particles then spontaneously form highly ordered, stable crystals that have the long-sought diamond structure (Fig. 1b).

The authors have so far produced crystals containing only about 100,000 particles and weighing less than one microgram. However, scaling up their process should be straightforward. Then, all that remains to form large 3D PBCs is to chemically fill the empty space in these crystals with pure silicon or titanium dioxide (for use with infrared or visible light, respectively) and then dissolve the building blocks.

One of the most exciting possible applications of PBCs is for quantum computers. In these devices, the digital bits that store values of '0' or '1' in a conventional computer are replaced with quantum bits (qubits) that can be both '0' and '1' at the same time. This replacement enables impressively faster computation of many difficult combinatorial problems that can be encountered in code-breaking. The challenge of building practical quantum computers lies in connecting many qubits together, typically using photonic signals, as well as isolating the qubits so that they do not get scrambled by interference from the outside world.

The piping around of photons in a PBC microcircuit is a solution to the first problem, and 2D PBCs have already been used to build prototype quantum devices[6]. But because current quantum photonic circuits are thin 2D sheets, their performance is limited – photons can leak out and disturbances can leak in. A simple solution to both problems would be to sandwich these circuits between two slabs of 3D PBC. More generally, bulk PBCs will enable a broad range of technologies in the production of large quantum systems[7], their controlled manipulation using light, and interfacing with conventional electronics[8]. The ultimate potential and applications of such technologies challenge our imagination.

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Plant biology

Calcium channel helps shut the door on intruders

Keiko Yoshioka & Wolfgang Moeder

Disease-causing microorganisms can invade plants through leaf pores called stomata, which close rapidly in a calcium-dependent manner on detecting such danger. The calcium channels involved have now finally been identified. **See p.569**

In plants, calcium ions (Ca^{2+}) function as a central signal for diverse stimuli, ranging from internal developmental cues to physical or biological stresses such as infection. However, the transient nature of Ca^{2+} signals and the enigmatic identities of plant Ca^{2+} channels have made the role of these ions difficult to study. Moreover, the connection between Ca^{2+} channels and specific plant responses is often unclear. On page 569, Thor *et al.*[1] now clarify one such connection, and report their finding of a type of Ca^{2+} channel that is activated during a specific response against infection.

Two specialized, moon-shaped cells, called guard cells, form a leaf pore called a stoma (Fig. 1a). Stomata allow gas exchange, including the entry of carbon dioxide for the energy-generating process of photosynthesis. They are thus essential for plant survival. However, disease-causing microorganisms (pathogens) can use stomata as a gateway for invasion. To limit infection, plants close stomata on recognizing such an attack, in a defence response called stomatal immunity[2]. The surfaces of the cells of both plants and animals have receptor proteins containing regions called kinase domains, and these proteins can recognize evolutionarily conserved microbial molecular motifs called pathogen-associated molecular patterns (PAMPs) and initiate signalling pathways needed for defence.

In the model plant *Arabidopsis thaliana*, a receptor protein called FLS2, which has a kinase domain, binds to the bacterial protein flagellin, recognizing a region of this PAMP called flg22. This recognition event causes FLS2 to form an active receptor complex with another cell-surface receptor kinase called BAK1. The complex adds a phosphate group to a cytoplasmic kinase called BIK1. This phosphorylation of BIK1 activates immune responses[3], such as the production of reactive oxygen species by the protein RBOHD. BIK1 is required for stomatal immunity[4], and if guard cells contain a mutant version of the gene that encodes this kinase, the plant cannot respond to flg22. However, the link between PAMP recognition and Ca^{2+}-mediated stomatal closure regulated by BIK1 has been unclear.

To join the dots, Thor *et al.* speculated that, through direct phosphorylation, BIK1 controls the Ca^{2+} channel(s) required for stomatal immunity. The authors focused on an ion channel called OSCA1.3, which is phosphorylated on sensing flg22. Thor and colleagues report that OSCA1.3 is permeable to Ca^{2+}, and that phosphorylation of OSCA1.3 by BIK1 at serine amino-acid residue 54 (in the same type of motif as that phosphorylated by BIK1 in RBOHD) activates this channel on pathogen recognition (Fig. 1b). Furthermore, the authors' observation that the gene that encodes OSCA1.3 is specifically expressed in stomata is consistent with a role for the channel in stomatal immunity.

