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SALICYLIC ACID - AND METHYL JASMONATE - INDUCED PROTECTION ON PHOTOSYNTHESIS TO PARAQUAT OXIDATIVE STRESS

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Summary. In the present work it is demonstrated that Salicylic acid (SA) and Methyl Jasmonate (MeJA) provided protection of photosynthesis (A) against paraquat (Pq) stress and diminished the oxidative damage caused by Pq. Barley seedlings (12 d old) were supplied with 500 µM SA, 23 µM MeJA or 10 µM Pq via the transpiration stream and kept in the dark for 24 h. They were then exposed to 100 µmol.m^{-2}.s^{-1} PAR and samples were taken 1, 2, 3, and 6 h after the light exposure. Leaf gas exchange parameters, the activity of RuBPC and of the photorespiratory enzymes PG, GO, and CAT were determined. Treatment of seedlings with SA or MeJA alone resulted in decreased levels of Chl, A and Tr. Pq treatment led to a decrease in Chl and protein content and to a very strong inhibition of A. Pq-treatment did not affect the activity of RuBPC but greatly increased the activity of the photorespiratory enzymes. Pre-treatment of seedlings with SA or MeJA fully blocked the inhibitory effect of Pq on A and provided protection against subsequent Pq-induced oxidative damage. This observation was confirmed by gas exchange parameters, Chl and protein content and by changes in lipid peroxidation, H_2O_2 level, and electrolyte leakage. The relationship between SA, MeJA and Pq toxicity and the degree of oxidative damage was examined by measuring the activities of several antioxidative enzymes such as SOD, APX, GR and POX. Treatment with 10 µM Pq reduced the activities of APX and GR. Pre-treatment with 500 µM SA for 24 h in the dark greatly improved the capacity of the antioxidative defence system and increased Pq tolerance. Pre-treatment with 23 µM MeJA only partially improved the capacity of the antioxidative

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enzyme system. It is suggested that the observed protection of MeJA on A against Pq-toxicity was mainly due to improvement of membrane stability and composition, Chl and protein levels.

Key words: jasmonates, oxidative stress, photosynthesis, paraquat, salicylic acid

Abbreviations: A – photosynthetic rate, APX – ascorbate peroxidase, GR – gluthation reductase, MeJA – methyl ester of jasmonic acid, PSII- photosystem 2, Pq – paraquat, SA – salicylic acid, SOD – superoxide dismutase, RuBPC – ribulose-1,5-bisphosphate carboxylase, GO – glycolate oxidase, PGP – phosphoglycolate phosphatase, g_s – stomatal conductance, ROS – reactive oxygen species, CAT – catalase, POX – guaiacol peroxidase

Introduction

Salicylic acid (SA) is an ubiquitous plant phenolic that controls plant growth and development (Schettel and Balke, 1983), photosynthesis and transpiration rates (Pancheva et al., 1996), ion uptake and transport (Harper and Balke, 1981), and induces changes in leaf anatomy and chloroplast ultrastructure (Uzunova and Popova, 2000). SA has been recognised as an endogenous regulatory signal in plants mediating plant defence against pathogens (Malamy et al., 1990; Durner et al., 1997). It was reported that SA also accumulates during exposure to ozone or UV light (Yalpani et al., 1994; Sharma et al., 1996) and plays a role in the plant response to adverse environmental conditions, such as salt and osmotic stresses (Borsani et al., 2001). In recent years SA has been the focus of much attention because of its ability to induce thermotolerance in mustard seedlings (Chen et al., 1997; Dat et al., 1998 a,b) or to provide protection in maize plants against low-temperature stress (Janda et al., 1999).

Jasmonates (jasmonic acid, JA and its methyl ester, MeJA) occur in many plant species and are involved in various physiological processes (reviewed in Creelman and Mullet, 1995; 1997). In barley (*Hordeum vulgare* L.) plants exogenous treatment with JA or MeJA revealed changes in a number of photosynthetic parameters, such as a decrease in the rate of photosynthetic CO_2 fixation and the activity of RuBP carboxylase. There were considerable increases in the rates of dark respiration and photorespiration, in the CO_2 compensation point value, and in the stomatal resistance (Popova et al., 1988). A breakdown in the biosynthesis of Rubisco (Weidhase et al., 1987; Popova and Vaklinova, 1988), an inhibition of the Hill reaction activity and some changes in the kinetic characteristics of the flash-induced O_2 evolution (Maslenkova et al., 1990) have been reported to occur as a result of JA and MeJA treatments. Furthermore, jasmonates induced accumulation of a number of proteins (JIPs) in many plant species, including barley (Muller-Uri et al.,1988). Maslenkova et al. (1992) ad-

duced experimental data showing an induction of JIPs mainly belonging to the thylakoid-bounded polypeptides. Most of the JA-induced polypeptides were identical to ABA- and NaCl-induced ones, leading to the assumption that exogenously applied jasmonates act as stress agents.

The intensive research on plant response to various environmental stresses during the last few years has revealed a role for jasmonates as signalling molecules or stress-modulating compounds. They have been involved in plant response to wounding (Farmer and Ryan, 1992), UV irradiation (Conconi et al., 1996), and pathogen infection (Creelman and Mullet, 1997). Increased endogenous levels of jasmonates have been found in plants suffering from drought and osmotic stress (Creelman and Mullet, 1995), wounding (Creelman et al., 1992; Baldwin et al., 1997), after treatment with fungal elicitors (Gundlach et al., 1992) or upon pathogen attack (Farmer and Rayan, 1992). In recent years jasmonates have been the focus of much attention because of their ability to provide protection to salinity stress (Tsonev et al., 1998), UV irradiation (Mackerness et al., 1999), or to increased freezing tolerance in bromegrass (Wilen et al., 1994), leading to the suggestion that jasmonates could mediate the defence response to various environmental stresses.

The common link among different stresses is that they all produce an oxidative burst. Chloroplasts, a major source of activated O_2 in plants (Foyer et al., 1994; Iturbe-Ormaetxe et al.,1998), and antioxidants, which may play an essential role in preventing oxidative damage, are greatly affected by environmental stress (Bowler et al., 1994).

Bipyridyl herbicides such as paraquat (Pq) and diquat are non-selective contact herbicides that act by intercepting electrons from the photosynthetic electron transport chain at PSI. This reaction results in the production of bipyridyl radicals that readily react with O_2 to produce superoxide and then, through a series of reactions, produce H_2O_2 and the hydroxyl radical. These toxic oxygen species cause extensive lipid peroxidation (Babbs et al., 1989), chlorophyll breakdown (Shaaltiel et al., 1988), loss of photosynthetic activity (Fedtke ,1982), leakage of electrolytes (Harris and Dodge, 1972), and loss in cell membrane integrity (Kunert and Dodge, 1989).

Cross tolerances were found between oxidant generating herbicides and environmental oxidants in different plant biotypes constitutively tolerant to paraquat, SO_2, O_3 (Shaaltiel et al., 1988), to photoinhibition (Jansen et al., 1989), or drought stress (Malan et al., 1990).

This study was undertaken to determine the physiological and biochemical changes in barley plants treated by SA or MeJA during Pq-induced stress, to investigate whether these plant regulators are involved in the induction of defence response, to elucidate the underlying mechanisms by which SA and MeJA alleviate the Pq-induced inhibition on photosynthesis, and to test the hypothesis that those treatments might reduce the Pq injury on photosynthesis through their effect on detoxification of AOS.

