Calcium-Dependent Voltage Transients Evoked by Illumination in the Liverwort Conocephalum conicum

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The liverwort Conocephalum conicum with anion channels blocked by anthracene-9-carboxylic acid (A-9-C) and potassium channels blocked by tetrodoylaminium (TEA) generates dose-dependent responses to illumination further called voltage transients (VTs). Unlike the action potentials in untreated Conocephalum thalli, VTs do not propagate and cannot be evoked by electrical stimuli. Except A-9-C, two other anion channel inhibitors: ethacrinic and niflumic acids were effective in inducing VTs. These responses were blocked by DCMU, diethylstilbestrol and vanadate, which indicates that the photosynthetic electron transfer chain and the proton pump mediate in their generation. Light-induced VTs were considerably suppressed by calcium channel inhibitors: Mn2+, Gd3+, verapamil and nifedipine, and to a less extent by La3+ and diltiazem, provided that the incubation lasted more than 2 h. The participation of voltage-independent Ca2+-permeable channels in ionic mechanism of VTs is postulated.

Key words: Action potential — Calcium channels — Conocephalum conicum — Light-induced potential changes — Proton pump.

The liverwort Conocephalum conicum generates action potentials (APs) in response to different stimuli, among them illumination and depolarizing current (DC) pulses. The APs have typical all-or-none characteristics. Subthreshold light stimuli evoke solely receptor or generator potentials (GPs), transient depolarizations whose magnitude (no higher than 20 mV) depends on the stimulus strength, whereas overthreshold stimuli trigger APs of constant amplitudes (typically approx. 100 mV) (Dziubinska et al. 1983, Trebacz and Zawadzki 1985). The APs are blocked by calcium channel inhibitors: La3+, Mn2+ and verapamil, K+ channel inhibitor, TEA (Trebacz et al. 1989) and by anion channel inhibitors: A-9-C, (5-nitro-2-3-phenylpropyloamino) benzoic acid and Zn2+ (Trebacz et al. 1997). The investigations with the application of ion-selective microelectrodes confirmed the participation of the three main ion fluxes (Ca2+, Cl− and K+) in the electrogenesis of APs in Conocephalum (Trebacz et al. 1994). Additionally, an important role of the proton pump in restoring the resting potential (RP) was postulated (Trebacz et al. 1994). The ionic basis of APs in Conocephalum is similar in many aspects to that in giant Charophyta cells (Lunevský et al. 1983, Kikuyama et al. 1984, Kourie 1994, Thiel et al. 1997).

APs in Conocephalum play an important role in regulation of respiration. The respiration rate increases by up to 100% several seconds after the passage of an AP (Dziubinska et al. 1989).

A new phenomenon was recently observed in Conocephalum after application of A-9-C (Trebacz et al. 1997). The amplitude of APs evoked by electrical stimuli was gradually reduced and occasionally completely blocked. Meanwhile, light stimuli evoked sharp VTs which no longer fulfilled the all-or-none principle. Their amplitudes depended on the stimulus strength (light intensity) and duration of a dark period preceding illumination. Their halftime of membrane potential changes (t1/2) was 2-4 times reduced in comparison to that in APs. Unlike APs, VTs could be evoked within refractory periods or even during APs, being superimposed on them. The effect of A-9-C was accelerated and the amplitude of light-induced VTs increased after additional treatment with TEA, which completely blocked APs (Trebacz et al. 1997). Attempts were undertaken to clarify the nature of VTs. The amplitude of VTs depended on a Ca2+ concentration in the medium in the range 0.1–10 mM with the slope close to Nernstian. This pointed to the involvement of Ca2+ currents in VTs formation. On the other hand, calcium channel inhibitors: La3+, Gd3+, nifedipine, verapamil and diltiazem did not influence significantly VTs within 2 h of treatment. It was concluded that the VT is one of the constituents of the light-triggered AP which in untreated plants is screened by currents blocked by A-9-C and TEA. The currents are supposed to be responsible for the all-or-none character of APs.

The aim of the present study was further characterization of VTs. A special attention was given to the role of Ca2+ in the ion mechanism of VTs. Calcium channel inhibitors were applied longer and at higher concentrations than previously (Trebacz et al. 1997). Except A-9-C, two other anion channel inhibitors: niflumic acid and ethacrinic...
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acid were tested as possible factors inducing VTs. The role of the photosynthetic electron transfer chain and the proton pump in mediating between light absorption and VTs was examined. We also checked the ability of VTs to propagate and the possibility to evoke them by electrical stimulation.

