Simulation of action potential propagation in plants

Vladimir Sukhov*, Vladimir Nerush, Lyubov Orlova, Vladimir Vodeneev

Department of Biophysics, N.I. Lobachevsky State University of Nizhny Novgorod, Nizhny Novgorod, Gagarin Avenue, 23, 603950, Russia

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**A B S T R A C T**

Action potential is considered to be one of the primary responses of a plant to action of various environmental factors. Understanding plant action potential propagation mechanisms requires experimental investigation and simulation; however, a detailed mathematical model of plant electrical signal transmission is absent. Here, the mathematical model of action potential propagation in plants has been worked out. The model is a two-dimensional system of excitable cells; each of them is electrically coupled with four neighboring ones. Ion diffusion between excitable cell apoplast areas is also taken into account. The action potential generation in a single cell has been described on the basis of our previous model. The model simulates active and passive signal transmission well enough. It has been used to analyze theoretically the influence of cell to cell electrical conductivity and H⁺-ATPase activity on the signal transmission in plants. An increase in cell to cell electrical conductivity has been shown to stimulate an increase in the length constant, the action potential propagation velocity and the temperature threshold, while the membrane potential threshold being weakly changed. The growth of H⁺-ATPase activity has been found to induce the increase of temperature and membrane potential thresholds and the reduction of the length constant and the action potential propagation velocity.

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1. Introduction

Action potential (AP) generation and propagation are considered to be one of the primary responses of a plant upon the actions of various environmental factors. AP induces a number of changes in physiological processes such as photosynthesis, respiration, phloem transport, gene expression and others (Fromm and Spanswick, 2000; Blackman and Overall, 2001). So, the application of theoretical approach here, in particular, the mathematical model of plant electrical signal transmission is absent. The problem, however, is quite complicated for an experimental analysis methodologically (changes are to be required of the cell to cell conductivity and the H⁺-ATPase activity over a lengthy stem part) as well as due to considerable lability of these parameters (Michelet and Boutry, 1996; Fleurat-Lessard et al., 1997). Conducting vascular bundles are believed to be AP propagation pathways in plants (Fromm and Lautner, 2007; Sibaoka, 1991; Opritov et al., 1991; Dziubinska, 2003). There are two possible channels to transmit electrical signals: symplast of parenchyma cells (Sibaoka, 1962, 1991; Opritov et al., 1991) and sieve elements (Fromm and Lautner, 2007; Volkov, 2000). AP was registered in both types of cells (Fromm and Lautner, 2007; Sibaoka, 1991; Koziolek et al., 2003). Parenchyma cells and sieve elements are quite different electrophysiologically; in particular, they differ in cell to cell electrical conductivity (Fromm and Lautner, 2007; Dziubinska, 2003) and content of H⁺-ATPase (Michelet and Boutry, 1996; Fleurat-Lessard et al., 1997), which is a basic primary active electrogenic ion transporter in the plasmalemma (Spanswick, 2006). The study of the influence of these two factors on the AP propagation may clear up the problem of electrical signal transmission in plants. The problem, however, is quite complicated for an experimental analysis methodologically (changes are to be required of the cell to cell conductivity and the H⁺-ATPase activity over a lengthy stem part) as well as due to considerable lability of these parameters (Michelet and Boutry, 1996; Fleurat-Lessard et al., 1997; Holdaway-Clarke et al., 2000; Blackman and Overall, 2001). So, the application of theoretical approach here, in particular, the mathematical model approach, seems to be quite topical.

There are some models that consider the AP generation under external stimuli (Mummert and Gradmann, 1991; Beilby, 1982, 2007; Sukhov and Vodeneev, 2009) or deal with the electrical...
processes developing on plasmalemma without stimulation (Gradmann et al., 1993; Gradmann and Hoffstadt, 1998; Gradmann, 2001a,b; Tyerman et al., 2001; Shabala et al., 2006). However, there are only a few works (Pietruszka et al., 1997; Garkusha et al., 2002; Sukhov et al., 2011), which describe theoretically the AP propagation in plants, but they do not take into account in detail an ion mechanism of the electrical response generation. Structure of the excitable cell symplast in plants is qualitative similar with the electrically coupled cardiomyocyte syncytium one in animals (Trebacz et al., 2006). This excitable structure can be described by a two-dimensional system of excitable elements with local electrical connections (Beaumont et al., 1998; Wohlfart and Ohlen, 1999; Ten Tusscher and Panfilov, 2006; Ten Tusscher et al., 2006; Shajahan et al., 2009), which is a simplified model of the real three-dimensional tissue. Therefore, it can be used in the theoretical description of the AP propagation in the three-dimensional plant symplast (Garkusha et al., 2002; Sukhov et al., 2011).