Materials and Methods

Model System

Barley plants (*Hordeum vulgare*. L., cv. Alfa) were grown for 12 days in soil in a growth chamber. The environmental conditions were: irradiance, 160 µmol.m^{-2}.s^{-1} PAR, 12 h-photoperiod, temperature 24±2 ºC, and relative humidity of 60±5%. Twelve-day-old seedlings were cut at their basal end and incubated on solutions containing: distilled water (control), 500 µM SA , 23 µM MeJA, and 10 µM Pq. They were kept for 24 h in the dark at room temperature. Thereafter, half of the SA-and MeJA-treated seedlings were transferred to a beaker containing 10 µM Pq and all variants were exposed to 100 µmol.m^{-2}.s^{-1} PAR, provided by cool-white fluorescent tubes and were sampled 24 h after dark and 6 h after light exposure.

The following physiological and biochemical parameters were determined: leaf gas exchange parameters, chlorophyll and protein content, activity of carboxylating and photorespiratory enzymes, PS II activity and O_2 evolution, stress markers: H_2O_2 level, lipid peroxidation and electrolyte leakage and ROS scavenging enzyme activity.

Results and Discussion

Treatment of barley seedlings with 500 µM SA or 23 µM MeJA for 24 h in the dark followed by 6 h light exposure did not cause wilting or irreversible damage to photosynthesis. Our previous experiments demonstrated that concentrations of JA or MeJA ranging from 10 to 50 µM or lower than 1 mM SA are very relevant for physiological studies, they did not cause visible damage symptoms after long-term treatment (up to 7 days), but provided well-reproducible and reversible effects on photosynthesis, growth and biochemistry of barley plants. In most of the experiments the effect of Pq on the studied physiological parameters was tested using Pq concentrations of 1, 10 and 100 µM. When seedlings were treated with 100 µM Pq for 24 h they were wilted and had no measurable photosynthesis. On the other hand, 1 µM Pq had no effect on the studied parameters (data not shown).

Gas-exchange parameters

Treatment of barley seedlings with 500 µM SA or 23 µM MeJA for 24 h caused an inhibition in the rate of photosynthesis, the effects were clearly expressed during the first hour after light exposure and then remained approximately constant. Incubation of seedlings with 10 µM Pq under the same experimental conditions led to a significant decrease in A in a time-dependent manner. Two hours after the light exposure the rate of A decreased by 62% compared with the control values. The rate of A was fully

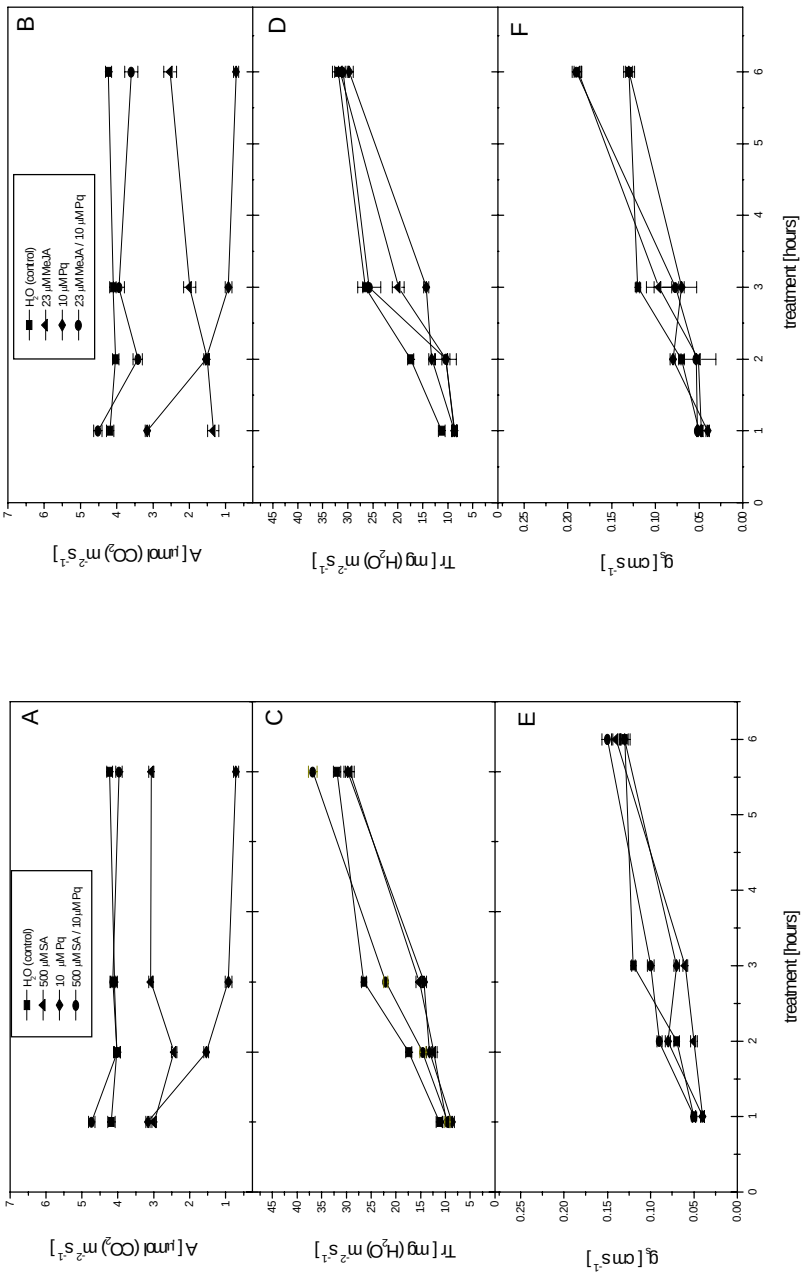

Fig. 1. Time-dependent changes in gas exchange parameters in barley leaves treated with SA, MeJA and Pq. A, net CO_2 assimilation rate (A,B); Tr, transpiration rate (C,D); g_s, stomatal conductance (E,F). Barley seedlings were cut at their basal end and incubated with distilled water (control), 500 µM SA, 23 µM MeJA, and 10 µM Pq for 24 h in the dark. Thereafter, half of the SA-and MeJA-treated seedlings were transferred to a beaker containing 10 mM Pq and all variants were exposed to 100 µmol.m^{-2}.s^{-1} PAR, and sampled 1, 2, 3, and 6 h after the light exposure. The data are averages from four separate experiments ± s.e.