**Materials and Methods**

*Conocephalum conicum* L. was collected together with a soil in a forest near Zwierzyniec, Poland, grown in a greenhouse being covered with a transparent foil. Young thalli were detached from the soil, thoroughly rinsed and mounted in an experimental chamber.

Electrophysiological experiments were performed as previously described (Trebacz et al. 1997). The transmembrane potential was measured with 3 M KCl filled glass microelectrodes (Hilgengen, Malsfeld, Germany) connected to VF-4 buffer amplifier (World Precision Instruments, Sarasota, FL, U.S.A.). The output signals were digitized by a custom made AD converter and registered on a hard drive of a PC. Light stimuli were applied by a xenon lamp (XBO 101. Wetron, Germany) equipped with interference and water filters. Electrical stimulation was applied from a regulated DC source through thin (0.2 mm) silver wires inserted into the thallus.

The experiments started after 2–4 h incubation of the thallus in light in standard solution containing 1 mM KCl, 0.1 mM CaCl$_2$, 50 mM sorbitol, 2 mM MES/Tris pH 7.0. Then, 4–8 APs were evoked every 10 min either by light or electrical stimulation in order to determine the threshold of excitation. Next, the standard solution was quickly exchanged to that containing additionally 2 mM A-9-C and 10 mM TEA. In separate series of experiments 50 μM niflumic acid or 0.5 mM ethacrynic acid was used instead of A-9-C. After VTs had been fully expressed which lasted approx. 2 h, calcium channel inhibitors: 5 mM La$^{3+}$, 2 mM Gd$^{3+}$, 5 mM Mn$^{2+}$, 0.5 mM diltiazem, 0.2 mM nifedipine, 0.2 mM verapamil were applied. DCMU, diethylstilbestrol (DES) and vanadate were used at 25 μM, 25 μM and 5 mM concentration, respectively. VTs were evoked by light stimuli of irradiance 300 μmol m$^{-2}$ s$^{-1}$. If not otherwise stated, they were applied every 10 min after 5 min in darkness. In such conditions the amplitudes of VTs reached maximal values. Each experiment was repeated at least three times. The values of parameters are given as mean ± SE.

**Results**

*General characteristics of VTs in Conocephalum*—Light-induced VTs appeared in nearly 100% of *Conocephalum* cells treated with A-9-C and TEA. We registered VTs in all 48 examined thalli. In order to check the capability of VTs to propagate thalli. In order to check the capability of VTs to propagate over the thallus, we covered its part where the microelectrode was inserted with an aluminium foil and illuminated the remaining part. We never registered VTs in cells located further than approx. 1 mm from the foil edge (Fig. 1).

VTs were induced by light but not by electrical stimulation. This might be caused either by incapability of DC pulses to evoke VTs or by inability of VTs to propagate from the vicinity of the cathode, the stimulating electrode at which cells are depolarized, to the cell where the microelectrode was located. We checked the other possibility by inserting the microelectrode as close to the cathode as possible (below 0.5 mm). We did not register VTs even after applying stimuli exceeding up to 5 times the threshold of excitation in untreated plants (Fig. 1).

*Effects of calcium channel inhibitors on light-induced voltage transients in Conocephalum*—In the previous paper (Trebacz et al. 1997) we demonstrated that amplitudes of VTs evoked by light in A-9-C and TEA treated cells of *Conocephalum conicum* depended on Ca$^{2+}$ concentration in the medium. This pointed to the involvement of calcium or calcium-dependent currents in ion mechanism of VTs. On the other hand, calcium chan-

![Fig. 1](http://pcp.oxfordjournals.org) Membrane potential changes evoked by light and electrical stimulation in cells of *Conocephalum conicum*. Traces: (a), (b) and (c) were obtained in the standard solution; traces (d), (e) and (f) in cells treated with 2 mM A-9-C and 10 mM TEA. (a) Hyperpolarization caused by darkening. (b) and (c) APs evoked by light and electrical stimulation, respectively. (d) Light-induced VT. (e) Stimulation artefact registered during an attempt of VT triggering by a DC current pulse. (f) A residual change of the membrane potential registered in a covered part of the thallus after illumination of the exposed part.
nel inhibitors: La$^{3+}$ (2 mM), Gd$^{3+}$, diltiazem, nifedipine and verapamil (all at 0.1 mM concentration) did not cause a significant reduction of VT amplitudes within two hours of treatment. In this study we used a little higher concentration of the inhibitors and incubation time up to 5 h to exclude the possibility that it was a diffusion rate which limited the influence of the inhibitors. Most of the inhibitors tested caused a significant reduction preceded by a temporary increase of VT amplitudes (Fig. 2A).