The aim of this work is to develop a mathematical model of AP propagation in plants and to carry out a simulation analysis of the influence of the cell to cell electrical conductivity and plasmalemma H−-ATPase activity on the electrical signal transmission in these objects.

2. The model

2.1. The action potential generation model

The AP generation has been simulated on the basis of our previous model (Sukhov and Vodeneev, 2005, 2009), with modifications. This model describes well the AP generation in higher plants as well as the ion fluxes during electrical response (Sukhov and Vodeneev, 2009) that allows to avoid a repeated model verification. Also, detailed description of the number of ion transport processes offers the challenge for the model using in analysis of interaction between cells, the electrical signals functional role, etc.

![Diagram of a plant cell electrophysiological model](Image)

**Fig. 1.** Scheme of a plant cell electrophysiological model (Sukhov and Vodeneev, 2009, with modifications). \(E_m\) is the electrical potential of the plasma membrane; \([K^+]\), \([Cl^-]\) and \([Ca^{2+}]\) are the concentrations of potassium, chloride and calcium ions, respectively (M); \(B_{in}\) and \(B_{out}\) are free and \(H^+\) bound proton-buffer molecules in the cell; \(B_{BMA}\), \(B_{BMA}^{out}\) and \(B_{BMA}^{in}\) are free; \(H^+\) and \(K^+\) bound buffer molecules in the apoplast; \(P_{max}\) is the maximum permeability; \(p_a\) and \(p_n\) are the open state probability and the inactivated state probability of the ion channel; \(k_{f_{in}}\) and \(k_{f_{out}}\) are the velocity constants of ion channel transitions from the closed state to the open one and vice versa (subscript "in" or from the open state to the inactivated one and vice versa (subscript "out"); \(E_{ATP}\) is the ATPTase concentration; \(k_1, k_2, k_3, k_4\) are velocity constants of forward (\(\rightarrow\)) and reverse (\(\leftarrow\)) transitions between two states of the pump; \(V^f\) and \(V^r\) are the total velocity constants of 2H+/Cl−-symporter and H+/K+-antiporter, respectively.
and $2 \text{H}^+/\text{Cl}^-$ symporter, respectively; $E_{PH} = (\Delta G_{\text{ATP}}/F) + E_H$, $E_{PCA} = (\Delta G_{\text{ATP}}/F) + E_{Ca} - E_H$ and $E_{Cl} = E_{Cl} + 2E_H$ are equilibrium potentials of $\text{H}^+$-ATPase, $\text{Ca}^{2+}$-ATPase and $2 \text{H}^+/\text{Cl}^-$ symporter fluxes, $E_K$, $E_{Cl}$, $E_{Ca}$ and $E_H$ are equilibrium potentials of $K^+$, $\text{Cl}^-$, $\text{Ca}^{2+}$ and $\text{H}^+$. Eq. (2) has been used for the electrical conductivity:

$$g_k = \frac{j_k}{E_m - E_k}$$

where $j_k$ and $E_k$ are the flux and the equilibrium potential for the process $k$, respectively.

Eq. (1) has been used because the AP development in the majority of higher plants is a slow process (from seconds to tens of seconds) (Sibaoka, 1991; Opritov et al., 1991; Trebačz et al., 2006), so the difference between the current membrane potential value and the stationary one is assumed to be very small for the conductivities used. Fig. 2 shows that cold-induced $E_m$ changes do not depend on the type of description (differential or stationary). What is really important in the AP generation model with the stationary $E_m$ is it can be numerically calculated (Euler’s method) at $\Delta t = 0.1 \text{ s}$ or more, whereas the model with the differential membrane potential requires $\Delta t \leq 0.005 \text{ s}$. This substantial increase in $\Delta t$ simplifies the analysis of multi-cellular complexes used for the AP propagation simulation.