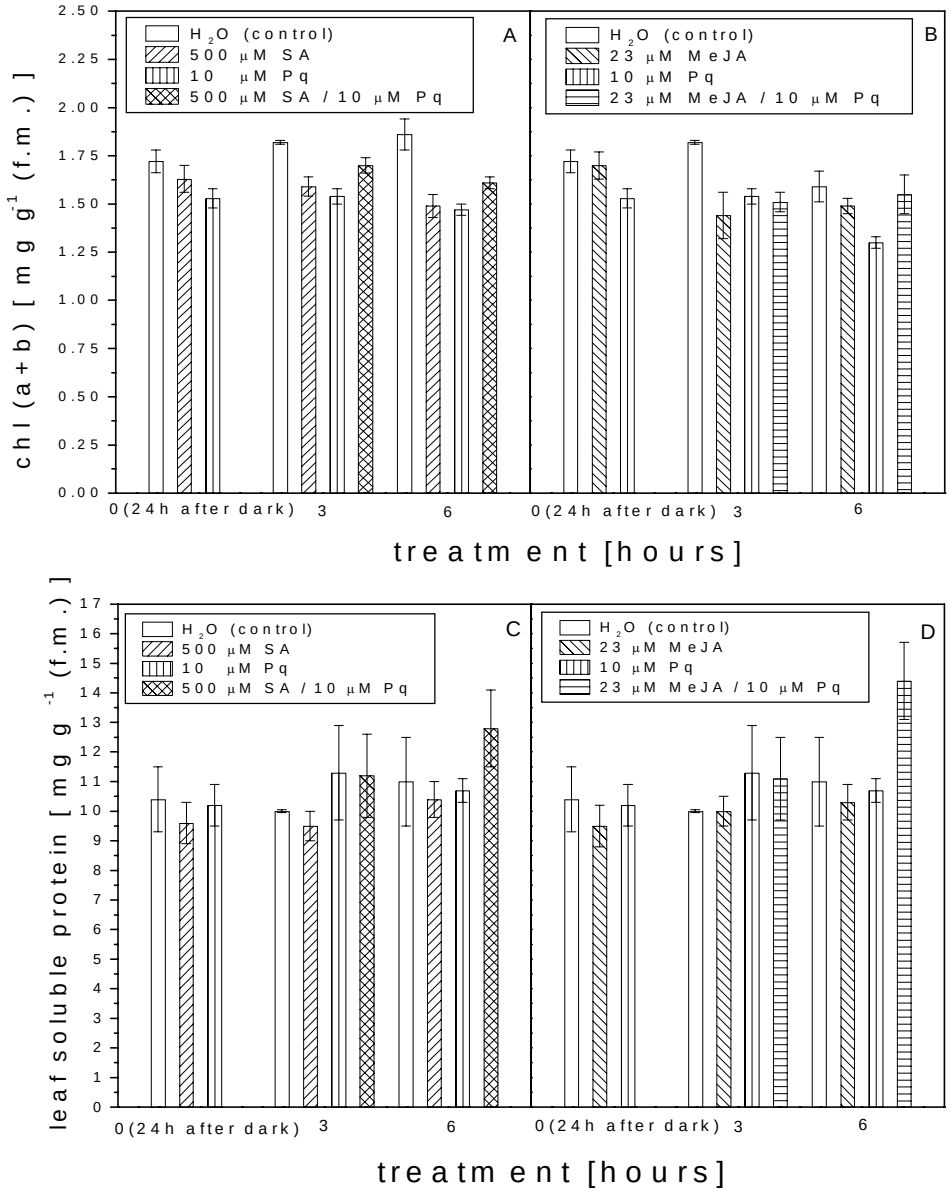

Fig. 2. Effect of SA, MeJA and Pq treatment on chlorophyll (A, B) and protein content (C,D). Variants and treatments are as described in Fig.1. All measurements were done 24 h after the dark (0 h) and 3 and 6 h after the light exposure. The data are averages from four separate experiments ± s.e.

recovered by pretreatment of seedlings with SA or MeJA for 24 h in the dark before the next exposure to Pq and light (Fig. 1, A, B). The same tendency was observed and for the changes in the transpiration rate (Tr) (Fig.1, C, D). In the presence of SA

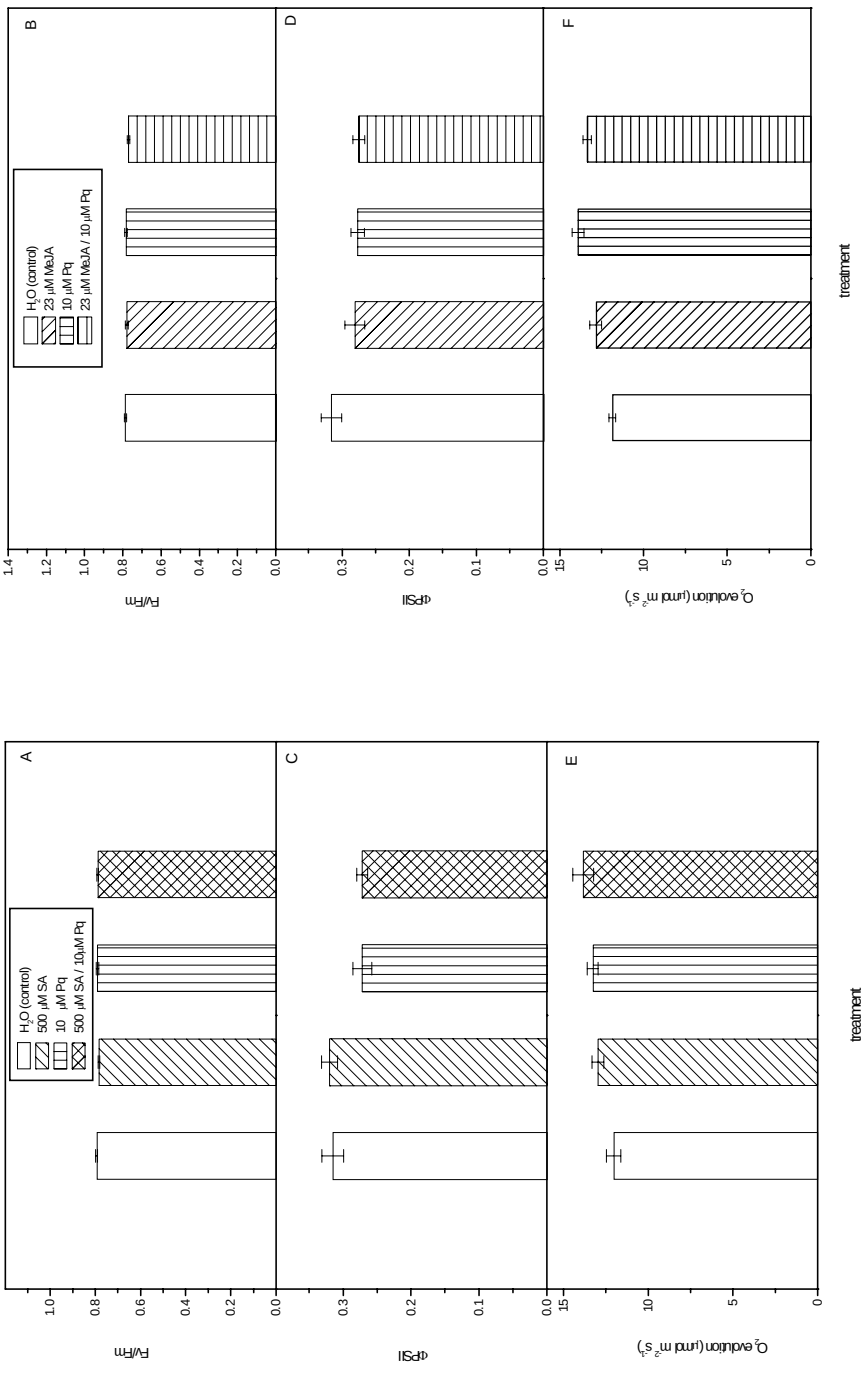

Fig. 3. Effect of SA, MeJA and Pq treatment on changes in variable to maximal fluorescence ratio (Fv/Fm) (A, B), quantum yield fPS II (C, D) and O_2 evolution (E, F). Variants and treatments are as described in Fig. 1. All measurements were done 24 h after the dark and 6 h after the light exposure. The data are averages from four separate experiments ± s.e.

the values of g_s were lower than respective control values. Pretreatment of seedlings with SA before the application of Pq resulted in an equal response of g_s in control and treated seedlings. MeJA treatment alone or pretreatment with it before the application of Pq led to an increase in the values of g_s (Fig. 1, E, F).

