Potassium (K^+) gradients serve as a mobile energy source in plant vascular tissues

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The essential mineral nutrient potassium (K^+) is the most important inorganic cation for plants and is recognized as a limiting factor for crop yield and quality. Nonetheless, it is only partially understood how K^+ contributes to plant productivity. K^+ is used as a major active solute to maintain turgor and to drive irreversible and reversible changes in cell volume. K^+ also plays an important role in numerous metabolic processes, for example, by serving as an essential cofactor of enzymes. Here, we provide evidence for an additional, previously unrecognized role of K^+ in plant growth. By combining diverse experimental approaches with computational cell simulation, we show that K^+ circulating in the phloem serves as a decentralized energy storage that can be used to overcome local energy limitations. Posttranslational modification of the phloem-expressed *Arabidopsis* K^+ channel AKT2 taps this "potassium battery," which then efficiently assists the plasma membrane H^+-ATPase in energizing the transmembrane phloem (re) loading processes.

channel gating | energy limiting condition | phloem reloading | posttranslational regulation | potassium channel

The genome of the model plant *Arabidopsis thaliana* contains nine genes that encode subunits of voltage-gated potassium channels. Four of these subunits must assemble to form homomeric or heteromeric channels that mediate either K^+ uptake (by so-called inward-rectifying K^+ channels, K_{in}) or K^+ release (by outward-rectifying K^+ channels, K_{out}). Due to their diverse functionalities, voltage-gated K^+ channels play important roles in the uptake of K^+ from the soil and in its distribution within the plant (1). A yet-unsolved role in this context is played by the K^+ channel subunit AKT2. Although intrinsically a K_{in} channel, it was shown in heterologous expression systems that AKT2 can be converted by phosphorylation into a nonrectifying channel mediating both K^+ uptake and release (2–4). Interestingly, AKT2-like channels are found only in higher plants (5–13) (Fig. S1). AKT2 is expressed in guard cells, phloem tissues, and root stellar tissues (9, 10, 14–16), and AKT2 loss-of-function plants were shown to exhibit a reduced reuptake of photoassimilates leaking from the phloem (15). Potassium is the major cation in the phloem and stimulates sugar loading into the phloem sap. It has been speculated that AKT2-like channels participate in this process by regulating sucrose/H^+ symporters via the membrane potential of phloem cells (17) (http://atted.jp/data/locus/At4g22200.shtml; http://arabidopsis.org/servlets/TairObject?id=129087&type=locus). However, the contribution of the unique phosphorylation-dependent features of AKT2 to K^+-stimulated sugar loading is unknown. Here, we provide evidence that posttranslational modification of AKT2 switches on a "potassium battery" that efficiently assists the plasma membrane H^+-ATPase in energizing transmembrane transport processes. K^+ ions, which are taken up in source tissues into the phloem by energy (ATP) consumption and then circulate in the phloem, serve as decentralized energy storage. This energy source can be exploited by AKT2 regulation to overcome local energy limitations.

Results

Development of *akt2-1* Plants Is Affected Under Short-Day Conditions. To obtain a better understanding of AKT2 function, we tested the effect of loss of AKT2 function in plants [*akt2-1* knockout plants (18)] (Fig. S2) grown at different day lengths. Upon cultivation in normal soil in a 16-h day/8-h night cycle (in the greenhouse or in growth chambers), no phenotypic differences were detectable between the *akt2-1* mutant and the wild type at any developmental stage (Fig. 1*A*). However, when the day length was reduced to 12 or 8 h, the plants displayed pronounced phenotypic differences. Compared with the wild type, the *akt2-1* line developed fewer leaves and showed a delay in bolting (Fig. 1 *B* and *C*). The wild-type phenotype was restored when the *akt2-1* line was complemented with the wild-type AKT2 allele, indicating that the phenotypic effects are indeed due to a disruption of the *AKT2* locus (Fig. S2; Fig. S3, *A*–*C*).

AKT2 Plays an Important Role in Phloem Tissues. The *akt2-1* line was also rescued when AKT2 was expressed under the control of the phloem companion cell-specific *AtSUC2* promoter (19) (Fig. S3, *D* and *E*). Thus, the observed phenotype of the *akt2-1* plants can be attributed to a loss of AKT2 function in phloem tissues rather than, for example, in guard cells.