2.2. The action potential propagation model

A two-dimensional array of identical excitable cells (monolayer of ones) has been used to simulate the AP propagation (Fig. 3a). The length ($L$) of the array is 800 cells, and the width ($W$) is 30 cells. Each cell with its surrounding apoplast is considered as a single cubic element with dimensions $a \times a \times a = 100 \times 100 \times 100 \mu\text{m}^3$.

Each cell is electrically connected with four neighboring ones (Fig. 3b) in accordance with the symplast structure where neighboring cells are electrically connected via plasmodesmata (Fromm and Lautner, 2007; Sibaoka, 1991). Taking into account relatively small cell sizes and high intracellular conductivity it has been assumed that the AP propagation depends only on cell to cell conductivity in our model. Electrical currents depending on intercellular interactions are represented by

$$i_{hw} = \sum_{ks} g_{hwk}(E_m^{ks} - E_m^{hw})$$

where $l \in [1,L]$, $w \in [1,W]$, $E_m^{hw}$ and $E_m^{ks}$ are cell membrane potentials with positions $(l;w)$ and $(k;s)$, respectively. $g_{hwk}$ is the electrical conductivity between these cells and $(k;s)$ is equal to $(l-1;w)$, $(l+1;w)$, $(l;w-1)$ or $(l;w+1)$.

Fig. 2. Cold-induced membrane potential ($E_m$) changes simulated by differential (a) and stationary (b) descriptions $E_m$ changes have been calculated by Euler’s method at $\Delta t = 5 \text{ ms}$ for the differential description and $\Delta t = 100 \text{ ms}$ for the stationary one.

Fig. 3. Scheme of a two-dimensional array of identical excitable cells simulating the AP propagation (a), and its single cell fragment (b) i is cell to cell electric current.
Using Eq. (1), we can find Eq. (4), which describes the stationary membrane potential for \((l,w)\) cell:

\[
E_{m_{lw}} = \frac{g_{x}E_{x} + g_{c1}E_{c1} + g_{c2}E_{c2} + g_{h}E_{h} + g_{fca}E_{fca} + g_{sy}E_{sy} + \sum_{k,s}g_{k,s}E_{m_{ks}}}{g_{x} + g_{c1} + g_{c2} + g_{h} + g_{fca} + g_{sy} + \sum_{k,s}g_{k,s}}
\]

(4)

where electrical conductivities of all plasmalemma ion transport systems and their equilibrium potentials relate to \((l,w)\) cell. In this work \(g_{k,s}=g\), where \(g\) is constant, i.e. cell to cell electrical conductivities are equal.

Diffusion changes of apoplastic ion concentrations (without \(\text{Ca}^{2+}\)) relating to \(\text{Ca}^{2+}\) are calculated every \(3\) ms in plants (Etherton, 1977; Fromm and Lautner, 2007; Sibaoka, 1991; Opritov et al., 1991). As a result, \(\Delta V\sim\) tens \(\mu\)s is used for the adequate description of the AP propagation, which is becoming unrealistic for too long calculations.

This problem can be solved using the method described in work of Qu and Garfinkel (1999) with some modifications. Model equations have been calculated (Euler’s method) using three values of \(\Delta V\) simultaneously: \(\Delta V_{1}=100\) ms, a basic time step, has been used for the processes in the single cell without intercellular interactions, i.e. transmembrane fluxes, concentration changes, ion channel state transitions, etc.; \(\Delta V_{2}=25\) \(\mu\)s has been used for the passive membrane potential change propagation (in Eq. (4) only membrane potentials are calculated every \(\Delta V_{2}\), other values are calculated every \(\Delta V_{1}\)); \(\Delta V_{3}=10\) ms has been used for ion diffusion between apoplast regions of neighboring cells (Eq. (5) has been calculated every \(\Delta V_{3}\)). These three values of \(\Delta V\) have been chosen as indicated since their further reduction is not essential for the calculation results.

2.3. Gradual cooling simulation

The AP generation is induced by the simulation of the gradual cooling used in a number of experimental works (Opritov et al., 1991, 2002, 2005; Vodeneev et al., 2006, 2007), as well as in our previous model (Sukhov and Vodeneev, 2009). First hundred lines of cells (Fig. 3, cells with \(l\leq 100\)) have been subjected to simulation stimulated as a linear decrease in the temperature (4 °C min \(^{-1}\)) from 25 to 10 °C. Cells with \(l>100\) have not been cooled.