Mn$^{2+}$ is an efficient inhibitor of APs in Conocephalum (Trebacz et al. 1989). MnCl$_2$ added to the medium at 5 mM concentration caused initially an increase of VT amplitudes followed by a gradual inhibition of VTs starting from 2 h after its application. The decrease of VT amplitude (to 38±7%, n=3 of the control) resulted partially from RP decrease (by 27±9 mV) and partially from shifting the peaks of VTs towards more negative potentials (Fig. 2B). The response to light off changed its character after Mn$^{2+}$ application. Darkening caused delayed transient depolarizations whose amplitude reached occasionally 40 mV (typically 5-10 mV) (Fig. 2B). In no case these responses mediated the evoking of VTs.

Nifedipine was applied at 0.1 and 0.2 mM concentration. The inhibitor caused temporal increase of VT amplitudes followed by a significant inhibition, to 43±4% (n=3) of the control. It caused also depolarization of the RP by 25±10 mV. Nifedipine at 0.2 mM but not 0.1 mM concentration delayed the repolarization phase of VTs which was manifested by 42% increase in t1/2. Delayed slow depolarizations after light off were observed after 2-3 h of treatment (Fig. 2B).

Verapamil (0.2 mM) reduced the amplitudes of VTs to 45±9% (n=3) of the control after 3-4 h of incubation. The process of VT inhibition, consisting mainly in decrease of the membrane potential difference (by 38±4 mV), began after 1.5-2 h of treatment. This was accompanied by a slow delayed depolarization in response to darkening.

5 mM La$^{3+}$ reduced VT amplitudes to 78±13% (n=3). The beginning of inhibition became obvious between 2.5 and 3 h from its application. In single experiment 2 mM La$^{3+}$ caused 81% reduction of VT amplitude after 4 h of treatment. La$^{3+}$ caused a slight depolarization of the RP, on average by 17±2 mV. The half-time of VTs increased by 40% in La$^{3+}$ treated cells.

Another lanthanide, Gd$^{3+}$ applied at 2 mM concentration produced similar effects. After 1.5-2 h of treatment the amplitude of VTs began to be hindered and reached 66±15% (n=3) of the control value after another 2 h. Gadolinium, similarly as La$^{3+}$, caused a slight depolarization of the RP by 12±6 mV and an increase in t1/2 of VTs by 37%.

Diltiazem (0.5 mM) caused relatively weak inhibition of VTs (67±17%, n=4). Only in one experiment of four it lowered the VT amplitude to 17% of the control and caused depolarization of the RP by 65 mV after 4 h of treatment. In the remaining cases it reduced the amplitudes to 84±5% of the control. Transient delayed depolarizations were registered after light off even in those cells where reduction of VT amplitudes was only partial. Control experiments showed that in plants treated with A-9-C
and TEA, but without calcium channel inhibitors, VTs remained unchanged during 6 h of incubation (data not shown).

**Influence of ethacrinic and niflumic acids on the membrane potential in Conocephalum**—We demonstrated previously that from the anion channel inhibitors tested: A-9-C, NPPB and Zn$^{2+}$ only A-9-C was active in exposing VTs (Trebacz et al. 1997). Here, we examined the influence of two other inhibitors, ethacrinic and niflumic acids on light-induced membrane potential changes. Ethacrinic acid is an effective inhibitor of inward Cl$^{-}$ current responsible for a depolarization phase of APs in Charophyta cells (Lunevsky et al. 1983). Niflumic acid efficiently inhibits S-type anion channels in guard cells (Schwartz et al. 1995) and red light-induced AP-like responses in the moss Physcomitrella patens (Ermolayeva et al. 1996).

Ethacrinic acid (0.5 mM) caused depolarization of the RP by 20-30 mV and in consequence lowering of AP amplitudes. Additional treatment with TEA resulted in total inhibition of APs within approx. 30 min. Responses to illumination changed gradually into VTs after 1.5-2 h of treatment with ethacrinic acid. Maximal amplitude of VTs was reached when illumination had been preceded by 15-20 min of darkness (Fig. 3A).