Posttranslational Regulation of AKT2 Is Essential for Its Proper Function in the Plant. We next wanted to test whether the unique phosphorylation-dependent characteristics of AKT2 are important for its proposed physiological role. To do this, we complemented the *akt2-1* knockout plant with mutant versions of the AKT2 channel protein that had been previously identified as being affected in posttranslational modifications (3, 4) (Fig. S4) (20): Whereas the mutant AKT2-S210N-S329N can be more easily converted into a nonrectifying channel than the wild type,

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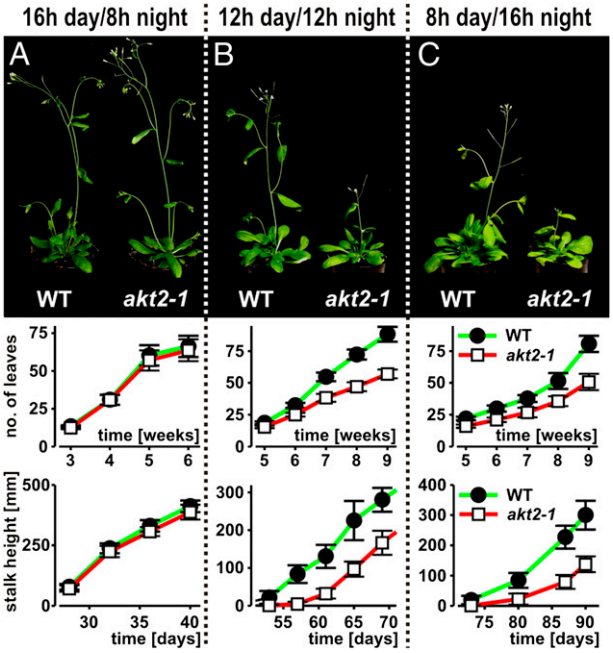

Fig. 1. Day-length–dependent phenotype of *akt2-1* plants. Phenotypical analysis of wild-type and *akt2-1* knockout plants grown under three different photoperiod regimes: (*A*) 16 h day/8 h night, (*B*) 12 h day/12 h night, and (*C*) 8 h day/16 h night (150 $\mu E \cdot m^{-2} \cdot s^{-1}$ in all three conditions). Photos show 5-wk-old (*A*), 6-wk-old (*B*), and 6.5-wk-old plants (*C*). At these time points, the Wassilewskija wild-type plants showed similar developmental stages. Time courses of number of leaves (*Middle panels*) and height of the main inflorescence stalk (*Bottom panels*) are shown as means ± SD of $n \geq 25$ plants.

the mutants AKT2-S210A-S329A and AKT2-K197S cannot be converted into a nonrectifying channel, so they function as inward-rectifying channels only.

The different mutant alleles of the channel had distinct effects on the physiology of the plants. Plants expressing inward-rectifying mutant AKT2 channels (AKT2-S210A-S329A or AKT2-K197S) behaved very similarly to the *akt2-1* knockout. They produced fewer leaves and showed a delayed development under short-day conditions. In contrast, plants expressing the mutant AKT2-S210N-S329N were, like wild-type plants, not negatively affected by the applied stress (Fig. 2).

Mathematical Simulations Reveal That K^+ Serves as an Energy Carrier for Phloem (Re)loading. Our wet-lab experimental data indicate that the importance of AKT2 in phloem tissues is tightly connected to its ability to be converted into a nonrectifying channel. To investigate further the role of this posttranslational modification in phloem transport, we carried out dry-lab experiments. Previous studies have proposed a very detailed model for a sieve element/companion cell (SE/CC) complex that expresses a transporter network of H^+-ATPase, sucrose/H^+ carriers, and AKT2-like K^+ channels (5, 21). We took this model as the basis and also included the leakage of sucrose from the phloem (e.g., by pH-independent facilitators) (22), as well as the sequestration of K^+ from the apoplast (Fig. 3*A*) to better reflect the in vivo situation. The features of all transporters were described mathematically (Fig. S5) (21, 23–25) and computational simulations were carried out using Virtual Cell software.

First inspection of the thermodynamical flexibility of the ohmic network revealed that it can act in three different states with respect to values of the membrane voltage V_m. In only one of them ($E_K < V_m < E_{H/Suc}$) did regulation of AKT2 have a clear physiological impact (*SI Text*). To achieve such an intermediate membrane voltage, the activity of the H^+-ATPase needs to be reduced compared with that of the other transporters. We therefore started our detailed analyses with an intermediate H^+-ATPase activity and allowed the system to equilibrate when all AKT2 channels were inward-rectifying (Fig. 3 *B–F*, i). Under this condition, the apoplastic sucrose concentration remained stable (Fig. 3*B*, i), and there was no net transport of sucrose across the membrane (Fig. 3*C*, i). This indicates that all sucrose molecules leaking from the phloem were retaken up by the sucrose/H^+ carrier, fueled by the H^+-ATPase (Fig. 3*E*, i). Next, AKT2 was switched from an inward-rectifying into a nonrectifying channel (Fig. 3 *B–F*, arrows). This change resulted in a rapid short-term net uptake of sucrose (Fig. 3*C*, ii), causing a decrease of the apoplastic sucrose concentration (Fig. 3*B*, ii). The energy driving this additional sucrose uptake was not provided by the H^+-ATPase; the switch of AKT2 caused a small hyperpolarization (Fig. 3*D*, ii) that, in turn, actually reduced the H^+-ATPase activity (Fig. 3*E*, ii). Instead, the energy for the additional sucrose uptake was provided by the electrochemical K^+ gradient (Fig. 3*F*, ii). The newly opened K^+ channels allowed a rapid flux of K^+ along the outwardly directed electrochemical gradient. This charge transport was largely compensated by a rapidly increased H^+/sucrose influx (Fig. 3*C*, ii). The fraction that is not compensated affects the membrane voltage, causing the slight hyperpolarization (Fig. 3*D*, ii). Thus, our mathematical simulations reveal that K^+ serves as an energy carrier for phloem (re)loading processes and that posttranslational regulation of AKT2 taps this potassium battery.