2.4. Numerical solution of model equations

The model equations have been numerically calculated by Euler’s method using the special computer program, which has been worked out with Borland Delphi 7. The values of model parameters have been described in previous work (Sukhov and Vodeneev, 2009). The values of the cell to cell electrical conductivity \((g)\) and the plasma membrane \(\text{H}^{+}\text{-ATPase}\) activity \((\text{A}^{\text{H}^{+}\text{-ATPase}})\) have been varied. Variations of \(\text{H}^{+}\text{-ATPase}\) activity have been described as relative changes in enzyme concentration \((\text{C}/\text{C}_{0})\) where \(\text{C}_{0}\) is the concentration corresponding to \(E_{m}=-180\) mV (see the AP generation model in work (Sukhov and Vodeneev, 2009)).

3. Results and discussion

3.1. Influence of cell to cell electrical conductivity and plasmalemma \(\text{H}^{+}\text{-ATPase}\) activity on the membrane potential without stimulation

At the first stage we have studied the influence of the cell to cell electrical conductivity and plasma membrane \(\text{H}^{+}\text{-ATPase}\) activity on the membrane potential without stimulation. In this case the changes in conductivity did not modify the membrane potential at rest (Fig. 4a), which is equal to \(-166\) mV at \(\text{A}^{\text{H}^{+}\text{-ATPase}}=0.7\). This magnitude is in a good accordance with the membrane potential in plant cells usually ranging between \(-80\) and \(-200\) mV (Fromm and Lautner, 2007).

The dependence of the resting membrane potential on \(\text{H}^{+}\text{-ATPase}\) activity is shown in Fig. 4b \((g=0.04\ S\ cm^{-2})\). \(\text{H}^{+}\text{-ATPase}\) activity decrease has been accompanied with the resting potential reduction at \(\text{A}^{\text{H}^{+}\text{-ATPase}}=0.1\) from 2 to 0.68 and from 0.25 to 0, while the stationary potential is not established at \(\text{A}^{\text{H}^{+}\text{-ATPase}}=0.25; 0.68\).

Fig. 4c shows the dynamics of the membrane potential at different \(\text{H}^{+}\text{-ATPase}\) activities (in 300,15 cell). Simulation of low activity \((\text{A}^{\text{H}^{+}\text{-ATPase}}=0.1)\) causes the spike and formation of the depolarized resting potential \((-90\ mV)\). Such changes of the membrane potential are in good agreement with its experimental dynamics at \(\text{H}^{+}\text{-ATPase}\) inhibition by external factors; in particular, the gradual cooling (Opritov et al., 1991; Pyatygin et al., 1999).

Simulation of a moderate \(\text{H}^{+}\text{-ATPase}\) activity \((\text{A}^{\text{H}^{+}\text{-ATPase}}=0.45)\) disturbs the resting potential formation: the membrane potential depolarization, which is developed in this case, activates potential-dependent ionic channels and thereby induces oscillations of \(E_{m}\) (Fig. 4c). This result corresponds to experimental and theoretical studies (Gradmund, 2001a,b; Tyerman et al., 2001; Shabala et al., 2006; Thiel et al., 1992), which show the appearance and development of membrane potential and ionic flux oscillations in connection with \(\text{H}^{+}\text{-ATPase}\) activity. Simulation of a high \(\text{H}^{+}\text{-ATPase}\) activity \((\text{A}^{\text{H}^{+}\text{-ATPase}}=1)\) induces a monotonous formation of the high resting potential \((-180\ mV)\) (Fig. 4c), which is also in a good agreement with the membrane potential values in higher plant cells (Fromm and Lautner, 2007; Sibaoka, 1991; Opritov et al., 1991).

So, our model can describe the depolarized membrane potential, oscillations and the normal resting potential in plant cells. Transitions between different modes depend on \(\text{H}^{+}\text{-ATPase}\) activity, which correlates very well with experiments (Shabala et al., 2006; Pyatygin et al., 1999; Thiel et al., 1992). The cell to cell electrical conductivity does not affect the resting potential. Theoretical analysis of electrical signal propagation in plants is not possible to be carried out in the oscillatory mode; therefore we use \(\text{H}^{+}\text{-ATPase}\) activities more than 0.68 or less than 0.25 in what follows.