Niflumic acid (50 μM) when added without TEA caused slight reduction of AP amplitudes and slowing down the depolarization phase. Light-triggered APs were blocked after 4-5 h but electrical stimuli were able to evoke APs even after 6 h of treatment. The generator potential was prolonged with superimposed VTs of low amplitudes (5±1 mV, n=4) appearing just after illumination (Fig. 3B). Simultaneous application of niflumic acid and TEA (10 mM) evoked similar effect to A-9-C plus TEA and ethacrinic acid plus TEA. TEA blocked APs completely. After approx. 2 h of incubation in niflumic acid VTs of gradually increasing amplitudes appeared. As in the case of ethacrinic acid, a long darkness period preceding illumination (15 min as compared to 5 min in A-9-C) was required for complete development of VTs.

**Effect of DCMU, diethylstilbestrol and vanadate on light-induced voltage transients in Conocephalum**—Light-triggered APs in Conocephalum are blocked by DCMU, the inhibitor of photosystem II. DCMU acts primarily on GPs, which in untreated plants play the role of AP triggers. DCMU treated plants generate normal APs after electrical stimulation (Trebacz and Zawadzki 1985).

DCMU (25 μM) added to plants preincubated in A-9-C plus TEA with fully expressed VTs caused total disappearance of VTs within approx. 30 min (Fig. 4A). This was preceded by a gradual decrease of VT amplitude. The RP...
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Fig. 4 Inhibition of light-induced VTs by 25 μM DCMU (A), 25 μM DES (B), and 5 mM vanadate, (C). (a) Control VTs evoked 10 min before application of the inhibitors. Traces (b), (c) and (d) in part (A), VTs obtained 10, 20 and 30 min after solutions exchange, respectively. Traces (b), (c) and (d) in part (B), VTs evoked after 30, 90 and 210 min of treatment, respectively. Traces (b), (c) and (d) in part (C), VTs evoked after 20, 40 and 60 min of treatment, respectively.

depolarized meanwhile by 20 mV.

DES and vanadate are inhibitors of the proton pump in the plasmalemma (Shimmen and Tazawa 1982, Nishizaki 1994, Michelet and Boutry 1995). DES applied at 25 μM concentration reduced the amplitude of VTs to 19 ± 3% (n = 4) of the control (Fig.4B). The reduction was partially caused by depolarization of the RP (by 30 mV) and partially by shifting the peak towards more negative potential.

Application of 5 mM vanadate caused a relatively fast decrease of VTs amplitudes (Fig.4C). The amplitudes of VTs were partially restored when a prolonged period in darkness (more than 15 min) preceded illumination (data not shown). No significant change of the RP was registered.

Discussion

Action potentials in *Conocephalum* seem to have the same characteristics and ion mechanism either when evoked by light or electrical stimulation. Both kinds of stimuli trigger APs of the same shape, though those induced by light are usually wider, i.e. their t1/2 is longer and are preceded by a slow phase, the GP (Fig.1). The APs exclude each other during refractory periods; it is impossible to evoke the AP electrically within an absolute refractory period after the AP triggered by light and vice versa (Trebacz and Zawadzki 1985). The dependence between a relative stimulus strength and duration of the refractory period is the same irrespective of the kind of the stimulus used (Trebacz 1989). Light and electrically evoked APs are equally susceptible to ion channel inhibitors (except those of anion channels) (Trebacz et al. 1989).

The study with the application of ion selective microelectrodes showed that changes of basic ion activities were the same during light and electrically-triggered APs (Trebacz et al. 1994). It was pointed out that identical sequence of events was the case during the APs but not before exceeding the excitation threshold. Before that, substantial differences in the electrical properties occurred. Slowly increasing, ramp electrical stimuli evoked APs at the same voltage as when instantly rising, rectangular pulses were used. On the other hand, responses to gradually increasing light stimuli showed accommodation. APs were not evoked even after considerable exceeding of the excitation threshold determined by sudden illumination (Trebacz 1989). Here, we show another difference between electrically and light triggered APs—occurrence of VTs masked by APs in the case of light but not electrical stimulation.