Many different causes could be responsible for the observed protection of photosynthesis by SA and MeJA against Pq: both plant growth regulators (1) could affect the photosynthetic light reactions leading to better adjustment of the rate of electron transport, (2) improve the rate of the carboxylating enzyme activity or increase the rate of photorespiration, (3) prevent the further chloroplast destruction caused by Pq by protection on chlorophyll and protein breakdown, (4) activate the antioxidative enzymes in chloroplasts, which in turn would increase Pq tolerance or trigger various defence-related genes. The characterisation of these specific changes was followed in the next experiments.

Changes in chlorophyll and protein levels

Dark- treated barley seedlings with 500 µM SA or 23 µM MeJA did not show loss in chlorophyll content, whereas 10 µM Pq-treated seedlings had a lower chlorophyll level. Light exposure led to a progressive decrease in chlorophyll content in Pq- treated seedlings. A decrease in chlorophyll level was found in MeJA-treated plants. Pretreatment of plants with SA or MeJA for 24 h before Pq application caused a protection against Pq-induced chlorophyll losses (Fig. 2, A, B).

No significant changes in the protein levels were observed in SA-, MeJA- and Pq- dark treated seedlings. A decrease of about 25% was measured in Pq-treated plants exposed to light for 6 h. Pre-treatment with SA or MeJA before Pq prevented the protein loss (Fig. 2, D).

PS II activity and O_2 evolution

The effect of paraquat on photosynthetic electron transport can be measured by the chlorophyll fluorescence induction method, because bipyridinium compounds quench fluorescence by accepting electrons from photosystem I (PSI), thereby keeping the plastoquinone pool oxidized. Our results showed that the primary photochemical efficiency of PSII, estimated by the ratio Fv/Fm was not influenced by Pq treatment (Fig. 3, A,B). However Pq decreased the quantum yield of whole-chain electron transfer, fPSII (by 12-14%, $p<0.05$) and pretreatment with SA or MeJA did not show any protective effect (Fig. 3, C, D). We found that the photochemical activity was less sensitive to Pq than the CO_2 fixation, characterizing the functioning of the whole photosynthetic apparatus. Since paraquat is an electron acceptor of the reducing side of PSI its presence in the chloroplast can deviate electrons from their normal physiological way. The electrons are attracted by Pq itself and NADPH can not be formed which will result in a strong inhibition of CO_2 assimilation. Under conditions of dimi-

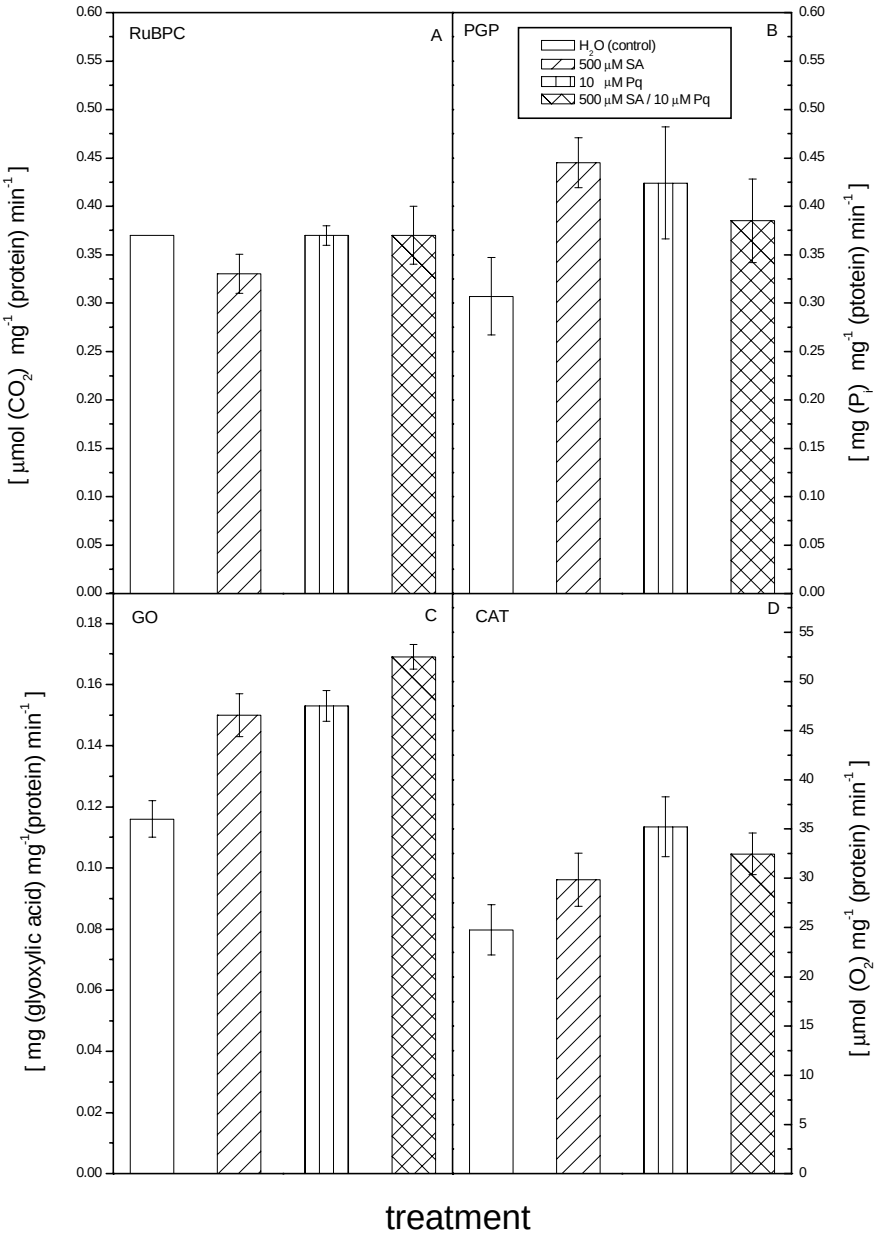

Fig. 4. Effect of SA and Pq treatment on the activity of RuBPC (A), PGP (B), GO (C) and CAT (D). Variants and treatments are as described in Fig.1. The activity of the enzymes was measured 6 h after the light exposure. The data are averages from four separate experiments ± s.e.

nished CO_2 reduction, the consumption of electrons by pathways other than the Calvin cycle can maintain noncyclic electron transport. One such pathway is the Mehler-per-

oxidase cycle. On the other hand H_2O_2 generation and degradation may act as an alternative electron sink maintaining the oxidized state of the primary PSII electron acceptor Q_A. This may be responsible for the increased rate of O_2 evolution we found after Pq treatment (Fig. 3, E, F). Oxygen evolution needs only a functional, intact PSII. If the water splitting system is intact and electron acceptor (Pq) is present O_2 evolution could not be influenced. If the Pq treatment was longer or its concentration was higher, the active oxygen forms generated by Pq can destroy the proteins, lipids and other important cell components, which effect can lead secondarily to damage of PSII machinery too.

