 and 5/250 and resistant to PSI photoinhibition exhibited a greater proportion of closed PSII reaction centers measured as 1−qP and approximately a 2-fold lower yield of PSII electron transport (Φ_e) (0.375 and 0.339, respectively) than plants acclimated to 20/50 (0.694), 20/250 (0.538) and 5/50 (0.670). Thus, plants exhibiting the highest values of Φ_e were most susceptible to PSI photoinhibition. This is in general agreement with the assumption that PSII down regulation may well serve as one of the main protective mechanisms against photoinactivation of PSI proposed by Sonoike [18].

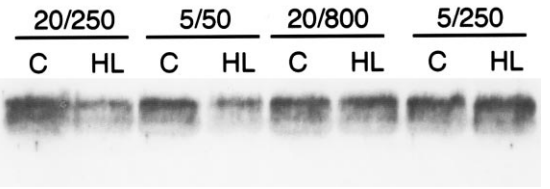

Fig. 5. Western blot of SDS-PAGE probed with an antibody raised against the PsaA/psaB heterodimer of thylakoids from control (C) and photoinhibited (HL) winter rye. Each lane was equally loaded with 6 μg Chl. Plants were grown under various temperature (°C)/light (μmol m^{-2} s^{-1}) regimes indicated. The high light treatment was as in Fig. 4.

Induction of PSII independent PSI-driven electron flow was suggested as another possible protective mechanism against photodamage of PSI [36]. In order to assess this possibility, kinetic measurements of dark re-reduction of $P700^+$ after turning off the far red light were performed (see Fig. 2). This is believed to reflect primarily the extent of cyclic electron flow around PSI [37]. It was found that the half-time of $P700^+$ re-reduction in darkness is accelerated significantly ($P < 0.05$) in 20/800 plants (2.02 ± 0.14 s, $n = 7$) as compared to plants acclimated to low excitation pressure growth conditions, i.e. 20/250 (2.77 ± 0.31 s, $n = 7$), and 20/50 (3.68 ± 0.45 s, $n = 6$). Although acclimation to low temperature causes an overall decrease in the rate of $P700^+$ re-reduction, the differences between 5/250 (2.73 ± 0.18 s, $n = 10$) and 5/50 (5.27 ± 0.42 s, $n = 8$) are significant ($P < 0.0001$) and are of a similar magnitude to that observed between rye grown at 20/800 and than grown at 20/50. These data are in good correlation with the stimulation of light saturated PSI electron transport reported earlier for thylakoids isolated from plants acclimated to low temperature [35]. Thus, we suggest that increased PSI mediated cyclic electron transport in plants grown under conditions of elevated excitation pressure (20/800 and 5/250) might be also involved in the protective mechanism(s) of PSI against photoinactivation.

It is evident that non-acclimated, cold hardy species such as winter rye are not only susceptible to low temperature dependent photoinhibition of PSII but also to photoinhibition of PSI. Although growth at either 20/800 or 5/250 decreases significantly the susceptibility of PSII to photoinhibition,

Table 1
Effects of high light (HL) treatment (4 h) at 5°C on the steady state oxidation of P700 ($\Delta A_{820}/A_{820}$) and electron donor pool size to PSI (e^-/P700) in winter rye leaves acclimated to various temperature (°C)/irradiance (μmol photons m^{-2} s^{-1}) growth conditions

Variant (°C/μmol m^{-2} s^{-1})	$\Delta A_{820}/A_{820}$ ($\times 10^{-3}$)			e^-/P700 (MT_{AREA}/ST_{AREA})		
	Control	+HL	%	Control	+HL	%
20/50	9.2 ± 0.3	6.0 ± 0.3	65.6	9.1 ± 0.4	30.7 ± 3.5	335.4
20/250	12.8 ± 0.3	8.8 ± 0.4	69.1	11.6 ± 0.5	32.3 ± 4.2	278.5
20/800	14.3 ± 0.5	13.1 ± 0.9	92.2	25.0 ± 1.2	27.8 ± 2.1	110.9
5/50	10.3 ± 0.2	6.1 ± 0.2	59.4	9.4 ± 0.5	26.7 ± 2.4	284.6
5/250	8.1 ± 0.6	8.7 ± 0.6	107.7	22.0 ± 1.2	22.0 ± 0.8	100.0

The photon fluence rate of the photoinhibitory light was 1600 μmol photons m^{-2} s^{-1}. All measurements were performed at the growth temperature. Mean ± S.E. values were calculated from 12–20 measurements in 3–5 independent experiments.

growth under the same conditions selectively imparts complete resistance to photoinhibition of PSI. Since rye grown at either 20/800 or 5/250 exhibited comparable excitation pressure (1−qP), we suggest that the development of resistance to photoinhibition of PSI reflects a response to growth under increased PSII excitation pressure rather than to temperature or light per se. Our results support the thesis that one role of the down regulation of PSII may be to protect PSI from photoinhibition. Furthermore, our results indicate that the susceptibility of PSI to photoinhibition is dependent upon the developmental history of the plant. We suggest that this may in part, explain the variability in the published literature regarding the susceptibility of PSI to photoinhibition [11–16,38–41].

Acknowledgements: This work was supported by a Natural Sciences and Engineering Research Council of Canada (NSERCC) Research Grant to N.P.A.H. and in part by the Institute of Biophysics, Bulgarian Academy of Sciences (A.G.I. and M.Y.V.). G.R.G. was supported, in part, by an Ontario Graduate Scholarship (OGS). R.M.M. was supported by OGS and a NSERCC Postgraduate Award.

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