What is the nature of VTs? It was demonstrated in many Charophyta cells that the plasmalemma and tonoplast generate APs upon stimulation (Findlay and Hope 1964, Kikuyama and Tazawa 1976, Kikuyama and Shimmen 1997). They differ significantly in a time-course: plasmalemma APs are usually much narrower than tonoplast ones. This becomes obvious when a microelectrode tip is located in a cytoplasm or in a vacuole. In the first case only a plasmalemma AP is registered, whereas both superimposed APs are recorded in the other case. In *Conocephalum* it is hard to determine the localization of the microelectrode tip solely by microscopic observation. However, on the basis of dozens of experiments with the application of ion selective microelectrodes, especially H, Ca, and Cl-selective, we could conclude that the microelectrode tip was localized either in the cytoplasm or in the vacuole (Trebacz et al. 1994). We never observed VTs in untreated plants, thus the possibility that A-9-C uncouples the link between the plasmalemma and tonoplast should be excluded.

In the previous paper the possible ion basis of the VT
was discussed (Trebcz et al. 1997). Two main ion species: Ca^{2+} and Cl^- were taken into consideration as responsible for the depolarization phase of the VT because the equilibrium potentials for both these ions are positive in the standard solution. VTs with peaks at up to +50 mV were registered in the presence of 10 mM TEACl. The addition of 10 M TEACl shifted the equilibrium potential for Cl^- from +46 to -10 mV. The experiments in which 1 or 10 mM Ca^{2+} was added as CaCl_2 showed an increase in VT amplitude, which indicated that the Ca^{2+} influx to the cytoplasm is the most probable reason of depolarization during the VT (Trebcz et al. 1997). On the other hand, calcium channel inhibitors: La^{3+}, Gd^{3+} verapamil, nifedipine and diltiazem did not influence significantly VT amplitudes within 2 h of treatment. Here we show that at a slightly increased concentration and longer incubation times inhibition of VTs occurs in many cases. A problem of plant electrophysiology is lack of sufficiently specific Ca^{2+} channel inhibitors (Pineros and Tester 1997). Recently, Lewis and Spalding (1998) reported inhibition of a blue light-induced opening of anion channels in Arabidopsis by La^{3+} at a high (10 mM) concentration. Verapamil (10 μM) was used as an inhibitor of outward rectifying K+ channels in tobacco mesophyll protoplasts (Blom-Zandstra et al. 1997). On the other hand, Shimazaki et al. (1997) demonstrated that varapamil at as high concentration as 1 mM did not evoke side effects having no influence on the proton pump in the plasmalemma of guard cells from Commelina and Vicia, although it prevented a blue light-induced stomatal opening known to be Ca^{2+} and calmodulin dependent. Despite these limitations, comparison of effects of chemically different inhibitors justifies the conclusion on the involvement of the ion channels in question. A similar influence of many Ca^{2+} channel inhibitors supports the hypothesis about the participation of Ca^{2+} permeable channels in the depolarization phase of the VT. The lack of the specificity of the inhibitors was probably the reason that the reduction of the VT amplitude was partially caused by depolarization of the RP. We hope that application of the patch-clamp technique will allow definitive checking of the hypothesis upon the involvement of Ca^{2+} permeable channels in VTs generation.

In the recent article (Trebcz et al. 1997) we reported that A-9-C but not NPPB and Zn^{2+} affected light-triggered membrane potential changes in Conocephalum. In this study we show that not only A-9-C but also two other anion channel inhibitors, ethacrinic and niflumic acids are able to expose light-induced VTs. This confirms the explanation that application of anion channel inhibitors, especially in combination with TEA, blocks or suppresses Cl^- and K^+ conductance which shunts presumably weak Ca^{2+} currents responsible for VT generation. The lack of the effect of NPPB and zinc on light-induced voltage responses might be caused either by a too low concentration of the inhibitors (100 μM and 500 μM, respectively) or by independence of the anion channels in the plasma-lemma of Conocephalum on these inhibitors.