The OSCA family of proteins are evolutionarily conserved ion channels, and *A. thaliana* contains 15 members of this family. Each ion channel is probably formed of two OSCA proteins. The largest group of these proteins, clade 1, includes OSCA1.1, OSCA1.2 (also known as OSCA1) and OSCA1.3 (refs 5–7). OSCA1.1 and OSCA1.2 are Ca^{2+}-permeable channels that are also permeable to several other types of positively charged ion (cations), and they are activated by an ionic imbalance known as osmotic stress[5,7].

Thor *et al.* observed no clear effect on the immune response to flg22 in a mutant plant in which the gene *OSCA1.3* was disabled. However, in a plant engineered also to have a mutant version of another clade 1 member – the gene *OSCA1.7* – stomatal closure on perceiving flg22 was impaired and susceptibility to bacterial infection was enhanced, compared with the response in the wild-type plant. OSCA1.7 has a similar protein motif to the one phosphorylated on OSCA1.3 by BIK1, and is activated through phosphorylation by BIK1 to generate a Ca^{2+} influx into cells. Thus, it seems that OSCA1.3 and OSCA1.7 are Ca^{2+} channels that regulate stomatal immunity and they probably function in a redundant manner,

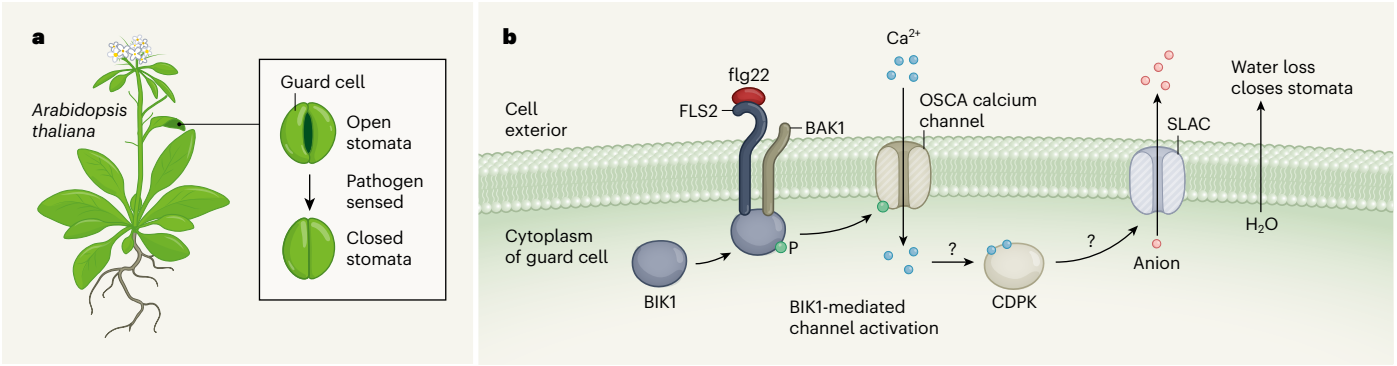

Figure 1 | A calcium channel that regulates closure of stomata. a, Plants, such as the model species *Arabidopsis thaliana*, have leaf pores called stomata. If the plant senses a disease-causing agent (termed a pathogen), stomatal guard cells rapidly close in response. **b**, Thor *et al.*[1] describe the identification of the calcium-ion (Ca^{2+}) channel in the pathway that leads to stomatal closure. Pathogens are sensed by the receptor protein FLS2, which forms a complex with the BAK1 protein. When this complex senses a bacterial-protein fragment, termed flg22, it adds a phosphate group (P) to the protein BIK1, thereby activating it. BIK1 then phosphorylates the Ca^{2+} channel. Thor *et al.* report that two proteins of the OSCA family, OSCA1.3 and OSCA1.7, can function as Ca^{2+} channels during this response (whether one or both of these together fulfil this role is unknown). How an influx of Ca^{2+} through the channel causes stomatal closure is unclear. One possibility is that enzymes called calcium-dependent protein kinases (CDPKs) activate S-type anion channels (SLACs). SLACs enable anions (negatively charged ions) to exit the cell, which leads to the water loss that drives stomatal closure.

such that if OSCA1.3 is absent, OSCA1.7 can fulfil its role. Whether just one or both of these proteins together form Ca^{2+} channels that act in stomatal immunity is unknown.