3.2. Simulation of the passive and active electrical signal propagation

The electrical signal propagation in plants can be active, which is connected with the AP generation in unstimulated cells, as well as passive, where membrane potential changes in unstimulated cells are electrotonic and the active electrical response is absent in these cells (Opritov and Retivin, 1982; Opritov et al., 1991). For the description of electrotonic membrane potential propagation, the calcium concentration has been decreased, as well as in work (Sukhov and Vodeneev, 2009), from \([\text{Ca}^{2+}]_{\text{out}}=0.5\ mM\) to \([\text{Ca}^{2+}]_{\text{out}}=0.5\ \mu M\) in the apoplastic regions of unstimulated cells \((l>100)\), since the decrease of \(\text{Ca}^{2+}\)
suppresses the AP generation in higher plants in experiments (Felle and Zimmermann, 2007; Vodeneev et al., 2006; Iijima and Sibaoka, 1985; Hodick and Sievers, 1988). It should be noted that internal sources of Ca^{2+} can plays role in the AP generation (Biskup et al., 1999; Trewaves, 1999; Wacke and Thiel, 2001), but these mechanisms have not been taken into account here. Also, the model does not take into account Ca^{2+} influence on the whole plasma membrane (changes of membrane stability, fluidity, etc.) (Opritov et al., 1991), i.e. the external Ca^{2+} decrease is not stressor in our model. As a result, the external Ca^{2+} drop fully suppresses the active AP propagation in unstimulated cells in the model; at that calcium influence on the whole plasmalemma is not simulated.

**Fig. 4.** Dependence of the membrane potential ($E_m$) at rest on the cell to cell electrical conductivity ($g$) (a) and H^{+}-ATPase activity ($A_H^{\text{ATPase}}$) (b) and examples of $E_m$ dynamics in cell (300; 15) at different H^{+}-ATPase activities (c).

**Fig. 5.** Passive (a) and active (c) electrical signal propagation and a dependence (b) of the electrical response amplitude ($\Delta E_m$) on a distance ($x$) from the stimulated region at the passive propagation. The cell to cell electrical conductivity ($g$) equals to 0.04 S cm$^{-2}$, the plasmalemma H^{+}-ATPase activity ($A_H^{\text{ATPase}}$) equals to 0.7; ($l$; 15) is the position of a cell, cells with $l \leq 100$ are stimulated, cell size equals to $100 \times 100 \times 100$ μm$^3$, $\lambda$ is the length constant, solid line is an exponential trend.
The simulation of the passive electrical signal propagation (Fig. 5a) shows that the response in stimulated cells (electrical reactions in cells (50; 15) and (100; 15) are shown) includes subthreshold changes of the membrane potential, the depolarization phase and the repolarization phase formed by two stages. This dynamics is consistent with experimental membrane potential changes in the cooling zone of plant (Opritov et al., 1991, 2002, 2005; Vodeneev et al., 2006, 2007; Pyatygin et al., 1999; Krol et al., 2003, 2004). However, as the distance from the stimulation zone increases, the amplitude of electrical responses decreases exponentially (electrical reactions in cells (150; 15), (200; 15), (300; 15) and (600; 15) are shown) that corresponds to the passive electrical reaction propagation in experiments (Opritov et al., 1991).

Fig. 5b shows the electrical response amplitude dependence on the distance from the stimulation zone simulated by the model, and the length constant calculation. In this variant, the length constant is equal to 0.29 cm, which is in a good agreement with the experimental magnitude in the range of 0.28–0.55 cm obtained for Cucurbita pepo L. Pyatygin (2008). Besides, the cell to cell electrical conductivity (0.04 S cm$^{-2}$) is close to the experimental one for Elodea canadensis (0.02 S cm$^{-2}$) (Spanswick, 1972). All these confirm the correctness of our description.

The simulation of the active electrical signal propagation (all cells are excitable) shows the workability of the model as well (Fig. 5c). In this case APs are generated in the unstimulated zone (electrical reactions are shown in cells (150; 15), (200; 15), (300; 15) and (600; 15)) as well as in the cold-stimulated zone (cells (50; 15) and (100; 15)); AP parameters in the unstimulated region are not dependent on the distance from the stimulation zone. The electrical signal propagation velocity is constant. This result is in a good qualitative agreement with experimental data on AP propagation in higher plants (Fromm and Lautner, 2007; Opritov et al., 1991; Trebczak et al., 2006; Davies, 2006).

