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Turgor Pressure Regulation in *Valonia utricularis*

EFFECT OF CELL WALL ELASTICITY AND AUXIN[1]

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ABSTRACT

The electrical membrane resistance ρ_0 of the marine alga *Valonia utricularis* shows a marked maximum in dependence on the turgor pressure. The critical pressure, P_C, at which the maximum occurs, as well as its absolute value, ρ_0^{max}, are strongly volume-dependent. Both P_C and ρ_0^{max}, increase with decreasing cell volume. It seems likely, that these relationships reflect the elastic properties of the cell wall, because the volumetric elastic modulus, ϵ, is also volume-dependent, increasing hyperbolically with cell volume. Both P_C and ρ_0^{max} can be affected by external application of indole-3-acetic acid at concentrations of $2 \cdot 10^{-7}$ M to $2 \cdot 10^{-5}$ M. The critical pressure is shifted by 1.2 to 6 bars toward higher pressures and the maximum membrane resistance increased up to 5.6-fold. During the course of the experiments (up to 4 hours), however, IAA had no effect on the volumetric elastic modulus, ϵ.

The maximum in membrane resistance is discussed in terms of a pressure-dependent change of potassium fluxes. The volume dependence of P_C and ρ_0^{max} suggests that not only turgor pressure but also ϵ must be considered as a regulating parameter during turgor pressure regulation. On this basis a hypothesis is presented for the transformation of both, a pressure signal and of changes in the elastic properties of the cell wall into alterations of ion fluxes. It is assumed that the combined effects of tension and compression of the membranes as well as the interaction between membrane and cell wall opposingly change the number of transport sites for K^+ providing a turgor-sensing mechanism that regulates ion fluxes. The IAA effects demonstrated are consistent with this view, suggesting that the basic mechanisms for turgor pressure regulation and growth regulation are similar.

Any relation connecting growth rate with turgor pressure should be governed by two parameters, *i.e.* by a yielding pressure, at which cell growth starts, and by the critical pressure, at which it ceases again.

Changes in salinity have a dual effect on plant tissues and on single plant cells, *i.e.*, salt stress influences both the water relations and the ionic balance of the plant. In general, water exchange between the plant and its environment is much more rapid than the exchange of ions. Two distinct phases can be distinguished in regard to osmotic adaptation of halophytic higher plants and of some algae. In the first rapid phase lowering of the external water potential will also lower the water potential and the cell turgor pressure in the tissue. This process will be controlled by the hydraulic conductivity, L_p, of the cells and by the elasticity of the cell walls expressed by the volumetric (bulk) elastic modulus, ϵ. The half-time of water exchange is given by (6, 17):

$$T_{1/2} = \frac{V \cdot \ln 2}{A \cdot L_p(\epsilon + \pi)} \quad (1)$$

where V and A are the cell volume and the cell surface area, respectively, and π is the osmotic pressure of the cell sap. As pointed out by Dainty (6) and discussed by us elsewhere (22) plants may have a useful mechanism which protects against short term osmotic stress, if their hydraulic conductivities and their ϵ values are low. In this case the half-times for water exchange might be in the order of up to a few hours (7). However, this is not the situation in osmoregulating plants subjected to high salinity for a longer period. These species have to perform a long term osmotic adjustment induced by salt uptake from the environment and by considerable changes in their ionic relations. The question is how these plants transform changes in the external salinity into changes in membrane permeability and in active ion uptake and how they maintain their turgor pressure over considerable salinity ranges.

It has often been postulated (9, 12, 13, 24, 25) that variations in ion transport in osmoregulating plants are induced by a turgor-sensing mechanism. Salt uptake or extrusion is regulated so that after a certain time the original cell turgor pressure is restored. Such a mechanism has been demonstrated by us in the marine alga *Valonia utricularis*. Simultaneous measurements of the hydrostatic pressure, electrical membrane resistance, and electropotential show that the membrane resistance as a measure of salt permeability has a marked maximum in its dependence on pressure (24, 25). Below this threshold pressure, required for the maximum resistance, a net uptake of KCl should result. Beyond this critical value excretion of KCl should take place. These results are consistent with the finding of Gutknecht (9) that pressure strongly inhibits influx and promotes efflux of K^+ in *Valonia*.