Development of *akt2-1* Plants Is Affected Under Energy-Limiting Conditions. Our dry-lab experimental data indicate that regulation of AKT2 has a clear physiological impact on phloem reloading when the activity of the H^+-ATPase is reduced, especially when the local energy provided by the H^+-ATPase is no longer sufficient for K^+ loading. Consequently, the observed plant phenotype (*akt2-1* plants are affected under short-day but not under long-day conditions) may be interpreted as resulting from the different energy status of the plants in the two tested conditions. Indeed, the light dependence of the *akt2-1* phenotype was not due to altered photosynthetic performance, as shown by measurements of electron transport rates based on chlorophyll fluorescence (Fig. S6). Interestingly, phenotypic differences between the *akt2-1* mutant and the wild type could also be induced when, instead of day length, light intensity was reduced. Whereas under normal light conditions (100 $\mu E \cdot m^{-2} \cdot s^{-1}$ for 16 h), knockout and wild-type plants were indistinguishable, a reduction of light intensity to 15 $\mu E \cdot m^{-2} \cdot s^{-1}$ (low light) was less well tolerated by the *akt2-1* mutant. The knockout plants developed fewer leaves and siliques, and the bolting time was not affected (Fig. S7).

To challenge the "energy status hypothesis" independently from light conditions, we tested plant growth at different atmospheric oxygen concentrations. With decreasing oxygen availability, the respiratory energy (ATP) production declines. This has severe effects on highly metabolically active tissues such as the phloem (26). Wild-type and *akt2-1* plants did not differ under normal atmospheric conditions (21% O_2, 16 h day, 100 $\mu E \cdot m^{-2} \cdot s^{-1}$). However, when the oxygen concentration was reduced to 10%, the knockout plants developed a similar phenotype to that observed under short-day conditions: they had fewer leaves, exhibited a delayed bolting time, and produced less biomass (fresh weight and dry weight) (Fig. 4). Here, also, the importance of AKT2 could be correlated to its convertibility into a nonrectifying channel. Similar to the *akt2-1* knockout plants, *akt2-1* plants expressing inward-rectifying mutant AKT2 channels (AKT2-S210A-S329A or AKT2-K197S) were affected by the O_2 reduction, whereas plants expressing the mutant AKT2-