The simulated AP propagation velocity is equal to 0.6 cm s$^{-1}$, which is in a good quantitative accordance with corresponding experimental values for most plants without motor activity: from several mm s$^{-1}$ or less (Stankovic and Davies, 1996; Stankovic et al., 1998; Grams et al., 2009; Felle and Zimmermann, 2007; Dziubinska, 2003; Zawadzki et al., 1991, 1995; Volkov and Haack, 1995; Favre et al., 2001; Favre and Degli Agosti, 2007) to several cm s$^{-1}$ (Fromm and Spanswick, 1993; Fromm and Bauer, 1994; Fromm et al., 1995; Grams et al., 2007). Moreover, the length constant 0.3 cm corresponds to the cold-induced AP velocity of 0.5 ± 0.2 cm s$^{-1}$ in Cucurbita pepo L. (calculated on the basis of data in Pyatygin (2008)) that additionally supports the model correctness.

Magnitudes of other AP parameters also correlate with the experimental ones. The membrane potential threshold, calculated as the difference between resting $E_m$ and $E_m$ at the beginning of rapid depolarization (Opritov et al., 1991), are equal to 36 mV in the cold-stimulated zone center (cell (50; 15), the first spike); this lies in the range obtained in the experiment (30 to 70 mV (Pyatygin, 2008)). The membrane potential threshold in the unstimulated zone (cell (300; 15)) decreases up to 20 mV that correlates with 12–30 mV in the experiment (Pyatygin, 2008).

The total amplitude of the simulated electrical response (subthreshold $E_m$ changes + AP) in stimulated cells is equal to 115 mV (cell (50; 15)) that corresponds to experimental amplitudes of electrical reactions induced by the gradual cooling in Cucurbita pepo L. (100–120 mV and more (Opritov et al., 1991, 2002, 2005; Pyatygin et al., 1999)). The simulated AP amplitude in the unstimulated zone (cell (300; 15)) is equal to 105 mV that lies in the AP amplitude range of most plants without motor activity (from 30–50 mV (Stankovic et al., 1998; Grams et al., 2009; Zawadzki et al., 1991) to 100 mV and more (Opritov et al., 1991; Wacke and Thiel 2001; Favre et al., 2001; Favre and Degli Agosti 2007; Fromm et al., 1995).

The simulated AP duration in stimulated (cell (50; 15)) and unstimulated (cell (300; 15)) zones is equal to 30 and 26 s, respectively; for most plants without motor activity, it ranges in the experiment from several seconds (Fromm et al., 1995) to tens of seconds and more (Opritov et al., 1991, 2002; Vodeneev et al., 2006, 2007; Krol et al., 2003, 2004; Favre and Degli Agosti, 2007).

It should be noted that the gradual cooling induces the series of repetitive APs in the stimulated zone; however, only the first spike propagates actively, while the second one is transmitted passively. This result also correlates well with the series of AP under gradual cooling in the experiment (Opritov et al., 1991, 2002, 2005; Pyatygin et al., 1999), together with the ability to propagate for only a single AP (Opritov et al., 1991, 2005).

Thus, simulated electrical responses in passive and active electrical signal propagation in plants are in a good qualitative and quantitative agreement with the experimental data that confirms the validity of our model. So, it can be used in the subsequent theoretical analysis of the influence of $g$ and $A_0$-ATPase on the AP propagation.