However, experimental results show that the threshold pressure varies widely for different cells from about 1 to 4 bars (24). This variability is not compatible with a simple turgor pressure transducing mechanism, which should sense only one distinct critical pressure for all cells.

In this paper we will demonstrate that the threshold pressure depends on the volume of the cell. Since the elastic properties of the cell wall are also strongly dependent on cell volume, we shall develop a hypothesis which incorporates both the hydrostatic pressure and the volumetric elastic modulus as important parameters for the osmoregulation in *Valonia*. Furthermore, we show that the threshold pressure of a given cell is affected by the application of growth hormones such as auxin. This suggests that regulation of the osmotic content of this species, as well as the regulation of growth, are additionally under hormonal control.

MATERIALS AND METHODS

Cells of *Valonia utricularis*, originally obtained from Naples, Italy, were grown in natural seawater at a salinity of 1300

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milliosmol (= 31.7 bar) under continuous illumination (25-w Osram-Fluora lamps) (25). For experiments elliptically shaped cells, which varied from about 2 to 9.5 mm in diameter and from 20 to 200 μl in cell volume, were used. The cells were fixed under a binocular microscope in a small Plexiglas chamber filled with seawater. The external medium was vigorously stirred by a pump and could be rapidly exchanged for artificial seawater.

Cell turgor pressure was measured directly by introducing a pressure probe (23) into the cell vacuole with the aid of a micromanipulator. The function of the probe has been described in previous papers (23, 25) and these papers should be consulted for details. The probe permitted a continuous measurement of cell turgor and could be used to change the cell turgor pressure, P, artificially, and to measure the corresponding changes in cell volume, V. Thus, with this device the volumetric elastic modulus, ϵ, of the cell wall could be determined; ϵ is defined by the following equation (17):

$$\epsilon = V \cdot \frac{\Delta P}{\Delta V} \qquad (2)$$

Such measurements have been performed previously on giant algal cells such as *Valonia* and *Characean* internodes (23, 26, 27) and on cells of a higher plant (22) to determine the hydraulic conductivity of these cells and the rate of water exchange between cell and environment. In this paper we will report ϵ values for *Valonia* measured over a wide range of cell volumes. The setup for the electrical measurements was essentially the same as used previously (24, 25). The membrane PD[2] and resistance were determined by conventional internal and external microelectrodes filled with 3 M KCl, which had resistances of 1 to 5 MΩ, (tip diameter <15 μm; tip potential <4 mv). To prevent any leakage from the cell via the internal current and voltage electrodes, they were tightly connected to Ag/AgCl electrodes in 3 M KCl/agar. The membrane PD was recorded by a microelectrode amplifier (model P16 from Grass Instruments). Current pulses of a duration of 10 to 20 sec were applied to the cell from a constant current source (Keithley 225) in order to determine the membrane resistance. The responses in potential were linear at the current densities used (up to $\pm$20 μamp/cm^2) according to Ohm's law. They did not depend on the polarity of current, *i.e.*, no rectification was observed in this range of current density.

Cell turgor pressures of the *Valonia* cells were altered by changing the osmotic pressure of the external seawater by adding stepwise aliquots of distilled H_2O or NaCl. In this way the membrane PD and resistance were measured as a function of pressure in the range from 0 to 10 bars. During this procedure the ion concentrations in the seawater (main contents in mM: Na^+, 560; K^+, 12; Ca^{2+}, 11; Mg^{2+}, 55; Cl^-, 665; SO_4^{2-}, 30) were reduced by an amount up to 30%, but this had no effect on the direction of PD changes and on the absolute value of the resistance measured (25).

The effect of auxin on the pressure dependence of the membrane PD and resistance was determined by adding IAA of $2 \cdot 10^{-8}$ to $2 \cdot 10^{-5}$ M to the outer solution. Auxin effects were recorded from about 5 min after application to a maximum of 4 hr.

RESULTS

As mentioned above the resistance of the cell membranes of *Valonia utricularis* reveals a biphasic response with increasing pressure. For a given cell the resistance increases up to a critical value (ρ_0^{max}) and then decreases again with increasing pressure. Simultaneously, in the range of the critical pressure (P_C) the membrane potential drops by an amount of 2 to 15 mv to more negative values. Larger changes, up to 40 mv in the potential drop have also been observed (25). The critical pressure and the corresponding changes of the electrical membrane parameters depend on the cell volume (Fig. 1). With decreasing cell volume P_C shifts to higher values. With pressure the corresponding changes of the electrical membrane parameters were reproducible for a given cell as described elsewhere (25). The relationship between critical pressure and cell volume becomes more evident where values of P_C are summarized for 41 cells (Fig. 2), the volume of which varied between 20 and 200 μl. The function $P_C = f(V)$ appears to be an inverse hyperbola reaching an asymptotic value for P_C at about 0.7 bar for large cells.