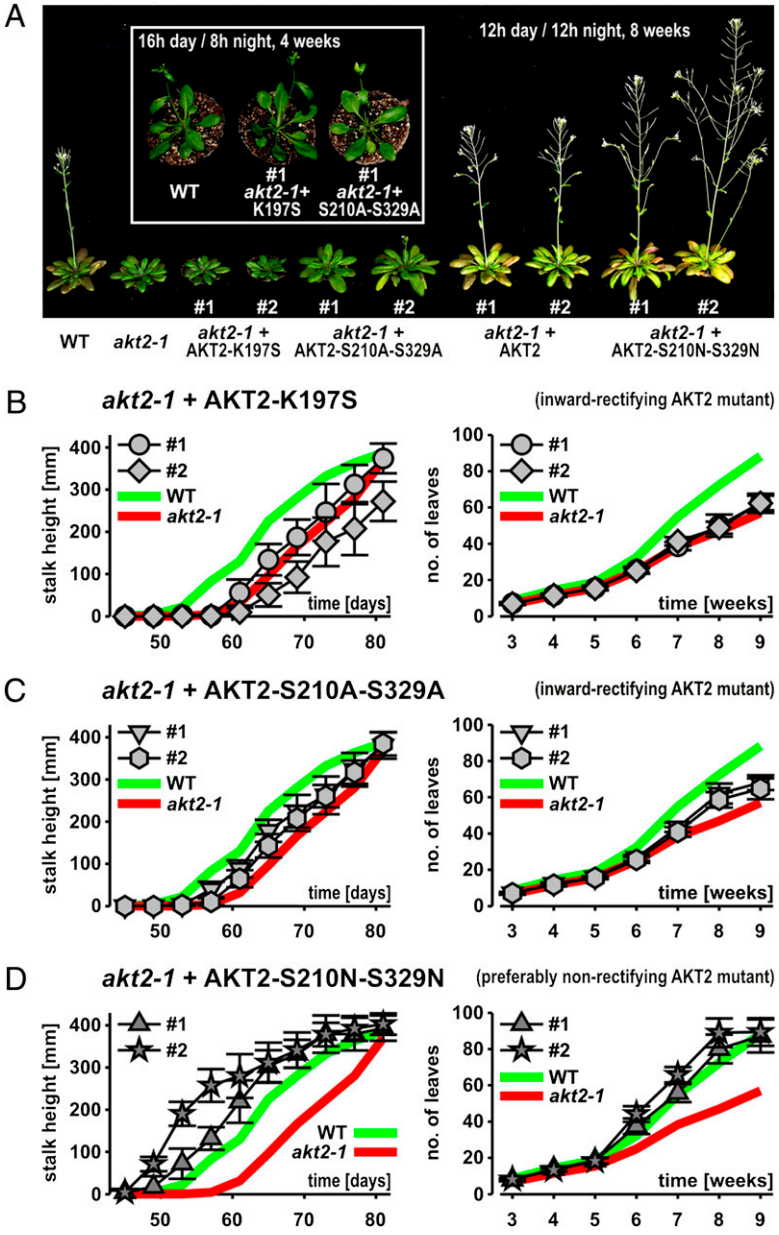

Fig. 2. Inward-rectifying AKT2 channel mutants do not complement the *akt2-1* knockout plant. Ten *Arabidopsis* lines were analyzed: WT, *akt2-1*, and eight *akt2-1* supertransformants expressing the AKT2 wild-type channel or channel mutants under control of the native *AKT2* promoter. Two independent lines of *akt2-1*+AKT2-K197S (inward-rectifying AKT2 mutant), two independent lines of *akt2-1*+AKT2-S210A-S329A (inward-rectifying AKT2 mutant), two independent lines of *akt2-1*+AKT2, and two independent lines of *akt2-1*+AKT2-S210N+S329N (preferentially nonrectifying AKT2 mutant). (*A*) Representative 8-wk-old plants grown under 12-h day/12-h night conditions. Note that some plants already show signs of senescence (WT, *akt2-1*+AKT2, and *akt2-1*+AKT2-S210N+S329N) whereas others have not yet started bolting. (*Inset*) Four-week-old plants of the representative lines *akt2-1*+K197S#1 and *akt2-1*-S210A-S329A#1 and the Wassilewskija wild type grown in long-day conditions (16 h day/8 h night). Under these conditions, all plants started flowering at the same time. (*B–D*) Time courses of the height of the main inflorescent stalk (*Left panels*) and the number of leaves (*Right panels*) of the plants *akt2-1*+AKT2-K197S (*B*), *akt2-1*+AKT2-S210A-S329A (*C*), and *akt2-1*+AKT2-S210N-S329N (*D*) compared with the wild type (green curves, no symbols) and the *akt2-1* knockout (red curves, no symbols). Detailed analyses of complementation lines (*akt2-1*+AKT2 and *akt2-1*+pSUC2:AKT2) are presented in Fig. S3. Data are shown as means ± SD of $n \geq 20$ plants.

S210N-S329N were not significantly affected (Fig. 4). Thus, even without knowing the underlying signaling cascade, this experiment shows that Arabidopsis reacts on low O_2 concentrations by posttranslational control of the AKT2 channel.

K^+ Gradients Combine the Energy Supply of Different Cells. Encouraged by the congruence between dry-lab predictions and wet-lab observations, we simulated another scenario that allowed us to also expose long-term effects of AKT2 channel regulation on phloem loading (Fig. 5; Fig. S8). As in the first scenario, we started again with an equilibrated system with only inward-rectifying AKT2 channels (Fig. 5, i). Then the activity of the H^+-ATPase was slightly reduced (by as little as 10%) to simulate, for example, varying ATP levels in the phloem or varying H^+-ATPase regulation (Fig. 5, arrow 1), and the system was again allowed to relax (Fig. 5, ii). The reduction in H^+-ATPase activity resulted in an increase of the apoplastic sucrose concentration. This indicates a net sucrose leakage from the phloem that lasted until a new equilibrium between efflux and influx at a higher apoplastic sucrose concentration had been reached. Following this, AKT2 was switched from an inward-rectifying into a non-rectifying channel (Fig. 5, arrow 2). This change resulted in a rapid short-term net uptake of sucrose (Fig. 5, iii), which, interestingly, was followed by a long-term stimulatory effect on sucrose uptake. This became more apparent when rerunning the simulation with a different sequestration rate of K^+ from the apoplast (red and blue curves in Fig. 5). This rate had no influence on phloem loading when the AKT2 channels were inward-rectifying (Fig. 5, i and ii). However, when the channels were posttranslationally modified into nonrectifiers, the K^+-sequestration rate had a determining influence on the apoplastic sucrose concentration. A higher K^+-sequestration rate resulted in an increased long-term loading of sucrose into the phloem (Fig. 5, iv). Also here, switching the operation mode of AKT2 channels taps a potassium battery, thus providing a rapidly available, albeit rapidly exhausted, energy source for phloem loading. Thereafter, surrounding cells can stimulate the (re)loading of sucrose into the phloem by sequestering K^+ from the apoplast, thus restoring a chemical K^+ gradient