Effect of SA, MeJA and Pq on RuBPC, PGP, GO and CAT activities

RuBPC activity was almost unaffected when plants were treated with 500 µM SA or 10 µM Pq for 24 and then exposed for 6 h in the light. Pretreatment with SA before application of Pq had no effect on the enzyme activity (Fig. 4, A).

Treatment of plants with 500 µM SA or 10 µM Pq alone or pretreatment with SA before exposure to 10 µM Pq caused increases in the activity of the photorespiratory enzymes: PGP, GO, and catalase. The effects of combined treatment of 500 µM SA and 10 µM Pq on the activity of the studied photorespiratory enzymes resembled those obtained with SA and Pq alone (Fig. 4, B–D).

Treatment with 23 µM MeJa or pretreatment with MeJA before application of Pq led to a small decrease in the activity of RuBPC (Fig. 5, A). MeJA alone did not affect the activity of the photorespiratory enzymes PGP and GO, but caused an increase in catalase activity. Pretreatment of barley seedlings with 23 µM MeJA before exposure to 10 µM Pq caused a decline in the activity of the photorespiratory enzymes studied (Fig. 5, B–D).

Effects of SA, MeJA and Pq on H_2O_2 level, lipid peroxidation and electrolyte leakage

No major changes were observed in H_2O_2 levels in plants treated with SA or MeJA alone. However, treatment with 10 µM Pq enhanced H_2O_2 levels by 35% compared with control leaves. Pretreatment with SA before next exposure to Pq and light fully suppressed the Pq-induced increase in H_2O_2 level. Pretreatment with MeJA failed to decrease the effect of Pq on this parameter. (Fig. 6, A, B).

Because Pq-treatment enhanced H_2O_2 level in leaves, we investigated the damage to membranes by monitoring MDA content and electrolyte leakage. The observed changes in MDA followed the same tendency as for H_2O_2 level. Pretreatment with SA before Pq application led to a decrease in the level of lipid peroxidation caused by Pq. Again pretreatment with MeJA did not suppress the high rate of lipid peroxidation (Fig. 6, C, D).

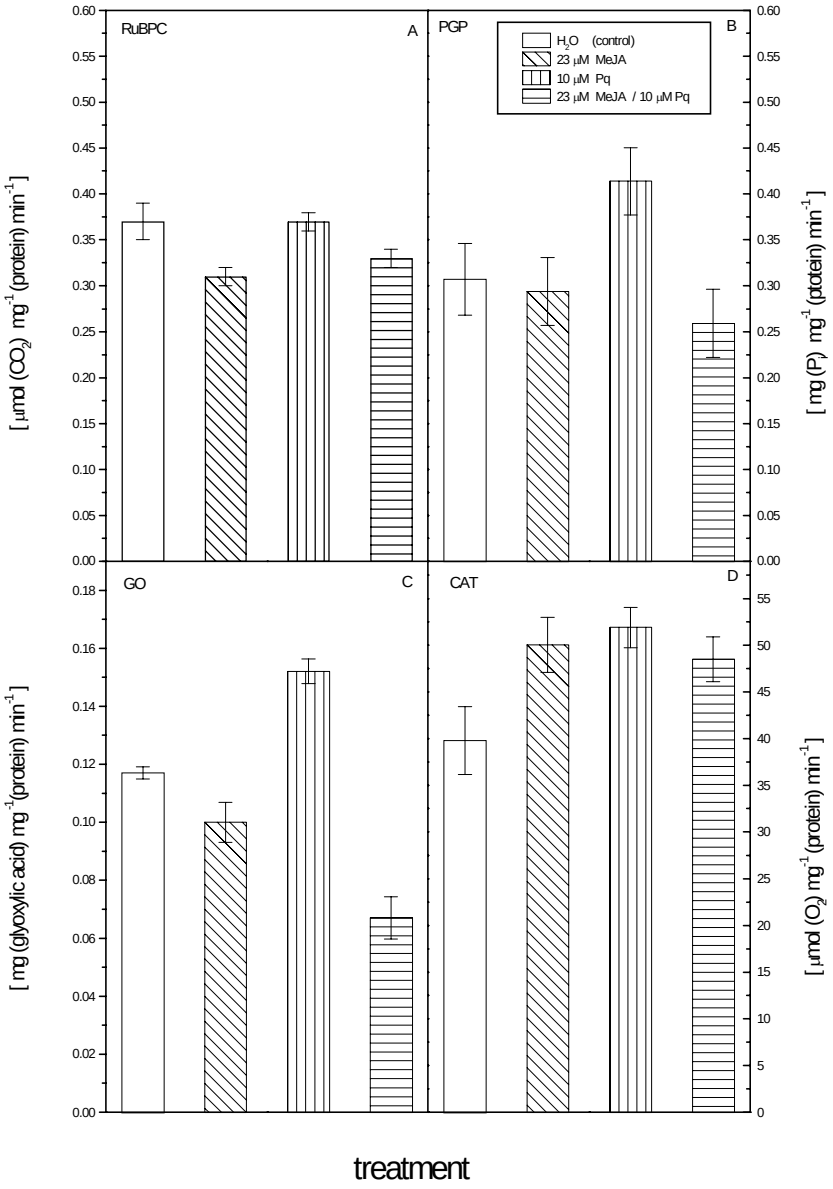

Fig.5. Effect of MeJA, and Pq treatment on the activity of RuBPC (A), PGP (B), GO (C), and CAT (D) . Variants and treatments are as described in Fig.1. The activity of the enzymes was measured 6 h after the light exposure. The data are averages from four separate experiments ± s.e.

Electrolyte leakage was much more pronounced in plants supplied with Pq than in those which were treated with SA or MeJA. Pretreatment with SA or MeJA greatly decreased the values of Pq-induced increase in electrolyte leakage (Fig. 6, E, F).