The absolute value of the membrane resistance of *Valonia* in the low pressure range (*i.e.*, at pressures below 1 bar) was of the order of 300 to 1000 $\Omega \cdot cm^2$ and did not vary with cell volume. Conversely, the absolute value of the maximum resistance,

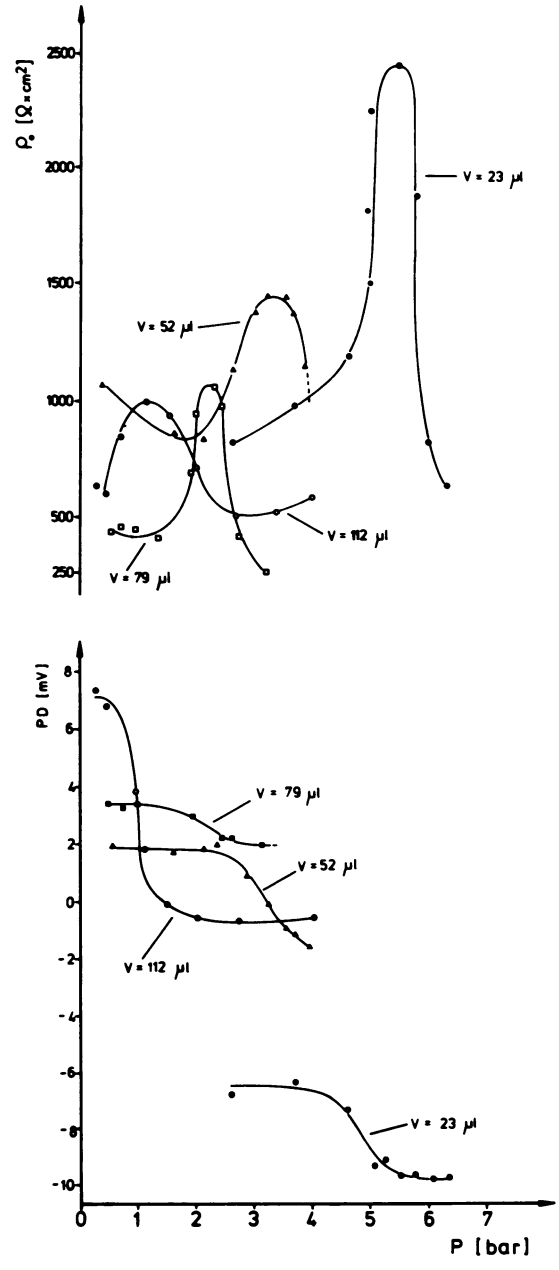

FIG. 1. Effect of cell turgor pressure, P, on the membrane resistance, ρ_0, and on the membrane PD of four different cells of *Valonia utricularis*. The critical pressure required for maximum resistance, ρ_0^{max}, and the absolute value of ρ_0^{max} increase with decreasing cell volume, V.

[2] Abbreviation: PD: potential difference.

ρ_0^{max}, increases with decreasing volume (Fig. 1). This increase of resistance at the critical pressure is also shown in Figure 3 for a larger number of cells by plotting the maximum membrane resistance as a function of cell volume. As with the relationship between critical pressure and cell volume, there is also a pronounced increase in membrane resistance with decreasing volumes. This finding indicates that the membrane resistance of the same species may vary dramatically with cell volume.