The question remains which way the light influences ion fluxes across the plasmalemma. The experiments with application of DCMU indicate that photosynthetic pigments but not phytochrome (Racusen and Satter 1975, Ermolayeva et al. 1996, 1997) or blue light absorbing system (Nishizaki 1988, 1994, Spalding and Cosgrove 1989) are photoreceptors in the case of light-induced VTs. The coupling between the light absorption and light-induced membrane potential changes is not yet fully understood. The presence of Cl^- and cation permeable channels in the thylakoid membrane constitutes a good starting point for such considerations (Pottosin and Schönknecht 1995, 1996). Cl^- and cation, mainly K^+ and Mg^{2+}, fluxes counterbalance protons taken up into thylakoids after the light onset (Bulychev and Vredenberg 1976, Pottosin and Schönknecht 1995, 1996). The role of Ca^{2+} in this process is still a matter of discussion. Miller and Sanders (1987) reported changes of cytoplasmic Ca^{2+} activity in Nitellopsis after light on and off measured with Ca-selective microelectrodes. Lewis et al. (1997) demonstrated no change of cytoplasmic Ca^{2+} concentration after blue light pulses. They used Arabidopsis seedlings expressing aequorin, a protein emitting photons depending on Ca^{2+} concentration. In Conocephalum cytoplasmic Ca^{2+} activity remains fairly constant during illumination/darkening transitions (Trebcz et al. 1994). Thus, it seems rather improbable that Ca^{2+} fluxes across chloroplast membranes mediate between light absorption and opening of Ca^{2+} permeable channels in the plasmalemma.

Except ion channels, the proton pump in the plasmalemma is often regarded as the structure participating in the photoresponse (Felle and Bertl 1986, Nishizaki 1996). The regulation of its activity may occur in different ways. It may respond to cytoplasmic pH changes (Shimazaki and Kondo 1987, Olivari et al. 1993), and changes in a cytoplasmic ATP content (Mimura and Tazawa 1986). An increase of the cytoplasmic pH after illumination and the decrease upon darkening seem to be common among green plants including Conocephalum (Felle and Bertl 1986, Yin et al. 1990, Okazaki et al. 1994, Trebcz 1994). Bulychev and Vredenberg (1995) proposed that light-triggered APs in liverworts, like Conocephalum, growing in poor habitats were caused by a temporal break-down of the plasmalemma proton pump within several seconds after illumination because of ATP deficit. They argued that just after light onset chloroplasts consume rather than produce ATP. The decisive role of the plasmalemma proton pump in light-induced membrane potential changes was also postulated by Nishizaki (1994). Blue light-induced transient depolarizations resembling VTs were blocked by proton pump inhibitors.
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VTs in Conocephalum are considerably suppressed by DES and vanadate. Thus, it seems that the proton pump plays an important role in evoking VTs. Transient suppression of the electrogenic proton pump upon cell illumination may, however, explain only a part of the depolarization occurring during VTs. The membrane potential in Conocephalum cells treated with proton pump uncouplers depolarizes to the "diffusion" level of approx. −70 mV (Trebacz et al. 1989). To explain depolarizations up to +50 mV, observed frequently at the peak of VTs, one has to consider, besides the proton pump suppression, the opening of ion channels.

It is often postulated that the depolarization caused by temporal suppression of the proton pump mediates in opening the voltage-dependent ion channels participating in light-induced APs (Trebacz 1989, Bulychev and Vredenberg 1995). The Ca\(^{2+}\) permeable channels in the plasmalemma, which are probably responsible for VTs, seem to be voltage independent. This was concluded after checking that VTs are not evoked by electrical stimulation and that they are limited to the spot directly illuminated, which precluded the propagation on the principle of local circuits engaging voltage-gated ion channels. Transient depolarizations appearing upon darkening after prolonged incubation in A-9-C and TEA resemble the generator potentials in untreated plants. These responses, although exceeding 40 mV, were unable to evoke VTs, which additionally confirms voltage-independence of the ion channels taking part in VT electrogensis. One can postulate that the proton pump and ion channels might be regulated by the same cytoplasmic factors whose levels change after illumination/darkening.

VTs and related phenomena may be a more common type of plant responses to illumination than APs. Light-triggered APs of all-or-none characteristics are restricted to a relatively narrow group of plants (Sinyukhin 1973, Gradmann 1976, Roblin 1982, Trebacz and Sievers 1998). In a vast number of plant species non-propagating, depending on the stimulus strength voltage changes resembling VTs are recorded (Bulychev and Turovetsky 1983, Spalding and Cosgrove 1989, Nishizaki 1987, 1988, Lewis et al. 1997). These plants probably either do not have functional voltage-dependent Cl\(^{-}\) channels in the plasmalemma as other excitable plants have, or, which is more probable, the coupling between factors like cytoplasmic pH, ATP, etc., whose levels are changed by illumination, and these channels is disconnected. Propagating signals like APs are not necessary when light stimulates the whole plant at the same time, but they may play a significant role in plants subjected to temporal insufficiency of light.

The investigation was supported by the State Committee for Scientific Research.

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(Received July 14, 1998; Accepted October 16, 1998)