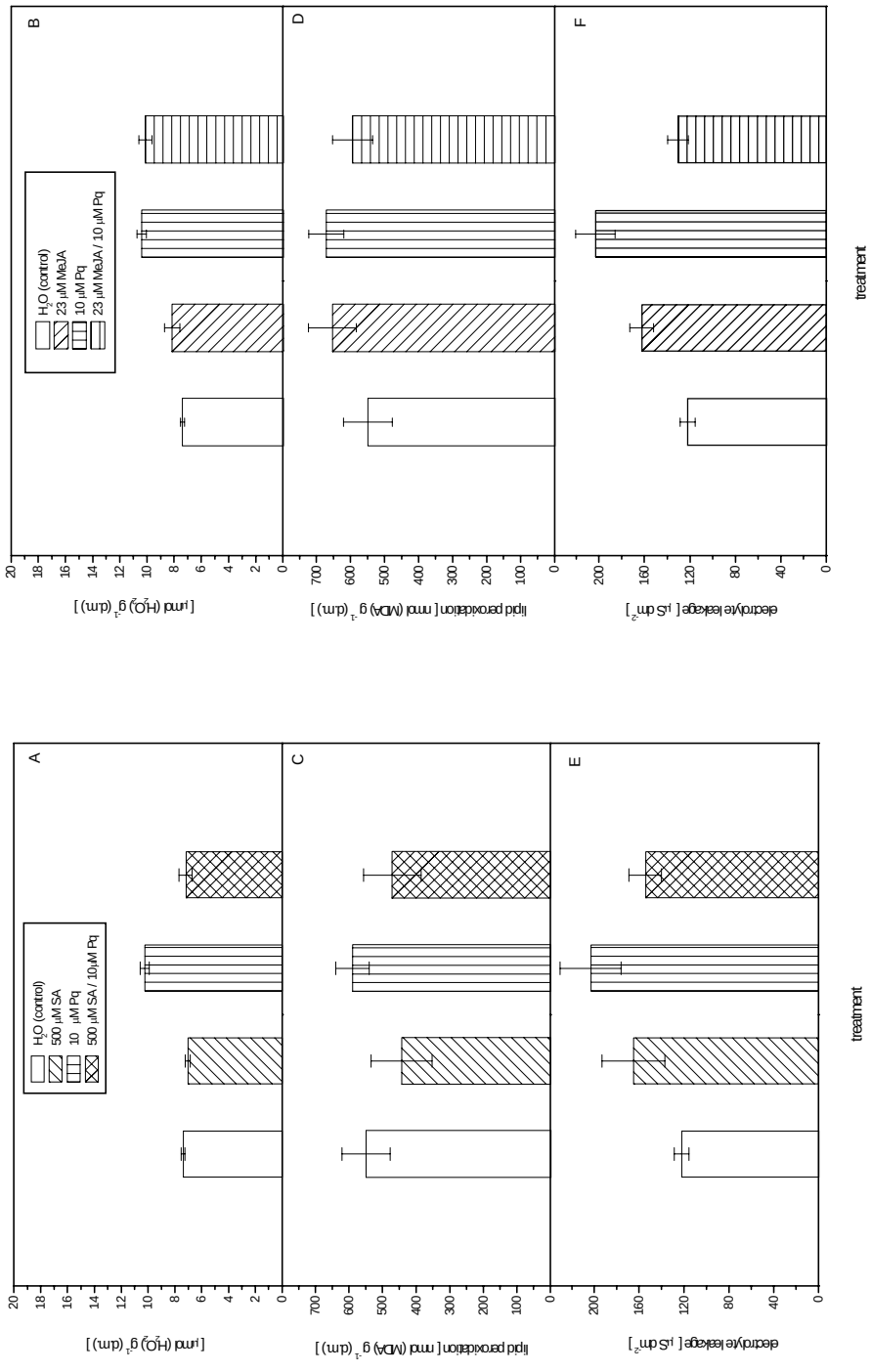

Fig.6. Effect of SA, MeJA and Pq treatment on H_2O_2 production (A,B), lipid peroxidation (C,D) and electrolyte leakage (E,F) in barley leaves. All measurements were done 6 h after the light exposure. Variants and treatments are as described in Fig.1. The data are averages from four separate experiments ± s.e.

ROS- scavanging enzyme activities

In order to assess the role of SA and MeJA in Pq-induced oxidative damage, enzyme activities of some antioxidative enzymes were evaluated in leaves of barley seedlings at the 6th hour of the light exposure. Plant cells presumably regulate H_2O_2 levels by co-ordinating the activities of H_2O_2-metabolising enzymes such as SOD, CAT, POX, APX and GR.

No changes were observed in the activity of SOD in barley seedlings treated with Pq, SA or MeJA and kept for 24 h in the dark. Six h after the light exposure, treatment with 500 µM SA alone caused an increase in SOD activity by 17%, whereas treatment with 10 µM Pq only slightly affected its activity. Pretreatment with SA before Pq application enhanced by approximately 20% the activity of SOD, compared with both control and Pq-treated leaves (Fig. 7, A,). No changes in the activity of total SOD were observed in plants treated with MeJA alone or in pretreated with MeJA before the application of Pq (Fig. 7, C).

After native PAGE, two major bands with SOD activity were detected. The lower and more active band *a* was previously characterized as cytosolic Cu, Zn-containing SOD, while the upper and less active band *b* was characterized as chloroplstic Fe-containing SOD. Pq treatment slightly increased the activity of cytosolic SOD. Treatment with SA increased the activity of both SOD bands but its effect on chloroplastic SOD was more significant. The effect of pretreatment with SA before Pq application on SOD isoforms was preserved and even greater activation of chloroplastic SOD was observed (Fig. 7, B).

The presence of MeJA did not influence the activity of cytosolic SOD and slightly decreased the activity of chloroplastic SOD (Fig. 7 D).

No significant changes were observed in APX activity in leaves treated with 500 µM SA or 10 µM Pq for 24 h in the dark (Fig. 8, A). Under the same experimental conditions treatment with MeJA led to a small decrease (about 20%) in APX activity (Fig. 8, B). For GR activity an increase of about 20% occurred in leaves treated with SA, MeJA and Pq compared to the control leaves after 24 h in the dark (Fig.8, E, F).

Light exposure for 6 h influenced differently the activities of APX, GR and DHAR in all treated plants (Fig. 8, A-F). SA treatment alone caused a slight increase (by 10%) in the activity of DHAR and changed insignificantly the activity of APX and GR, compared to the control leaves. Pq treatment alone had no effect on DHAR activity but caused almost 25% and 20% decrease in the activities of APX and GR, respectively. Pretreatment with SA before exposure to Pq and light had a protective effect on the enzyme activities, the effect being highly expressed on DHAR (approximately 60% over the control values) (Fig. 8, A, C, E). MeJA treatment alone or pretreatment with MeJA before application of Pq led to decrease in the activities of all three studied enzymes (Fig. 8, B, D, F).