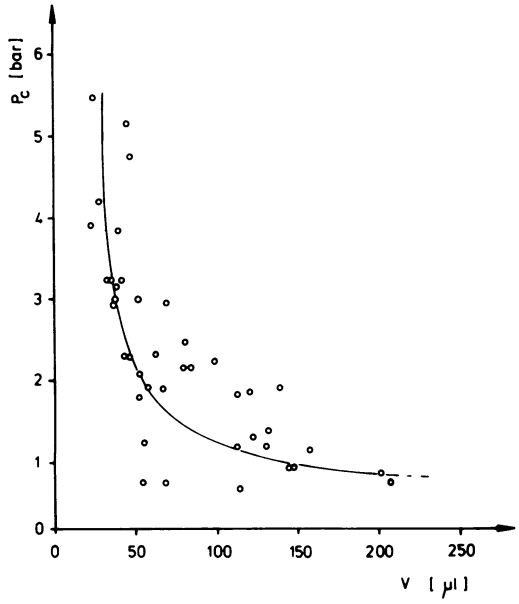

FIG. 2. Shift of critical pressure, P_C, in dependence on cell volume, V. Critical pressures were obtained from pressure-resistance curves such as in the upper part of Fig. 1. Cumulative data for 41 *Valonia* cells.

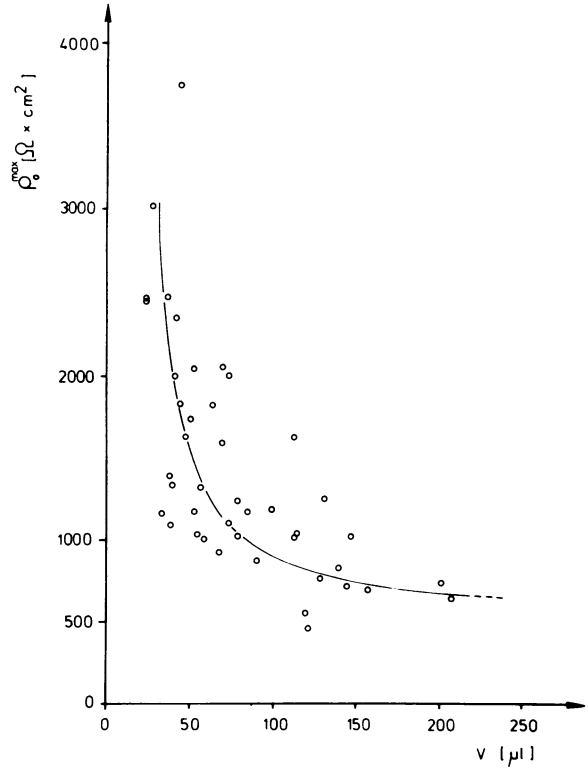

FIG. 3. Volume dependence of the maximum resistance, ρ_0^{max} (see Fig. 1) of the same cells as in Fig. 2.

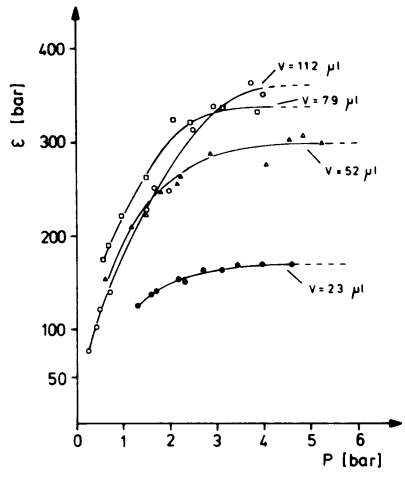

FIG. 4. Volumetric elastic modulus, ϵ, as a function of cell turgor pressure for different cell volumes (cells as in Fig. 1). At pressures >3 bar ϵ practically becomes independent of pressure, but not of cell volume.

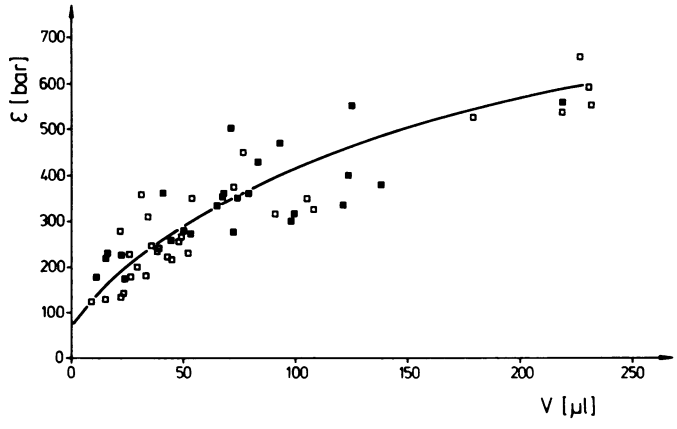

FIG. 5. Dependence of the volumetric elastic modulus ϵ on cell volume in high pressure range (P = 4 bars). Cumulative data for 53 cells. ■: ϵ values measured in seawater as well as subsequently in seawater $+2 \cdot 10^{-5}$ M IAA. Both values were identical.