### 3.3. Influence of cell to cell electrical conductivity and plasmalemma H$^+$-ATPase activity on the passive electrical signal propagation

Fig. 6 shows the influence of cell to cell electrical conductivity and plasmalemma H$^+$-ATPase activity on the length constant ($\lambda$). The growth of $g$ induces the nonlinear rise of $\lambda$ (Fig. 6a). As it is known (Cotterill, 2002), $\lambda$ can be calculated using (6) derived from the cable equation:

$$\lambda = \sqrt{\frac{G_m}{G_m + G_i}}$$

where $G_m$ is the total plasmalemma conductivity per unit length, $G_i$ is the total inner conductivity of the excitable structure per unit length depending on intracellular and cell to cell conductivities. In our model the intracellular resistance is not taken into account; so we have assumed that $G_m$ depends only on $g$, $G_m$ and $G_i$ have been calculated by $G_m = 6g_m W a$ and $G_i = 4g_i W a^2$, where $W = 30$ cells is the width of the system, $a = 10^{-2}$ cm is the array

![Fig. 6. Dependence of the length constant ($\lambda$) on the cell to cell electrical conductivity ($g$) and plasmalemma H$^+$-ATPase activity ($A_0$). Solid lines are trends (see the text).](image-url)
element dimension, $g_m$ is the total plasmalemma conductivity per unit area. “6” reflects six surfaces of cubic cells, which contain transporters of ions, and “4” corresponds with four ones, which contain plasmodesmata in our model (Fig. 3b). Therefore, Eq. (6) is transformed into

$$\lambda = \sqrt{\frac{4g_o^2}{6g_m}}$$

(7)

It should be noted that $g_m$ is not changed in this variant of analysis ($A_e^{H^-\text{ATPase}}=0.7$, $g$ is varied) and is equal to $3.2 \times 10^{-5}$ S cm$^{-2}$. The last result is in agreement with data of literature $(3 \times 10^{-6} - 8 \times 10^{-5}$ S cm$^{-2}$ in plants (Spanswick, 2006; Holdaway-Clarke et al., 2000; Beilby, 2007; Pyatygin, 2008)) and supports correctness of the model. Fig. 6a shows that Eq. (7) well describes dependence of $\lambda$ on $g$ at these conditions (trend line). Thus, it can be suggested that growth of $g$ increases $\lambda$ according to the cable equation.

The length constant dependence on the plasmalemma H$^+$-ATPase activity is shown in Fig. 6b. It should be noted that $\lambda$ has only been calculated for $A_e^{H^-\text{ATPase}} \geq 0.68$, since at low values (less than 0.25) the AP generation has been suppressed, which fits experimental data reasonably well (Vodeneev et al., 2006), and the moderate ones have induced membrane potential oscillations. When $A_e^{H^-\text{ATPase}} \geq 0.68$, the growth of proton pump activity has caused a weak decrease of $\lambda$ (from 0.29 to 0.21 cm).

It should be noted that the experimental confirmation of the plasmalemma H$^+$-ATPase activity influence on the length constant in higher plants is absent. However, this theoretical result can be well explained using the cable equation. In this case $g$ does not change and equals to 0.04 S cm$^{-2}$, while $g_m$ increases from $2.9 \times 10^{-5}$ S cm$^{-2}$ ($A_e^{H^-\text{ATPase}}=0.68$) to $6.8 \times 10^{-5}$ S cm$^{-2}$ ($A_e^{H^-\text{ATPase}}=2.00$) (data no shown). The H$^+$-ATPase conductivity rises from $6 \times 10^{-6}$ S cm$^{-2}$ ($A_e^{H^-\text{ATPase}}=0.68$) to $2.3 \times 10^{-5}$ S cm$^{-2}$ ($A_e^{H^-\text{ATPase}}=2.00$) (data no shown), that supports direct H$^+$-ATPase participation in growth of $g_m$. Fig. 6b shows that Eq. (7) well describes dependence of $\lambda$ on $A_e^{H^-\text{ATPase}}$ under different activities of H$^+$-ATPase (trend line).

Thus, our theoretical analysis shows that the cell to cell electrical conductivity and the plasmalemma H$^+$-ATPase activity can influence on the passive electrical signal propagation. In particular, the changes of these parameters may induce the length constant modifications. However, the parameters of the passive electrical signal transmission influence the AP propagation (Cotterill, 2002; Pyatygin, 2008); therefore, the simulated active electrical signal transmission also possibly depends on the electrical conductivity between cells and the plasmalemma H$^+$-ATPase activity.