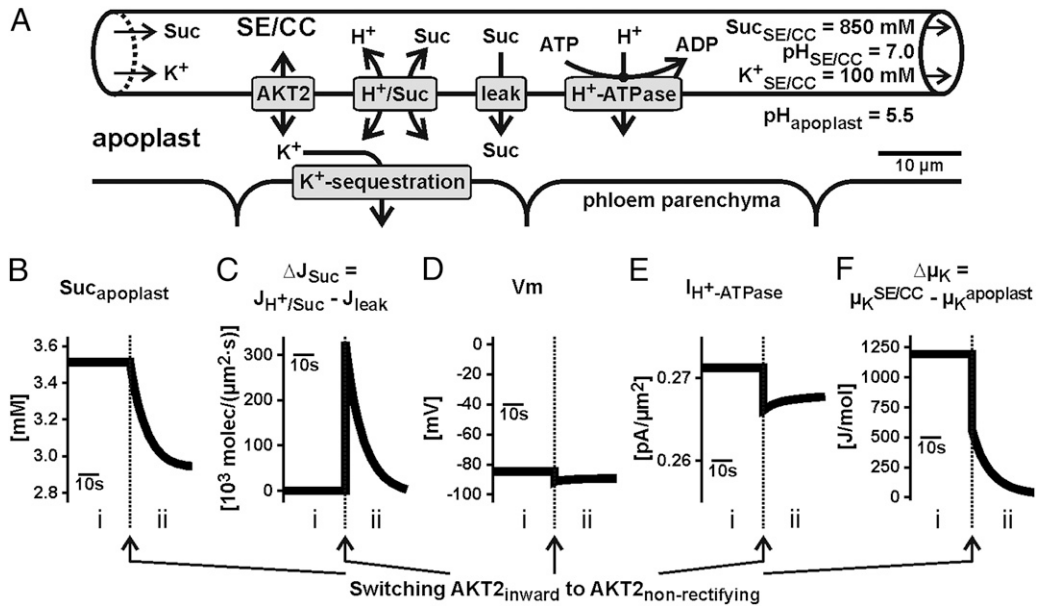

Fig. 3. Computational simulation of phloem (re)loading processes. (*A*) A SE/CC complex was modeled as a cylinder with a surface-to-volume ratio of 0.4 μm^{-1} and placed in a three times larger environment (apoplast). Note that different cell/environment values do not qualitatively change the obtained results. The continuous flux of the phloem sap was approximated by keeping $pH_{SE/CC}$, $Suc_{SE/CC}$, and $K^+_{SE/CC}$ constant. Likewise, $pH_{apoplast}$ was kept constant to reflect the buffer capacity of the apoplast. Transport of K^+, sucrose, and H^+ into or out of the phloem was mediated by the H^+-ATPase, the AKT2 K^+-channel, the H^+/Suc cotransporter, and a sucrose leak. Additionally, K^+ was removed from the apoplast by adjacent cells. For further details, see Fig. S5. (*B–F*) Simulation of the network behavior. First, AKT2 was set as an inward-rectifying channel (i). Next, AKT2 was switched from an inward-rectifying channel into a nonrectifying channel (dotted lines, arrows). Time courses of the apoplastic sucrose concentration ($Suc_{apoplast}$: *B*), the transmembrane sucrose flux (ΔJ_{Suc}: *C*), the membrane voltage (V_m: *D*), the current pumped by the H^+-ATPase ($I_{H+\text{-}ATPase}$: *E*), and the electrochemical K^+ gradient ($\Delta\mu_K$: *F*) are shown.

between the phloem and apoplast (Fig. S8). In this way, the energy needed for the loading process is supplied in a decentralized manner by the K^+ ions pumped from source tissues into the phloem sap and flowing with it and by the surrounding cells that invest energy (ATP) to take up K^+ from the apoplast for their own use.

Root Growth of *akt2-1* Knockout Plants Is Affected When Sufficient K^+ Is Available. In addition to tapping a potassium battery, the simulated scenario also points toward a role for AKT2 in K^+ supply of growing tissues (*SI Text*, ii). We therefore tested the dependence of *akt2-1* and wild-type plants on K^+ availability in early development. Indeed, compared with the wild type, *akt2-1* mutants showed decreased root growth, lower total fresh weight, and a decreased root-to-shoot biomass ratio when sufficient K^+ was in the medium (2,500 µM) (Fig. 6*A*). Surprisingly, in contrast to observations on knockout plants of inward-rectifying K^+ channels in roots (27–30), this phenotypical difference between wild type and *akt2-1* knockout disappeared when we lowered the external K^+ concentration. Under K^+-deficient conditions (10 µM; 100 µM), root growth, fresh weight, and root-to-shoot ratio were almost identical in mutant and wild-type plants (Fig. 6 *B* and *C*). Thus, although both wild type and *akt2-1* performed better in high K^+ than in low K^+, the knockout plant was not able to use the additional resources as efficiently as the wild type. Controlling the potassium battery and K^+ supply therefore represents two sides of the physiological role of AKT2.