The phenomena described in Figures 1 to 3 seem to be directly correlated with the elastic properties of the cell wall. This becomes evident from Figures 4 and 5. In Figure 4 the volumetric elastic modulus, ϵ, is plotted as a function of turgor pressure for the same cells as in Figure 1. As indicated by the figure ϵ depends on both pressure and volume. As discussed in previous papers [(26, 27) Steudle and Zimmermann, in preparation] the pressure dependence of ϵ is more pronounced in larger than in smaller cells and the relationship $\epsilon = f(P)$ results in hyperbola-like curves. Furthermore, the absolute value of ϵ increases with cell volume. This volume dependence is more pronounced at higher pressures, where ϵ becomes independent of pressure. Figure 5 demonstrates that the volume dependence of ϵ in this range of pressure (*i.e.* for P = 4 bar) is also hyperbolic. The volume dependence of ϵ seems to be a general phenomenon in plant cells and was also found for the internodal cells of several *Characean* species [(26, 27) Steudle and Zimmermann, in preparation] and for the epidermal bladder cells of *Mesembryanthemum crystallinum* (22). The effect has been interpreted in terms of two different cell wall regions with different elasticity. The analysis of the data obtained from elasticity measurements on *Nitella flexilis* and on *Nitellopsis obtusa* suggests that one of these regions has a very low ϵ value and exhibits a high wall extensibility. It is obvious that the finding of a volume-dependent overall

elastic modulus is important for both phenomena, *i.e.* osmotic and growth regulation. Osmoregulation might sense not only the turgor pressure but also the overall rate of cell extension; such a mechanism was shown previously in growth regulation at least for *Nitella* (8) and for *Avena* coleoptiles (see ref. 3). If it is true that similar mechanisms are involved in both processes, we should expect that osmoregulation can be influenced also by growth hormones. In this paper we report the influence of IAA on the osmoregulation of *Valonia,* which we tested by measuring the pressure dependence of membrane resistance and electropotential after application of IAA. IAA had a dramatic effect on both the critical pressure, P_C, and on the maximum resistance, ρ_0^{max} (Fig. 6, Table I). In a concentration range down to $2 \cdot 10^{-7}$ M IAA, P_C was increased by 3 to 6 bar, while ρ_0^{max} was increased by a factor of 1.2 to 5.6 (Table I). The earliest onset of the IAA effect could be measured about 5 min after application of IAA to the external medium in concentrations ranging from $2 \cdot 10^{-5}$ down to $2 \cdot 10^{-7}$ M IAA. Within that range no dependence on the IAA concentration occurred. At a concentration of $2 \cdot 10^{-8}$ M IAA no changes in ρ_0^{max} and P_C were observed.

During the experiments with auxin, lasting up to 4 hr, no influence of IAA on the cell wall elasticity was observed. The $\epsilon = f(P)$ curves remained unchanged for cells bathed in seawater and seawater + IAA, respectively (see also Fig. 5).

DISCUSSION

An interesting result of this paper is the finding that membrane resistance and, to a lesser extent, the membrane potential of a plant cell are not only dependent on pressure, but also on cell volume. Furthermore, our results seem to show that the volume dependence of the membrane electrical parameters reflects indirectly the elastic properties of the cell wall. The pressure and volume dependence of electrical membrane parameters may attract further interest in the determination of membrane resistance and potential in algal cells and cells of higher plants providing that the relationships found in *Valonia utricularis* are generally valid. The data quoted in the literature point to different transport properties of *Valonia utricularis* compared with those of other cells. However, with respect to pressure-controlled processes, which are considered in this paper, *Valonia* seems to behave like other plant cells. It is reasonable to assume similar pressure and volume effects in other cells. As indicated in Figures 1 and 3 the changes in membrane resistance with pressure can be remarkably high depending on the cell volume and on the pressure range.