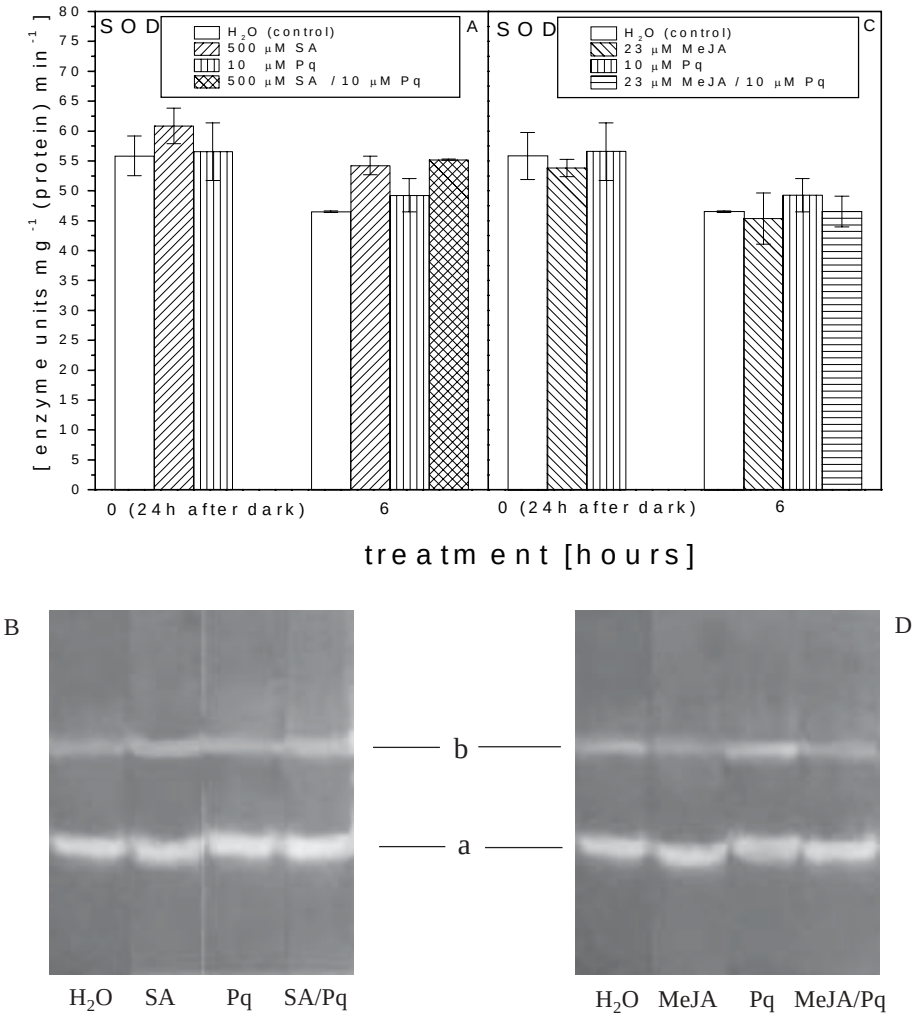

Fig.7. Effect of SA, MeJA, and Pq treatment on the activity of SOD (A,C), and on individual SOD isoforms, estimated by separating in nondenaturing gels (B,D). Variants and treatments are as described in Fig.1. The activity of the enzymes was measured 24 h after the dark and 6 h after the light exposure. The data are averages from four separate experiments ± s.e.

POX and CAT activities and their isoforms

No changes were observed in POX activity in leaves treated with SA, MeJA or Pq for 24 h in the dark (Fig. 9, A, C).

6 h after the light exposure, treatment with 10 μM Pq alone had no effect on POX activity. However, both treatment with SA or MeJA alone and pretreatment with SA before next exposure to Pq and light enhanced POX activity (Fig. 9, A, C).

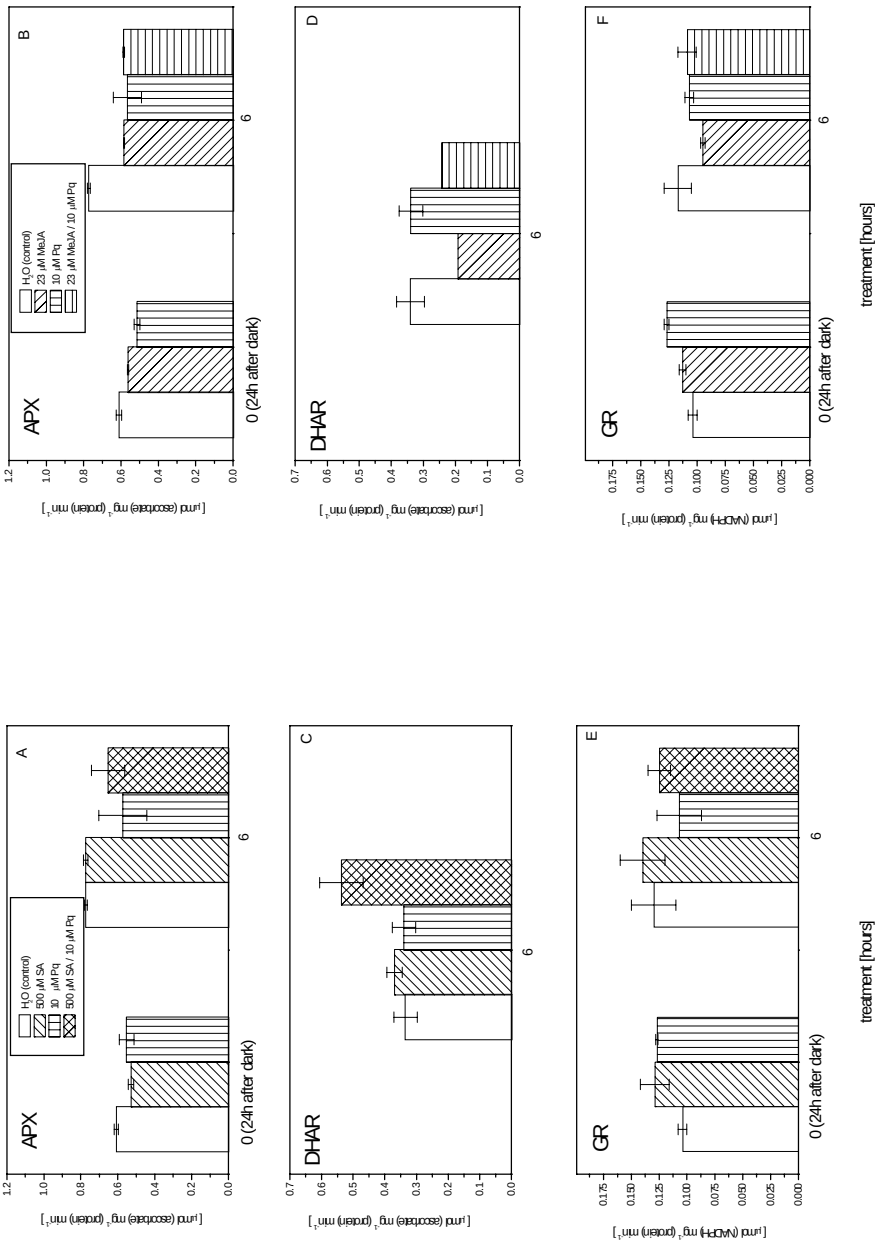

Fig.8. Effect of SA, MeJA, and Pq treatment on the activity of APX (A,B), DHAR (C,D) and GR (E,F). Variants and treatments are as described in Fig.1. The activity of the enzymes was measured 24 h after the dark and 6 h after the light exposure. The data are averages from four separate experiments ± s.e.