Discussion

The results presented in this study suggest that mobile K^+ gradients are used by plants as an energy source suitable for temporarily bridging locally occurring energy limitations (Movie S1). Here "energy-limitation" should be considered as a reduced *relative* activity of the H^+-ATPase with respect to that of the other transporters. Such a reduction could be induced by lower ATP levels (as proposed in Fig. 5 and Fig. S8) or by a downregulation of the H^+-ATPase, but also by an increase of the apoplastic sucrose concentration due to other processes. In all these conditions, AKT2 would have optimal conditions to act as shown in Figs. 3 and 5. Therefore, the concept of mobile K^+ gradients as an energy source may apply to more physiological conditions than those presented here exemplified. K^+ is loaded into the phloem in source tissues where sufficient chemical energy is available. The established transmembrane K^+ gradient is then transported with the phloem stream to other parts of the plant. The energy stored in this K^+ gradient can be harvested to fuel transport processes by opening a gateway (i.e., the AKT2-like channels) for the passage of K^+ ions through the membrane. When cells of the surrounding tissues take up the effluent K^+, this potassium battery can be kept active over longer time periods. Additionally, these tissues are supplied with the important nutrient K^+. In its extreme, this network would allow phloem (re)loading even without any energy contribution by the SE/CC complex. The energy could be exclusively provided from remote sources by the K^+-loading process in source tissues and the K^+-sequestering activity of the surrounding tissues. In conclusion, our study proposes that plants establish local energy security by a sophisticated mix of energy sources.

Materials and Methods

General Methods and Construct Generation. Molecular biology methods were performed according to standard procedures. Further details are provided in the legend of Fig. S2 (31–33).

Plant Cultivation and Phenotypic Analyses. Seeds were cold-treated for 24 h at 4 °C. Plants were grown on a standardized soil substrate (type GS-90, Einheitserde). After 2 wk, seedlings were transferred to individual pots. For all experiments, plants were grown in 60% relative humidity at 21 °C during the day and 18 °C during the night. For analyses under different light regimes, plants were grown in three different diurnal cycles with day lengths of 8, 12, or 16 h. The response to different light intensities was observed at 100 $\mu E \cdot m^{-2} \cdot s^{-1}$ and 15 $\mu E \cdot m^{-2} \cdot s^{-1}$ (16 h day/8 h night). The illumination system was composed of Philips TLD58W840 or Philips HPI-T+ 400W/645 and Philips Master Son-T PIA Agro 400-W bulbs. All phenotypical analyses were carried out in the middle of the day. For experiments under different oxygen concentrations, 2-wk-old plants were transferred into plastic containers, sealed, and supplied with a gas mixture containing 21% and 10% oxygen, respectively. The experiment was performed under standard light conditions (16 h day/8 h night). For dry weight measurements, shoots were dried for 24 h at 60 °C. Root-to-shoot ratios were calculated on the basis of fresh weight measurements.