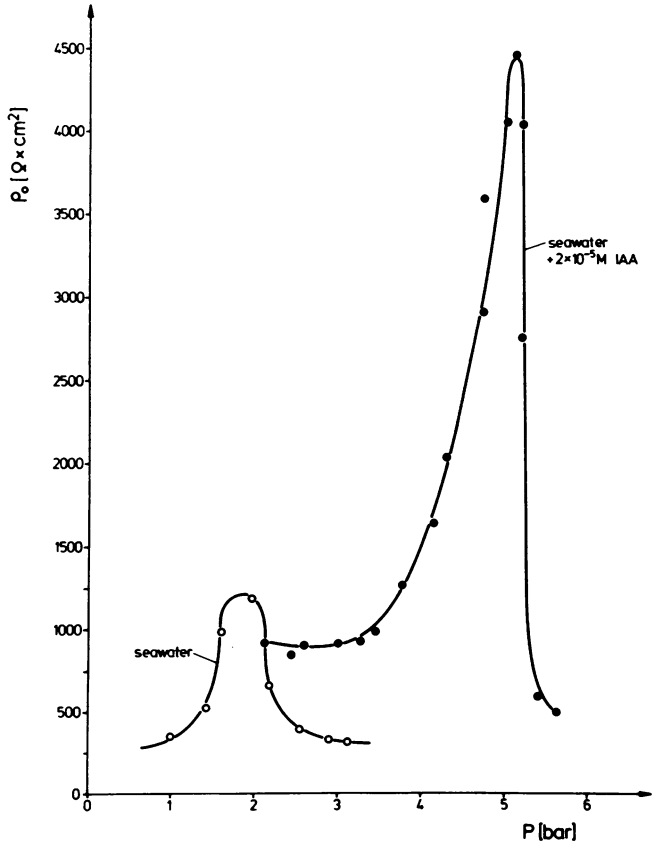

FIG. 6. Effect of IAA on the pressure-resistance curve of a *Valonia* cell (cell No. 9 in Table I). Addition of IAA to the seawater shifts the critical pressure and the maximum resistance to higher values (see also Table I).

Table I. *Effect of IAA on Critical Pressure, P_C, and on Maximum Resistance, ρ_0^{max}, for Different Cells of Valonia utricularis*

Pressure-resistance curves in the presence of IAA were measured beginning about 5 min after external application of IAA.

Cell No.	Cell Volume	SEAWATER P_C	SEAWATER ρ_0^{max}	SEAWATER + IAA IAA Conc.	SEAWATER + IAA P_C	SEAWATER + IAA ρ_0^{max}
	µl	bar	Ωcm²	mole	bar	Ωcm²
1	46.8	1.9	1675	$2 \cdot 10^{-8}$	2.2	1700
1	46.8	1.9	1675	$2 \cdot 10^{-7}$	3.1	2450
2	91.9	4.5	1070	$2 \cdot 10^{-7}$	7.1	1840
3	57.5	4.4	1340	$2 \cdot 10^{-6}$	8.3	4230
4	89.6	2.6	1240	$2 \cdot 10^{-6}$	6.4	2470
5	57.9	1.8	1170	$2 \cdot 10^{-5}$	8.8	2100
6	53.4	0.8	1030	$2 \cdot 10^{-5}$	6.7	5750
7	119.1	1.8	550	$2 \cdot 10^{-5}$	5.2	2940
8	128.2	1.5	760	$2 \cdot 10^{-5}$	5.7	2080
9	138.9	1.9	825	$2 \cdot 10^{-5}$	5.1	4450
10	146.5	0.9	1020	$2 \cdot 10^{-5}$	3.6	3700

It might be argued that the observed pressure effect is an artifact of the experimental procedure by which the turgor pressure was increased, because in this procedure not only the osmotic pressure of the medium, but also the ionic concentrations in the seawater were decreased by an amount up to 30%. This argument can be rejected by the following experimental observations. First, in the concentration range between 5 and 12 mM K^+ in the external medium the resistance and potential were found to be independent of the external K^+ concentration. Secondly, increasing the turgor pressure by the addition of 12 mM KCl to the seawater led to the same relationship between pressure and the electrical membrane parameters as reported previously (25). It is true that under these conditions the Na^+ and Cl^- concentration also changed. The effect of the external concentrations of both ions on the membrane conductance and potential seems to be low under these conditions since the replacement of NaCl of the seawater in this range of concentration by an osmotically equivalent concentration of a nonelectrolyte (*e.g.* glucose) resulted in the same membrane resistance and potential (25). On the other hand, lowering the chloride concentration in the seawater by 30% at constant Na^+ concentration shifted the membrane PD by about 3 mv to more positive values, *i.e.* the potential changed in the opposite direction to that found during the increase of pressure (unpublished results). These results point to a pressure-regulated process rather than to concentration effects. Considering the data of Gutknecht (9) on pressure-dependent K^+ fluxes in the pressure range between 0 and 1 bar it is reasonable to assume that the pressure depend-