In addition to identifying these Ca^{2+} channels, Thor *et al.* explored the role of the plant hormone abscisic acid (ABA), which regulates stomatal closure when the plant senses a water deficit. This hormone also controls stomatal defences, because stomata of ABA-deficient plants do not close effectively on perceiving pathogens[2]. However, the authors found that a plant with mutations in the genes encoding both OSCA1.3 and OSCA1.7 is fully responsive to ABA, indicating that these channels are not involved in ABA-mediated stomatal closure. This observation corroborates previous evidence[4] that the regulation of stomatal immunity by BIK1 does not require ABA. Furthermore, Thor *et al.* report that the overall Ca^{2+}-signal activation by flg22 in leaves that had mutations in the genes encoding both OSCA1.3 and OSCA1.7 was not impaired; guard cells make up only a small fraction of leaf cells. This result strongly supports the specific role of these channels in stomatal immunity, rather than general immunity, even though BIK1 is required for both types of response.

How changes in Ca^{2+} concentration deliver stimulus-specific cellular responses is a central question in this area of research. One proposed idea is that stimulus-specific temporal patterns of cytoplasmic Ca^{2+} levels might provide a key cue, and that these 'Ca^{2+} signatures' might be generated and decoded by specific Ca^{2+}-binding components, such as calmodulin proteins or calcium-dependent protein kinases[8]. Thor and colleagues' results suggest instead that the Ca^{2+} channels themselves might determine specificity, at least for stomatal immunity.

Although OSCA proteins allow stomata to close independently of ABA involvement, closure mediated either by ABA or in response to infection probably involves the same mechanism, which eventually closes stomata through water movement out of guard cells. Therefore, both pathways should converge at some point. The activation of channels that enable negatively charged ions (anions) to exit the cell, such as S-type anion channels, termed SLACs, is a crucial step in stomatal movement[9]. The protein kinase OPEN STOMATA1, which is a component of an ABA-mediated signalling pathway, activates SLACs and has been proposed[2] as a point of convergence for defence responses and ABA signalling. However, some calcium-dependent protein kinases also activate SLACs[10], and such Ca^{2+}-signal decoders, or perhaps even the anion channels themselves, might be the convergence point instead.

"An emerging theme in studies of plant calcium-ion channels is their regulation by phosphorylation."

An emerging theme in studies of plant Ca^{2+} channels is their regulation by phosphorylation. Previous studies[11,12] reported that BIK1 and BAK1 phosphorylate members of another group of plant Ca^{2+} channels, the cyclic nucleotide-gated ion channels, to regulate their function or stability. The phosphorylation of OSCA1.3 and OSCA1.7 by BIK1 underscores the connection between receptor kinases and Ca^{2+} channels, presumably to generate stimulus-specific Ca^{2+} signals. It will be interesting to determine whether OSCA-family proteins interact with other components on the surface of cells to form a structure called a channelosome — a group of signalling molecules surrounding an ion channel[13].

OSCA proteins have so far been linked mostly to the sensing of osmotic stress. They are categorized as a type of mechanosensing channel, one that converts physical forces into biochemical signals[14,15]. Are OSCA1.3 and OSCA1.7 activated by osmotic stress or mechanical stimulation, in addition to their activation by BIK1? Did the two proteins evolve a defence-specific role, or do they also have other functions in stomata? Understanding the biological function of each OSCA and the Ca^{2+} signals they generate will shed light on stomatal biology. Such insights could be crucial for the bioengineering of plants to meet future challenges in crop production.

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