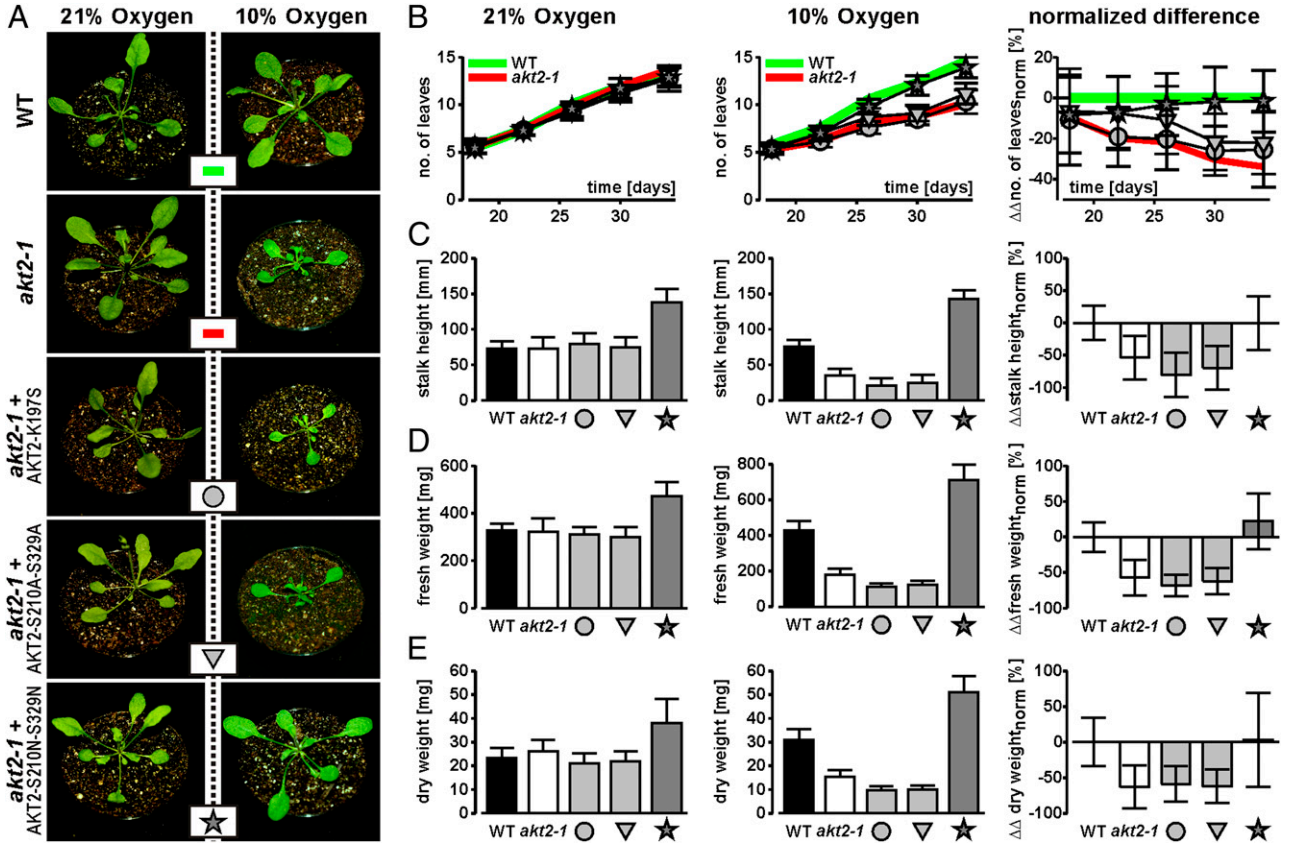

Fig. 4. Limited oxygen supply is less tolerated by plants expressing no or only inward-rectifying AKT2 channels. Two-week-old plants were transferred from the greenhouse into plastic boxes supplemented with atmospheric gas mixtures containing 21% and 10% oxygen, respectively (16-h day/8-h night). (*A*) Representative 32-d-old plants grown under normal (21%; *Left*) and reduced (10%; *Right*) atmospheric oxygen. (*B*) Time courses of number of leaves in 21% (*Left*) and 10% (*Center*) O_2 atmospheres. Symbols between panels are cross-referenced with *A*. (*C–E*) Height of the main inflorescent stalk (*C*), fresh weight (*D*), and dry weight (*E*) of 42-d-old plants of wild type (black bars), *akt2-1* knockout (open bars), *akt2-1*+AKT2-K197S#1 (circles), *akt2-1*+AKT2-S210A-S329A#1 (triangles), and *akt2-1*+AKT2-S210N-S329N#2 plants (stars). (*Right panels*) To determine whether the reduced O_2 treatment induced significant changes in the different plants, the data were referenced to the wild type. Normalized differences were calculated as $\Delta\Delta_{norm} = (X^{10\%} - mean_{WT}{}^{10\%})/mean_{WT}{}^{10\%} - (X^{21\%} - mean_{WT}{}^{21\%})/mean_{WT}{}^{21\%}$, where $X^{10\%}$ and $X^{21\%}$ denote the measured values in 10% and 21% O_2, respectively, and $mean_{WT}{}^{10\%}$ and $mean_{WT}{}^{21\%}$ are the mean values obtained for the wild type in the two O_2 conditions. Data are shown as means ± SD of $n \geq 10$ plants. The reaction of *akt2-1* was significantly different from the wild type under O_2-reduced conditions (Student's *t* test, $P < 2e\text{-}06$), as was the response of the plants expressing only inward-rectifying AKT2 mutants: *akt2-1*+AKT2-K197S ($P < 2e\text{-}08$) and *akt2-1*+AKT2-S210A-S329A ($P < 4e\text{-}08$). In contrast, there was no significant difference between the plant *akt2-1*+AKT2-S210N-S329N and the wild type (*B*: $P = 0.43$; *C*: $P = 0.98$; *D*: $P = 0.03$; *E*: $P = 0.84$).