ence of the membrane resistance reflects the pressure dependence of K^+ fluxes in *Valonia*. The pressure has an opposite effect on K^+ influx and efflux (9). Consider now the case that at low pressures the influx decreases more rapidly with pressure than the increase in efflux, with increasing pressure. The membrane resistance will then increase with pressure. If at high pressures the efflux rises more rapidly than the influx falls with increasing pressure, the resistance will then decrease again with increasing pressure. The different effects of pressure on K^+ influx and efflux provide the *Valonia* cell with an efficient osmoregulating mechanism in response to long term osmotic stresses. This model, proposed by Gutknecht (9) and extended by us (24, 25), implies that turgor pressure only is the regulating factor in a simple negative feedback mechanism (5, 13), but it fails to explain the volume dependence of the critical pressure and the maximum resistance. The volume dependence of the critical pressure requires an additional parameter in the regulation of K^+ fluxes. On the basis of the demonstrated volume dependence of the volumetric elastic modulus of the cell wall it seems reasonable to assume that the elastic properties of the cell wall are also involved in the regulation mechanism. The data presented here show that with small cells, which exhibit a small volumetric elastic modulus, the inhibition of K^+ uptake and the acceleration of potassium extrusion occur at higher pressures compared with large cell volumes. From a physiological point of view this is plausible, because it provides small and still growing cells with a useful mechanism for establishing the higher cell turgor pressures which are required to initiate extension growth (3, 8).

The question arises as to how a pressure signal and changes in the elastic properties of the cell wall can be transformed into changes of ion fluxes. A cell with a low ϵ will increase more in volume and in surface area than a cell with a high ϵ. Therefore, a larger strain will be applied to the cell membranes of small cells, if we can assume that the incorporation of new material into the stretched membrane is a very slow process. The stretching of the membrane could result in changes of the interactions of the membrane proteins and lipids and therefore lead to a change in the active and passive sites of the K^+ transport system. On the other hand with increasing turgor pressure the plasmalemma components will be strongly pressed against the negatively charged cell wall and the resulting change in the surface charge pattern of the membrane may also induce structural changes of the membrane (12). It seems also likely that the first step in the transformation of the pressure signal into the changes of the electrical parameters is based on the compression of the cell membrane. From dielectric breakdown measurements Coster and Zimmermann (4) concluded that the cell membrane may have compressible properties depending on the transverse elastic modulus of the membrane. These authors estimated a value of 50 bars for the transverse elastic modulus from the breakdown voltage interpreting the breakdown phenomenon in terms of an electromechanical model (Coster and Zimmermann, in preparation). Using this value it can be easily calculated that a change of the turgor pressure of 1 bar could change the membrane thickness by 0.17 nm assuming that the thickness of the unstressed membrane is about 9 nm. A model of osmoregulation based on compression of the cell membrane will be presented elsewhere (Coster, Steudle, and Zimmermann, in preparation). It will be shown there that changes in membrane thickness could regulate the transport properties. In this context, a mechanochemical model of enzyme mobilization in biomembranes which had been recently proposed by Berezin *et al.* (2) should be considered. Studying the activation of chymotrypsinogen in polyacrylamide gel these authors found a pronounced, reversible increase in the reaction rate at unidirectional compression of the gel. They interpreted their results as being due to an increased protein diffusibility resulting from structural changes of the gel. Obviously, such effects may play a role in our system and may result in changes of the permeability coefficients not only for K^+, but also for other ions such as Cl^- and Na^+. Furthermore, active K^+ uptake might be inhibited by pressure or the active K^+ pump might reverse its direction, as it was postulated for *Valonia* (24, 25).