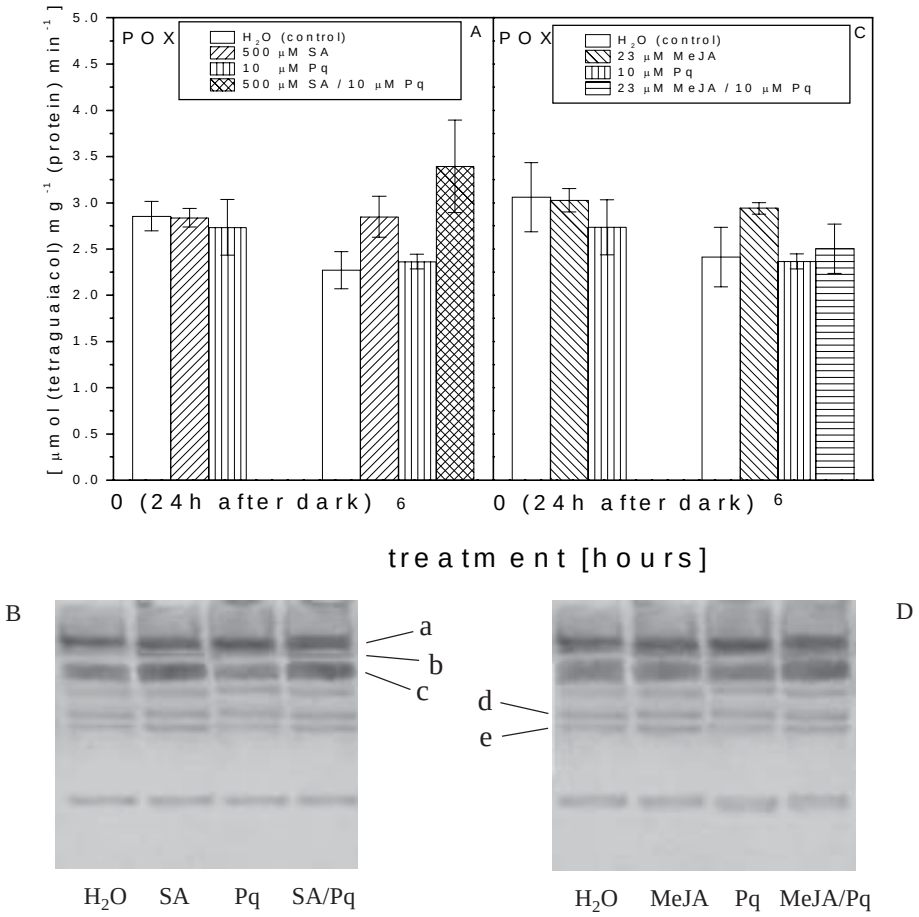

Fig.9. Effect of SA, MeJA, and Pq treatment on the activity of POX (A,C), and on individual POX isoforms, estimated by separating in nondenaturing gels (B,D). Variants and treatments are as described in Fig.1. The activity of the enzymes was measured 24 h after the dark and 6 h after the light exposure. The data are averages from four separate experiments ± s.e.

The electrophoretic profile of peroxidase in barley leaves consisted of 7 different POX isoenzymes. In the course of treatment with SA and pretreatment with SA before Pq application but not in the case with Pq alone, the activities of slow moving and highly active isoperoxidases – bands *a*, *b* and *c* were remarkably increased, while the activities of the other isoperoxidases were not changed (Fig. 9 B). Treatment with MeJA but not pretreatment with MeJA before exposure to Pq caused an increase of the activities of bands marked as *d* and *e* (Fig. 9, D).

After native PAGE on 7.5% gel, two different bands with catalase activity appeared. The activity of the both bands was increased especially on the highly active one in the course of treatment with SA and pretreatment with SA before Pq application

but it was less pronounced in the case with Pq (Fig. 10, B). A similar effect was found in the course of treatment with MeJa and pretreatment with MeJA before subsequent exposure to Pq – MeJa alone did not exert a specific influence on CAT isoforms while in the presence of MeJA before the application of Pq the activity of the both isoforms was increased (Fig. 10 D).

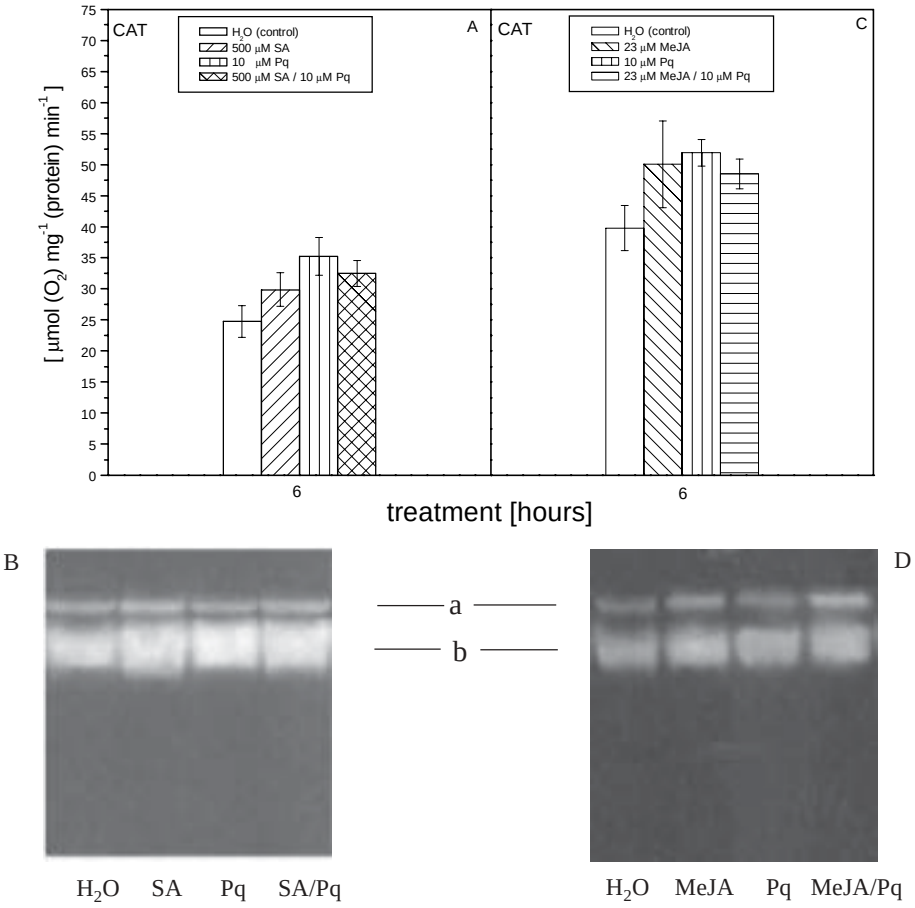

Fig.10. Effect of SA, MeJA, and Pq treatment on the activity of CAT (A,C), and on individual CAT isoforms, estimated by separating in nondenaturing gels (B,D). Variants and treatments are as described in Fig.1. The activity of the enzymes was measured 6 h after the light exposure. The data are averages from four separate experiments ± s.e.

Conclusions

In conclusion, we have shown that pretreatment of barley plants with SA or MeJA induced protection on photosynthesis against Pq.

Considering the enhanced activity of the antioxidative enzymes and the decrease in the levels of H_2O_2, lipid peroxidation and electrolyte leakage in pretreated with SA barley plants we could assume that the observed protection on photosynthesis was probably due to increased antioxidant capacity and improved cell permeability.

Pretreatment with MeJA also caused protection of photosynthesis against Pq stress but its action differed from that of SA. It provided protection of membrane integrity but did not affect the activity of the ascorbate-gluthatione cycle. Most probably this protection was due to an involvement of the other antioxidative components like enzymes catalysing the detoxification of high H_2O_2 production, namely catalase and POX, or lypoxigenase.

There is a need to understand how wide a range of adverse environmental factors provoke the same changes in physiological processes, reflecting the changed expression of a common or overlapping set of biochemical reactions.

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