Growth Tests in Different Potassium Concentrations. Sterile seeds were plated on Arabidopsis medium (AM) containing 50% Murashige and Skoog salts supplemented with 1% sucrose and solidified with 1% agar. Seven-day-old seedlings were transferred to a synthetic medium [2.5 mM $NaNO_3$, 2.5 mM $Ca(NO_3)_2$, 2 mM $NH_4(H_2PO_4)$, 2 mM $MgSO_4$, 0.1 mM FeNaEDTA, 25 μM $CaCl_2$, 25 μM H_3BO_3, 2 μM $ZnSO_4$, 2 μM $MnSO_4$, 0.5 μM $CuSO_4$, 0.2 μM Na_2MoO_4, 0.01 μM $CoCl_2$, 0.5% sucrose, pH 5.7, solidified with 1% agar] with 0.01, 0.1, or 2.5 mM KCl. Plants were vertically grown under standard light conditions (16-h day/8-h night; 100–150 $\mu E \cdot m^{-2} \cdot s^{-1}$).

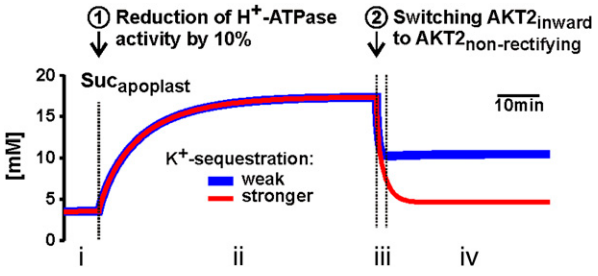

Fig. 5. Computational simulation of a realistic scenario. First, the activity of the H^+-ATPase was reduced to 90% of its original value (arrow 1). After equilibration, AKT2 was switched from an inward-rectifying channel into a nonrectifying channel (arrow 2). Time courses of the apoplastic sucrose concentration ($Suc_{apoplast}$) for two different sequestration rates of K^+ from the apoplast are displayed (red and blue curves). Further details on the simulation results are provided in Fig. S8.

Computational Simulations. The behavior of the transporter networks was mathematically simulated using Virtual Cell Modeling and Analysis Software developed by the National Resource for Cell Analysis and Modeling, University of Connecticut Health Center. Biophysical properties of the transporters were mathematically modeled to reflect their measured in vivo characteristics. Details are outlined in the legend of Fig. S5.

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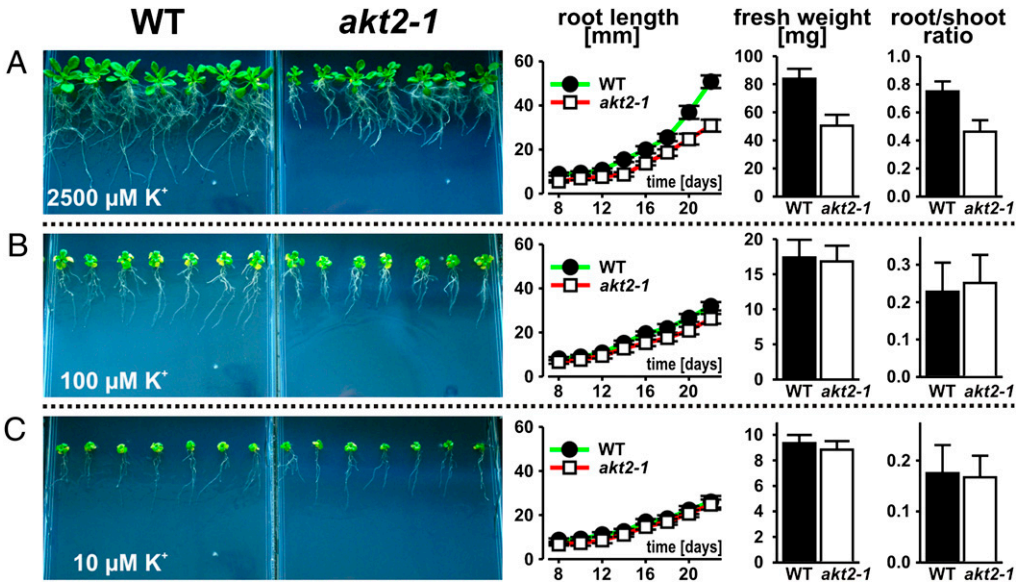

Fig. 6. *akt2-1* plants differ from WT plants under sufficient potassium supply but not in K^+-limited conditions. Root growth of *akt2-1* and WT plants on synthetic media supplemented with 2,500 µM (*A*), 100 µM (*B*), and 10 µM (*C*) potassium. (*Left*) Representative 22-d-old plants grown vertically on plates (16 h light, 150 $\mu E \cdot m^{-2} \cdot s^{-1}$). (*Right panels*) Time course of root growth as well as fresh weight and root-to-shoot ratio of 22-d-old plants. Data are means ± SD of n = 15 plants of each genotype. Note that in normal soil *akt2-1* and WT plants did not show developmental differences in long days, possibly indicating that K^+ is not abundantly available under these conditions.

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