Therefore, with the combined effect of tension, compression, and interaction between membrane and cell wall induced by pressure we can put forward the following hypothesis for the explanation of the shift in the critical pressure and the increase in membrane resistance with cell volume. If it is true that the maximum resistance is caused by the pressure-controlled K^+ fluxes, extension of the cell membranes might increase the transport sites for K^+ uptake at the plasmalemma; on the other hand wall-membrane interactions and/or the compression of the membrane might compensate to some extent the increase in uptake sites. Since the relative extension of the cell/unit pressure is given by $1/\epsilon$ (equation 2) the extension of a small cell exhibiting a nearly pressure independent and low ϵ should be greater than for a larger cell for which the extension decreases with increasing pressure. We can expect that in small cells tranport sites can be created over a wider pressure range by extension than in larger ones. The compensation (or overcompensation) of this increase in transport sites by the effect of the two other mechanisms may result in a maximum of the membrane resistance at higher pressures for smaller cells. This means that a low ϵ inhibits (or delays) the decrease of K^+ influx with pressure.

This view is consistent with the IAA effect on turgor pressure regulation in *Valonia,* which shifts the critical pressure to higher values and increases the absolute height of the maximum resistance (Fig. 6 and Table I). There is considerable evidence that the rapid effects of auxin on ion transport are related to a stimulation of transport sites (or carrier systems) in the cellular membranes [see review by Ray (18)]. IAA is known to enhance the uptake of cations such as K^+ and Rb^+ (14–16) and to influence also anion fluxes (1, 21). Interestingly, there are some hints that the action of IAA on ion uptake is dependent on the age and, therefore, probably on the wall extensibility of plant cells. Lüttge *et al.* (16) found a different action of IAA on the K^+ (Rb^+) uptake in young and old *Mnium* leave cells, whereas the uptake of Cl^- and SO_4^{2-} remained unchanged. The electropotential of the cells was not changed by IAA, but the uptake of K^+ (Rb^+) was strongly reduced by a slight increase of external osmolarity. This finding might point to an effect of cell turgor pressure on the K^+ fluxes of these cells similar to that reported here.

Furthermore, the action of IAA on elongation growth is coupled with an K^+-H^+ exchange mechanism, which results in a cell wall acidification and a loosening of the cell wall (10, 18, 20). The internal pH is proposed to be regulated by a mechanism based on the synthesis of organic acids (11). Such an exchange mechanism may also take place in *Valonia,* but does not (up to 4 hr after application) lead to a wall loosening effect, *i.e.* to a reduction of the volumetric elastic modulus, which represents the "instantaneous" elastic extension (3) and, therefore, includes the elastic and, to a lesser extent, also plastic properties of the cell wall (22). It seems very likely that wall loosening is not involved in the turgor pressure regulation and in the short term growth responses of this alga. If we assume a linear relationship between growth rate, r, and turgor pressure, P, as it was found for *Nitella* (8, 19) and for *Avena* coleoptiles (see ref. 3), *i.e.* if

$$r = m(P - Y) \quad (3)$$

where m is the wall extensibility or yielding compliance and Y a yield threshold, then the extensibility of the wall should be a function only of pressure and cell volume (Figs. 4 and 5). The wall extensibility should not be controlled by auxin, whereas the yielding threshold may vary with cell size (*i.e.* with ϵ), pressure range, and auxin content (see ref. 3). This means that the

relationship between growth rate and turgor pressure as expressed by equation 3 should probably be extended to a relation containing two threshold parameters, *i.e.*, the yielding pressure, Y, and the critical pressure, P_C. Growth rate should increase with pressure in a hypotonic solution at pressures larger than Y and should cease again at pressures larger than P_C due to an extrusion of solutes. If this model is correct, the ability of osmoregulating plants such as *Valonia* to perform pressure-induced growth should depend on the difference between P_C and Y. A large pressure range for growth induction would be found for small, still growing plants and for cells stimulated by IAA. In large, old cells having a high ϵ the difference should be low or practically zero, so that high turgor pressures required for growth cannot be maintained for a longer time.

Although this hypothesis appears to be coherent it should be pointed out that we cannot completely exclude the possibility that the effects described in this paper, particularly the maximum in resistance, may reflect a superposing effect produced by the two membranes, tonoplast and plasmalemma. In any case, the similarity in the response of the electrical membrane parameters to changes in the elastic properties, and to auxin, suggest that the basic mechanisms of turgor and growth regulation are similar.

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