3.4. Influence of cell to cell electrical conductivity and plasmalemma H$^+$-ATPase activity on the active electrical signal propagation

Fig. 7 shows the influence of cell to cell electrical conductivity and plasmalemma H$^+$-ATPase activity on the simulated AP propagation velocity. The growth of electrical conductivity induces a nonlinear rise of AP velocity (Fig. 7a) from 0.17 cm s$^{-1}$ (0.003 S cm$^{-2}$) to 0.8 cm s$^{-1}$ (0.1 S cm$^{-2}$). This result is a good illustration of the connection between the AP propagation and the cell to cell electrical conductivity, in particular, the electrical conductivity of plasmodesmata (Sibaoka, 1991; Opritov et al., 1991; Dziubinska, 2003). It confirms our previous data (Sukhov et al., 2011), which showed that growth of conductivity between cells accelerated the AP propagation in a simple two-dimensional model on base of the FitzHugh–Nagumo equations. The increase in plasmalemma H$^+$-ATPase activity induces the reduction in AP propagation velocity, which has been simulated by the model (Fig. 7b). It should be noted that the AP generation has not been observed at $A_e^{H^-\text{ATPase}} < 0.68$ and the AP propagation has been suppressed at $A_e^{H^-\text{ATPase}} > 1$.

Dependences of temperature and membrane potential thresholds on $g$ and $A_e^{H^-\text{ATPase}}$ are shown in Fig. 8. The temperature threshold has been calculated as the difference between initial temperature and temperature at the beginning of rapid depolarization, $\Delta T_m$ is the membrane potential threshold in the stimulated cell (50; 15), $\Delta E_m^i$ is the membrane potential threshold in the unstimulated cell (600; 15). The membrane potential thresholds have been calculated as the difference between resting $E_m$ and $E_m$ at the beginning of rapid depolarization.

The increase in conductivity has induced an essential rise of the temperature threshold (from 1.2 to 3.5 K) simulated by the model, whereas membrane potential thresholds in the stimulated
induced by decrease of the plasmalemma H\(^+\)-ATPase activity induces the rise of the temperature threshold as well as the membrane potential one (Fig. 8b). This process is possibly connected with the reduction of the resting membrane potential (Fig. 4a).

Taken together, these results theoretically show that growth of the cell to cell electrical conductivity accelerates AP propagation. It is rather connected with increase in the length constant, which strongly depends on \(g\) (Fig. 6a), than with change in the membrane potential threshold weakly depended on \(g\) (Fig. 8a). It should be noted that the temperature threshold (stimulated growth of AP propagation velocity and the AP generation threshold, which strongly depends on \(Ae\text{H-ATPase}\) (Fig. 8b), whereas \(\lambda\) change is weak under these conditions (Fig. 6b). However, there is some minimal H\(^+\)-ATPase activity, which is necessary for generation and propagation of AP (\(Ae\text{H-ATPase} = 0.68\)).

Thus, our results show that optimal conductivity for the AP generation (the low temperature threshold) can differ from the optimal conductivity for the AP transmission (low velocity). The H\(^+\)-ATPase activity decrease can compensate this effect; but the low enzyme activity suppresses the AP generation.

### 4. Conclusion

The elaborated model of the AP propagation describes the basic features of the passive and active electrical signal transmission. The values of signal propagation parameters (the length constant, the AP velocity, electrical response amplitude and threshold) are in a good quantitative accordance with the experimental data. From all this it follows that the model can be used for the theoretical investigation of the AP propagation under different conditions.

The model has shown that the cell to cell electrical conductivity and the plasmalemma H\(^+\)-ATPase activity influence AP generation and propagation parameters. This influence can rather intricate that the conductivity rise induces simultaneously the growth of AP propagation velocity and the AP generation threshold; the H\(^+\)-ATPase activity rise can induce the AP propagation velocity reduction and the threshold growth, but a considerable drop in the activity (\(Ae\text{H-ATPase} < 0.68\)) disrupts the electrical response generation. These results provide a theoretical ground for understanding that cell to cell electrical conductivity and the plasmalemma H\(^+\)-ATPase activity are essential factors influencing on AP generation and propagation. In particular, they show that sieve elements possessing the high electrical conductivity between cells (Fromm and Lautner, 2007) and the low plasma membrane H\(^+\)-ATPase content (Fleurat-Lessard et al., 1997) can be a good system of the AP transmission, but not optimal for the AP generation.

A further analysis of the AP propagation mechanisms requires the investigation of heterogeneous systems consisted of the elements with weak electrical coupling (parenchyma cells) and the elements with strong electrical coupling (sieve elements).

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### References


